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# Intra-individual variability in adult ADHD: an exploration of the viability of distinct purely inattentive condition

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INTRA-INDIVIDUAL VARIABILITY IN ADULT ADHD: AN EXPLORATION OF THE  
VIABILITY OF A DISTINCT PURELY INATTENTIVE CONDITION

A Dissertation

Submitted to the Graduate Faculty of the  
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by  
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## LIST OF NON-TEST ACRONYMS

ACC	Anterior Cingulate Cortex
ADHD	Attention-Deficit/Hyperactivity Disorder
BAS	Behavior Activation System
BIS	Behavior Inhibition System
DA	Dopamine
DAT	Dopamine Transporter
DLPFC	Dorsolateral Prefrontal Cortex
DSM	Diagnostic and Statistical Manual of Mental Disorders
DV	Dependent Variable
EM	Expectation Maximization
FSIQ	Full-Scale IQ (from the WAIS-III)
IIV	Intraindividual Variability
ISD	Intraindividual Standard Deviation
ISI	Inter-Stimulus Interval
IV	Independent Variable
MANOVA	Multivariate Analysis of Variance
MAR	Missing at Random
MCAR	Missing Completely at Random
MRI	Magnetic Resonance Imaging
NE	Norepinephrine
PET	Positron Emission Topography
PST	Processing Speed Index (from WAIS-III)
RT	Reaction Time
RTSD	Reaction Time Standard Deviation
RTSE	Reaction Time Standard Error (from Conners' CPT)
SCT	Sluggish Cognitive Tempo
SD	Standard Deviation
SE	Standard Error

## LIST OF TEST ACRONYMS

CPT	Continuous Performance Test
TMT	Trail Making Test
WAIS-III	Wechsler Adult Intelligence Scale, 3 <sup>rd</sup> Edition
WJ-III	Woodcock-Johnson Tests of Achievement, 3 <sup>rd</sup> Edition
WMS-III	Wechsler Memory Scale, 3 <sup>rd</sup> Edition
WURS	Wender Utah Rating Scale

## ABSTRACT

Researchers have suggested that intraindividual variability (IIV), or variation in cognitive testing performance within an individual across a measure or group of measures, may be an endophenotypic marker of Attention-Deficit/Hyperactivity Disorder (ADHD). However, not all studies have consistently demonstrated significant differences in IIV between individuals with and without ADHD. One potential explanation for this ambiguity is experimental group heterogeneity owing to Sluggish Cognitive Tempo (SCT). Individuals with SCT exhibit behavioral characteristics dissimilar from individuals with ADHD; rather than being impulsive, hyperactive, and aggressive, they tend to be shy, day-dreamy, and cognitively slow. Researchers have hypothesized that the presence of SCT in the absence of hyperactivity may reflect a distinct purely-inattentive condition that is currently diagnosed as ADHD. If these purely inattentive individuals are included in ADHD experimental groups, they could obfuscate between-groups differences. Unfortunately, few studies have attempted to separate purely inattentive individuals from those with ADHD, with none having examined cognitive functioning after such a separation and in adults. The purpose of the current study, then, was to attempt to identify and separate adults with “pure inattention” from adults with ADHD, and to then compare the groups’ performances on a large neuropsychological test battery. Of particular interest were measured group differences in IIV—operationalized as both reaction time standard deviation and intraindividual standard deviation (ISD)—and SCT as assessed by objective cognitive testing. Cluster analysis was used to identify experimental groups via responses to twelve items on the Wender Utah Rating Scale (WURS). Individuals were also grouped, in separate analyses, by DSM-IV-TR ADHD subtype, and by their degree of endorsed SCT-like symptoms on the WURS. Results indicated that the use of the selected items from the WURS, combined with



cluster analysis, was not an effective method of delineating a purely inattentive group. No significant between-groups effects were identified across any of the three grouping methods with respect to IIV or SCT. The implications of these results are discussed, and future research directions are suggested.

## INTRODUCTION AND LITERATURE REVIEW

George Still is credited with first reporting on the concept of Attention-Deficit/Hyperactivity Disorder (ADHD) more than 100 years ago (Still, 1902). In a series of lectures, Still described a subset of children seen in his clinical experiences who exhibited disordered “moral consciousness and moral control” (p. 128; Still, 2006). Since that time, the condition Still described has undergone multiple nominal imputations, including brain-injured child syndrome (Strauss & Lehtinen, 1947) and minimal brain damage/dysfunction (Barkley, 1999b; Rie & Rie, 1980). The disorder was first included in the Diagnostic and Statistical Manual of Mental Disorders in its 2<sup>nd</sup> revision as Hyperkinetic Reaction of Childhood (American Psychiatric Association[APA], 1968). DSM-III would then change the name of the condition to Attention-Deficit Disorder while including two possible subtypes: “with Hyperactivity” and “without Hyperactivity” (APA, 1980). Diagnosis was based on checklists of symptoms divided into the three clusters of Hyperactivity, Inattention, and Impulsivity. Somewhat confusingly, the “without hyperactivity” subtype was abandoned and the three previously-discrete symptom categories were amalgamated into a single checklist for DSM-III-R (APA, 1987). This marked change in diagnostic concept likely increased the variability of the associated patient group (Quay, 1999). Perhaps owing in part to this decreased diagnostic specificity, field trials were conducted in the late 1980s and early 1990s to determine those symptom types and thresholds most central to the disorder. Based heavily on initial findings by these field trials (Lahey et al., 1994), DSM-IV assigned the condition its current name of Attention-Deficit/Hyperactivity Disorder and implemented a two-factor symptom structure list—one involving hyperactivity/impulsivity and the other inattention (APA, 1994). DSM-IV also delineated the following three distinct subtypes: Predominantly Inattentive (ADHD-PI), Predominantly

Hyperactive/Impulsive (ADHD-HI), and Combined (ADHC-C), as well as a Not Otherwise Specified category.

Despite it being the topic of these multiple conceptual changes and tens of thousands of journal articles, there is still much that is not yet known or understood about ADHD. What can be said is that ADHD is thought to be the most prevalent childhood neurodevelopmental disorder (Rowland, Lesesne, & Abramowitz, 2002). The condition is estimated to affect approximately 4.5 million, or roughly 6% of, children aged five to seventeen years in the United States (Bloom & Cohen, 2007; Dey & Bloom, 2005), and 5.29% of children and adolescents worldwide (Polanczyk, De Lima, Horta, Biederman, & Rohde, 2007), with estimated annual per-child treatment-related costs totaling at least \$15,000 (Pelham, Foster, & Robb, 2007). Longitudinal studies show that anywhere from 30% to 70% of these children report persistence of some ADHD-related symptoms and/or impairment into adulthood, leading to the roughly 4.4% to 5.2% of the adult population in the U.S. that meets diagnostic criteria for the disorder (Barkley, Fischer, Smallish, & Fletcher, 2002; Faraone, Biederman, & Mick, 2006; Fayyad et al., 2007; Kessler et al., 2006; Mannuzza et al., 1991; Rasmussen & Gillberg, 2000; Weiss & Hechtman, 1993). The variability in documented persistence rates may be in part due to the inappropriateness of DSM-IV-TR diagnostic criteria when applied to adults (Adler, Barkley, & Newcorn, 2008; Conners et al., 1999; Faraone et al., 2006; Kessler et al., 2010; McGough & Barkley, 2004); changes in diagnostic criteria from base-line assessment to follow-up (Lara et al., 2009); non-random attrition of healthier vs. more-debilitated participants (Weiss, Hechtman, Milroy, & Perlman, 1985); and cross-study differences in reporting source, assessment methods, and selected diagnostic criteria (Barkley et al., 2002; Mannuzza, Klein, & Moulton III, 2003). Regardless, persistence of the disorder from childhood into adulthood does not appear to be

related to gender or ethnicity, but is significantly correlated with severity of childhood symptoms (Kessler, Adler, Barkley et al., 2005). While gender may not predict persistence of symptoms into adulthood, ADHD is nonetheless diagnosed at much higher rates in young boys than in young girls, with ratios ranging from 3:1 (Offord et al., 1987; Wang, Chong, Chou, & Yang, 1993; Wolraich, Hannah, Pinnock, Baumgaertel, & Brown, 1996) to 4:1 (Cantwell, 1996) in community samples. Gender discrepancies in prevalence rates then appear to decrease with age, declining to 2:1 in early- to mid-adolescence (P. Cohen et al., 1993; Offord et al., 1987) and becoming nearly equal across genders in young- to mid-adulthood (P. Cohen et al., 1993; DuPaul et al., 2001; Kessler, Adler, Barkley et al., 2005). This reduction in gender differences may be due in part to the fact that hyperactive/impulsive symptoms, which boys exhibit more than girls (Biederman et al., 2002; Gershon, 2002), decline, or are at least “internalized,” with age while inattentive symptoms do not (Barkley, Murphy, & Fischer, 2008; Gittelman, Mannuzza, Shenker, & Bonagura, 1985; Lubke, Hudziak, Derks, van Bijsterveldt, & Boomsma, 2009), possibly causing more males to believe the condition has remitted and thus not seek treatment as adults.

Another issue possibly related to the disappearance of the gender discrepancy in ADHD by adulthood, as will be discussed in significant detail later in this paper, is the posited existence of a distinct purely-inattentive condition (e.g., Barkley, 1997; Quay, 1997). This condition, which entails inattention occurring in the lifelong absence of significant hyperactivity, may not initially spawn concern in teachers and parents due to a lack of exhibited disruptive behaviors in much the same way that girls with ADHD less often attract the attention of adults than do boys with ADHD (Stefanatos & Baron, 2007). However, as individuals age and the “costs” of inattention, particularly in the workplace, potentially become more apparent, so too might this

purely-inattentive condition be more frequently identified by clinicians. It will be the purpose of the current paper to explore the viability of this purely-inattentive condition, recount its history, catalogue its associated interpersonal and cognitive characteristics, and finally, attempt to separate individuals with pure inattention from those with ADHD via data from specific personality and neuropsychological assessment instruments.

### **Patient Outcomes (Childhood to Adulthood)**

The examination of both the personal impact of ADHD, as well as the course of its symptoms over the lifespan, has been a research area of fervent interest. Multiple studies have shown that children with ADHD experience significant detriments to overall quality of life (see: Danckaerts et al., 2010 for review). Socially, adolescents with childhood-diagnosed ADHD have fewer close friendships and greater amounts of rejection by peers as reported by their parents (Bagwell, Molina, Pelham, & Hoza, 2001). Given that this rejection is a predictor of such later-life adverse outcomes as criminality and dropping out of school (Parker & Asher, 1987), it is not surprising that when followed into adulthood, many of these childhood-diagnosed individuals continue to have various difficulties in multiple life domains. Adults with persisting ADHD diagnoses exhibit higher rates of divorce/separation, antisocial personality disorder, substance use, depression, and anxiety (Biederman et al., 1993; Biederman, Faraone, Monuteaux, Bober, & Cadogan, 2004; Kessler et al., 2006; Kevin R. Murphy, Barkley, & Bush, 2002; Weiss et al., 1985). Adult ADHD is also correlated with lower IQ and socioeconomic status (Biederman et al., 1993), unemployment (Faraone & Biederman, 2005; Kessler et al., 2006), lower educational attainment (Faraone & Biederman, 2005; Mannuzza, Klein, Bessler, Malloy, & Hynes, 1997; Kevin R. Murphy et al., 2002), and work underperformance (Kessler, Adler, Ames et al., 2005; Mannuzza et al., 1997).

Regarding cognitive functioning, research suggests that many children with ADHD eventually “outgrow” their symptoms after an initial period of delayed maturation, with deficits in areas such as alertness, reaction time, and working memory exhibited in late childhood largely remitting by adolescence or adulthood (Drechsler, Brandeis, Foldenyi, Imhof, & Steinhausen, 2005; Faraone et al., 2006; Fischer, Barkley, Smallish, & Fletcher, 2005; Halperin, Trampush, Miller, Marks, & Newcorn, 2008). These findings are consistent with those involving the progression of behavioral symptoms, which show declines in overt hyperactive-impulsive symptoms as children age (Hart et al., 1995). However, a significant minority of childhood-diagnosed individuals continue to display measurable deficits well into adolescence and adulthood. Research has shown such persisting disturbances in executive functions (e.g., planning, set-shifting, working memory), visual attention, variability, and behavioral hyperactivity/fidgeting (Fischer et al., 2005; Halperin et al., 2008; Hinshaw, Carte, Fan, Jassy, & Owens, 2007). Additionally, some authors suggest that part of the decrease in prevalence of ADHD with age may be due to individuals outgrowing the diagnostic criteria, which are static, rather than outgrowing the disorder (Barkley et al., 2008; Stefanatos & Wasserstein, 2001).

Persistence of ADHD into adulthood, as previously mentioned, is associated with neither gender nor ethnicity (Kessler, Adler, Ames et al., 2005). Somewhat surprisingly, persistence also does not appear to be related to receipt of treatment in childhood (Lara et al., 2009). Conversely, continuation of the disorder in adolescents and young adults is more likely when childhood symptoms are more severe/debilitating, are of the combined type, are familial (i.e., probands have close relatives also diagnosed with ADHD), and exist in the presence of comorbid psychiatric disorders (Biederman, Faraone, Milberger, & Curtis, 1996; Hart et al., 1995; Kessler, Adler, Ames et al., 2005; Lara et al., 2009).

## **Treatment**

Despite, or perhaps because of, the lack of data supporting a strong link between childhood treatment type and later-life persistence of symptoms, much time has been spent developing and assessing the viabilities of various ADHD interventions. The only treatment types to display consistent evidence-based efficacy in reducing ADHD symptoms are psychosocial/behavior interventions, pharmacological interventions, and combined interventions (i.e., psychosocial intervention combined with medication; (P. S. Jensen et al., 2001; NIH, 2000; Pelham & Waschbusch, 1999; Schachar & Ickowicz, 1999). With respect to medication treatment, short-term results have been consistently and significantly positive, with improvements observed in multiple ADHD symptom areas, including teacher and parent ratings of disruptive behaviors, time on task, and attentiveness (Biederman & Spencer, 2008; Dulcan & Benson, 1997; P. S. Jensen et al., 2001). Perhaps owing to these promising findings, as well as to increased public awareness of ADHD, stimulant medication utilization rates in U.S. children increased steeply (from 0.6% to 2.4%) in the decade from 1987 to 1996 (Olfson, Marcus, Weissman, & Jensen, 2002). While usage rates in children and adolescents appear to have leveled off since then (2.7% in 1997 and 2.9% in 2002, neither of which represents a significant increase over 1996 data; Zuvekas, Vitiello, & Norquist, 2006), concerns have nevertheless been raised over long-term adverse consequences. These concerns may indeed have merit; studies on the side-effects of long-term psychostimulant use in children indicate statistically-significant reductions in height and weight (Zhang, Du, & Zhuang, 2010) and rare-but-significant increases in suicidal ideation (Bangs et al., 2008). Increased blood pressure and heart rate are also associated with stimulant medication use in children and adolescents, although generally not to a clinically-significant degree (Rapport & Moffitt, 2002; Wernicke et al., 2003). Sustained

improvement may represent another potential concern in pharmacological interventions—while some data suggest that childhood medical treatment is associated with higher employment rates later in life (Halmøy, Fasmer, Gillberg, & Haavik, 2009), there is relatively little evidence supporting long-term efficacy of these medications. Additionally, while response rates to psychopharmacological interventions are generally high, approximately 30% of children and adolescents who take ADHD medications report little or no symptom improvement at any time (Biederman & Spencer, 2008). Data have not supported early suspicions of links between childhood stimulant use and adult-onset substance abuse problems, however, as much of the significant variance in this relationship is accounted for by comorbid conduct disorders (Biederman et al., 2008; Harty, Ivanov, Newcorn, & Halperin, 2011; Mannuzza et al., 2008). Additionally, children diagnosed with ADHD who were not on medication were found to have a higher risk of obesity (odds ratios of 1.42 and 1.85 in boys and girls, respectively); this heightened risk was not found in those children who were taking medications to treat their ADHD (Kim, Mutyala, Agiovlasitis, & Fernhall, 2011). Thus, while the long-term effectiveness for remediation of ADHD symptoms is unclear, and associated health risks with long-term stimulant medication usage have been found, short-term effectiveness and potential health benefits (e.g., reduced obesity risk) of ADHD medications appear to continue to drive prescription and use of these substances.

Non-pharmacological treatments for ADHD predominately involve behaviorally-based psychotherapeutic programs. Indeed, research suggests that the only effective, evidence-based psychosocial interventions for ADHD are behavioral parent training, behavioral school/classroom management, and behavioral peer summer treatment/recreational setting paradigms (Diamond & Josephson, 2005; Knight, Rooney, & Chronis-Tuscano, 2008; Pelham &



Fabiano, 2008; Young & Amarasinghe, 2010). Tentative, recent support also exists for child-centered behavioral activation therapy, although limited data are available for its effectiveness when used alone rather than being coupled with behavioral parent training (Curtis, 2010). These behavioral interventions have all been shown to produce symptom- and functioning-related treatment effect sizes similar to medication and significantly greater than non-behavioral methods (e.g., cognitive therapy, nondirective counseling), the latter of which show little-to-no effectiveness in treating ADHD (Pelham & Fabiano, 2008).

Combined psychotherapeutic and pharmacologic treatments for children and adolescents with ADHD have also shown some promise. Indeed, these types of programs have tentatively been shown to reduce symptom severity in ADHD with comorbid oppositional defiant disorder to a greater degree than medication alone (So, Leung, & Hung, 2008). In a review of data from the NIMH Multimodal Treatment Study of Children with ADHD (the MTA study), Jensen and colleagues (2005) also found that combined treatments provided significant symptom remediation. However, these researchers also pointed out important data regarding cost-effectiveness, which suggested that in many instances, medication alone may be the cheapest option. This did not apply to all circumstances, though, as particularly in children with comorbid conditions, combined interventions displayed the relative best cost-effectiveness value. Thus, in more complicated cases, combined treatment may be a better option than either medication or psychotherapy alone.

Much less research exists that explores treatment types and effectiveness in adults. This might in part be fueled by an underutilization of services by adults—survey data indicate that only 10.9% of adults with ADHD reported actually receiving treatment specifically for ADHD in the past year (Kessler et al., 2006). This low rate of service usage is particularly troubling given

the finding that in children, successful treatment of ADHD symptoms also resulted in significant reduction of symptoms of comorbid conditions (Jensen et al., 2001). Thus, lack of treatment of adult ADHD-related difficulties may result in a lack of reduction of difficulties associated with comorbid psychological conditions. In general, as with children, the most common first-line intervention of choice in adults is medication; specifically, treatment guidelines recommend stimulant medication or atomoxetine when not otherwise contraindicated (Dulcan & Benson, 1997; Kooij et al., 2010; Nutt et al., 2007). Recent studies have also begun indicating that as with children and adolescents, combined pharmacotherapy and cognitive-behavioral therapy may also be effective in adults, providing incremental benefits beyond medication alone (see: Ramsay & Rostain, 2007 for a thorough review).

### **Neurophysiology of ADHD**

Concomitant with the continued development and study of ADHD interventions has been the improvement of methods for attaining neurophysiological data related attentional disturbances, as this information may inform both treatment and theory. Accordingly, with increases in sophistication of neuroimaging technologies over the past two decades, researchers have increased their efforts in evaluating the neurophysiology of ADHD. An early examination of ADHD-related neurophysiology via positron emission tomography (PET) scan found globally-depressed cerebral glucose metabolism in currently-symptomatic adults with histories of childhood hyperactivity (Zametkin et al., 1990). Of 60 brain regions examined, Zametkin and colleagues (1990) found that 30 exhibited this glucose hypometabolism, with particularly abnormal findings observed in the premotor and superior prefrontal areas. Later structural studies using magnetic resonance imaging (MRI) then supported and elaborated on these results. Multiple teams of researchers have shown that children with ADHD exhibit lower overall

cerebral, cerebellar, and both white and gray matter volumes (Castellanos et al., 2001; Castellanos, Giedd et al., 1996; Filipek et al., 1997; Makris et al., 2007; Mostofsky, Cooper, Kates, Denckla, & Kaufmann, 2002). As with the work by Zametkin et al. (1990), these later studies suggested particular involvement of prefrontal and premotor brain regions, especially in the right cerebral hemisphere. Other implicated regions include the anterior cingulate and prefrontal cortices (Makris et al., 2007), right hemisphere globus pallidus and caudate nucleus (Castellanos, Giedd et al., 1996), left hemisphere total caudate and caudate head volumes (Filipek et al., 1997), and posterior-inferior cerebellar vermis (Castellanos et al., 2001; Castellanos, Giedd et al., 1996; Mostofsky, Reiss, Lockhart, & Denckla, 1998). Meta-analysis of these and other imaging studies has helped to sort through the large variety of implicated cerebral areas. In general, such analysis has revealed that disruptions of frontostriatal (dorsal anterior cingulate cortex, striatum, and dorsolateral and inferior regions of the prefrontal cortex) and frontoparietal neural networks are the most robust functional imaging-related discoveries (Bush, Valera, & Seidman, 2005; Dickstein, Bannon, Castellanos, & Milham, 2006). However, as is often the case with imaging research, many of these studies made use of relatively small sample sizes (i.e., most included fewer than 40 total participants), failed to make comparisons to control groups, did not report results in standardized fashion, and/or limited result disclosure to only those areas hypothesized to show dysfunction rather than focusing on the entire brain (see Durston, 2003 and Dickstein et al., 2006 for comprehensive reviews).

