Information Processing Speed in Adults with ADHD

by

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Abstract

The Computerized Tests of Information Processing (CTIP) and the Adjusting-PASAT (A-PASAT) are two recently developed tests of Information Processing Speed (IP speed). The purpose of this study was to determine if these two new tests could be useful in the identification of Attention Deficit/Hyperactivity Disorder (ADHD) in adults. The CTIP and the A-PASAT were administered to three groups of undergraduate students, Pure ADHD, ADHD plus a coexisting Learning Disability (ADHD/LD) and nonclinical Controls. Also administered were the Conners Continuous Performance Test (CCPT-II) and a number of other traditional neuropsychological tests of attention. Significant differences were observed on the CTIP, A-PASAT, the Visual Matching subtest from the Woodcock Johnson Tests of Cognitive Ability (WJ-III), and the Digit Symbol Coding and Digit Span tests from the Wechsler Adult Intelligence Scales (WAIS-III). However, these tests were not sensitive to ADHD. Instead, the performance of only the ADHD/LD group was significantly impaired, with the Pure ADHD and nonclinical Controls performing equivalently. Results underline the importance of separating ADHD groups according to the presence of LD. Future research should include a fourth, Pure LD group in order to determine if the observed IP speed impairment in the ADHD/LD group was solely a consequence of having LD, or was due to the presence of both ADHD and LD.

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Introduction

What is ADHD?

Attention Deficit Hyperactivity Disorder (ADHD) is the latest in a series of diagnostic terms for a behavioural syndrome first described in 1902. While the population is highly heterogeneous, all individuals with ADHD exhibit some combination of symptoms of inattention, impulsivity, and hyperactivity: the so-called "holy trinity" of ADHD (Barkley, 1998). ADHD first occurs in childhood, and, has been recently acknowledged, continues into adulthood in a significant proportion of cases (American Psychiatric Association [APA], 1994). According to the fourth edition of the Diagnostic and Statistical Manual of the American Psychiatric Association (DSM-IV; 1994), along with Conduct Disorder (CD) and Oppositional Defiant Disorder (ODD), ADHD is classified as a Disruptive Behaviour Disorder, which is in turn a sub-class of Disorders Usually First Diagnosed in Infancy, Childhood, and Adolescence. There are three types of ADHD: Inattentive (ADHD-IA), Hyperactive/Impulsive (ADHD-H/I) and Combined (ADHD-C), diagnosed on the basis of the presence of six or more of eleven core symptoms of inattention and/or at least six of nine core symptoms of hyperactivity-impulsivity. There must also be evidence that present symptoms are highly maladaptive, inconsistent with developmental level, and have persisted for at least six months prior to diagnosis. Symptoms must be manifest in more than one area (e.g. school, work, and home), and pervasive developmental disorder or any psychotic, mood, anxiety, dissociative, or personality disorder must be ruled out as the primary cause of symptoms. Finally, there must be evidence that significant ADHD symptoms first appeared before age seven for diagnosis to occur (APA, 1994).

Because symptoms of inattention, hyperactivity and impulsivity are commonly observed in individuals with brain injury or disease, and because early research samples of

children with what is today called ADHD included individuals with known brain injury and disease, ADHD and its taxonomic predecessors (described below) have always been assumed to reflect an underlying CNS damage or dysfunction, with the role of the environment in most scientific models limited to a mitigating factor (Barkley, 1998). Current empirical support for this view includes twin and adoption studies, which have demonstrated that ADHD is genetically transmitted in a significant proportion of cases (Barkley, 1998; Wender et al., 2001). Research using functional brain imaging has implicated the dopaminergic "reinforcement" pathway linking the pre-frontal cortex to midbrain basil ganglian structures (Barkley, 1998; Biederman & Faraone, 2002; Giedd et al., 2001; Riccio & Reynolds, 2001). These findings are consistent with the knowledge that stimulant medications that increase brain levels of dopamine (such as Ritalin®) have been shown to reduce symptoms of ADHD in children and adults (Barkley, 1998; Biederman & Faraone, 2002; Wender et al., 2001).