Nonetheless, imaging research has done much to inform the neuroscience of ADHD, leading to a handful of neurotransmitter-focused theories of the disorder. The high concentration of dopamine (DA)- and norepinephrine (NE)-producing neurons populating and innervating midbrain and forebrain areas implicated in ADHD (Mendoza & Foundas, 2008), as well as the

observed effects on these neurotransmitter systems of the predominantly stimulant-based medications most-frequently used to treat ADHD (Pliszka, McCracken, & Maas, 1996; Shafritz, Marchione, Gore, Shaywitz, & Shaywitz, 2004; Vaidya et al., 1998; Zametkin & Rapoport, 1987), have caused these two neurochemicals to become focal points of theory and research. Such theories, then, have begun focusing on the neural networks in which these transmitters typically operate, and in what ways neurotransmitter function therein might be disturbed.

Evidence suggests that top-down, goal-directed vs. bottom-up, transient, response-related attentional resources are marshaled by two distinct neural networks, with the former tied to dorsolateral prefrontal cortex (DLPFC) and the latter to the more medially-located dorsal anterior cingulate cortex (ACC; Banich et al., 2009; Banich, Milham, Atchley, Cohen, Webb, Wszalek, Kramer, Liang, Wright et al., 2000; Banich, Milham, Atchley, Cohen, Webb, Wszalek, Kramer, Liang, Barad et al., 2000; Milham, Banich, & Barad, 2003; Milham, Banich, Claus, & Cohen, 2003). Both types of attention, and thereby both associated neural networks, are thought to be dysfunctional in individuals (children and adults) diagnosed with ADHD. In their extensive work using the Stroop task, for example, Banich and colleagues (2009) found reduced DLPFC and dorsal ACC activity in college-aged students with ADHD vs. carefully-matched controls. Additionally, these researchers also observed decreased activity in the right inferior frontal cortex, a region that is associated with late-stage response inhibition (Aron, Robbins, & Poldrack, 2004; Forstmann et al., 2008; Sharp et al., 2010). Similarly, Schneider et al. (2010) observed, via fMRI, dysfunction in ACC and prefrontal cortex in adults diagnosed with ADHD who completed a continuous performance test (CPT). These researchers point out that many of the implicated ACC and frontostriatal neural networks are modulated predominately by DA, potentially implicating this neurotransmitter in the development of ADHD.

Despite many studies, such as those above, implicating DA and DA-modulated networks in ADHD, much is still unknown regarding the actual neural mechanisms in effect. Examinations of DA transporter (DAT) function and quantity in animals and humans have attempted to reduce this uncertainty. Early pharmaceutical research initially prompted much of this focus on DAT, as it suggested that striatal DAT blockade leading to increased synaptic DA concentration was the primary mechanism of action of methylphenidate (Castellanos, Elia et al., 1996; Schwenker et al., 1985; Volkow et al., 1998). Genetic knockout and knockdown studies that eliminated or reduced, respectively, DAT function in mice reported, as is the case with humans, that administration of psychostimulants reduced ADHD-like symptoms in these mice (namely disinhibition and hyperactivity), presumably as a function of increased extracellular DA levels (Gainetdinov et al., 1999; Giros, Jaber, Jones, Wightman, & Caron, 1996; Zhuang et al., 2001). Also as with humans, Gainetdinov et al. (1999) found that administration of psychostimulants in wild-type/normal mice actually increased activity in direct proportion to the amount of extracellular dopamine present. Thus, it was hypothesized that disrupted DAT functioning in these mice may have caused ADHD-like symptoms which were remediated by psychostimulant medication. Such tentative support for the role of the DAT in ADHD induced a transition from mice to human paradigms. Spencer and colleagues (2005) reviewed many of the subsequent DAT-related studies in children and adults, finding that the majority (six of eight) reported statistically-significant increases in striatal DAT binding levels, which would conceivably decrease extracellular DA. Although the findings were encouraging, they led to many questions regarding the specifics of DAT/DA levels and activity in neural attention systems, such as whether the observed increased DAT binding was due to trait-level variables (e.g., reduced neurodevelopmental pruning of

dendritic trees) or state-level variables (e.g., neurophysiologic responses to persistently high or low levels of extracellular DA; Madras, 2005; Volkow et al., 2007).

Volkow et al. (2007) attempted to clarify the roles of DAT and DA in ADHD by directly examining the effects of psychostimulant (methylphenidate) administration on striatal DA levels in humans. Their findings indicated reduced striatal (particularly caudate) and limbic (amygdala and hippocampus) DA release in adults with ADHD vs. controls when administered methylphenidate. This reduced DA release was significantly related to measured symptoms of inattention, suggesting that striatal DA activity influences this aspect of ADHD symptomatology. The researchers also observed reduced amounts of striatal D<sub>2</sub>/D<sub>3</sub> receptor availability which, when coupled with the previously-reported reduced striatal DA release, suggests lower overall numbers of these receptors in the striatum of individuals with ADHD, further implicating striatal DA function in disrupted attention. Finally, the results suggested increased reinforcement effects from drugs in adults with ADHD, potentially linking DA functioning in this population not only to attentional dysfunction, but also to their increased likelihood of substance abuse. Such a finding might help to explain early, but eventually unsubstantiated, ideas that stimulant medication treatment in children may lead to adult substance abuse disorders; these and other treatment-related concerns will be discussed in more detail later.

Genetics studies have lent further support to the hypothesis that DAT plays an important role in ADHD pathology. Multiple sets of results have implicated the DAT gene (DAT1) in ADHD. For example, Bellgrove and colleagues (2004) found greater amounts of response variability, a cognitive phenomenon often associated with attentional disturbance, in children and adolescents with two copies of the 10-repeat DAT1 allele versus those with one or two copies of the 9-repeat DAT1 allele. Loo et al. (Loo et al., 2003) also examined children with two copies of

the 10-repeat DAT1 allele, finding that homozygosity for the allele was associated with poorer vigilance than heterozygosity or homozygosity for the 9-repeat allele. These researchers also found that homozygosity for the 10-repeat allele mediated methylphenidate effects as measured by EEG. The 10-repeat allele variant is thought to influence attentional functioning by altering the expression of the DAT in humans when compared with other variants (i.e., 7-, 9-, and 11-repeat alleles) (Fuke et al., 2001). Yet while most data seems to indicate a connection between DAT1 and ADHD, some research has failed to find any such link in children with the disorder versus those without (Simsek, Al-Sharbati, Al-Adawi, & Lawatia, 2006). This inconsistency in these studies could potentially be related to small sample leading to reduced statistical power, differences in sample characteristics (e.g., comorbidity, degree of impairment) and diagnostic criteria, and differences in genetic polymorphism detection techniques.

In addition to DAT and DA, NE has also been implicated in ADHD. Specifically, a NE-affiliated posterior attentional neural network involving the parietal lobes, thalamus, and precuneus has shown dysfunction in individuals with ADHD (Posner & Petersen, 1990; Schneider et al., 2010; Smith, Taylor, Brammer, Toone, & Rubia, 2006; Tamm, Menon, & Reiss, 2006). This posterior attention network is thought to be involved in the orienting response (Fan, McCandliss, Fossella, Flombaum, & Posner, 2005), and is also believed to play a role in attentional shifting and disengagement (Jackson, Swainson, Mort, Husain, & Jackson, 2009; Posner, Inhoff, Friedrich, & Cohen, 1987; Rushworth, Nixon, Renowden, Wade, & Passingham, 1997). Lesion and brain injury studies involving the phenomenon of left hemispatial neglect following right parietal lobe damage initially spurred, and have since lent support to, conceptualizations of this brain region's involvement in attentional functions (see: Proto, Pella, Hill, & Gouvier, 2009 for a review). Children and adolescents with ADHD have shown less

activation of the parietal lobes than control children on tasks of simple visual attention (Booth et al., 2005), alerting and reorienting (Konrad, Neufang, Hanisch, Fink, & Herpertz-Dahlmann, 2006), and attention switching (Smith et al., 2006). These findings of decreased parietal lobe activation have since been extended to adults with childhood diagnoses of ADHD, including those in whom the condition had since partially remitted (Schneider et al., 2010), suggesting potential developmental abnormalities in the posterior attentional system. Pharmacological research showing dose-dependent clinical effectiveness of substances that influence NE, such as atomoxetine (brand name: Strattera), a non-stimulant norepinephrine reuptake inhibitor, in reducing ADHD symptoms lends further support to the involvement of NE systems in the disorder (Michelson et al., 2001; Spencer, 2004). These results have prompted the initiation of a randomized controlled atomoxetine trial in children and adolescents (Tsang et al., 2011).

**Neurobiological Theories and Hypotheses of ADHD.** Castellanos and colleagues (2005) extrapolated from this line of thinking to develop a hypothesis of catecholaminergic (particularly DA and NE) deficit in individuals with ADHD. These researchers posit that in individuals with ADHD, there may be a catecholaminergically-related deficiency that interferes with these neurochemicals' abilities to properly regulate neuronal activity fluctuations that occur at a very low frequency. These fluctuations, then, could be responsible for the behavioral difficulties associated with ADHD via the brief, frequent attentional lapses they are proposed to cause. Research involving Castellanos et al.'s hypothesis, particularly in relation to the concept of intra-individual performance variability in ADHD and as it would evolve into a more complete hypothesis by Sonuga-Barke and Castellanos (2007), will be discussed in greater depth later in this paper.



Sagvolden et al. (2005) also built upon research involving the catecholaminergic-regulated (and specifically DA-regulated) frontostriatal neural network to propose a “dynamic developmental theory” of ADHD. These authors limit their theory to the predominantly hyperactive/impulsive and combined subtypes, and further narrow their focus to behavioral symptoms in particular. They theorize that the behaviorally-based causes of these subtypes of ADHD are changes in the reinforcement of new behaviors coupled with dysfunctional extinction of previously learned and reinforced behaviors, both of which are hypothesized to be linked to deficiencies in DA regulation of mesolimbic systems. Specifically, the researchers state that the window of opportunity to properly link a behavior and its consequence(s) will be smaller in individuals with ADHD. This reduced behavior-consequence association ability will then result in reduced/restricted stimuli for influencing behavior and an inclination to enact short sequences of motor responding, subsequently bringing about inattention and impulsivity, respectively. Concomitantly, dysfunctional extinction processes will result in an overabundance of learned and primed behavioral responses, thereby inducing hyperactivity. Research indicating alterations of delayed discounting behaviors (i.e., choices regarding immediate versus delayed reward), reward sensitivity, and effects of incentives in children with ADHD (Hurst, Kepley, McCalla, & Livermore, 2011; Luman et al., 2009; Uebel et al., 2010) would seem to lend tentative initial support to the dynamic developmental theory.

A somewhat older theory of ADHD that has spurred a large response in the ADHD community is that proposed by Barkley (1997). This model was informed by the prior work of Quay (Quay, 1988a, 1988b, 1997), whose work in turn adapted to ADHD the neuropsychological theory of anxiety involving the Behavioral Inhibition System (BIS) and Behavioral Activation System (BAS) as originally proposed by Gray (1982). Quay hypothesized

that the main dysfunction in the hyperactive/impulsive and combined-types of ADHD (like many other researchers, both he and, later, Barkley posited that the purely inattentive subtype may reflect a separate disorder, an idea which will be discussed in greater depth later in this dissertation) was underactivation of the BIS, which lead to poor and improper behavioral inhibition. Barkley (1997) also theorized that response inhibition was the central deficit in ADHD. However, he then linked this response inhibition deficit to a subsequent breakdown in four separate executive self-control functions whose successful implementation relies on intact inhibition. As Barkley (1997) put it, response inhibition “permits a delay in the decision to respond that is used for further self-directed, executive actions” (p. 68). Disruption of these executive functions then results in subsequent disruption of motor output due to the decreased control of this output by the self-regulation executive functions. Much research conducted since this model’s inception has supported the existence of response inhibition and moderate executive functioning deficits in ADHD (Barkley, 1997, 1999a; Fischer et al., 2005; Kevin R. Murphy, Barkley, & Bush, 2001). However, meta-analytic studies have shown that neuropsychological deficits beyond, and potentially unrelated to, executive functioning also occur in ADHD (Boonstra, Oosterlaan, Sergeant, & Buitelaar, 2005; Woods, Lovejoy, & Ball, 2002). Thus, a response inhibition deficit in ADHD, while supported by much research, may not necessarily reflect the disorder’s central dysfunction.

### **Diagnostic Factors and Controversies**

DSM diagnostic criteria for ADHD, as mentioned earlier in this paper, changed significantly from DSM-III to DSM-III-R, and again from DSM-III-R to DSM-IV. Currently, the DSM-IV-TR, which made no substantive changes to its ADHD section compared with DSM-IV, requires that individuals experience at least six symptoms of inattention (criterion A) or

hyperactivity/impulsivity (criterion B) to receive an ADHD diagnosis (APA, 2000). Additionally, these symptoms must have persisted for at least six months, must have begun causing impairment to some degree before age seven years, must be present in at least two separate settings (such as school and home), must currently be causing clinically-significant impairment, and must not be better accounted for by another psychological disorder such as schizophrenia, a pervasive developmental disorder (e.g., intellectual disability, autism), or a psychotic disorder. The DSM-IV-TR, for its A and B criteria, provides three separate symptom lists—inattention, with nine entries; hyperactivity, with six entries; and impulsivity, with three entries. These latter two categories, hyperactivity and impulsivity, are both grouped under the B criteria, while the inattention symptom list alone comprises criteria A. Subtype diagnosis is then determined by whether the individual meets the full six-symptom threshold within the past six months for criteria A but not B (predominantly inattentive type), B but not A (predominantly hyperactive/impulsive type), or both A and B (combined type). If the individual meets neither criteria A nor B, or if the clinician is unsure regarding other diagnostic issues (e.g., if the individual began displaying symptoms before age seven), but the clinician feels an attention disorder is present, there exists an ADHD Not Otherwise Specific category as well.

Given its high degree of diagnostic flux across DSM revisions, it is likely not surprising that the ADHD symptom lists and criteria have received their fair share of criticism. One of the more frequently-mentioned of these criticisms relates to the addition of the age of onset criteria in DSM-III, which required that symptoms begin manifesting themselves before age seven (APA, 1980). Shockingly, despite this criteria having been implemented in all future revisions and editions of the DSM since then (APA, 1987, 1994), there was little to no evidentiary basis supporting it when it was originally proposed (Barkley, 2010). A review of the DSM-IV field

trials data for ADHD indicated that the age of onset criteria actually reduced diagnostic reliability and clinician agreement ratings, in part owing to it not being met in a substantial minority (up to 43%, depending on subtype) of cases (Applegate et al., 1997). Barkley and Biederman (1997) also reported that age of onset less than or greater than seven years, while being associated with an average of 1.2 more reported symptoms in childhood, has not been shown to significantly relate to measures of severity, impairment, achievement, or psychological adjustment, and thus may not relate to any true differences in etiology or outcome. The age of onset criteria can become even more troublesome when it is applied to adults making retrospective report of ADHD onset and symptoms, as will be discussed later.

Also controversial are the behavioral symptoms themselves. As mentioned earlier, boys are diagnosed with ADHD at significantly higher rates than girls. Some of this diagnostic discrepancy may be due to gender differences in expressions of the disorder. Boys diagnosed with ADHD tend to show more externalizing-type behaviors, such as hyperactivity, oppositionality, and aggression (Biederman et al., 2002; Gershon, 2002). Conversely, girls with ADHD exhibit more internalizing-type behaviors such as inattention and anxiety, display fewer problems behaviors, and have lower likelihoods of comorbid learning and conduct disorders than boys (Biederman et al., 2002; Gershon, 2002; Keenan & Shaw, 1997). Girls may then less-often come to the attention of referral sources, resulting in disproportionate numbers of referrals and diagnoses in boys (Stefanatos & Baron, 2007). This referral bias may have contributed to previous findings indicating that over three-quarters of research participants in ADHD studies were male (Hartung & Widiger, 1998), resulting in many of the DSM-IV ADHD symptoms proposed by expert opinion, literature review, and field trials to be based on experimental data

obtained predominantly with boys (Stefanatos & Baron, 2007). Thus, in addition to referral source biases, the symptom lists themselves may be predisposed to underdiagnosing females.

Despite the controversial nature of the age of onset criteria and the DSM symptom lists, there are facets of the diagnostic characteristics of DSM-defined ADHD that research findings support. When progressing from DSM-III to DSM-IV, as alluded to above, field trials were conducted to obtain a better idea of the construct of ADHD itself. These trials revealed that three distinct disorder subtypes, named predominantly hyperactive/impulsive, predominantly inattentive, and combined, emerged based on significant variations between them in client age, type of impairment, and sex (Lahey et al., 1994). Since that time, the predominantly hyperactive/impulsive and combined subtypes have held up relatively well to scientific scrutiny, albeit with some struggle. For example, Barkley (1997) has suggested, based on DSM-IV field trial data indicating that the predominantly hyperactive-impulsive subtype was found largely in preschoolers while the combined and predominantly inattentive types were more prevalent in older children (Applegate et al., 1997), that the hyperactive-impulsive condition is actually the predecessor of the combined-type condition. Thus, he proposes that the two are in fact the same disorder, simply in different stages of development and presentation. Research showing that behaviors associated with hyperactivity/impulsivity, such as disruptiveness and oppositionality, arise earlier than signs of inattention (Hart et al., 1995; Loeber, Green, Lahey, & Christ, 1992) would appear to support Barkley's proposal. Regardless, recent data continues to support the viability of hyperactive and impulsive symptom clusters, which are generally considered to belong to the same underlying behavioral factor, and which often co-occur with inattention as a second factor, in individuals diagnosed with ADHD (Glutting, Youngstrom, & Watkins, 2005;

Kessler et al., 2010; Lahey, Pelham, Schaughency, & Atkins, 1988), strengthening their cases as behavioral manifestations of the disorder.

The predominantly inattentive subtype, on the other hand, has received much scrutiny regarding its relationship to ADHD. While researchers such as Barkley (1997) contend that the hyperactive/impulsive and combined subtypes are not independent diagnostic entities, and instead may reflect a single ADHD condition as it progresses through development, the opposite is true of the predominantly inattentive type when in the absence of hyperactivity—there are many who believe it to be a fundamentally different disorder from ADHD, brought about by potentially distinct etiological factors (Barkley, 1997, 1999a; Carlson & Mann, 2002; Hinshaw, 2001; Sagvolden et al., 2005; Taylor et al., 1998; Willcutt, Pennington, & DeFries, 2000). Indeed, the DSM-IV field trial data suggests that the types of inattention symptoms exhibited by some children with the predominantly inattentive type appear fundamentally different than those observed and reported in hyperactive/impulsive and combined-type children (Applegate et al., 1997; Lahey et al., 1994). These children seem to display a cognitive and interpersonal dynamic disparate from hyperactive/impulsive individuals, coming across as socially withdrawn/introverted, cognitively and behaviorally sluggish, and day-dreamy rather than overly talkative, aggressive, and impulsive (Carlson & Mann, 2000; McBurnett, Piffner, & Frick, 2001; Milich, Balentine, & Lynam, 2001). These observations led some researchers to begin exploring the quantitative differences between children who are primarily inattentive without hyperactivity and those who display a more “typical” ADHD, with one such finding being that inattentive-only type deficits in particular may be consistent with dysfunction in right cerebral hemisphere neural networks (Stefanatos & Wasserstein, 2001). One of the more consistent discoveries from this research involves the characteristic of cognitive sluggishness, a phenomenon which has been

termed Sluggish Cognitive Tempo (SCT; Carlson & Mann, 2000; Lahey et al., 1988). This deficit in cognitive speed is particularly important because it may reflect a central dysfunction in the predominantly inattentive/non-hyperactive subtype, given that much like with the combined subtype, basic attention mechanisms do not appear to be disrupted in this condition (Huang-Pollock, Nigg, & Carr, 2005; Weiler, Bernstein, Bellinger, & Waber, 2002).