Despite empirical evidence for an underlying organic pathology, however, ADHD cannot be definitively diagnosed through physiological indicators (Barkley, 1998; Conners & Lett, 1999; Hallowell & Ratey, 1994; Wender et al., 2001). Furthermore, while an increasing number of clinicians use objective neuropsychological tests to supplement ADHD assessment, no test has emerged as consistently reliable (Barkley & Grodzinsky, 1994; Gordon & Barkley, 1998; National Institutes of Health [NIH], 1998). Hence, clinical diagnosis of ADHD continues to be accomplished through highly subjective methods such as interviews and rating scales (Conners & Lett, 1999; Hallowell & Ratey, 1994; NIH, 1998; Wender et al., 2001).

Besides the lack of an objective physiological or neuropsychological test, other factors add uncertainty to ADHD diagnosis. As mentioned above, ADHD encompasses a highly

diverse population. Children and adults with ADHD are highly heterogeneous with respect to unique combinations of individual DSM-IV symptoms, along with their particular impact at work, home, and school. Furthermore, symptoms of inattention and impulsivity/hyperactivity, while assumed to be organic in etiology, tend to interact with the environment, appearing and reappearing to a greater or lesser extent with ever-changing behavioural consequences as the child, adolescent, and adult with ADHD confronts new challenges, including a greater-than-normal risk of dysfunctional outcomes, such as academic and vocational underachievement, poor peer relationships, marital instability, and substance abuse (Hallowell & Ratey, 1994; Weiss et al., 2002).

Another major factor contributing to the uncertainty associated with ADHD diagnosis is its resemblance to many other medical, learning, and psychiatric disorders (Barkley, 1998; Pearl et al., 2001). As stated in a review of this issue, the following subsume enough ADHD symptoms to satisfy diagnostic criteria: schizophrenic spectrum disorders, anxiety disorders (including social phobia, Post-Traumatic Stress Disorder, and Obsessive Compulsive Disorder), alcoholism and drug addiction, head injury, chronic fatigue syndrome, HIV, bulimia, and mental retardation (Taylor & Miller, 1997).

In addition to disorders that may mimic its symptoms, some disorders frequently occur concomitantly with ADHD, adding still more uncertainty to diagnosis. The most common is Learning Disability (LD): It is estimated that 80% of Canadian children with ADHD also have LD (Learning Disabilities Association of Canada [LDAC], 2001). While considered separate disorders with distinct etiologies, ADHD and LD have many common outcomes, including disorganization, poor metacognitive functioning, and inefficient use of strategies (Learning Disabilities Association of Ontario [LDAO], n.d.), and an increased risk of underachievement at school and in the workplace (Barkley, 1998; Mirsky et al., 1999).

Other clinical conditions with a potential impact on attention are frequent ADHD comorbidities, particularly CD and ODD in children (Barkley, 1998) and major depression and anxiety disorders in adults (Downey et al., 1997). Other common comorbid disorders are Antisocial Personality Disorder, Somatization Disorder (APA, 1994), Tourette's syndrome, Autism (Ontario Ministry of Health, 2002), and Borderline Personality Disorder (Wender, 1995).

Diagnostic uncertainty notwithstanding however, it is generally acknowledged that ADHD is one of the most prevalent child psychiatric disorders in North America today: the most common reason children are referred to mental health practitioners (Barkley, 1998; Conners & Lett, 1999; NIH, 1998; Wender et al., 2001). It is estimated that between five and ten percent of Canadian schoolchildren have ADHD (Human Resources Development Canada [HRDC], 2002). ADHD is far more common in boys than girls (HRDC, 2002; NIH, 1998) although the precise ratio varies across studies, with an average 6:1 ratio across clinical samples, and an average ratio of 3.4:1 among non-referred, community samples (Barkley, 1998). Consensus-based estimates are unavailable for adults, both here (HRDC, 2002) and in the U.S. (Barkley, 1998; Wender et al., 2001). Independent adult prevalence estimates are highly variable, with anywhere from 30 to 60 percent of childhood cases believed to persist into adulthood (e.g. Murphy & Gordon, 1998; Wender, 1995).

Brief history of ADHD[1]. The first published report of a condition resembling ADHD is attributed to English physician George Still (1902), who described 43 children from his clinical practice. Still attributed their disruptive behaviours (e.g., hyperactivity, aggression, defiance, cruelty, spitefulness, dishonesty, and insensitivity to punishment) to a defect in "moral control", an active cognitive process whereby an individual compares intended

behaviours to past experience, speculates as to their potential impact on the "greater good", and then behaves accordingly. Still's subjects included many children who would not likely be identified as having ADHD today, as many had a history of brain injury or disease. That being said, in this earliest precursor to today's "holy trinity" of ADHD symptoms, Still specifically described his patients as "hyperactive" and "inattentive". While he did not use the term "impulsivity", two defining features of the disorder, "heightened emotionality" and an "uncontrollable need for immediate gratification" are consistent with this symptom category.

The next major advance came from descriptions of Postencephalitic Behaviour Disorder in children who had survived the 1917-1918 encephalitis epidemic in North America. This further strengthened the association of what is today termed ADHD to underlying brain pathology, and while the range of symptoms was extremely broad, many symptoms identified were similar to contemporary ADHD symptoms; in particular, attentional impairments, poor regulation of activity, and impulsivity. During the 1930s and 1940s, investigators studied children with other known brain pathologies, such as brain infection, lead toxicity, epilepsy, and head injury. They, too, described what they believed to be permanent impairments in attention, impulsivity, and hyperactivity (along with more severe behavioural and cognitive impairments). Terminology from this period reflects an assumption of underlying neuropathology, along with and emphasis on hyperactivity. "Organic Driveness", "Restlessness Syndrome" and "Brain Injured Child" were coined to describe a broad population of children with significant and chronic inattention, hyperactivity and impulsivity (along with more severe behavioural and cognitive impairments in many cases). When actual documentation of brain injury or disease was

[1] This discussion draws heavily from Barkley (1998) to which the reader is directed for a thorough review.

unavailable, and when hyperactivity was prominent, the disorder was assumed to result from non-localized brain damage, possibly due to birth trauma, brain infection, lead toxicity, or epilepsy. Milder cases were attributed at the time to environmental causes, such as poor parenting.

During the 1950s, the term "Minimal Brain Dysfunction" (MBD) emerged to describe "homogenous populations of cognitive, learning and behavioral disorders, such as dyslexia, language disorders, learning disabilities, and hyperactivity" (Barkley, 1998, p.8). MBD was assumed to reflect CNS pathology even in the absence of confirming documentation. Also during the 1950s, a theory appeared that for the first time, speculated on the specific neurological mechanisms for the disorder. Based on their study of motor stimulation thresholds, Laufer and colleagues posited that children with ADHD had a lower threshold for neural stimulation based in impaired filtering in the thalamus, which in turn resulted in overstimulation of the cortex, and hence, hyperactivity. During the late 1950s and early 1960s, prominent authors advocated the use of the term "Hyperactive Child Syndrome". This described the hyperactive child as "one who carries out activities at a higher than normal rate of speed than the average child, or who is constantly in motion, or both" (Barkley, 1998, p.8). It was believed that the syndrome was caused by impaired brain mechanisms; however, it could occur in the absence of brain damage or disease. In 1968, the newly-published DSM-II reflected this trend toward a focus on hyperactive symptoms and away from implied brain pathology with its description of Hyperkinetic Reaction of Childhood Disorder: "The disorder is characterized by overactivity, restlessness, distractibility, and short attention span, especially in young children; the behaviour usually diminishes by adolescence" (APA, 1968, p.50).

During the 1970s, Paul Wender repopularized the term Minimal Brain Dysfunction, which like its earlier version, assumed an underlying brain pathology. His six symptom clusters included the modern ADHD triad, along with other symptoms indicative of learning difficulties, impaired interpersonal relations, and emotional difficulties (including aggressive and defiant behaviours). Inattention, another symptom cluster, incorporated impairments in concentration, attention span, distractibility, daydreaming, and organization of thoughts. Hyperactivity was conceptualized as only one of a number of possible impairments in the category "motor behaviour", which also included hypoactivity (lethargy) and poor physical coordination. Impulsivity, or "impulse-control problems" was also precisely defined, and included low frustration tolerance, antisocial behaviour, poor planning and judgment, disorganization, and recklessness. Wender posited that symptoms from these six categories were the result of "three primary deficits: (1) a decreased experience of pleasure and pain, (2) a generally high and poorly modulated level of activation, and (3) extroversion" (Barkley, 1998, p.11).