Before the particularities of SCT are explored, however, it is important to mention a key diagnostic tenet related to ADHD predominantly inattentive-type. Clinicians and researchers have proposed that this subtype may in fact reflect three distinct groups of individuals—those with true inattentive-type ADHD, which may be similar to DSM-III's ADD without hyperactivity; those with combined-type ADHD whose hyperactive/impulsive symptoms are subthreshold but nonetheless present; and, in the case of adults, those former combined-type children who have “outgrown” their hyperactivity in later life (Barkley et al., 2008; Hinshaw, 2001; Milich et al., 2001). Purely inattentive individuals, then, are only those whose inattention occurs in the lifelong absence of hyperactive/impulsive symptoms. Thus, when studying the predominantly inattentive subtype, it is very important to somehow identify and separate out purely inattentive individuals from those who are closer in disorder characteristics to individuals with the combined type. This subtype heterogeneity may have been one contributing factor in producing inconsistent cognitive testing results across ADHD studies, as many such studies have not parceled out lifelong inattentive/non-hyperactive (i.e., “purely inattentive”) children from their experimental groups. Such heterogeneity could also have led to an obfuscation of findings related specifically to the predominantly inattentive type, which may have often been composed of both purely inattentives and individuals with subthreshold combined-type ADHD, thereby delaying recognition of non-hyperactive inattention as a potentially separate diagnostic entity.

Nonetheless, the field of ADHD study has now begun examining the differences and similarities between purely inattentive individuals and those diagnosed with the ADHD hyperactive/impulsive and combined-types, as well as other diagnostic groups such as individuals with reading disorder (Weiler et al., 2002). It is interesting to note that despite this relatively recent interest in purely inattentive individuals, and particularly in the associated concept of SCT, three items assessing SCT behavioral symptoms were actually included in the DSM-IV ADHD field trials after being identified by Lahey et al. (1988) as a separate factor specific to the DSM-III diagnosis of ADD without hyperactivity subtype. However, the items were eventually excluded from consideration due to their poor negative predictive power in the predominantly inattentive subtype; that is, although positive self-report responses on the items were associated with the presence of inattentive symptoms, negative self-report responses on the items did not often indicate the absence of disordered attention (Frick et al., 1994). Researchers have since pointed out that poorer negative predictive power would be expected if the items are used in a sample consisting of both true inattentives and individuals with hyperactive/impulsive symptoms who display their own “brand” of reported inattention (McBurnett et al., 2001; Penny, Waschbusch, Klein, Corkum, & Eskes, 2009).

Poor negative predictive power notwithstanding, researchers have continued to study the concept of SCT as it relates to purely inattentive individuals. SCT itself may represent an inability to consistently muster cognitive alerting and orienting resources, thereby leading to the observed and reported characteristics of sluggishness, day dreaming, and drowsiness (Lahey et al., 1988; McBurnett, Pfiffner, & Frick, 2000). These characteristics have been substantiated with the development of observational informant-report items and measures that validly and reliably relate to SCT (Carlson & Mann, 2002; Garner et al., 2010; Hartman et al., 2004;



McBurnett, Pfiffner, & Frick, 2000; Penny et al., 2009). Unfortunately, owing to much of the existing SCT research having been conducted with children, few studies have examined the use of rating scales in adults to assess SCT symptoms. The limited data that does exist is promising, suggesting that some widely-used self-report inventories such as the Personality Assessment Inventory (PAI; Morey, 1991) may be linked to ADHD and/or SCT characteristics in adults. These data include significant correlations between elevations on the Antisocial Behaviors, Activity, Aggression, and Schizophrenia scales of the PAI and either a diagnosis of ADHD, or results on measures of retrospective adult report of childhood inattention symptoms (Billingsley-Jackson, 2009; Hill et al., 2009; Sadler, 2009).

The Wender Utah Rating Scale (WURS; Ward, Wender, & Reimharr, 1993) is another adult self-report inventory that, for reasons to be discussed shortly, might be useful in assessing SCT symptoms. Thus far, however, research has only focused on its utility in diagnosing ADHD. As mentioned earlier in this paper, the measure itself is a retrospective self-report questionnaire of childhood interpersonal, cognitive, and behavioral characteristics. It was initially developed in an attempt to help clinicians establish the presence of a childhood onset of ADHD symptoms (Ward, Wender, & Reimharr, 1993). The measure's possible utility with respect to identifying SCT in adults lies in the distinct qualitative similarities between a handful of the individual WURS items and the aforementioned qualitative behavioral characteristics of individuals with SCT. These items include, "inattentive daydreaming;" "shy, sensitive;" and, to address the sluggish component of SCT, "slow reader." To date, though, no research exists identifying these items' relationship to SCT. Indeed, only one study was found that has assessed SCT in adults using self-report scales at all (Barkley, 2011b), with its authors having added a list of nine SCT symptoms to the existing Adult ADHD Rating Scale-IV (Barkley, 2011a). Results from this

study indicated that adults with SCT symptoms reported greater difficulties with self-reported disorganization and problem solving than adults with ADHD and adults with both ADHD and SCT symptoms. Additionally, adults with SCT symptoms reported more educational/academic and employment problems, and had a lower income, than adults with ADHD only, and had fewer years of education than adults in the control group.

However, despite these relatively promising investigations of personal and interpersonal characteristics in SCT, very little work has been done to examine any objective cognitive/neuropsychological deficits in the condition. Those studies that have explored cognitive performance in individuals with SCT have reported promising results, suggesting deficits in speeded visual processing, especially with increased/parallel cognitive demands, compared to children with reading disorder (Weiler et al., 2002) and, relatedly, early selective attention when compared to other children with ADHD (Huang-Pollock, Nigg, & Carr, 2005). This latter study in particular included a variety of neuropsychological measures, although additional deficits may not have been discovered due to significantly limited statistical power owing to a very small SCT group (initially seven individuals, and expanded to twelve for further analyses; Huang-Pollock, Nigg, & Carr, 2005). Additionally, both studies focused exclusively on children.

**Diagnostic Criteria in Adults.** Much as with questions regarding the appropriateness of subsuming the predominantly-inattentive subtype of ADHD under the same diagnostic entity as the predominantly hyperactive/impulsive and combined subtypes, the applicability of existing DSM-IV-TR diagnostic criteria as a whole to adults has been repeatedly questioned by researchers and clinicians (Barkley, 2010; Barkley et al., 2008; Conners et al., 1999; McGough & Barkley, 2004; Roy-Byrne et al., 1997). Conners and colleagues (1999) voice concerns that

these criteria were developed only on children and adolescents, and were not informed regarding any phenomenological, functional, or threshold changes in symptom type or expression with age. Barkley (2008) later evaluated this last tenet regarding symptom threshold in adults in the context of his own and others' research, and in light of the DSM's own wording that ADHD reflects developmentally-inappropriate symptom expression (APA, 2000). He found that the number of symptoms needed to separate adults diagnosed with ADHD from their non-diagnosed peers, using the often-employed criteria for clinical impairment of 1.5 and 2 standard deviations' difference, was fewer than that currently required by the DSM-IV-TR. In keeping with his own theory of executive dysfunction/behavioral disinhibition in ADHD, Barkley also found that executive-related behavioral symptoms assessed via self- and informant-report (e.g., parent, spouse) were able to contribute significantly to diagnostic accuracy in identifying adults with ADHD. Kessler and colleagues (2010) further supported the inclusion of executive functioning symptoms in the diagnosis of ADHD by showing that they represented one of three key factors in adult ADHD (the other two being inattention/hyperactivity and impulsivity), and that they were more specific to adult ADHD versus comorbid conditions than either of the other two factors. These findings have since caused Barkley and others to suggest changes in the symptom lists that are used for diagnosis in adult ADHD (Barkley, 2010; Kessler et al., 2010).

Another point of contention in the diagnosis of ADHD in adults is the age of onset criteria. Given that this concept has, nearly since its inception, been the recipient of multiple criticisms in children and adolescents, it is understandable that its footing is even more tenuous with respect to adults. Indeed, researchers have shown that the number and type of recalled symptoms reported in adults can vary by reporting source (Barkley et al., 2002) and age of the individual with ADHD (Barkley et al., 2008). In a 16-year prospective follow-up study using a

semi-structured interview with trained interviewers, Mannuzza et al. (2002) found that of 176 adults diagnosed by this research group with ADHD in childhood, 22% would not have received a diagnosis of ADHD based on their retrospective self-report of childhood symptoms. Additionally, 11% of control participants were falsely classified as having ADHD based on retrospective self-report. The authors indicate that with untrained or less-trained raters, or in a lower base-rate setting of ADHD (such as the national prevalence of approximately 5% in adults, which would be applicable to non-clinic-referred individuals), predictive rates would have been even lower. These findings cast considerable doubt on retrospective adult recall of childhood symptoms as a whole, especially when one is considering a time frame restricted to seven years of age or less. However, other researchers have found that younger adults (i.e., early twenties) tend to recall childhood symptoms less-accurately than older adults (i.e., late twenties and early thirties; Barkley et al., 2008), potentially explaining how some studies have found that adults do accurately recall childhood ADHD symptoms (albeit in a sample not necessarily known to have had diagnoses of ADHD as children; Murphy & Schachar, 2000). Additionally, research suggests that current self-report of symptom severity by adults with ADHD is consistent with levels of symptom severity observed by independent observers (Downey, Stelson, Pomerleau, & Giordani, 1997).

Despite the ambiguous findings regarding the accuracy of adult self-report of childhood ADHD symptoms, the current age of onset criteria have necessitated the development of retrospective self-report measures, particularly in cases where collateral reports (e.g., parents, teachers, school records) are sparse or non-existent. The Wender Utah Rating Scale represents one such measure. In its initial development and validation study, it was found to correctly classify 86% of individuals with ADHD, 99% of controls, and 81% of individuals with

depression (included by the authors because their symptoms were reportedly sometimes similar to those exhibited or reported by individuals with ADHD; Ward, Wender, & Reimherr, 1993). However, with a patient group of 81 individuals, a control group of 100 individuals, and a depression control group of 70 individuals, the experimental base-rate of ADHD would have been approximately 33%, which is markedly higher than that in the general population, and thus may have inflated predictive accuracy rates. Some later work continued to find higher WURS scores in adults with ADHD versus those not diagnosed (Roy-Byrne et al., 1997), although other studies found that despite having high sensitivity, the WURS displayed rather low specificity (i.e., it incorrectly identified 42.5% of experimental controls as having ADHD; McCann, Scheele, Ward, & Roy-Byrne, 2000) and that WURS scores were actually more strongly-correlated with responses on a measure of personality/psychological functioning than with neuropsychological measures of attention (Hill, Pella, Singh, Jones, & Gouvier, 2009). Thus, while the WURS and other such measures reflect admirable efforts to establish childhood impairment and thereby appease DSM diagnostic requirements, they have done little to quell the debate surrounding assessment of ADHD in adults, especially those without established childhood diagnoses.

### **Neuropsychological Assessment**

Between ambiguities regarding the accuracy of retrospective self-report of childhood ADHD symptoms by adults, and controversies relating to the appropriateness of applying current DSM-IV-TR diagnostic criteria in adults, then, adult ADHD remains a rather contested sub-field of study and practice. Owing in large part to the subjective, and potentially-inaccurate, nature of retrospective self-report, as well as the idea that adults may exhibit ADHD symptoms in ways inconsistent with DSM-IV-TR symptoms, some researchers have begun pushing for more

objective measures of assessment. The direct testing of cognitive abilities, such as that afforded by neuropsychological assessment, has thus received increased attention in recent years. However, the neuropsychological assessment of ADHD is not without its own series of controversies and uncertainties.

Perhaps the largest uncertainty regarding neuropsychological assessment in ADHD is its actual utility, based in large part on research findings of limited or non-specificity. Owing to only modest between-groups comparison effect sizes on some neuropsychological tests, up to half of the ADHD and control group score distributions of various cognitive measures may overlap (Nigg, Willcutt, Doyle, & Sonuga-Barke, 2005). Thus, on some such instruments, the ADHD and control groups may nearly be as alike as not. However, effect sizes on some of the more consistently-demonstrated neuropsychological deficits in childhood and adolescent ADHD do at least fall in the moderate range (Nigg, 2005). However, until recently, relatively few neuropsychological studies have evaluated performance in adults diagnosed with ADHD, with fewer still focusing on the predominantly-inattentive subtype (Nigg, 2005), resulting in a large amount of scientifically-unexplored territory.

Nonetheless, neuropsychological assessment provides the opportunity to objectively assess cognitive domains thought to be central to ADHD, such as executive functioning and behavioral inhibition (Barkley, 1997, 1998), effort and motor output organization (Sergeant, 2000; Sergeant & Van der Meere, 1990), and intra-individual performance variability (Castellanos & Tannock, 2002; Douglas, 1999). Such objective assessment is particularly important given that these cognitive functions likely cannot be adequately assessed via behavioral rating scales, observation, or subjective self-report measures. Additionally, behaviorally-based symptoms of ADHD, such as those listed by the DSM-IV-TR, are

subjectively reported by large proportions of non-diagnosed (i.e., healthy) individuals, sometimes even to diagnostically-significant levels, suggesting that symptom report alone is not a sufficient diagnostic criteria (DuPaul et al., 2001; Lewandowski, Lovett, Coddington, & Gordon, 2008; K. R. Murphy, Gordon, & Barkley, 2000; Weyandt, Linterman, & Rice, 1995). Thus, objectively establishing some marker of symptom presence and severity, such as via neuropsychological testing, is crucial to proper diagnosis. Neuropsychological testing can also provide unique insights into potential ADHD endophenotypes, defined by Castellanos and Tannock (2002) as, “quantitative indices of disease liability or risk” (p. 619). The term endophenotype itself originated in insect biology (John & Lewis, 1966) and was applied to mental illness, and particularly schizophrenia, by Gottesman and Shields (1972, 1973). Gottesman and Gould (Gottesman & Gould, 2003) described the purpose of endophenotypes as, “[marking] the path between the genotype and the behavior of interest” (p. 637). Endophenotypes, then, can provide distinctive views on the connections between complex disease concepts and genetics (Gottesman & Gould, 2003).

With respect to cognitive domains that hold promise as endophenotypes in ADHD, reaction time (RT) has been an area of frequent, and relatively fruitful, study in various areas of the cognitive neurosciences, and particularly in neuropsychology. An increase in RT variability, or increased fluctuations of RT across a particular task or series of tasks, has been one of the longest-standing and most consistently-demonstrated findings in all of ADHD-related neuropsychological research (N. J. Cohen & Douglas, 1972; Douglas, 1999; Huang-Pollock et al., 2005; Hurks et al., 2005; Kaufmann et al., 2010; Klein, Wendling, Huettner, Ruder, & Peper, 2006; Leth-Steensen, Elbaz, & Douglas, 2000; Sergeant & Van der Meere, 1990; Zahn, Kruesi, & Rapoport, 1991). One rationale driving the multitude of research into RT has been its

purported role as a directly-measurable proxy for attention in general, and sustained attention in particular, which some theorists posit is one core area of dysfunction in ADHD (Douglas, 1999; Van der Molen, 1996). However, despite the multitude of studies having demonstrated various RT variability phenomena in ADHD, the broader concept of attention has not always been observed as deficient in individuals diagnosed with the disorder (Loo et al., 2003; van der Meere & Sergeant, 1988; van der Meere, Wekking, & Sergeant, 1991). Some feel this inconsistency suggests that attention, or at least specific attention components such as selective attention, may not be the core deficit of ADHD (Barkley, 1997; Huang-Pollock et al., 2005), while others believe the ambiguity may be due to variations in experimental paradigms such as testing instruments, study designs (e.g., length of sustained attention task, frequency of response targets), and sample characteristics (e.g., age, gender, ADHD subtype) (Douglas, 1999).

Ambiguity of attention deficits notwithstanding, the neural underpinnings of sustained attention and, perhaps more specifically, of task consistency are somewhat more agreed-upon. The ability to adequately and consistently maintain task engagement has been associated with the top-down attentional control system and its dorsolateral prefrontal and frontal-striatal cortical networks mentioned earlier in this paper (Banich et al., 2009; Barkley, 1997; Bellgrove et al., 2004; Stuss, Murphy, Binns, & Alexander, 2003). It is this attentional network that Castellanos et al. (2005) thought might be influenced by the brief, frequently-occurring attentional lapses induced by disrupted catecholaminergic regulation in individuals with ADHD. These frequent, short lapses then may lead to the occurrences of inconsistent performance that are captured by measures of RT variability.

Castellanos and colleagues' theory regarding RT variability was informed by early work examining RT in ADHD. This initial research focused primarily on measures of central tendency



and dispersion when collecting and examining sample data. In so doing, researchers found deficits in overall RT (i.e., slowed responding) as well as RT standard deviation (RTSD; a measure of RT variability) in individuals diagnosed with ADHD (N. J. Cohen & Douglas, 1972; Epstein, Conners, Sitarenios, & Erhardt, 1998; Zahn et al., 1991). Information has also been gathered from non-ADHD research. Indeed, intraindividual variability (IIV) is not, as an overarching concept, unique to ADHD. Research has indicated that IIV is associated with a variety of neurodegenerative diseases such as Alzheimer's dementia and Parkinson's disease (Hultsch, Strauss, Hunter, & MacDonald, 2008), HIV infection and associated cognitive decline (Morgan, Woods, Delano-Wood, Bondi, & Grant, 2011), and brain injury/insult (Stuss et al., 2003; Stuss, Pogue, Buckle, & Bondar, 1994; Stuss et al., 1989).

In the wake of these and other ADHD and non-ADHD studies, greater attention was paid to RT and variability data to determine the exact nature of the deficits and mechanisms involved in ADHD. Consequently, slowed RT findings in ADHD have not always held up to this increased scrutiny. Several subsequent studies have failed to show significant mean RT differences between experimental and control groups, especially when fitting data to the ex-Gaussian curve in an attempt to control for the positive skew frequently found in ADHD RT data (Buzy, Medoff, & Schweitzer, 2009; Geurts et al., 2008; Hervey et al., 2006; Leth-Steensen et al., 2000). Conversely, RT standard deviation (RTSD) differences data have fared much better. As mentioned earlier in this paper, RTSD disruptions have since gone on to become some of the most consistently-replicated findings in ADHD literature (Nigg, 2005), with recent studies still choosing to examine this value and/or use it as a proxy of IIV (e.g., Klein et al., 2006; Wahlstedt, 2009).

Many neuroscience researchers such as Castellanos and colleagues, on the other hand, chose a slightly different analytic route. Rather than focusing on RTSD, these researchers have spent much time examining variability in smaller time increments and on a trial-by-trial basis. Using a data transformation technique known as the fast Fourier transform and subsequent wavelet analyses that allow for examination of the power of selected frequency bands, Castellanos et al. (2005) examined the performance of boys with and without diagnoses of ADHD. They not only found that children with ADHD exhibited greater overall RT variability (RTSD), but also that these children showed a specific increase in RT variability in the .05 Hz frequency band, and that variability in this frequency band was amenable to methylphenidate administration. Two teams of researchers (Hervey et al., 2006; Vaurio, Simmonds, & Mostofsky, 2009) expanded on these findings by evaluating larger samples and varying the complexity of experimental task demands. In both cases, the data indicated that, as with the Castellanos et al. (2005) study, children with ADHD exhibited greater overall amounts of RT variability regardless of task complexity (i.e., simple or complex). Vaurio, Simmonds and Mostofsky (2009) concluded that on simple tasks, children with ADHD exhibit occasional (every 15-40 seconds, or in the 0.027 to 0.075 Hz frequency band) excessively-slow responses corresponding to attentional lapses. Conversely, on complex tasks, children with ADHD exhibit slower, more variable responses throughout the entirety of the analyzed RT spectrum, which may implicate a fault in the top-down attentional system that allows for maintaining a readiness to respond to stimuli.

Other groups of neuroscientists have, in studying “resting” brain activity, made exciting discoveries that appear to be highly related to both ADHD and intraindividual variability. In a small series of articles and reviews, Raichle, Gusnard, and Snyder (Gusnard & Raichle, 2001;

Raichle & Gusnard, 2005; Raichle & Snyder, 2007) examined the decreases in brain activity that often accompany all variety of cognitive tasks. Interestingly, they found that while the location of some of these decreases was task-dependent, other decrease locations were task-independent, continually occurring in the same brain regions regardless of the type of cognitive task undertaken (Gusnard & Raichle, 2001). These authors proposed a hypothesis that this network of brain structures, which consists of the posterior (parietal) medial and lateral cortices, the ventromedial prefrontal cortex, the precuneus-posterior cingulate cortex, and the retrosplenial cortex, is actually responsible for maintaining a continuous brain resting state. This continuous resting state then allows for humans to maintain a constant, active connection with the surrounding world via orienting, monitoring, and extracting emotionally-salient information, which in turn helps to develop and sustain a stable sense of self with relation to the external (and internal) environment. With onset of goal-directed behavior, this network decreases in activity to “shut off” the continual, broad environmental monitoring in favor of directed attention and specific problem solving. Simultaneously with this decrease in task-independent network activity occurs an increase in activity in a separate task-active neural network containing structures that play integral roles in goal-directed activities, including the dorsolateral and ventral prefrontal cortices, the pre-motor or supplementary motor area, and the intraparietal sulcus (Fox et al., 2005; Fransson, 2006). Thus, there appear to be two networks, working in concert, that allow for both a resting-state level of baseline attentional activity and a more focused and sustained level of increased attention necessary for goal-directed behaviors.