Virginia Douglas was another pioneer in the understanding of ADHD who first advanced her ideas during the 1970s. Using a variety of neuropsychological and cognitive tests in children with what was then called Hyperkinetic Reaction of Childhood Disorder, Douglas concluded that hyperactivity is not the most crucial element, noting that while the hyperactive children displayed more frequent irrelevant movements overall, it had no adverse affect on their performance compared to controls (Douglas, 1972). In her view, inattention and impulsivity were more important symptoms, manifested as an inability to "stop, look, and listen": "These youngsters are apparently unable to keep their own impulses under control in order to cope with situations in which care, concentrated attention, or organized planning are required" (Douglas, 1972, p.275).

Fad theories of childhood ADHD first became popular during the 1970s, occurring simultaneously with the beginnings of a broad public awareness and an increase in the use of medication to control symptoms in children. Generally unfounded in empirical research, such theories first advanced the notion that the disorder and its symptoms were caused by food additives, then later, refined sugar. Others blamed socio-cultural factors, including rapid technological and cultural change, inconsistent social standards for child behaviour, and poor parenting. Still others claimed that ADHD was a myth perpetuated by the likes of greedy drug companies, lazy teachers, and a substandard educational system.

The DSM-III, published in 1980, replaced the term Hyperkinetic Reaction of Childhood with Attention Deficit Disorder (ADD). For the first time, significant hyperactive symptoms were not a requirement for diagnosis; rather, subtypes were specified based on the presence or absence of hyperactivity (ADD-H and ADD+H). However, in 1987, with the publication of the DSM-III-R, this emphasis on inattention as a defining feature was completely reversed, and only diagnostic criteria for ADD with hyperactivity were included. The disorder was renamed ADHD, with the formerly Inattentive subtype (ADD-H) replaced by to the category of "Undifferentiated ADHD". Today, as mentioned above, DSM-IV diagnostic criteria reflect the complete ADHD symptom triad: Inattention, hyperactivity, and impulsivity.

Contemporary explanations of the nature of ADHD. As discussed above, Virginia Douglas originally formulated her ideas during the 1970s on the basis of the performance of hyperactive children on a variety of laboratory tests, concluding that the deficits stemmed from a single underlying dimension: the inability to "stop, look, and listen". Inattention and impulsivity, but not hyperactivity, were considered key factors (Douglas, 1972). Douglas later expanded and refined her views, describing four primary deficits

stemming from an underlying impairment in what she now termed "self-regulation: impaired attention and effort, poor inhibitory control, arousal modulation problems, and an inclination to seek immediate reinforcement (Douglas, 1983). Still later, these were reconceptualized as three pervasive, "macro-level" control problems: failure to allocate adequate attention to meet task demands, high variability in performance, and, impulsive (or nonreflective) response style (Douglas, 1999). In addition, Douglas identified "micro-level" control problems, evident in tasks that require "consistent and sustained allocation of effort and attention, inhibition of inappropriate responding, preparation to process and respond to task stimuli, and flexible adaptation to changing task demands" (Douglas, 1999, p.130).

Russell Barkley, a second major theorist in the area of ADHD, developed his "Unifying Theory of ADHD" (1997, 1998) in response to what he perceived as a glaring need for a new, more comprehensive paradigm. He criticized the work of his contemporaries as being overly exploratory, descriptive, and atheoretical, or at the very least, driven by theory that inadequately accounted for the variety of associated cognitive impairments seen in ADHD, that did not generate specific testable hypotheses, and/or that provided "explanatory" concepts that were not themselves explained (for example, Douglas' concept of self-regulation) (Barkley, 1997, 1998). According to Barkley, ADHD[2] is caused by is an impairment in a process he terms "behavioural inhibition", itself comprised of three intermediate processes that occur in response to a stimulus in order to create and maintain a "delay in the decision to respond" (Barkley, 1997, p.5). The first is the inhibition of the prepotent response to an event, defined as "that response for which

[2] As Barkley considers ADHD-IA a distinct disorder (1997, 1998) his theory applies only to ADHD-C and ADHD-H/I.

immediate reinforcement (positive or negative) is available or has been previously associated" (Barkley, 1997, p.5). The second is the cessation of an ongoing response, and the third is "the protection of this period of delay and the self-directed responses that occur within it from disruption by competing events and responses" (Barkley, 1997, p.5). An impairment in behavioural inhibition, in turn, prevents the implementation of a group of executive functions that together would normally result in adaptive, goal-directed behaviour and task persistence: "the organization of behavior relative to time and the future" (Barkley, 1998, p.244). That is, while behavioral inhibition does not directly *cause* the executive functions to exert their adaptive influence on behaviour, through direct connections with the motor system it provides a "window" during which the opportunity to do so exists. An impairment in behavioral inhibition can account not only for impulsivity and hyperactivity, but also sustained attention. That is, in a normal individual, behavioral inhibition enables them to resist responding to distractions, and the executive functions are thus free to help them to "formulate and hold in mind the goal of the task and the plan for attaining that goal" (Barkley, 1998, p.246), and hence stay attentive to that task for a sufficient period of time.

Adult ADHD. In addition to a lack of awareness of its prevalence, mentioned above, relatively little is known about the unique nature of adult ADHD (Barkley, 1998; Wender et al., 2001). This is party because it has only recently been widely acknowledged that it could continue into adulthood; hence, most research has always focused on children (Hynd, 2001; Murphy & Gordon, 1998; Wender et al., 2001). Only since the 1970s did an interest in the adult outcomes of childhood ADHD plus the recognition of similar problems in the parents of children with ADHD lead to the first follow-up studies of adolescents and adults who had ADHD in childhood (Weiss et al., 2002; Wender et al., 2001). These and other

studies since completed have demonstrated that most children with ADHD do not "outgrow" the disorder; rather, they report the presence of at least one ADHD symptom severe enough to cause impairment in adulthood (Weiss et al., 2002). Another consistent observation is that unlike childhood ADHD, in which hyperactive symptoms tend to predominate, symptoms of inattention and impulsivity are more prevalent in adult ADHD (Douglas, 1972; Mirsky et al., 1999; Murphy & Gordon, 1998; Weiss et al., 2002). In addition, a large body of evidence has accumulated supporting the use of medication in treating adult ADHD (Barkley, 1998; Weiss et al., 2002).

In addition to scientific evidence, increased exposure in the popular media has contributed to a more recent awareness of adult ADHD among the general public. This includes television coverage and numerous best-selling books, some with provocative titles like "Driven to Distraction" (Hallowell & Ratey, 1994) and "You Mean I'm not Lazy, Stupid, or Crazy?"(Kelly & Ramundo, 1996). Advocacy organizations, such as Children and Adults with Attention Deficit Disorder (CHADD), have helped publicize adult ADHD, and, more recently, a multitude of websites and online discussion groups has appeared (Barkley, 1998; Weiss et al., 2002).

Occurring in parallel with an increasing scientific and public awareness of adult ADHD have been revisions of the DSM allowing for more applicability to the adult population. As described in the DSM-II (APA, 1968) released in 1968, "Hyperkinetic Reaction of Childhood" was an exclusively childhood disorder that individuals were believed to outgrow by puberty. In the DSM-III (APA, 1980), the possibility that symptoms might persist into adolescence and adulthood was stated for the first time, with the term "ADHD-Residual State" offered as a diagnostic category for adults (Barkley, 1998; Weiss et al., 2002). Later, some exclusively child-centered symptoms were rewritten in the DSM-III-R

(APA, 1987), thereby increasing the applicability of diagnostic criteria to adults (Weiss et al., 2002). For example, "calls out in class" became "blurts out answers to questions", and "butts into other children's games" was replaced with "butts into conversations or games". In addition, criteria of hyperactivity were expanded to include "subjective feelings of restlessness" from earlier exclusively behavioural symptoms. With the DSM-IV (APA, 1994), applicability to adults became still more enhanced. Specific mention was made of potential impairment in the workplace, in addition to home and school. Inattention was included as a separate diagnostic factor, thereby creating the predominantly inattentive subtype of ADHD. This provided "an important impetus for diagnosis in adults, since hyperactive symptoms decrease with age right through the life cycle while inattentive symptoms do not; many adults meet the criteria for the inattentive subtype but not for the combined subtype" (Weiss et al., 2002, p.100). Despite these changes, however, DSM diagnostic criteria of ADHD are not based on adult data: Field trials undertaken to validate diagnostic criteria for the DSM-IV included only children and adolescents as subjects (Lahey et al., 1994). Some suggest that ADHD is widely underdiagnosed among adults, in part because current symptom criteria remain largely child-centered, and require more radical modifications for adults (e.g. Weiss et al., 2002; Wender, 1995; Wender et al., 2001).