Sonuga-Barke and Castellanos (2007) then explored this neuroscientific finding as it might relate to ADHD and, specifically, to IIV-related dysfunction in the disorder. These authors took the above discoveries regarding the task-independent network (termed the Default Mode

Network; DMN) and integrated them with behavioral data showing that momentary attentional lapses are associated with incomplete deactivation of certain components of the DMN (Weissman, Roberts, Visscher, & Woldorff, 2006). In so doing, they developed a unique DMN hypothesis of ADHD. Their hypothesis theorizes that ADHD attentional disturbances are due to dysfunctional transitions from the DMN to the task-active network that allows for focused processing and goal-directed activity. Such dysfunction leads to interference in attention-laden processing via the incompletely-deactivated DMN, which results in short, frequent RT fluctuations (in the aforementioned 0.027 to 0.075 Hz range) that cause brief, recurrent attentional lapses. This theory scientifically bolsters itself by incorporating various data streams, including neural morphometric data, mentioned earlier in this paper, showing decreased retrosplenial cortical thickness (Makris et al., 2007) and reduced precuneus-posterior cingulate cortex grey matter volume (Carmona et al., 2005; Overmeyer et al., 2001) in adults with ADHD, as well as functional neuroimaging findings implicating heightened (and thus dysfunctional) DMN activity in children and adolescents with ADHD during active, attention-laden tasks; see: Castellanos, Kelly, & Milham, 2009 for a review).

Its significant promise as an endophenotypic marker of ADHD notwithstanding, IIV's current validity and applicability as a concept is not without its limitations, owing largely to the features of its supportive studies. One near-global shortcoming of existent IIV research, particularly concerning the efforts of neuroscience-focused methodologies, pertains to sample characteristics. As was once the case for the majority of ADHD research, current IIV data has been gathered largely in children and adolescents (primarily boys), with comparatively little data on adults. Of the studies that have evaluated IIV in adult ADHD, most (Adams, Roberts, Milich, & Fillmore, 2011; Epstein et al., 1998; Halperin et al., 2008; Hinshaw et al., 2007), but not all

(Drechsler et al., 2005), have found variability-related differences, and are thus supportive of persisting effects into adulthood. In the case of Drechsler et al.'s (2005) non-significant results, it is possible that a relatively small sample size may have led to reduced statistical power in detecting significant findings.

While IIV across a single task, such as that captured by RTSD and RT fluctuations at specific frequencies of occurrence, has shown great promise and consistency in the neuropsychological ADHD literature, another concept related to performance consistency has also recently been increasingly studied, albeit in a different context. For decades, neuropsychologists have practiced the technique of examining IIV across not just a single test, but across a battery of tests or subtests assessing different skills, as a means of evaluating acquired cognitive deficits. Anastasi (1954) provided an early and eloquent explanation of the process of assessing intra-individual subtest scatter on a variety of measures, including the Wechsler-Bellevue Intelligence Scale (Wechsler, 1946). Current work, which has sometimes labeled this concept of performance variability across an entire testing session or sessions “dispersion,” continues to build on and use those ideas espoused by Anastasi and others. Lindenberger and Baltes (1997), for example, evaluated inter- and intra-individual variability in older adults (aged 70+) to determine if dispersion across cognitive testing domains increased with age. In so doing, they developed a measure of dispersion known as the intraindividual standard deviation (ISD). By converting summarizing scores in each assessed cognitive area into  $z$  scores, Lindenberger and Baltes were able to then calculate the standard deviation across these  $z$  scores for each individual, with higher values indicating greater dispersion and lower values indicating less dispersion. Others have since adopted this same methodology in further study of dispersion amongst older adults with (Christensen et al., 1999) and without dementia (Hilborn,

Strauss, Hultsch, & Hunter, 2009), as well as older adults diagnosed with HIV (Morgan et al., 2011).

To date, however, no studies have examined ISDs in ADHD. One group of researchers has examined a similar concept, the coefficient of variation (SD divided by the mean), in children with ADHD (Klein et al., 2006). They found that, as with RTSD, children with ADHD exhibit significantly greater coefficients of variation than non-diagnosed children. Unfortunately, Klein and colleagues limited their study to only heavily attention-based measures, foregoing other areas of cognitive functioning such as memory and intelligence. Thus, while these results regarding dispersion as a deficit in ADHD are promising, further research is needed, as the spheres assessed could greatly influence the dispersion results obtained, perhaps even changing their interpretation.

### **Rationale for Current Study**

Neuroscience and neuropsychological researchers have frequently called for further study focused on scrutinizing and reducing heterogeneity in ADHD (Castellanos et al., 2009; Nigg, 2005; Nigg et al., 2005; Sonuga-Barke & Castellanos, 2007). These scientists hope that such reductions in heterogeneity will aid in producing more consistent and relevant research findings, thereby increasing our overall understanding of ADHD as a whole. One means by which some authors hope to accomplish these goals is a process Castellanos, Kelly and Milham (2009) have termed “endophenotypic fractionation” (p. 670). This fractionation involves breaking ADHD down from its overarching phenotype(s) into less-complex, more genetically- and neuroscientifically-linked endophenotypes (Castellanos, Glaser, & Gerhardt, 2006), thereby allowing for the development of more accurate pathophysiological models of the disorder (Sonuga-Barke & Castellanos, 2007). IIV, as often measured by RT variability and as also might

be captured by dispersion analysis, represents one such well-supported ADHD endophenotype that has been suggested for use in future research (Castellanos et al., 2009; Castellanos & Tannock, 2002; Rommelse et al., 2009; Rommelse et al., 2008; Sonuga-Barke & Castellanos, 2007; Uebel et al., 2010). Given that IIV is such a consistent finding in ADHD, one could posit that it may, as an endophenotype, represent an underlying dysfunction in the disorder. Individuals with ADHD could be then expected to universally exhibit IIV, suggesting its use as a neuropsychological marker for the condition. What remains to be examined, though, is if IIV or a particularly type of IIV is in fact unique to ADHD, or if it might instead simply reflect a general cognitive dysfunction in a variety of syndromes that affect brain functioning (e.g., brain injury, dementia, encephalitis, etc.).

SCT, a potential marker of another kind, has been suggested as a key characteristic of those individuals diagnosed with the predominantly-inattentive ADHD subtype who actually have a purely inattentive/non-hyperactive condition (Carlson & Mann, 2000; Carlson & Mann, 2002; Garner, Marceaux, Mrug, Patterson, & Hodgens, 2010; Hartmann, Willcutt, Rhee, & Pennington, 2004; McBurnett et al., 2001; Penny et al., 2009). Given that this purely inattentive condition may reflect a fundamentally different disorder than ADHD, the use of SCT to separate these two sets of disorders and thereby reduce group heterogeneity would be significantly beneficial. However, cognitive sluggishness is a concept without a clear link to any one particular neuropsychological testing deficit. Thus, unlike RT variability, its viability as a unique neuropsychological marker needs further research.

However, there does exist a key area of difference, with empirical support, between individuals with lifelong inattention/non-hyperactivity and individuals with ADHD (i.e., those with a history of hyperactivity/impulsivity, possibly in addition to attention): qualitative

interpersonal, behavioral, and emotional characteristics. Individuals with lifelong inattention tend to be described by parents and teachers as more apathetic, sluggish, socially introverted and withdrawn, day-dreamy, and anxious while children and adults with ADHD and a history of at least some hyperactive/impulsive symptoms are more outgoing, potentially aggressive, prone to interrupting, and have increased incidence of substance abuse and antisocial personality characteristics such as stimulus-seeking (Carlson & Mann, 2002; McBurnett, Pfiffner, & Frick, 2001; Milich, Ballentine, & Lynam, 2001). Therefore, an inventory containing items and/or scales assessing the qualitative social, behavioral, and emotional characteristics associated with SCT could be used to identify the subset of individuals diagnosed with ADHD who are actually lifelong inattentive. Such inventories have been created (e.g., Carlson & Mann, 2002; Penny et al., 2009), but have thus far been restricted to children and adolescents. Yet given that ADHD symptoms appear to change in type and severity as individuals age, it is possible that SCT and purely-inattentive symptoms may do the same, and thus SCT inventories developed with children may not be applicable to adults. Unfortunately, there exist no studies to date that have examined the longitudinal progression of purely-inattentive/SCT symptoms into adulthood. In fact, only one study has evaluated the construct of SCT in adults at all (Barkley, 2011); and while the author did assess a construct of cognition (executive functioning), this was done exclusively by self-report rather than objective cognitive test performance.

Thus, a battery of assessment instruments containing measures of both neuropsychological functioning and qualitative personality, behavioral, and emotional characteristics could uniquely add to the existing literature by using both variability (e.g., RT standard deviation, dispersion, etc.) and social functioning to explore endophenotypes and diagnostic heterogeneity in ADHD and pure inattention. While previous studies have examined



differences in neuropsychological functioning across ADHD subtypes, these results have been limited and equivocal regarding IIV. The majority of this research has examined IIV via RT variability, with some results showing significant differences between inattentive versus combined and hyperactive/impulsive types (Clarke et al., 2007; De Zeeuw et al., 2008; Mullins, Bellgrove, Gill, & Robertson, 2005) and others showing minimal or no subtype discrepancies (Epstein et al., 2011; Pasini, Paloscia, Alessandrelli, Porfirio, & Curatolo, 2007). Additionally, only one study to date has evaluated IIV as measured by dispersion in ADHD. However, none of these studies attempted to separate out purely inattentive individuals. Rather, they typically divided groups by DSM subtype diagnosis, which likely confounded their inattentive group due to its consisting of purely inattentive individuals as well as subthreshold combined-type individuals. Additionally, all of these studies recruited only children and adolescents. Thus, few studies have examined IIV (particularly via dispersion analysis) in ADHD, and even fewer studies in any realm of ADHD research have used an extensive cognitive testing assessment battery spanning multiple neuropsychological domains. Likewise, nearly all work evaluating SCT has been conducted using children and adolescents. Indeed, as mentioned earlier, only one study has attempted to identify adults with SCT to any degree, and it exclusively utilized self-report measures (Barkley, 2011). No study to date has examined RT variability or dispersion, or essentially any type of neuropsychological test performance, in a subgroup of adults suspected of having a purely inattentive condition as determined by self-reported interpersonal, behavioral, and emotional characteristics. Similarly, no study has evaluated the concept of dispersion in an adult ADHD sample as measured across a neuropsychological testing battery assessing multiple cognitive domains regardless of how the sample was grouped/divided.

The present study, therefore, aimed to address these deficits in existing research via three goals. First, the study attempted to evaluate the viability of using an existing retrospective self-report measure of childhood ADHD symptoms and associated characteristics (the WURS), rather than relying entirely on DSM diagnosis, to identify and separate out a “purely inattentive” group of adults. It was hypothesized that such classification would be possible, with one identified group reporting childhood characteristics similar to those associated with SCT (i.e., purely inattentive individuals), and another identified group reporting characteristics more typically associated with ADHD (hereafter referred to as “purified ADHD”). Second, the study aimed to compare these purely inattentive and purified ADHD groups on two measures of IIV, RT variability and battery-wide dispersion, with the hypothesis that if IIV reflects a unique endophenotype of ADHD, then the purified ADHD group should be significantly more variable than the purely inattentive group. Third, the study sought to explore possible neuropsychological correlates of SCT, with the hypothesis that the purely inattentive group would perform significantly more slowly on a variety of cognitive information processing speed-related tasks than the purified ADHD group.

**Rationale for Additional Grouping Methods.** It is of paramount importance to note here that had the current study failed with respect to the first of the three goals listed above, the second and third goals would be untestable. That is, if the WURS was unable to effectively distinguish purely inattentive and purified ADHD groups, the remainder of the study would be impossible to complete. Thus, two additional methods of grouping participants were added following the initial proposal of this project to ensure that variants of the second and third goals could be examined. These grouping methods will be described in greater detail in the Procedures section, but are briefly reviewed here. The initial classification method, using a variety of

ADHD-like and SCT-like WURS items to identify purely-inattentive and purified-ADHD groups, will hereafter be referred to as Method A. The first of the two new methods (Method B) involved classifying individuals by DSM-IV-TR ADHD subtype diagnoses, as has often been done in previous research. The second new grouping method (Method C) involved classifying individuals by their degree of SCT-like symptom endorsement on the WURS, and included the levels high-, moderate-, and low-SCT. As with ADHD subtype diagnosis, researchers have utilized similar grouping methods to this second option in prior work, although not nearly as extensively as DSM diagnostic category (e.g., Garner et al., 2010). The inclusion of these new methods resulted in the subsequent addition of a series of unproposed hypotheses, as listed below.

In reviewing prior research as related to Method B, I determined that existing data was too ambiguous to make *a priori* predictions regarding the directionality of results. Very few studies have examined the relationship between IIV and either DSM diagnostic category or symptom types (i.e., hyperactive/impulsive and inattentive). The vast majority of previous work in IIV either combined all individuals with ADHD into a single group, did not include any individuals with ADHD Predominantly Inattentive Type, or did not mention the subtype characteristics of the ADHD group (e.g., Castellanos et al., 2005; Drechsler et al., 2005; Geurts et al., 2008; Halperin et al., 2008; Hervey et al., 2006; Johnson et al., 2006; Kaufmann et al., 2010; Klein et al., 2006; Llorente et al., 2008; Rommelse et al., 2008). Only two studies were found that explored IIV (in the form of RTSD) in relation to ADHD subtype—one showed no significant differences between the Combined and Predominantly Inattentive types (Nigg, Blaskey, Stawicki, & Sachek, 2004), while the other indicated that the Predominantly Inattentive type was significantly more variable than Combined type and control groups (Desman,

Petermann, & Hampel, 2008). Thus, not enough data was present to predict directionality of any between-groups IIV effects for Method B. Conversely, the rationale for a directional hypothesis regarding SCT was readily apparent in previous research. Published findings have indicated that SCT symptoms are elevated and/or found more frequently in the Predominantly Inattentive type (Garner et al., 2010; Lahey et al., 1988; McBurnett, Pfiffner, & Frick, 2001), that SCT and Predominantly Inattentive type symptoms are much more strongly-correlated than SCT and Hyperactive/Impulsive type symptoms (Hartman et al., 2004), and that the presence of SCT predicted inattention symptoms (Frick et al., 1994).

The process of developing the hypotheses for Method C was a natural extension of that explored for the originally-proposed Method A hypotheses, particularly regarding performance on SCT-related neuropsychological measures. As Method C dealt with classification of groups based on degree of SCT-like WURS item endorsement, it would theoretically stand to reason that higher SCT symptoms would be associated with worse performance on SCT-sensitive neuropsychological variables. However, the hypotheses pertaining to IIV in Method C had to be treated slightly differently than in Method A, as the latter accounted for levels of ADHD-related symptoms (i.e., hyperactivity/impulsivity and inattention) while the former did not. That is, Method A attempted to identify and separate out those individuals with high SCT and low levels of ADHD symptoms from all other individuals with ADHD. Conversely, Method C did not attempt any such separation, and instead grouped all individuals by level of endorsed SCT-related symptoms regardless of level of ADHD symptoms. Thus, Method C in effect examined IIV as it related to SCT in a solitary state, separate from its connection to the purely inattentive condition. Only one relevant existing study was found that had explored this SCT-variability relationship, and it did so in a non-traditional and inadvertent sense (Skirbekk, Hansen, Oerbeck,

& Kristensen, 2011). Its authors discovered a significant correlation between SCT and variability in children/adolescents, but the measure of variability was one component of a spatial memory task rather than a well-founded measure of variability such as RTSD. I could therefore justify no directionality of predictions in the IIV-related hypotheses of Method C.

## **Hypotheses**

**Hypothesis 1: Identification of a Purely Inattentive Subgroup.** It was hypothesized that cluster analysis of adults diagnosed with ADHD would successfully identify two distinct groups based on retrospective self-reported interpersonal, cognitive, and behavioral characteristics in childhood as measured by selected items on the Wender Utah Rating Scale (WURS). These two groups would consist of one group interpersonally, behaviorally, and cognitively similar to individuals with SCT and a purely inattentive condition as defined earlier (i.e., a purely inattentive group), and one group consisting of all remaining individuals diagnosed with ADHD (i.e., a purified ADHD group). Specifically, the purely inattentive group should report higher scores on SCT-associated WURS items, as further specified in the Measures section, than the purified ADHD group. Conversely, the purified ADHD group should report greater amounts of ADHD-associated WURS symptoms such as Hyperactivity and Impulsivity, Inattention/Anxiety Symptoms, and Conduct Problems/Lability (also as further specified in the Measures section) than the purely inattentive group. A graphical representation of the hypothesized group formats is shown in Figure 1 below:

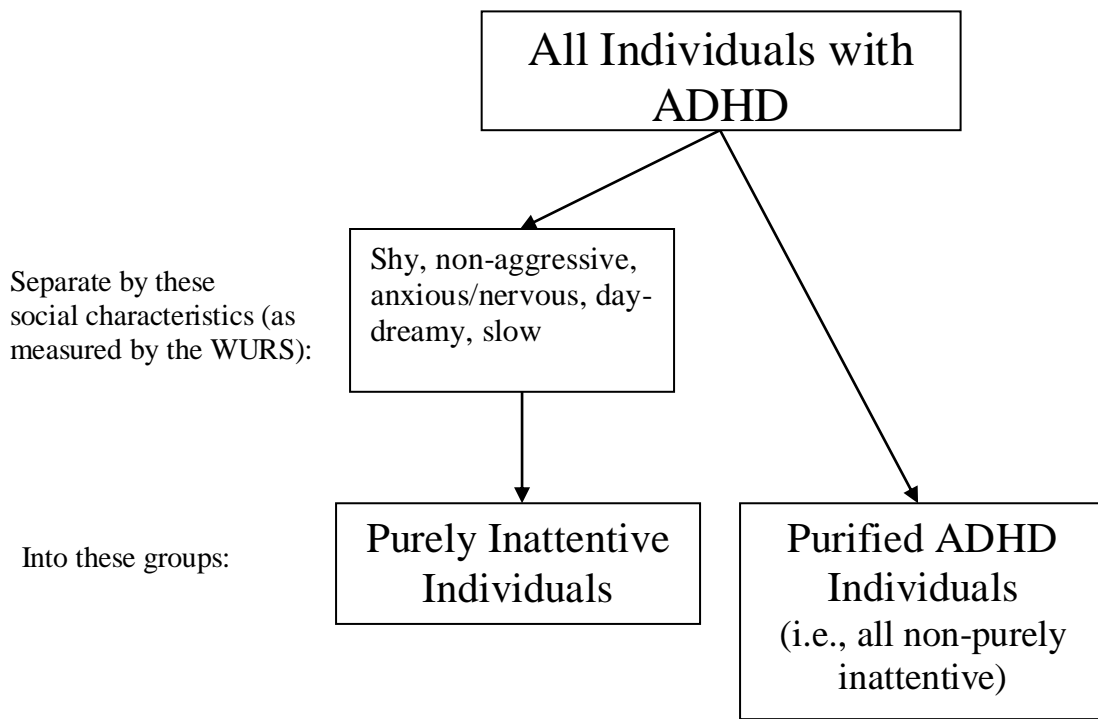


Figure 1: Schematic of Hypothesized Cluster Analyzed Groups

**Hypothesis 2a: Variability as Measured by RTSD/RTSE.** It was hypothesized that, as variability may reflect an endophenotype of ADHD, the purified ADHD group would display significantly more variability than the purely inattentive group. This variability would be reflected as larger RT standard error (RTSE; analogous to RTSD) scores on the Conners' Continuous Performance Test (Conners' CPT).

**Hypothesis 2b: RTSE Differences Across Method B Groups.** It was hypothesized that the two ADHD subtype groups, ADHD Predominantly Inattentive type and ADHD Combined type, would significantly differ with respect to IIV as measured by RTSE. No directionality of results was predicted.

**Hypothesis 2c: RTSE Differences Across Method C Groups.** It was hypothesized that the three Method C groups (high-SCT, moderate-SCT, and low-SCT) would differ significantly on RTSE scores. No directionality of results was predicted.

**Hypothesis 3a: Variability as Measured by Dispersion (ISD).** Similarly to RTSD/RTSE, it was hypothesized that the purified ADHD group would display significantly more variability as measured by battery-wide dispersion than the purely inattentive group. Dispersion was captured by the Intraindividual Standard Deviation (ISD; to be explained in the Procedures section below) scores for each group.

**Hypothesis 3b: ISD Differences Across Method B Groups.** It was hypothesized that the two ADHD subtype groups, ADHD Predominantly Inattentive type and ADHD Combined type, would significantly differ with respect to IIV as measured by ISD. No directionality of results was predicted.

**Hypothesis 3c: ISD Differences Across Method C Groups.** It was hypothesized that the three Method C groups (high-SCT, moderate-SCT, and low-SCT) would differ significantly on ISD scores. No directionality of results was predicted.

**Hypothesis 4a: Sluggish Cognitive Tempo (SCT).** Based on prior research showing SCT in individuals with a purely inattentive condition, it was hypothesized that the purely inattentive group would display significantly slower/worse scores on selected indices of SCT (to be detailed in Proposed Analyses section below) than the purified ADHD group. Indices of SCT were be selected based on their ability to assess affected cognitive areas as shown in prior research findings mentioned earlier in this dissertation (i.e., speeded visual processing and early selective attention).

**Hypothesis 4b: SCT Differences Across Method B Groups.** It was hypothesized that the two ADHD subtype groups, ADHD Predominantly Inattentive type and ADHD Combined type, would significantly differ with respect to scores on SCT measures. Specifically, the

Predominantly Inattentive type group should exhibit significantly slower performances across the SCT measures than the Combined type group.

**Hypothesis 4c: SCT Differences Across Method C Groups.** It was hypothesized that the three Method C groups (high-SCT, moderate-SCT, and low-SCT) would differ significantly with respect to scores on SCT measures. Specifically, the high-SCT group should produce scores indicating the slowest overall performances across SCT measures, the low-SCT group should exhibit the least cognitive slowing on SCT measure scores, and the moderate-SCT group should exhibit a degree of slowing on SCT measure scores that falls between the high- and low-SCT groups. This hypothesis is based on the same reasoning used in hypothesis 4; that is, individuals with a purely inattentive condition, which is associated with SCT, should perform worse on neuropsychological measures thought to assess cognitive abilities impacted by SCT. Higher levels of SCT, then, should be associated with higher levels of reduced performance on suspected SCT-related neuropsychological tests.



## METHODS

### Participants

All information for the current project was obtained from an archival clinical dataset. Participants included in this dataset were individuals who sought psychoeducational assessment services from the university's Psychological Services Center between 1999 and 2011. These clients were generally self-referred, often presenting for evaluation of academic difficulties secondary to suspected learning and/or attentional problems. A battery of psychological and neuropsychological measures was administered to all participants in an attempt to identify the presence and sources of any objective cognitive disturbance. While this battery was fixed, test substitutions or exemptions may have been made on an individual basis based on physical or time limitations, recent prior testing with these same measures, or voluntary termination of testing sessions by the client. Thus, not all participants had data for all evaluated tests. The testing battery was constructed so as to assess the areas of intelligence, attention/concentration, learning and memory, academic functioning, and psychological/emotional functioning. The typical session length, per standardized task instructions and including a clinical interview, ranged from six to eight hours. All assessment measures were administered and scored using standardized methods by appropriately-trained clinical psychology graduate students, after which client testing and interview data were evaluated on a case-by-case basis at weekly supervision and training meetings. Diagnostic decisions were made by consensus agreement during these meetings, with all final diagnoses being approved by a licensed clinical psychologist/neuropsychologist. In the event of a lack of consensus, ultimate diagnostic judgment rested with the licensed psychologist/neuropsychologist. All clients included in this

archival database were informed of the nature of the evaluation and provided signed consent prior to testing.

For the purposes of this study, only individuals with a stand-alone Axis I diagnosis of ADHD (any type) per DSM-IV-TR criteria were included in the experimental groups. Exclusionary criteria included the presence of any Axis I diagnosis other than ADHD (e.g., learning disorder, mood disorder, anxiety disorder), any formal Axis II diagnosis, age less than 18 years, and Full Scale IQ less than 70. While data on neurological conditions or injuries was not directly available in all cases, the exclusionary criteria of any axis I diagnosis other than ADHD should have captured most, if not all, individuals exhibiting objective impairment on cognitive testing due to any such condition or injury, as these findings would necessitate an appropriate diagnosis (e.g., Cognitive Disorder Not Otherwise Specified). In those cases where information on neurological conditions/injuries was available, this data was screened.

Initially, the database contained entries for 800 individuals. After implementing the above-mentioned exclusionary criteria, 120 individuals were identified with age  $\geq 18$  years and who had stand-alone diagnoses of ADHD. None of these individuals had a Full Scale IQ less than 70 or any listed neurological condition. Twelve individuals did not have any WURS data available, and were thus excluded from all further analyses. Prior to their exclusion, parametric (ANOVA) and non-parametric analyses were run to determine that complete absence of WURS data was not significantly correlated with age, race/ethnicity, gender, years of education, WAIS-III Full Scale IQ, or ADHD subtype diagnosis, which was indeed the case. Individuals with no WURS data were also not significantly different from individuals with complete WURS data on any of the experimental dependent variables. Exclusion of these individuals resulted in a sample of 108 participants.

In screening these remaining participants, ten individuals were found to have missing WURS data on the items to be used in the cluster analysis, with each of these ten individuals missing one WURS item value. These ten individuals were compared to the remaining 98 individuals with respect to age, race/ethnicity, gender, years of education, and ADHD subtype diagnosis as well as all experimental dependent variables. ANOVA and chi-square analyses revealed that the ten individuals missing data were significantly older (mean = 26.20 years) than individuals not missing data (mean = 21.26 years). No other significant differences between individuals with and without missing data were found. Given the significant difference in age across groups, the data did not appear to support an assumption of being missing completely at random (MCAR). Thus, listwise deletion of these individuals in most instances would not have been appropriate/valid (Schafer & Graham, 2002; Tabachnik & Fidell, 2007). Additionally, listwise deletion would have resulted in removing a sizable proportion (9.3%) of the sample to deal with a relatively small proportion (ten out of 1296 total WURS values, or 0.77%) of missing data. As no WURS item was missing more than two entries, removal of “problematic” WURS items was also not a practical solution.

I therefore evaluated the appropriateness of various methods of imputing missing values for use in the current project. Research indicates that with questionnaire data using Likert-type responses, a variety of imputation techniques can produce similarly-accurate results (Bono, Ried, Kimberlin, & Vogel, 2007; Raaijmakers, 1999). Research also suggests that with small amounts of missing data (e.g., < 5%), a variety of single imputation methods, including mean substitution, regression-based imputation, and expectation maximization (EM), can be used without having significantly deleterious effects on the structure of the data and without resulting in markedly different outcomes (Rubin, Witkiewitz, St. Andre, & Reilly, 2007; Saunders, Morrow-Howell,

Spitznagel, Dore, Proctor, & Pescarino, 2006; Shrive, Stuart, Quan, & Ghali, 2006; Tabachnik & Fidell, 2007). However, mean substitution is often viewed unfavorably for its effects on the variance structure of data in even small to moderate amounts of missingness, and it is not frequently recommended as a viable solution (Schaefer & Graham, 2002; Schlomer, Bauman, & Card, 2010; Tabachnik & Fidell, 2007). Therefore, given the small proportion of missingness in the current sample, EM was selected as the data imputation method. Data from all non-missing WURS items were used as predictors to impute missing data on the seven incomplete WURS items via the EM feature of SPSS version 19's Missing Values Analysis module (IBM, 2010).

The sample to be included in further analysis initially consisted of 108 individuals (47 males). I removed one of these individuals after he was found to be a significant univariate outlier on ISD, as described in greater detail in the Data Screening section below, resulting in a final sample size of 107 (46 males). 88 of the individuals were Caucasian, ten were African-American, one was Asian, six were Hispanic, one was Arab, and one did not report an ethnicity. 29 participants were diagnosed with ADHD combined type, 48 with ADHD predominantly inattentive type, and 30 with the not otherwise specified (NOS) type. The average age in years across all participants was 21.74 years ( $SD=6.36$ ) and ranged from 18 to 68, average years of education was 13.71 years ( $SD=1.65$ ) and ranged from ten to 18, and average WAIS-III Full Scale IQ was 105.72 ( $SD=11.39$ ) and ranged from 80 to 134.

## **Procedures**

**Additional Grouping Methods.** In planning for the possibility that the cluster analysis procedure might not result in the identification of an appropriate solution, I developed two additional grouping methods. The first strategy entailed separating groups based on ADHD subtype. The Predominantly Hyperactive/Impulsive type was collapsed into the Combined Type

group as has been done in prior SCT studies based on data suggesting that the two subtypes display relatively equivalent amounts of SCT symptoms (Carlson & Mann, 2002; Garner et al., 2010). Individuals diagnosed with the NOS subtype were not included in this grouping method. Their exclusion was based on the idea that the NOS group likely consisted of both subthreshold predominantly-inattentive and subthreshold-combined type individuals, and it could not be determined to which of the two ADHD subtypes I should most-appropriately assign these individuals.

The second non-cluster grouping strategy was based on previous work in identifying and evaluating SCT in children (Garner et al., 2010) and adults (Barkley, 2011a). Garner and colleagues (2010) identified individuals with high- and low-SCT by first calculating the summed raw score of all participants on only the SCT-related report items used in their study, and then conducting a median split using this summed score. The first portion of this method was adapted to the current study—the total scores of all participants on the three SCT-like items from the WURS, as listed in Table 3 as theoretically loading on the SCT factor, were calculated, with possible values ranging from zero to twelve. However, Garner et al. (2010) limited this median split to only individuals with the Predominantly-Inattentive type of ADHD, which nullified their ability to examine the full co-occurrence of SCT and ADHD. In light of recent data from Barkley (2011a) indicating that 65% of individuals with the Combined subtype also exhibited significant SCT, the SCT-like WURS summed score was calculated for the entire sample rather than just for Predominantly Inattentive individuals. For the second portion of this grouping strategy, methodology from the Barkley (2011a) study was adapted to the current project. In his study, Barkley (2011a) developed an SCT “diagnostic threshold” by determining the number of SCT symptoms reported by adults that corresponded to 1.5 standard deviations above the mean

number of reported symptoms in his entire sample. He then labeled any individual above this 1.5 SD cut-off as having SCT. For the current study, the 1.5 SD cut-off was developed based on the aforementioned summed score on the SCT-like WURS items across all individuals in the database, which included those with non-ADHD and no diagnoses as in the Barkley (2011a) study. Individuals at or above this 1.5 SD cut-off were identified as being high-SCT. A less-conservative 1 SD cut-off was also identified. Individuals whose summed scores were less than 1.5 SD above the mean, but were at or above 1 SD above the mean (i.e.,  $1.5 \text{ SD} > X \geq 1 \text{ SD}$ ), were identified as moderate-SCT. All individuals whose summed scores were less than 1 SD above the mean were identified as low SCT. For ease of reference, as mentioned earlier in this paper, the grouping method based on cluster analysis will hereafter be referred to as Method A; the method based on DSM-IV ADHD subtype will be referred to as Method B; and the method based on SCT-like WURS item summed score values will be referred to as Method C.

**Intraindividual Standard Deviation.** To assess intraindividual variability via dispersion analysis, methodology detailed in prior dispersion studies (Lindenberger & Baltes, 1997; Morgan et al., 2011) was used in the current project. The chosen methodology converted index scores from various measures to z-scores for each participant in the following cognitive domains: intelligence, memory, attention, processing speed, working memory, executive functioning, and academic functioning. The specific indices used in each domain were as follows: intelligence—Full-Scale IQ, Verbal Comprehension Index, and Perceptual Organizational Index, all from the WAIS-III; memory—Visual Immediate and Delayed Memory indices and Auditory Immediate and Delayed indices, all from the WMS-III; attention—commission errors, Hit RT Block Change, Hit SE Block Change, Hit RT ISI Change, and Hit SE ISI Change from the Conners' CPT; processing speed: Processing Speed Index from the WAIS-III; working memory—

Working Memory Indices from both the WAIS-III and WMS-III; executive functioning—Trail Making Test part B; and academic functioning—Broad Reading, Broad Written Language, and Broad Math, all from the WJ-III. The omission errors score from the Conners' CPT was initially proposed as an additional attentional component to be included. However, upon evaluation of the database, I discovered that nearly half of all participants were missing omission errors values. Thus, the omission errors scale was removed from the calculation of ISD. The standard deviations amongst the obtained z-scores for the included scores/indices were then computed for each participant, and the means of these SDs were calculated for each group. These means were termed each group's Intraindividual Standard Deviation (ISDs).

## **Measures**

**Wechsler Intelligence Scale—3<sup>rd</sup> Edition** (WAIS-III; Wechsler, 1997a): The WAIS-III is the most frequently used intelligence test by clinical neuropsychologists in North America (Rabin, Barr, & Burton, 2005). Its typical administration time is 60-90 minutes. The measure consists of thirteen subtests, twelve of which were administered to individuals included in the current clinical database: Vocabulary, Similarities, Arithmetic, Digit Span, Information, Comprehension, Letter-Number Sequencing, Picture Completion, Digit-Symbol Coding, Block Design, Matrix Reasoning, and Symbol Search. Scaled scores on these subtests are standardized with means of ten and standard deviations of three. From these subtests are derived seven indices: Full Scale IQ, Verbal IQ, Performance IQ, Verbal Comprehension Index, Perceptual Organization Index, Processing Speed Index and Working Memory Index. Scores on these indices are standardized with means of 100 and standard deviations of fifteen. The current project will include the following subtests and indices for analysis: Full Scale IQ (FSIQ), Verbal

Comprehension Index (VCI), Perceptual Organization Index (POI), Processing Speed Index, and Working Memory Index (WMI).

**Wechsler Memory Scale—3<sup>rd</sup> Edition** (WMS-III; Wechsler, 1997b): The WMS-III is the most widely-used multi-modal memory test by neuropsychologists in North America (Rabin, Barr, & Burton, 2005). The scale measures memory for auditory and visual information immediately and after a 30-minute delay via both cued (recognition) and uncued (free recall) procedures. Scaled scores are calculated for nine individual subtests: Logical Memory I and II, Verbal Paired Associates I and II, Faces I and II, Family Pictures I and II, and Spatial Span. As with the WAIS-III, these scaled scores are standardized with means of 10 and standard deviations of 3. These subtests then provide a total of eight index scores: Immediate Memory, General Memory, Auditory Immediate, Auditory Delayed, Auditory Recognition Delayed, Visual Immediate, Visual Delayed, and Working Memory. These indices, again as with the WAIS-III, are standardized with means of 100 and standard deviations of 15. The following indices will be included in the current project for analysis: Auditory Immediate, Auditory Delayed, Visual Immediate, Visual Delayed, and Working Memory.

**Woodcock-Johnson Tests of Achievement—3<sup>rd</sup> Edition** (WJ-III; Woodcock, McGrew, & Mather, 2001): The WJ-III is a frequently-used “omnibus” assessment measure of multiple areas of academic functioning. The measure consists of 22 total subtests, with twelve comprising the Standard Battery, and an additional ten making up the Extended Battery. Additionally, there are two equivalent forms of all subtests, Form A and Form B. Administration of the Standard and Extended Batteries allows for the calculation of a variety of subject-specific (e.g., Board Reading, Broad Math) and overarching (e.g., Total Achievement, Academic Fluency) academic indices. Of the available subtests, at least twelve were administered to the majority of individuals



in the current database as part of the standard psychoeducational battery. All indices reported in the WJ-III are shown as Standard Scores, which have a mean of 100 and standard deviation of 15. For the purposes of the current project, those indices included for analysis will be Broad Reading, Broad Math, Broad Written Language, Listening Comprehension, Reading Fluency, Math Fluency, and Writing Fluency.

**Conners' Continuous Performance Test** (Conners' CPT; Conners, 1994): The Conners' CPT is a computerized test of attention and concentration. The test requires that participants respond to all stimuli other than the infrequent target stimuli; it is therefore different from many other continuous performance tests in which the target stimuli are more frequent (Lezak, Howieson, & Loring, 2004). The Conners' CPT, therefore, is more similar to go/no-go and sustained attention to response tasks, and places greater demands on sustained attention and behavioral inhibition (Johnson et al., 2007; Strauss, Sherman, & Spreen, 2006). All Conners' CPT indices are reported as T scores, which are standardized to have a mean of 50 and standard deviation of 10. Indices used in the current project will include Reaction Time (RT), RT standard error (RTSE), commission errors, Hit RT Block Change, Hit SE Block Change, Hit RT ISI Change, and Hit SE ISI Change.

**Trail Making Test, Parts A and B** (TMT A and B; Reitan, 1955): In the TMT, participants are tasked with drawing lines to connect either numbers or alternating numbers and letters on a page as quickly and accurately as possible. TMT A is viewed as a measure of cognitive, motor, and visual scanning speeds, while TMT B is related to cognitive flexibility and set-shifting (Lezak, Howieson, & Loring, 2004; Strauss, Sherman, & Spreen, 2006). Individuals' performances are scored based on the raw time, in seconds, required to complete each of the two parts. Values for both TMT A and TMT B are reported in T scores, which are standardized to

have a mean of 50 and standard deviation of 10. As higher raw time-to-completion values on the test indicate poorer performance, T scores are reverse-scored so that lower (i.e., faster) time-to-completion results are reflected by higher T scores, and vice-versa.

**Wender Utah Rating Scale** (WURS; Ward, Wender, & Reimherr, 1993): The Wender Utah Rating Scale is a 61-item measure designed to assess adults' retrospective self-report of childhood ADHD and ADHD-like symptoms. Participants are asked to indicate the degree to which they were, had, or were bothered by each of the 61 items as a child by using a 0 to 4 scale, with 0 representing the response, "not at all or very slightly" and 4 representing, "very much." Of the 61 items, 25 were found to be diagnostically useful in identifying ADHD in adults, and thus only those 25 items are included in the scale's overall score. Factor analyses of these 25 items has supported the presence of three (McCann et al., 2000) to five (Norvilitis, Ingersoll, Zhang, & Jia, 2008; Stein et al., 1995; Suhr, Zimak, Bluelow, & Fox, 2009) factors. Of these factors, three (impulsivity, poor academic functioning, and inattention/anxiety symptoms) were useful in separating ADHD and control groups in females, but not in males (Suhr et al., 2009). Total scores on the measure can range from 0 to 100, with a score of  $\geq 36$  being suggestive of ADHD in the absence of unipolar depression, and  $\geq 46$  suggesting ADHD in the presence of unipolar depression (Ward, Wender, & Reimherr, 1993).

For the current study, a subset of twelve items was selected for inclusion in cluster analysis. These items were selected based on their similarity to characteristics associated with SCT; their high loadings on WURS factors that have been identified as potentially diagnostically useful in identifying ADHD (i.e., impulsivity and inattention/anxiety; Suhr et al., 2011); and/or their similarity to behavioral rating items that were significantly different across ADHD combined type, ADHD predominantly inattentive type with low-SCT, and ADHD predominantly

inattentive type with high-SCT groups of individuals (Carlson & Mann, 2002). While the “academic problems” WURS factor identified by Suhr et al. (2011) was also listed as potentially diagnostically-useful, data suggests that individuals with high and low SCT do not significantly differ from one another, nor does either group differ significantly from individuals with ADHD, with regard to academic/learning problems (Carlson & Mann, 2002); thus, these items were not included in the cluster analysis. Conversely, while Suhr et al. (2011) did not find the “conduct/lability” scale to be diagnostically useful in their sample, which did not contain any SCT groups, other data have shown that SCT tends to be significantly negatively correlated with teacher ratings of disruptive behavior symptoms/characteristics (Penny et al., 2009), and as such conduct/lability items were included in the present study. In order to reduce any one item-related factor (e.g., impulsivity, inattention, SCT) from unduly influencing the cluster analyses via uneven weighting, a relatively even number of items thought to load on each item-related factor were chosen. The hyperactivity/impulsivity factor was allowed to contain one more item than all other factors owing to previous data having shown that SCT symptoms are significantly negatively correlated with both parent and teacher ratings of hyperactivity/impulsivity symptoms (Penny, 2009), suggesting that these items may be particularly useful in differentiating purified ADHD and purely inattentive individuals. The selected items that were included in the cluster analyses, and their suspected associated symptom factors, are shown in Table 1 below:

Table 1: List of Wender Utah Rating Scale Items and Suspected Factors Included in Cluster Analyses

<b>Hyperactivity and Impulsivity</b>	<b>Inattention/Anxiety Symptoms</b>	<b>Conduct Problems/Lability</b>	<b>SCT Symptoms</b>
Active, restless, always on the go	Anxious, worrying (.70)	Short temper (.80)	Shy, sensitive
Tendency to be immature (.74)	Nervous, fidgety (.72)	Temper outbursts (.78)	*Inattentive daydreaming
Acting without thinking, impulsive (.67)	*Inattentive daydreaming (.64)	Angry (.68)	Slow reader
Incautious, dare-devilish, involved in pranks			

\*Item is included in this table more than once because it loaded/was suspected to load on more than one factor; Factor loadings from Suhr et al., 2009 are included in parenthesis when available

## Analyses

**Data Screening.** Prior to conducting any hypothesis-related analyses, all data to be examined were evaluated for potential violation of the assumptions inherent to parametric statistical techniques. Most basically, the sample size of each cell in the MANOVA analyses needed to exceed the number of included dependent variables (i.e., six); this was the case for Methods A and B, but not for Method C, which contained only five members in the high-SCT group. This finding led to the combining of the high- and moderate-SCT Method C groups (hereafter referred to as Method C combined), as will be described in the Results section below. Thus, no MANOVA analysis was run using high-SCT and moderate-SCT as separate grouping categories; however, I did conduct the ANOVA analyses examining ISD and RTSE with the

high-SCT and moderate-SCT groups remaining distinct. For data to be evaluated using independent-samples *t*-tests and ANOVAs (i.e., RTSE scores from the Connors' CPT and calculated ISD values), I screened for univariate outliers by examining individuals' z-scores on measured variables within each group. Z-scores exceeding  $\pm 3.29$  may indicate a potential outlier ( $p < .001$ ), although a few such scores might be expected in a larger data set (Tabachnick & Fidell, 2007); a z-score above  $\pm 4$  would be an even more conservative indicator of an outlier, which was the criteria used in the current study. One outlier was identified on ISD, having a z-score of 6.80, and this case was removed from further analysis. No outliers were noted on RTSE or any of the variables included in the MANOVA analysis of SCT, with all z-scores actually falling below the more-conservative  $\pm 3.29$  mark.

Skewness and kurtosis values were examined for group distributions to, in part, evaluate normality. However, actual significance tests for these values were not used, as they may be overly sensitive to small deviations from normality in larger ( $>100$ ) sample sizes (Tabachnick & Fidell, 2007). The overall (i.e., disregarding group membership) skewness value for ISD was .565 and for RTSE was .425. Overall kurtosis for ISD was .419 and for RTSE was .474. For the Method A groups (Less Symptomatic and More Symptomatic, as identified below), values on skewness and kurtosis on ISD, RTSE, and the six variables included in the SCT MANOVA (hypothesis 4) were between -1 and 1 in all instances except Connors' CPT RT, which exhibited a kurtosis of 1.106 in the Less Symptomatic group, and 1.406 in the More Symptomatic group. However, dividing these by each kurtosis value's standard error resulted in values  $< 2$ , which suggests that the distributions may not be non-normal to a degree that would interfere with parametric analyses. For the Method B groups (ADHD Combined type and ADHD Predominantly Inattentive type), the skewness and kurtosis values for all variables fell between -

1 and 1. For the Method C groups (high-SCT, moderate-SCT, and low-SCT), all of the skewness and kurtosis variables for the low-SCT group fell between -1 and 1. For the moderate-SCT group, the WJ-III Reading Fluency kurtosis was -1.142, with all other values falling between -1 and 1. For the high-SCT group, the WJ-III Reading Fluency kurtosis was -1.255, the WJ-III Math Fluency kurtosis was -2.256, and the Conners' CPT RTSE kurtosis was 1.305. For the Method C combined groups, only kurtosis for ISD in the high-and-moderate SCT group exceeded -1, with a value of -1.106. However, as with the Method A screening results, all of these kurtosis values that exceeded 1 in the Method C and Method C combined groups resulted in quotients  $< 2$  when divided by their standard error terms. Thus, no transformations were conducted.

To determine if assumptions of homogeneity of variance were violated for the univariate analyses, Levene's test for equality of variances results as produced by SPSS were evaluated. However, as Levene's test is highly conservative,  $F_{\max}$  was examined in coordination with sample-size ratios in instances when Levene's test was significant, as suggested by Tabachnick and Fidell (2007). In the univariate analyses for all of the included grouping methods (i.e., Method A, Method B, Method C, and Method C collapsed), neither of the Levene's test values (i.e., for ISD or for RTSE) was significant. Thus, the assumption of homogeneity of variance did not appear to be significantly violated.

For multivariate analyses, I initially screened for outliers by examining Mahalanobis distances and compared these values to the chi-square significance table with  $p = .001$  and  $df = 6$  (i.e., the number of variables included in the calculation) as per recommended guidelines (Tabachnik & Fidell, 2007). No individual exhibited a significant Mahalanobis distance when examining the groups created via Methods A, B, C, and C collapsed. Multicollinearity was

evaluated by evaluating bivariate correlations between the analyzed variables. The highest such correlation was between WJ-III Reading Fluency and WAIS-III PSI ( $r=.573$ ), with  $r$  for most values being between  $|.1|$  and  $|.3|$ . Thus, no variables appeared to be correlated highly enough to interfere with the analysis via multicollinearity, which was supported by SPSS failing to provide any error messages while performing the MANOVAs. Homogeneity of variance-covariance matrices was evaluated with Box's M, although this test may be overly conservative in larger sample sizes (Tabachnick & Fidell, 2007). Regardless, for Methods A, C, and C combined, Box's M values did not reach or approach significance. For Method B, Box's M was significant at the  $p<.01$  level, but not a more-conservative  $p<.001$  level. Additionally, evaluation of variances in each of the three Methods indicated that the ratio of variance in the smallest to largest cell did not nearly approach suggested limits of 10:1 (Tabachnick & Fidell, 2007), with the largest such ratio being approximately 2.2:1 between Method B groups with respect to WJ-III Math Fluency. The univariate normality of distributions for the various dependent variables tentatively supported the presence of multivariate normality. Additionally, all MANOVA analyses had more than 20 degrees of freedom for error, suggesting robustness against violations of normality, (Tabachnick & Fidell, 2007) and no cell size discrepancy was greater than approximately 3.5:1. Thus, no data transformations were conducted.

**Cluster Analysis.** Once data was appropriately screened, analyses were conducted to test the proposed research hypotheses. To examine the ability to identify and separate out a purely inattentive subgroup of individuals diagnosed with ADHD (Hypothesis 1), all individuals in the current database who had a diagnosis of ADHD were collapsed across subtypes (i.e., predominantly inattentive, predominantly hyperactive/impulsive, combined, and NOS) into a single group. Cluster analysis was then used in an attempt to identify distinct groups of

participants, with Hypothesis 1 having proposed the existence of two such groups—purely inattentive and purified ADHD. The variables used were the twelve WURS items listed in Table 1. Per published methodological recommendations (Hair & Black, 2001; Mandara, 2003; McIntyre & Blashfield, 1980; Milligan, 1996), the cluster analysis was conducted in a series of steps using an initial sample and holdout sample, as well as both hierarchical and k-means clustering methods, in a replication analysis paradigm. This combination of clustering methods allows for the hierarchical clustering model to serve an exploratory role in evaluating the structure of the data, which allows for the potential identification and use of informed rather than random cluster centers, as the latter may lead to inaccurate clusterings (Mandara, 2003; Milligan & Sokol, 1980). However, as the hierarchical method is non-iterative, the k-means clustering analysis, which is an iterative technique, can then refine the cluster centers (Blashfield & Aldenderfer, 1988; Rovniak et al., 2010). The replication analysis then allows for a validation of the developed clusters via comparison between the initial and holdout sample results (McIntyre & Blashfield, 1980; Milligan, 1996). Validation is particularly crucial in cluster analysis owing to the procedures' identifying clusters in the sample even if no such clusters truly exist in the construct and/or population of interest (Aldenderfer & Blashfield, 1984; Hair & Black, 2001).

The first step of the cluster analysis procedure involved dividing the sample into two approximately equally-sized halves via the select cases feature of SPSS 19. This step resulted in one group (subsample A) of 57 individuals and a second group (subsample B) of 50 individuals. Next, subsample A was evaluated using a hierarchical cluster analysis. I chose the squared Euclidean distance method for determining the distances between individuals based on its commonality in prior research, and its ability to preserve many typological characteristics of the data (Mandara, 2003; Milligan, 1996). Given the tendency of WURS items to correlate to a



modest extent with one another within and across factors (Suhr et al., 2009), the clustering algorithm selected for all cluster analyses was Ward's method (Ward, 1963). This method attempts to cluster cases such that the within-cluster Sums of Squares (i.e., SSE) is minimized at each step. One drawback of this method with respect to the current study is that it tends to create relatively equally-sized clusters (Hair & Black, 2001), although recent research by Barkley (2011a) has indicated that roughly equally-sized groups may accurately reflect the appropriate cluster structure when examining ADHD and SCT in adults. Additionally, Ward's method is robust to multiple data types (Milligan, 1996; Mandara, 2003), and because it attempts to minimize SSE, its output is appropriate for use with multiple indices developed to evaluate the accuracy of the number of clusters produced, such as the Calinski Harabasz index (discussed below; Steinley & Brusco, 2011). The resulting hierarchical cluster analysis dendrogram, coupled with theoretical information, were considered in identifying the number of clusters apparent. As previously mentioned, it was hypothesized that two groups would emerge: a purely inattentive group and a purified ADHD group. However, research released after the proposal of this project indicated that in adults, a three-group solution might be more accurate, which would include an ADHD + SCT group in addition to the two aforementioned groups (Barkley, 2011a). This three-group solution reflects the high degree of comorbidity between ADHD symptoms and SCT/purely inattentive symptoms. Based on this ambiguity in prior research, three separate clustering solutions were saved: a two-group solution, a three-group solution, and a four-group solution. The group numbers for each participant under the identified solutions were then saved and used to calculate the cluster centers for each group on the clustering variables.

In the second step of the cluster analysis procedure, the calculated cluster centers from the hierarchical solution(s) were used as seeds for separate k-means cluster analyses, where k is

the number of clusters that the analysis is setup to identify. One k-means cluster analysis was run for each of the solutions to refine and identify the final cluster centers. To determine which of the k-means cluster analyses best fit the data, the Calinski Harabasz index (CHI; Calinski & Harabasz, 1974) was used to evaluate each of the different solutions. The CHI is calculated via the formula  $[\text{trace } B/(k-1)]/[\text{trace } W/(n-k)]$ , where trace B is the between-clusters sums of squares, trace W is the within-clusters sums of squares, k is the number of clusters, and n is the number of observations, and on which higher values represent numbers of clusters that more-accurately reflect the underlying structure of the data (Maulik & Bandyopadhyay, 2002; Steinley & Brusco, 2011). The CHI was chosen based on the relatively high accuracy with which it has been shown to identify the true number of clusters in known-groups data sets when compared with other such methods (Steinley & Brusco, 2011). Based on CHI results, the best number of groups was selected, and the cluster centers from this solution were saved.

For the next step of the cluster analysis procedure, the refined cluster centers identified in subsample A were used with subsample B. Per the external validation process (McIntyre & Blashfield, 1980; Milligan, 1996), participants in subsample B were assigned to groups using the cluster centers identified in subsample A. Next, a separate k-means cluster analysis was run on subsample B without using the cluster centers from subsample A. Finally, the agreement in group membership between these two solutions was calculated via Cohen's kappa statistic. Interpretation of the kappa statistic can be somewhat arbitrary, with some authors suggesting that a value  $>.80$  indicates high agreement (McGinn et al., 2004), while others suggest that  $k > .7$  or  $.75$  indicates good to strong agreement (Altman, 1991; Fleiss, 1981; Landis & Koch, 1977). Given the exploratory nature of the current study, I implemented a less-conservative cut-off of  $k > .7$  in determining whether sufficient agreement between the two subsample cluster solutions

existed to warrant further analysis. Should the kappa statistic exceed .70, the two subsamples would be collapsed into a single sample, and a final series of hierarchical and k-means cluster analyses would be run to identify each participant's group. I would then examine the cluster center information for all groups to determine their WURS-related characteristics (e.g., more shy and daydreamy, more aggressive and impulsive, etc.). If the kappa statistic did not exceed .70, then the data would not support the true existence of the identified cluster solution, and no further analyses using this solution would be conducted.

**Between-Groups Analyses.** Following the completion of the identification of groups via the three grouping strategies (i.e., Methods A, B, and C), chi-square and independent-samples *t*-test or ANOVA analyses were run on nominal/categorical and interval/ratio demographic variables, respectively and as appropriate, to determine if any significant between-group differences existed. These demographic variables included age at the time of evaluation, gender, ethnicity, and years of education. Handedness was also planned for inclusion, but was ultimately not evaluated owing to its not having been recorded for the majority of participants. Between-group Full-Scale IQ differences were also examined, as were differences across groups regarding ADHD subtype in Methods A and C. The use of Full-Scale IQ as a covariate, however, even in the presence of significant between-group differences (particularly if these differences exist between controls and the two experimental groups), was precluded based on Full-Scale IQ capturing certain cognitive phenomena that may reflect central deficits to ADHD and a purely inattentive condition, such as reduced processing speed and working memory.

To examine between-group RTSE and ISD differences (Hypotheses 2a, 2b, 2c, 3a, 3b, and 3c), I initially proposed one-tailed independent-samples *t*-tests (Methods A and B) and one-way ANOVAs (Method C), as appropriate. However, as discussed in more detail in the Results

section below, for Method C, a significant between-group age difference was revealed. This factor was subsequently selected for entry as a covariate into all between-group univariate analyses in Methods C. Thus, I ran ANCOVAs for Method C, and one-tailed independent-samples *t*-tests, as proposed, for Methods A and B. The level of significance for all univariate analyses was set at .05 (i.e.,  $\alpha < .05$ ) and the power at .80 (i.e.,  $[1-\beta] = .80$ ). With these values, the total sample needed to detect a significant difference via a one-tailed *t*-test was 21 for an effect size (*d*) of .5 and 64 for an effect size of .3, with these values representing the effect sizes commonly found in neuropsychological testing in ADHD (Nigg, 2005). For these same significance and power values in an ANCOVA, total sample size needed to detect a significant difference with three groups was 42 for an effect size of .5 and 111 for an effect size of .3. The current study, with a sample size of  $N=107$ , was adequately-powered to detect between-group differences for all analyses other than the three-group (i.e., Method C) ANCOVA if the effect size of any between-groups difference was 0.3 rather than 0.5.

Potential Sluggish Cognitive Tempo-related differences between groups (Hypotheses 4a, 4b, and 4c) were evaluated using MANOVA for all grouping methods (i.e., A, B, and C). Post-hoc analyses were not required for Methods A and B, as the independent variables only consisted of two levels. Post-hoc analysis would have initially been necessary for Method C, as the independent variable would have been divided into three levels. However, owing to the combining of the high-SCT and moderate-SCT categories into a single group, the independent variable in the MANOVA analysis of Method C only had two levels, and thus did not necessitate post-hoc testing. With  $\alpha = 0.05$  and Power = 0.80, a MANOVA analysis would require a total sample size of 36 individuals to detect a global effect at an effect size of 0.5 with two groups and six DVs, and 54 individuals to detect a global effect with an effect size of 0.3. The current study,

then, was adequately-sized to detect all anticipated between-group differences. A summary of the three grouping methods and their associated between-groups analyses is presented in Table 2:

Table 2: Grouping Methods Used for Between-Groups Analyses

<b>Grouping Method</b>	<b>Technique</b>	<b>Category 1</b>	<b>Category 2</b>	<b>Category 3</b>
Method A	Cluster analysis	*Less symptomatic	*More symptomatic	N/A
Method B	DSM-IV-TR ADHD Subtype	Combined type	Predominantly Inattentive type	N/A
Method C	SCT-related WURS items summed score	High-SCT ( $\geq 1.5$ SD)	Moderate-SCT ( $< 1.5$ SD, $\geq 1$ SD)	Low-SCT ( $< 1$ SD)

\*These cluster analysis groups, which do not align with those initially hypothesized, are described in greater detail in the Results and Discussion sections.

### **Independent Variables**

The independent variables for each of the hypotheses and analyses will be listed here for the sake of improved clarity. Demographic factors used in between-groups analyses for all hypotheses included age (coded in years) and education (coded in years). I also included ethnicity as an independent variable in demographic analyses, and in doing so, collapsed the number of possible categories from six to two (coded as 1 = Caucasian and 2=non-Caucasian) owing to the large proportion of Caucasian individuals in the sample, and the subsequently small numbers of individuals in the remaining ethnic categories.

For Hypotheses 1, 2, 3, and 4 the independent variable was group membership as determined by the cluster analysis, as reported in the results section below. For Hypotheses 2b, 3b, and 4b, the independent variable was DSM-IV-TR diagnostic category (coded as 1=ADHD-Combined type and 2=ADHD-Predominantly Inattentive type). For Hypotheses 2c and 3c, the independent variable was SCT-related WURS items summed score group (coded as 1 = low-

SCT, 2 = moderate-SCT, and 3 = high-SCT). For Hypothesis 4c, the independent variable was again SCT-related WURS items summed score group, but coded differently (1 = low-SCT and 2 = high-and-moderately SCT).

### **Dependent Variables**

The dependent variable evaluated in Hypotheses 2a, 2b, and 2c was RTSE (coded as a T score). For Hypotheses 3a, 3b, and 3c, the dependent variable was ISD (coded via the method described above in the Procedures section). For Hypotheses 4a, 4b, and 4c, the dependent variables included subtest scores or scales that are related to cognitive processing speed. These scores and scales were initially proposed to include Digit-Symbol Coding and Symbol Search from the WAIS-III, RT from the Conner's CPT (Hit RT), academic fluencies (Reading Fluency, Writing Fluency and Math Fluency) from the WJ-III, and TMT A. However, owing to sample size limitations (particularly in Method C as discussed in the Results section below), these variables were reduced in number by utilizing index scores rather than individual subtest scores or indices when available. This resulted in the following scores and indices being included in the analysis: WAIS-III Processing Speed Index (PSI; a combination of the Digit-Symbol Coding and Symbol Search subtests, coded as a Standard Score), Conners' CPT Hit RT (coded as a T score), the three academic fluency indices from the WJ-III (Reading Fluency, Writing Fluency, and Math Fluency, all coded as Standard Scores), and TMT A (coded as a T score). I also attempted to substitute the Academic Fluency index from the WJ-III for the three individual academic fluency scores to further reduce the number of dependent variables. However, only 38 individuals had Academic Fluency scores available, which prohibited its use.

## RESULTS

### Participant Information for Grouping Methods B and C

The use of grouping Method B resulted in the delegation of 29 individuals (14 male) to the ADHD Combined type group, and 48 individuals (19 male) to the ADHD Predominantly Inattentive type group. The remaining 30 individuals from the study sample had been diagnosed with ADHD NOS, and thus were not included in either group. Table 3 below lists the demographic information for both groups. ANOVA and chi-square tests indicated that the two groups were significantly different demographically with respect to ethnicity. No between-group differences were noted for gender, ethnicity, age, education, or FSIQ.

Table 3: Demographic Characteristics of Method B (DSM-IV-TR Diagnosis) Groups

<b>Group</b>	<b>n</b>	<b>Age</b>	<b>Gender</b>	<b>Ethnicity*</b>	<b>Education</b>	<b>FSIQ</b>
ADHD-Combined type	29	22.76(9.03)	14 male	21 Caucasian 7 AA 1 Hispanic	14.00(1.47)	105.17(12.62)
ADHD-Predominantly Inattentive type	48	21.13(3.74)	19 male	39 Caucasian 3 AA 1 Asian 5 Hispanic	13.65(1.70)	106.29(10.72)

\*Groups were significantly different,  $p < .05$ ; all non-Caucasian participants were coded as a single category for between-groups analyses

For Method C, as previously mentioned, the entire database was analyzed to determine +1.5 SD and +1.0 SD cut-points. Of the 800 individuals in the database, 684 (334 males) had complete data available for all three SCT-like WURS items; males and females did not differ significantly on total SCT-like WURS scores ( $p = .126$ ). The average combined SCT-like WURS item score for these 684 individuals was 6.66 (SD=2.56). This value resulted in a +1.5 SD cut-off of 10.5 for the High-SCT group (rounded up to 11, as summed scores for individuals were in

whole numbers) and a +1.0 SD cut-off of 9.22 for the Moderate-SCT group (rounded to 9). In applying these values to the study sample, six individuals (two male) were selected into the high-SCT group, 18 individuals (8 male) were selected into the moderate-SCT group, and all other individuals (n = 84; 36 male) were selected into the low-SCT group. Demographic and DSM-IV-TR diagnostic characteristics of the three groups are presented in Table 4 below. Chi-square and ANOVA analyses indicated that the High-SCT group was significantly older than both other groups; no significant between-group differences were observed on any other demographic factor. Age was thus entered as a covariate for the Method C univariate analyses.

Table 4: Demographic and Diagnostic Characteristics of Method C (SCT-related WURS Items Sum) and Method C Combined Groups

Group	n	Age	Gender	Ethnicity <sup>a</sup>	Education	FSIQ	DSM-IV-TR Diagnosis *
Low-SCT	83	21.42(4.88)	36 male	68 Caucasian 7 AA 1 Asian 5 Hispanic 1 Arab	13.67(1.64)	106.17(11.78)	23 ADHD-C 35 ADHD-I 25 NOS
Moderate-SCT	18	20.83(2.94)	8 male	14 Caucasian 3 AA 1 Hispanic	13.67(1.53)	103.00(9.26)	4 ADHD-C 10 ADHD-I 4 NOS
High-SCT	6	28.83 (19.29) <sup>b</sup>	2 male	6 Caucasian	14.33(2.25)	107.67(12.24)	2 ADHD-C 3 ADHD-I 1 NOS
High-and-Moderate SCT	24	22.83(9.99)	10 male	20 Caucasian 3 AA 1 Hispanic	13.83(1.71)	104.17(10.01)	6 ADHD-C 13 ADHD-I 5 NOS

<sup>a</sup>All non-Caucasian participants were coded as a single category for between-groups analyses; \*ADHD-C = ADHD, Predominantly Combined type; ADHD-PI = ADHD Predominantly Inattentive-type; NOS = ADHD Not Otherwise Specified; <sup>b</sup>Group was significantly different,  $p < .01$ ; All (parentetical) values are standard deviations.



Prior to conducting any hypotheses-testing analyses for Method C, a one-way ANCOVA with age as the covariate, Method C group as the independent variable, and total WURS total raw score as the dependent variable was run. This analysis was included to help determine if any potential Method C between-group differences would have been due to the high- and moderate-SCT groups globally endorsing more items at a higher level across the entire WURS than the low-SCT group. That is, did the high- and moderate-SCT groups have significantly higher total WURS scores rather than only having higher SCT-related scores? The results from this ANCOVA were not significant,  $F(2, 94)=1.521, p=.224$ , indicating that the three levels of Method C did not significantly differ with respect to WURS total raw score.

### **Collapsing of Groups in Method C Combined**

With only six individuals in the high-SCT group, the use of MANOVA with six dependent variables would be precluded, as the smallest cell size must be greater than the number of included DVs. Thus, while the three separate groups were retained for all univariate analyses, the high-SCT and moderate-SCT groups were combined into a single category for the multivariate analysis of SCT-related neuropsychological test performances. This high-and-moderate-SCT category consisted of 24 individuals (10 male). Demographic and DSM-IV-TR diagnostic characteristics for this combined group are presented along with those of the three other Method C groups in Table 8. ANOVA and chi-square tests revealed no significant differences between the combined high-and-moderate SCT and low-SCT groups on any demographic factor.

## **Hypothesis 1: Identifying Purified ADHD and Purely Inattentive Groups via Cluster Analysis**

The hierarchical cluster analysis was run such that group membership for three separate solutions would be saved in subsample A: a two-group solution, a three-group solution, and a four-group solution. The cluster centers from these three solutions were saved and subsequently entered into separate k-means cluster analyses for refinement. The CHI values for these three solutions were then calculated, with CHI = 17.77 for the two-group solution, CHI = 14.02 for the three-group solution, and CHI = 12.35 for the four-group solution. Based on these CHI values, the two-group solution was selected as best-fitting the data.

The cluster centers from this two-group k-means cluster analysis were then used in the second subsample (subsample B) for the next step of the cluster validation process. The kappa statistic between group membership assignments using subsample A cluster centers in subsample B, and group membership using the independent cluster analyses of subsample B without the subsample A cluster centers, was  $k = .714$ . Although not excellent/very strong, this  $k$  value indicated good/high agreement between groups. I thus decided to evaluate the characteristics of the two-group solution in determining its theoretical value and viability, and to include the cluster results from this grouping method (i.e., Method A) in further analyses. Table 5 provides the cluster/group characteristics for demographic and diagnostic information and Table 6 lists average scores on the twelve WURS items used in the cluster analyses. ANOVA and chi-square tests revealed no significant differences between the two cluster analysis-identified groups on any demographic factor. Based on these results, and as more thoroughly-described in the Discussion section, the two groups were identified as less symptomatic and more symptomatic rather than purely inattentive and purified ADHD.

Table 5: Demographic and Diagnostic Characteristics of Method A (Cluster Analysis) Groups

Group	n	Age	Gender	Ethnicity <sup>a</sup>	Education	FSIQ	DSM-IV-TR Diagnosis*
Less Symptomatic	70	21.44(7.26)	26 male	57 Caucasian 6 AA 6 Hispanic	13.50(1.78)	105.07(12.05)	14 ADHD-C 33 ADHD-I 23 NOS
More Symptomatic	37	22.30(4.20)	20 male	31 Caucasian 4 AA 1 Asian 1 Arab	14.11(1.28)	106.95(10.08)	15 ADHD-C 15 ADHD-I 7 NOS

\*ADHD-C = ADHD, Predominantly Combined type; ADHD-PI = ADHD Predominantly Inattentive-type; NOS = ADHD Not Otherwise Specified; <sup>a</sup>All non-Caucasian participants were coded as a single category for between-groups analyses

Table 6: Mean and Standard Deviations for WURS Item Values for Method A (Cluster Analysis) Groups\*

Group	Active, Restless, Always on the Go	Anxious, Worrying	Nervous, Fidgety	Inattentive, Daydreaming	Short-tempered	Shy, Sensitive
Less Symptomatic	2.70(1.23)	1.27(1.05)	1.76(1.23)	3.05(1.01)	.92(.91)	1.48(1.21)
More Symptomatic	3.54(.66)	2.21(1.44)	3.00(.98)	3.79(.42)	3.29(.69)	2.08(1.18)
Group	Outbursts, Tantrums	Incautious, Daredevilish	Angry	Impulsive	Immature Tendencies	Slow Reader
Less Symptomatic	.92(1.01)	1.42(1.22)	.48(.72)	2.12(1.26)	1.58(1.80)	1.82(1.47)
More Symptomatic	2.83(.76)	2.75(.99)	1.29(.91)	3.10(1.04)	2.92(1.10)	1.66(1.57)

\*Values were rounded to the nearest whole number for use in the actual k-means clustering procedure

### Hypotheses 2a, 2b, and 2c: Group IIV Differences as Measured by RTSE

**Hypothesis 2a: Examining Group RTSE Differences in Method A.** As the groups identified above in the Hypothesis 1 results portion could not be easily-identified as purely-inattentive and purified ADHD, the independent-samples *t*-test was run as a two-tailed rather than one-tailed analysis. The results from this *t*-test revealed no significant differences between

less symptomatic and more symptomatic groups,  $t(100)=.855, p=.395$ . Qualitatively, the less symptomatic group exhibited a slightly higher RTSE score ( $M=69.25, SD=18.06$ ) than the more symptomatic group ( $M=65.89, SD=20.31$ ).

**Hypothesis 2b: Group RTSE Differences in Method B.** Results from the two-tailed independent-samples  $t$ -test revealed that the Combined type group ( $M=72.69, SD=19.48$ ) and the Predominantly Inattentive type group ( $M=65.03, SD=16.42$ ) did not significantly differ with respect to RTSE,  $t(71)=1.796, p=.077$ . Qualitatively, the Combined type group exhibited a slightly, but again, not significantly higher mean RTSE score than did the Predominantly Inattentive type.

**Hypothesis 2c: Group RTSE Differences in Method C.** Results from the one-way ANCOVA revealed that the covariate of age was not significantly related with the dependent variable, RTSE,  $F(2, 98)=.459, p=.499$ . Similarly, the ANCOVA also indicated that there were no significant differences across any of the three Method C groups with respect to RTSE on the Conner's CPT,  $F(2, 98) = .316 (p=.730)$ . Owing to the lack of significant findings, no post-hoc analyses were necessary. Qualitatively, the Low-SCT and High-SCT groups had similar RTSE values (Low-SCT  $M=67.49, SD=19.30$ ; High-SCT  $M=67.40, SD=11.93$ ), while the Moderate-SCT group had the highest RTSE value ( $M=71.16, SD=18.76$ ).

In order to determine if the reduced statistical power associated with such a small High-SCT group ( $n = 5$ ) may have led to non-significant results, I ran an independent-samples  $t$ -test using the Method C combined groups. As with the one-way ANCOVA, results from the independent-samples  $t$ -test indicated no significant group difference between low-SCT ( $M=67.49, SD=19.30$ ) and high-and-moderate SCT ( $M=70.30, SD=17.26$ ) individuals,  $t(101)=-.701, p(\text{one-tailed})=.243$ .

### **Hypotheses 3a, 3b, and 3c: Group IIV Differences as Measured by ISD**

**Hypothesis 3a: Group ISD Differences in Method A.** The independent-samples *t*-test for Hypothesis 3, like that in Hypothesis 2, was run as a two-tailed rather than one-tailed analysis. The results of this *t*-test revealed no significant differences between the less symptomatic group ( $M=.78$ ,  $SD=.18$ ) and the more symptomatic group ( $M=.85$ ,  $SD=.21$ ),  $t(105)=-1.85$ ,  $p=.067$ .

**Hypothesis 3b: Group ISD Differences in Method B.** Results from the two-tailed independent-samples *t*-test indicated that the Combined type and Predominantly Inattentive type groups were not significantly different with respect to ISD,  $t(75)=.666$ ,  $p(\text{one-tailed})=.507$ . Qualitatively, the Predominantly Inattentive type group exhibited a lower average ISD amongst members ( $M = .797$ ,  $SD = .198$ ) than did the Combined type group ( $M = .829$ ,  $SD = .204$ ). However, again, these differences were not statistically significant.

**Hypothesis 3c: Group ISD Differences in Method C.** Results from the one-way ANCOVA indicated that there were no significant differences across any of the three SCT-like WURS Item summed score groups with respect to ISD,  $F(2, 104) = .857$  ( $p=.428$ ). Post-hoc analyses were therefore unnecessary. Qualitatively, the High-SCT group had the lowest ISD score ( $M=.755$ ,  $SD = .081$ ), the Low-SCT group had the highest ISD score ( $M=.818$ ,  $SD = .203$ ), and the Moderate-SCT group had a score that fell between these two values ( $M=.761$ ,  $SD = .176$ ).

As with the Hypothesis 2b results above, I ran an independent-samples *t*-test using the Method C combined groups to determine if limited statistical power may have played a role in the lack of discovered significant results. And, also as with the hypothesis 2b results, the independent-samples *t*-test did not indicate a significant between-group difference between low-

SCT ( $M=.82$ ,  $SD=.20$ ) and high-and-moderate SCT ( $M=.76$ ,  $SD=.16$ ) individuals,  $t(105)=1.313$ ,  $p(\text{one-tailed})=.096$ .

**Hypothesis 4, 4a, and 4b: Group SCT Differences**

**Hypothesis 4a: Group SCT Differences in Method A.** Using the Wilks’ Lambda criterion, the MANOVA for the Method A procedure revealed that the combined dependent variables were not significantly affected by group membership (i.e., less symptomatic vs. more symptomatic),  $F(6, 90)=.468$ ,  $p=.830$ . Similarly, no significant univariate differences between groups on any SCT variable were indicated. Mean scores for the Method A groups on the SCT measures are presented in Table 7.

Table 7: Mean Values for SCT Items in Method A (Cluster Analysis) Groups

Group	WAIS-III PSI*	TMT A†	Conners’ CPT RT†	WJ-III Reading Fluency*	WJ-III Math Fluency*	WJ-III Writing Fluency*
Less Symptomatic (n=70)	95.56(12.98)	42.35(9.43)	51.75(11.12)	93.32(12.63)	90.73(11.65)	102.23(11.72)
More Symptomatic (n=37)	94.60(13.88)	43.26(12.13)	50.24(13.07)	95.11(13.39)	92.97(12.98)	104.71(11.63)

\*Value is presented as a standard score (mean = 100, SD = 15); †Value is presented as a t-score (mean = 50, SD = 10)

**Hypothesis 4b: Group SCT Differences in Method B.** The MANOVA for the Method B procedure indicated, via the Wilks’ Lambda criterion, the combined dependent variables were not significantly affected by group membership (i.e., ADHD-Combined type vs. ADHD-Predominantly Inattentive type),  $F(6, 64)=.822$ ,  $p=.557$ . Similarly, no significant univariate between-groups differences were revealed. The mean scores for the Method B groups on the SCT measures are included in Table 8.

Table 8: Mean Values for SCT Items in Method B (DSM-IV-TR Diagnosis) Groups

Group	WAIS-III PSI*	TMT A†	Conners' CPT RT†	WJ-III Reading Fluency*	WJ-III Math Fluency*	WJ-III Writing Fluency*
Combined type (n=27)	92.89(15.25)	41.15(9.07)	52.20(13.67)	90.37(13.16)	91.33(14.74)	100.56(9.85)
Predominantly Inattentive type (n=44)	95.84(12.19)	42.57(12.20)	51.01(10.05)	94.98(13.47)	90.70(11.72)	105.27(13.47)

\*Value is presented as a standard score (mean = 100, SD = 15); †Value is presented as a t-score (mean = 50, SD = 10)

**Hypothesis 4c: Group SCT Differences in Method C.** The Wilks' Lambda value of the MANOVA for the Method C combined procedure indicated that the combined dependent variables were not significantly affected by group membership (i.e., low-SCT vs. high-and-moderate SCT),  $F(6, 90)=1.808, p=.106$ . Univariate analysis revealed one significant between-groups difference on WJ-III Reading Fluency,  $F(1,95)=8.542, p=.004, \eta^2=.082$ , with the low-SCT category exhibiting a higher WJ-III Reading Fluency score than the high-and-moderate SCT category. As there were only two levels to the independent variable, follow-up post-hoc analyses were not necessary. Mean scores for the Method C groups are provided on Table 9. For the sake of completeness, all Method C groups (i.e., low-SCT, moderate-SCT, high-SCT, and high-and-moderate SCT) are included in Table 9.

Table 9: Mean Values for SCT Items in Method C (Level of SCT) Groups

Group	WAIS-III PSI*	TMT A†	Conners' CPT RT†	WJ-III Reading Fluency*	WJ-III Math Fluency*	WJ-III Writing Fluency*
Low- SCT (n=75)	95.48(14.14)	42.75(10.70)	51.38(12.13)	95.96 <sup>a</sup> (13.30)	92.25(11.84)	103.79(12.19)
Moderate- SCT (n=17)	94.06(9.53)	41.76(10.18)	49.80(10.68)	85.82(8.58)	87.24(13.60)	99.00(9.70)
High- SCT (n=5)	95.20(11.88)	44.80(8.32)	53.40(12.46)	91.80(6.76)	95.40(9.53)	107.20(7.19)
High- and- Moderate SCT (n=22)	94.32(9.81)	42.45(9.68)	50.62(10.90)	87.18 <sup>a</sup> (8.44)	89.09(13.06)	100.86(9.69)

\*Value is presented as a standard score (mean = 100, SD = 15); †Value is presented as a t-score (mean = 50, SD = 10); <sup>a</sup>Scores were significantly different ( $p < .01$ )



## DISCUSSION

The goals of the current project were to attempt to distinguish individuals with a posited purely inattentive/non-hyperactive condition from those with ADHD, and to then compare these groups of individuals on measures of IIV and SCT. In the course of the project's development, additional components were added such that ADHD diagnostic categories, as defined by the DSM-IV-TR, were evaluated regarding IIV and SCT. Additionally, the construct of SCT potentially separate from its role as a marker of a purely inattentive condition was included, and its relationship with IIV partially examined. Novel aspects of this study compared with prior research included the use of the WURS, which assesses retrospectively-reported childhood ADHD-related difficulties rather than current symptoms, to identify purely inattentive adults; the inclusion of intraindividual standard deviation, evaluated across multiple cognitive functions rather than a single domain, as a measure of IIV in ADHD; the attempt to identify objective cognitive measures associated with self-reported SCT symptoms in adults; and the partial evaluation of the relationship between SCT and IIV in adults. Prior research examining the suspected purely inattentive condition, which has focused largely on the concept of SCT and its associated interpersonal characteristics, has typically only studied children/adolescents. Additionally, objective cognitive data related to SCT has often been limited to single test scores (e.g., reaction time) and/or single cognitive domains (e.g., attention). Only one study to date has examined SCT in adults (Barkley, 2011a), and in it, cognitive performance was assessed via self-report data of daily executive functioning. Likewise, few studies have examined IIV in adults, with none having evaluated ISD across an in-depth psychometric testing battery tapping multiple cognitive domains. Finally, only one study has evaluated the relationship between SCT and

variability, and it did so in children/adolescents and without the use of a well-supported measure of actual IIV (Skirbekk et al., 2011).

As a whole, the majority of the proposed hypotheses were not supported via statistically-significant results. However, the project was still able to answer questions regarding the viability of the WURS as a means of identifying individuals with a purely inattentive condition. Additionally, multiple observed trends in the data provided limited insights into the relationship between IIV and SCT. Similarly, trends revealed elsewhere in the project possibly supported the extension of previous work showing increased IIV in more-impaired vs. less-impaired individuals with ADHD, as measured by objective cognitive test performance, to more-symptomatic vs. less-symptomatic individuals as measured by self-report. These insights and possible extensions of prior findings allowed for the development of several recommendations for directed future research, not least of which involved the relationship between SCT and pure inattention. The experimental questions will now be addressed, and their relevant results and data trends discussed, in a topic (i.e., identification of a purely inattentive group, IIV, and SCT)-by-hypothesis (i.e., 1, 2, 3, and 4) format.

### **Identification of a Purely Inattentive Subgroup (Hypothesis 1)**

While cluster analysis has proven to be promising in other areas of psychological and social science research, its utility in the current project was relatively limited. The cluster analysis procedure was able to successfully identify two distinct and validated groups, and thus Hypothesis 1 was supported in this respect. However, the characteristics of the two groups, as evaluated by average WURS scores, were not entirely as was predicted. Neither group was consistently higher on all of the SCT-related WURS items while also being consistently-lower on the non-SCT-related WURS items. While one of the groups did have a slightly higher score

on the “Slow Reader” symptom (i.e.,  $M=1.82$  vs.  $M=1.66$ ), this same group exhibited lower scores on every other WURS item, including the other SCT-like symptoms (i.e., “shy, sensitive” and “inattentive, daydreaming”), which was not expected based on findings in previous literature regarding interpersonal behavioral factors associated with SCT (Carlson & Mann, 2000; McBurnett, Pfiffner, & Frick, 2001; Milich, Balentine, & Lynam, 2001). Rather than identifying purely-inattentive and purified ADHD groups, then, it appeared as though the cluster analysis procedure identified a more-symptomatic ADHD group and less-symptomatic ADHD group. This finding may be due to the cluster analysis procedure itself, but is also likely an artifact associated with the WURS, indicating its relatively poor ability to identify pure inattention as reflected by SCT characteristics in adults via retrospective self-report of childhood symptoms. Such a result is not entirely unexpected, as the WURS was never intended to measure SCT symptoms explicitly and so does not specifically include any such symptoms. Additionally, many of the WURS items that contain SCT-like characteristics also contain ADHD-like symptoms and behaviors (e.g., “inattentive” is combined with “day dreaming”). These items’ ability to discern SCT from ADHD, then, is compromised. Finally, the accuracy of young adults, particularly those in the age range of the groups in the current study (i.e., early-20’s), is questionable when they are asked to retrospectively recall childhood symptoms of ADHD (Barkley, Murphy, & Fischer, 2009). Thus, a variety of plausible explanations exist as to why the WURS’s was unable to identify SCT/pure inattention.

However, it is nonetheless interesting that the less-symptomatic group endorsed slightly higher levels on only the “slow reader” symptom than did the more-symptomatic group. This finding is somewhat paradoxical given the former group’s lower ratings on all of the other eleven items. It could possibly be the case that the WURS, coupled with the cluster analysis procedure,

did separate out purely inattentive individuals. However, owing to the imprecision of both methods, or perhaps to the greater sensitivity of the WURS to ADHD than to SCT/pure inattention, it was not able to fully distinguish purely inattentive participants from those with an overall less-severe ADHD. Thus, the less-symptomatic group may have been akin to a new “version” of the Predominantly Inattentive subtype—the latter has been posited to include both purely inattentive and subthreshold Combined type individuals (Barkley et al., 2008; Hinshaw, 2001; Milich et al., 2001), while the former may have included purely inattentive and less-severe ADHD participants. It is also possible that the “slow reader” item is negatively-correlated with ADHD pathology rather than being positively-correlated with SCT, perhaps owing to the former’s hyperactive component. This idea is somewhat rebutted by the “slow reader” symptom not having been included in the list of 25 items used to derive the WURS total score, as the scale’s authors selected into this group by choosing items with the strongest association, whether positive or negative, with ADHD diagnosis (Ward, Wender, & Reimherr, 1993). However, the item may have been excluded not because it failed to negatively correlate with ADHD, but because it might have done so too weakly to be included in the list of the 25 best items.

### **Examining Group IIV Differences in Method A (Hypotheses 2a and 3b)**

In Hypothesis 2a, I predicted that the two groups identified via cluster analysis would differ significantly on RTSE, with the purified ADHD group being significantly more variable than the purely inattentive group. This prediction was based on research indicating that heightened RTSE is one of the most frequently-replicated findings in all of ADHD-related neuropsychological research (Nigg, 2005). However, as the groups identified in Hypothesis 1 were not akin to purely inattentive and purified ADHD categories, it is not particularly surprising that no significant between-group difference was discovered. What is interesting, though, is the

non-significant trend that the less symptomatic group exhibited a greater mean RTSE than the more symptomatic group. On the other hand, this result could merely be an artifact/chance finding related to data, methodology, and analysis issues. For example, other than indirectly via the establishment of a clinical cut-off (Ward, Wender, & Reimherr, 1993), no existing data suggests that higher scores on the Wender correlate with greater ADHD-related impairment, and so it might not be expected that a WURS-based “more symptomatic” group would exhibit more ADHD-related cognitive disturbance such as heightened RTSE than a “less symptomatic” such group.

Similar to Hypothesis 2a, I initially determined Hypothesis 3a based on an assumption that cluster analysis using WURS items could successfully identify purely inattentive and purified ADHD groups. The failure of this assumption significantly detracted from my ability to hypothesize about the actual identified groups, with the analyses then becoming highly exploratory. The lack of a significant group difference on ISD between less symptomatic and more symptomatic individuals provided further support that, at least with respect to IIV, these two categories (as operationalized by WURS responses) were not particularly informative to the project at hand. What can be said is that a higher degree of reported WURS symptomology on the included items did not coincide with a significantly higher degree of ISD. However, as with RTSE, a non-significant trend showed that the more symptomatic group did exhibit a higher mean ISD than the less symptomatic group.

Taken together, the non-significant IIV variable trends observed in the results for both Hypotheses 2a and 3a could indicate that higher levels of ADHD symptoms may be associated with higher levels of IIV. This type of concomitant increase in degree of reported ADHD symptoms and level of observed IIV would tentatively support a relationship between these two

factors (i.e., ADHD and IIV), which would of course be necessary for the latter to be an endophenotype of the former. Nonetheless, the lack of statistical significance in both sets of analyses suggests that the size of this effect may be small, that the WURS may not be particularly adept at delineating severity groups and/or capturing ADHD symptom severity in a linear manner, or that the relationship may in truth be non-existent and simply an artifact of chance and/or error variability. Additionally, even if the relationship is genuine, it may be related to an unstudied factor or series of factors rather than the two directly studied in this dissertation.

Regarding the IIV measures themselves, the increased size of the non-significant between-groups difference for ISD versus RTSE from the Conners' CPT suggested that the former may actually be a more sensitive measure of IIV as related to reported ADHD symptom severity. This increased sensitivity may be due to the ISD measure's ability to capture variability across a range of tasks, particularly those with increased cognitive complexity, while the Conners' CPT is a somewhat simpler task, with previous research suggesting that IIV in ADHD may be related to and more apparent in tasks of increased complexity and/or higher working memory demand (Buzy, Medoff, & Schweitzer, 2009; Klein et al., 2006; Pollak et al., 2009; Vaurio, Simmonds, & Mostofsky, 2009). However, the above-mentioned caveats regarding the WURS as a measure of ADHD symptom severity are relevant here. That is, if the WURS is not measuring ADHD symptom severity, then ISD may instead be more related to whatever extraneous and/or unstudied characteristic the WURS is capturing.

### **Examining Group IIV Differences in Method B (Hypotheses 2b and 3b)**

I tentatively predicted in Hypothesis 2b that after grouping individuals based on DSM-IV-TR ADHD subtype, those with the Combined type would exhibit significantly different RTSE scores from those with the Predominantly Inattentive type. This hypothesis was based on

very limited and ambiguous existing research suggesting that the two examined ADHD subtypes might differ with respect to RTSE (Desman, Petermann, & Hampel, 2008). Additionally, previous researchers have suggested that the Predominantly Inattentive type consists of individuals with subthreshold ADHD along with those who have a purely inattentive/non-hyperactive condition that may be distinct from ADHD (Barkley et al., 2008; Hinshaw, 2001; Milich et al., 2001). One could then posit that if IIV is greater in individuals with ADHD than those without, and if individuals with a purely inattentive condition do not actually have ADHD, then the presence of these purely inattentive/non-ADHD individuals in the Predominantly Inattentive type group would change its overall level of IIV as measured by RTSE. The results from the independent-samples *t*-test did not support this hypothesis, though, as no statistically-significant between-group effect was revealed.

Similarly, the non-significant results from the independent samples *t*-test of ISD differences between Combined Type and Predominantly Inattentive type groups did not support the proposed Hypothesis 3b, which stated that the two ADHD subtype groups would significantly differ with respect to ISD. However, a non-significant trend was observed in the results of Hypotheses 2b and 3b, with the Combined Type group exhibiting a slightly higher mean ISD and RTSE than the Predominantly Inattentive type group in both instances. Such non-significant trends are highly tentative and must be interpreted with extreme caution, as they may simply reflect chance findings. Nonetheless, this trend is consistent with the aforementioned theory that the Predominantly Inattentive group consists of purely inattentive and subthreshold Combined type individuals. The presence of the trend, then, could be explained by the existence of a purely inattentive group in the Predominantly Inattentive category. Similarly, the non-significance of the results could be due to the presence of subthreshold Combined Type

individuals in the Predominantly Inattentive category, which would have decreased the size of the between-group differences in the current study. Such obfuscation of group differences is similar to that which may have occurred in the majority of prior ADHD research that included Predominantly Inattentive individuals without attempting to screen out or account for SCT/pure inattention.

### **Examining Group IIV Differences in Method C (Hypotheses 2c and 3c)**

With the failure of the cluster analysis methodology to successfully identify a purely inattentive category, Method C was the only grouping option remaining that could evaluate the relationship between IIV and SCT/pure inattention. Unfortunately, as Method C did not attempt to identify pure inattention by establishing an absence of hyperactivity/"typical" ADHD symptoms, it was only able to examine the effects of SCT level, independent of pure inattention, on IIV. Given that previous research has supported the presence of a high level of comorbidity between SCT and ADHD in adults (Barkley, 2011a), the two conditions could have very well co-occurred in Method C, and thus evaluation of IIV's status as an endophenotype of ADHD was not viable. Regardless, statistical analysis via ANCOVA did not indicate the presence of any significant between-group differences on either RTSE or ISD.

Qualitatively, review of the data indicated that the moderate-SCT group actually exhibited the highest degree of RTSE, while the low- and high-SCT groups scored similarly on this variable. As with any non-significant result, these trends could simply have been chance occurrences related to normal and error-related variability within the samples. However, the direction of the trends is very interesting in light of Barkley's (2011a) data. In his study, which is the only to date to examine SCT in adults, individuals with both ADHD and SCT showed generally greater self-reported executive dysfunction and poorer functional outcomes than did



controls and individuals with either of the two conditions in isolation. The trend in the current study, then, could suggest that my moderate-SCT group was akin to Barkley's ADHD+SCT group.

If this were the case, and my moderate-SCT category were the equivalent of a comorbid ADHD+SCT group, I would have expected these individuals to exhibit a trend of greater ISD values than both the low- and high-SCT groups. However, the Hypothesis 3b results did not indicate the presence of such a trend. Rather, the trend in ISD analysis results was one in which high-SCT individuals displayed the lowest ISD, low-SCT individuals displayed the highest ISD, and moderate-SCT individuals fell between these two extremes. This is the pattern of results that would have been expected were SCT mutually-exclusive with ADHD (i.e., high-SCT would equate to low-ADHD, etc.). To make matters even more ambiguous, while the three levels of SCT (i.e., low, moderate, and high) did not significantly differ with respect to total WURS score, there was a non-significant trend indicating increasing average WURS total scores with each increasing level of SCT. Therefore, it could be stated that the high-SCT group was not, in fact, only a high-SCT group; rather, these individuals were high on the WURS as a whole, just as the moderate-SCT individuals were moderate on the WURS as a whole, and low-SCT individuals were low regarding overall WURS total score. Thus, there might be an unexamined variable related to individual response style, such as over-/under-endorsing, that could potentially explain or contribute to both the WURS total scores and ISD levels.

#### **Examining Group SCT Differences in Method A (Hypothesis 4a)**

The results of the MANOVA analysis for Hypothesis 4a indicated that the less and more symptomatic groups did not significantly differ with respect to any of the six SCT-related cognitive test scores or to their multivariate combination. Qualitatively, there were no

immediately-identifiable trends across the results, as each of the two groups performed better on three of the six measures (see Table 9). Such a pattern could tentatively indicate that ADHD symptom severity was not significantly related in the current sample to performance on these six cognitive testing indices, and/or that these indices are not highly sensitive to symptom severity differences. However, as has been previously mentioned, there is no prior data to support the contention that higher WURS scores on the items included in the current project equate to higher degrees of ADHD symptom severity or impairment. Conversely, data do generally support the presence of significant neuropsychological deficits in ADHD on either the exact included cognitive measures, or measures that tap similar cognitive constructs (i.e., processing speed; Nigg, 2005; Woods, Lovejoy, & Ball, 2002).

As the present analysis was adequately powered to detect anticipated effect sizes, the most plausible conclusion of the null findings for Hypothesis 4a is that higher scores on the twelve selected WURS items do not adequately capture and/or are not appropriately sensitive to ADHD symptom/impairment severity. As not all of these items used for Method A were a part of the 25 symptoms from the WURS used to obtain a total score on the measure, and as these 25 scored items were selected by the scale's authors based on sensitivity to ADHD diagnosis/pathology (Ward, Wender, & Reimherr, 1993), it is not surprising that the twelve items in the current analysis were insensitive to ADHD symptom severity.

#### **Examining Group SCT Differences in Method B (Hypothesis 4b)**

Prior research generally does not extensively support significant between-group differences in neuropsychological functioning across ADHD diagnostic subtypes (Hinshaw et al., 2007; Nigg, 2005; Woods, Lovejoy, & Ball, 2002), leading some authors to propose that these subtypes are neuropsychologically-heterogenous in nature (Nigg, 2005). The results from

Hypothesis 4b did nothing to contradict these views, and were largely in agreement with previous findings. That is, the MANOVA testing Hypothesis 4a indicated that there were no significant multivariate or univariate effects for the independent variable of ADHD diagnostic category on the six included cognitive testing indices.

### **Examining Group SCT Differences in Method C (Hypothesis 4c)**

Perhaps the most potentially-informative of the three multivariate analyses in the current project was that for Method C, particularly after the failure of cluster analysis to delineate the anticipated/predicted purely inattentive and purified ADHD groups. Disappointingly, though, the overall multivariate effect of the MANOVA for Hypothesis 4c was not significant. Univariate analyses did indicate that the high-and-moderate SCT group was significantly slower on WJ-III Reading Fluency than the low-SCT group. Qualitative analysis of mean values on the various SCT variables indicated that as with the results for Hypothesis 2c, the moderate-SCT group actually performed slightly worse than both the low-SCT and high-SCT groups (see Table 9 above). These results would support a contention that my moderate-SCT category was similar to Barkley's (2011a) co-occurring ADHD+SCT group, which reported greater functional impairment and executive dysfunction relative to the two non-co-occurring groups (i.e., ADHD and SCT).

### **Conclusions and Future Directions**

The current project, while providing few statistically-significant findings in support of its predicted hypotheses, can nonetheless offer multiple novel insights into the relationships between ADHD, SCT, IIV, and pure inattention. First and foremost, the results do not support the use of the WURS in combination with cluster analysis in identifying purely inattentive individuals via SCT-like characteristics. Rather, such a methodology instead resulted in the

identification of more symptomatic and less symptomatic groups. Furthermore, subsequent analyses of IIV and suspected SCT-related cognitive testing performance indices suggested that the twelve selected WURS items were not sensitive enough to ADHD severity/pathology to result in identification of groups that significantly differed on any of the dependent variables, despite these dependent variables having prior experimental support showing their sensitivity to ADHD-related cognitive disturbances. Second, the current project was consistent with previous neuropsychological research in its inability to demonstrate significant group differences between the ADHD Combined type and Predominantly Inattentive type on six cognitive measures thought to be associated with SCT and two measures of IIV (including one, ISD, which had not previously been evaluated with respect to adult ADHD in any capacity). Third, the project was initially thought to have provided tentative support to the finding by Barkley (2011a) that a co-occurring ADHD+SCT group, akin to the current study's moderate-SCT group, might exhibit greater dysfunction than both the ADHD and SCT groups alone. However, Barkley's co-occurring group reported higher levels of SCT symptoms than did the stand-alone SCT group, while the current study's moderate-SCT group did not report the highest degree of SCT-like characteristics. Fourth, in evaluating ISD and RTSE as measures of IIV, the current project indicated that ISD may be more sensitive to variability when dividing—or attempting to divide—groups using SCT characteristics. Indeed, in both such Methods (i.e., A and C), the test statistics for ISD were higher than those for RTSE. Of note, this trend might instead reflect a greater sensitivity of ISD to varying levels of ADHD symptom severity, as in Methods A and C, the more symptomatic/high-SCT and moderate-SCT groups reported slightly (although not significantly) higher levels of ADHD symptoms overall.

There were multiple limitations to the current study, principal among them being the operationalization of SCT/pure attention as responses to selected items on the WURS. As was previously noted, the WURS was not developed to assess SCT/pure attention, and many of its SCT-like items also contain characteristics commonly associated with ADHD. Thus, it is entirely possible that the construct of SCT was never adequately measured, thereby precluding any opportunity to identify a purely inattentive group. The sample in the current study was relatively highly-educated, majority-Caucasian, and exhibited FSIQ scores that tended to fall in the upper-half of the average range. Thus, generalization of any of the current findings to different samples may be inappropriate, and at the least should be performed with significant caution. The tiering and dichotomizing of the quasi-quantitative SCT variable in Methods C and C combined, respectively, may have decreased the ability of the analyses to detect between-group differences due to loss of individual information, effect size, and power (McCallum, Zhang, Preacher, & Rucker, 2002). More basically, the selection of the SCT-like WURS items itself may have been flawed, as it was highly theoretically and with limited direct empirical support.

In light of these limitations, and in addition to the previous trends, multiple avenues of further research are warranted. A repetition of many of the included methods using a more well-established measure of SCT, such as the items chosen by Barkley (2011a) in his analysis of SCT in adults, would allow for greater consistency across studies, and may lead to more informative results. As has been suggested elsewhere, evaluation of IIV across time in conjunction with other cognitive task components, such as accuracy, could more thoroughly evaluate the ways in which IIV influences cognitive functioning (MacDonald, Li, & Backman, 2009). Perhaps even more ambitiously, research could begin examining the exact role SCT plays in the posited purely inattentive condition. Given the high degree of co-occurrence with ADHD (Barkley, 2011a), as

well as possible comorbidity with anxiety disorders (Skirbekk et al., 2011), SCT does not appear to be unique to pure inattention. Studies examining what other characteristics are associated with the subset of individuals who are diagnosed with ADHD yet who exhibit SCT along with a lifelong inattention could be exceedingly beneficial in determining better ways of diagnosing the condition. Additionally, further exploration of the cognitive underpinnings and effects of SCT is warranted, given the continued dearth of multi-domain assessments of these issues.

The purely inattentive condition remains a relatively elusive construct. Even its most highly-associated characteristic, SCT, does not appear to be exclusively in the domain of pure inattention. Further research is necessary to more thoroughly-understand this long-hypothesized, yet poorly-understood concept. As its boundaries are better-elucidated, those of ADHD will also become more distinct and specific, potentially leading to continued advances in diagnosis and treatment of both conditions.

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# APPENDIX A: INFORMED CONSENT FORM



College of Arts and Sciences  
Psychological Services Center,  
Department of Psychology

## CONSENT FOR PSYCHOLOGICAL SERVICES (ADULT)

I understand that my participation in psychological evaluation and/or treatment at the LSU Psychological Services Center (LSU PSC) is strictly voluntary. I understand that my service provider will be a psychologist or trainee under the direct supervision of a psychologist. I am requesting EVALUATION/COUNSELING services (circle one or both).

I understand that all information obtained in the course of my activities at the LSU PSC is confidential, and can be released only with written consent. Exceptions to this legal safeguard are as follows:

1. If I am a danger to myself or others
2. If my records are subpoenaed by a court of law
3. If my provider learns of the abuse or neglect of any child or adult who cannot physically or mentally protect themselves from harm.

I understand that the LSU PSC is a teaching and research clinic, and that my sessions may be observed or audio/videotaped for training purposes, and that the data from my case file may be used for research purposes. In the event of any such use, my identity will remain totally anonymous, and no record connecting my name with my data will be recorded in any research file. The LSU Institutional Review Board will review and approve in advance any projects conducted using data from LSU PSC case files. I understand that I am free to refuse the use of my data for research purposes at any time, without prejudice affecting my eligibility for clinical services. Any questions concerning clinical or research uses of my case file can be directed to the LSU PSC Director, Amy L. Copeland, Ph.D. at 225-578-1494.

My signature to this form indicates that I have read the information about voluntary consent to receive services and the potential use of my clinical data for research purposes. All of my questions have been answered to my satisfaction, and my consent is granted freely and without coercion.

\_\_\_\_\_  
Signature

\_\_\_\_\_  
Date

\_\_\_\_\_  
Research Consent Y/N

\_\_\_\_\_  
Witness

\_\_\_\_\_  
Date

## APPENDIX B: ITEMS OF THE WENDER UTAH RATING SCALE

### As a child I was (or had):

1. Active, restless, always on the go
2. Afraid of things
3. Concentration problems, easily distracted
4. Anxious, worrying
5. Nervous, fidgety
6. Inattentive, daydreaming
7. Hot- or short-tempered, low boiling point
8. Shy, sensitive
9. Temper outbursts, tantrums
10. Trouble with stick-to-it-tiveness, not following through, failing to finish things started
11. Stubborn, strong-willed
12. Sad or blue, depressed, unhappy
13. Incautious, dare-devilish, involved in pranks
14. Not getting a kick out of things, dissatisfied with life
15. Disobedient with parents, rebellious, sassy
16. Low opinion of myself
18. Outgoing, friendly, enjoyed company of people
19. Sloppy, disorganized
20. Moody, ups and downs
21. Angry
22. Friends, popular
23. Well-organized, tidy, neat
24. Acting without thinking, impulsive
25. Tendency to be immature
26. Guilty feelings, regretful
27. Losing control of myself
28. Tendency to be or act irrational
29. Unpopular with other children, didn't keep friends for long, didn't get along with other children
30. Poorly coordinated, did not participate in sports
31. Afraid of losing control of self
32. Well-coordinated, picked first in games
33. Tomboyish (for women only)
34. Running away from home
35. Getting into fights
36. Teasing other children
37. Leader bossy
38. Difficulty getting awake
39. Follower, led around too much
40. Trouble seeing things from someone else's point of view
41. Trouble with authorities, trouble with school, visits to principal's office
42. Trouble with police, booked, convicted

**Medical problems as a child:**

- 43. Headaches
- 44. Stomachaches
- 45. Constipation
- 46. Diarrhea
- 47. Food allergies
- 48. Other allergies
- 49. Bedwetting

**As a child in school, I was (or had):**

- 50. Overall a good student, fast learner
- 51. Overall a poor student, slow learner
- 52. Slow in learning to read
- 53. Slow reader
- 54. Trouble reversing letters
- 55. Problems with spelling
- 56. Trouble with mathematics or numbers
- 57. Bad handwriting
- 58. Able to read pretty well but never really enjoyed reading
- 59. Not achieving up to potential
- 60. Repeating grades (which grades?)
- 61. Suspended or expelled (which grades?)



## VITA

Daniel Proto was born in Plano, Texas in January, 1980 to mother Denise Proto and father Vincent Proto. He spent his early years in a variety of states, including Texas, New York, and Massachusetts, before finally settling in Georgia in the fall of 1985.

Educationally, Daniel performed well, entering the gifted program in early elementary school, and continuing with advanced courses through middle and high schools. Daniel's first long-standing career goal, formed in late junior high, involved astronomy and astrophysics. However, this aspiration began shifting toward psychology by the time he had entered college at the University of Georgia in 2001, and was further established and entrenched with each additional course taken.

One year after obtaining his Bachelor of Science degree in 2004, Daniel enrolled in the clinical psychology doctoral program at Louisiana State University in Baton Rouge, where he studied under Professor Wm. Drew Gouvier. Daniel successfully completed his master's thesis on the malingering of intellectual disability, and general examination involving a case report of the neuropsychological assessment of progressive supranuclear palsy, under Dr. Gouvier's tutelage. Mr. Proto then moved to Birmingham, Alabama, where he is currently completing a one-year pre-doctoral internship in clinical psychology with the University of Alabama—Birmingham Department of Psychiatry and Behavioral Neurobiology and Birmingham Veterans Affairs Medical Center. Following the conclusion of this internship, Daniel will be moving to Houston, Texas, where he has accepted an offer for a post-doctoral fellowship in clinical neuropsychology at the Michael E. DeBakey VA Medical Center.