Other factors compound the uncertainty associated with ADHD diagnosis in adults. Adults are more likely to have a comorbid psychiatric disorder and/or to have accumulated dysfunctional life outcomes and stressors that may overshadow or confound ADHD symptoms (Weiss et al., 2002). In addition, informants who can report first-hand as to the presence, degree and extent of symptoms are far less likely to be available for adults than for children (Conners & Lett, 1999; Weiss et al., 2002), and the diagnosing clinician must

often rely on only one source of data: the adult client. Informant data, provided it is available, may be limited to recent history, as in the case of a spouse, friend, or roommate. As a consequence, clinicians cannot always confirm onset of ADHD symptoms before age seven, as stipulated by the DSM-IV. Furthermore, the integrity of retrospective historical data from the patient, and from informants who knew the patient as a child (if they are available) is compromised by the need to recall distant memories, and hence an increased risk of under- or over-reporting of childhood symptoms (Murphy & Gordon, 1998; Weiss et al., 2002). Thus with respect to the diagnosis of adult ADHD, reliance on subjective and child-centered DSM-IV symptom criteria are is further compromised by the need to satisfy the DSM-IV age of onset criterion through even less reliable methods (Wender, 1995).

The CPT

Clearly, objective tests to assist in the diagnosis ADHD, particularly adult ADHD, are needed. One of the most widely used tests in the context of ADHD assessment in adults and children is the Continuous Performance Test (CPT), originally developed as a test of sustained attention (Rosvold et al., 1956). Many different CPTs exist, all with a common task paradigm. This entails monitoring a relatively lengthy sequence of continuous stimuli, such as letters, numbers, words, pictures, or tones over an extended period of time (Lezak, 1995). Stimuli are presented visually (e.g. on a computer monitor) or aloud (e.g. on a recording). The goal is to distinguish pre-specified targets from non-targets, and to respond only for targets. For example, an individual might monitor a series of single letters on a computer screen and tap the spacebar only upon seeing an "A".

Various computerized CPTs are currently available to researchers and clinicians. Besides mode of presentation (visual or auditory), specific CPTs differ in terms of how a "target" is defined. A simple target task, often referred to as an "X-type" task, is one in

which a response is required for each presentation of a pre-specified stimulus; for example, "hit the space bar every time you see an X" (Rosvold et al., 1956). A complex, or "A-X type" task, on the other hand, requires that the target stimulus be responded to only if it is preceded by another specific stimulus, for example, "Respond every time you see a 1 followed by a 9" (Lezak, 1995). Finally, a "Not-X type" CPT task requires that subjects respond to all but a pre-designated stimulus, for example, "Respond every time you see a letter, except if it's an X" (Conners' Continuous Performance Test; Conners, 1995, 2002). Besides mode of presentation and target complexity, CPTs vary in terms of duration and rate of presentation of stimuli, length and consistency of inter-stimulus intervals (long versus short, fixed versus variable, subject-versus experimenter-controlled), quality of stimuli (e.g. degraded versus intact), and finally, ratio of targets to non-targets (e.g., mostly non-targets versus mostly targets) (Riccio & Reynolds, 2001).

Brief history of CPTs. Cancellation Tasks (CTs) are the immediate antecedents to CPTs. Indeed, some researchers refer to CTs as "paper-and-pencil" CPTs (e.g. Barkley, 1998). They assess "...visual selectivity at a fast speed on a repetitive motor response task" (Lezak, 1995, p. 548), by requiring that a subject rapidly scan printed rows of digits, letters, symbols, or pictures in order to mark pre-specified targets interspersed throughout the array. For example, the Visual Matching Test from the Woodcock-Johnson III Tests of Cognitive Abilities (Woodcock et al., 2001) requires the scanning of printed rows of six numbers of increasing digit length in order to circle the two identical numbers dispersed within each row. The CT dependent variable is typically the number of correct items (rows) completed within a time limit (Lezak, 1995).

Cancellation Tasks have been demonstrated to be sensitive to response slowing and inattentiveness as a function of diffuse cerebral damage or acute brain conditions, and, like

CPTs, they are classified as basic vigilance tests (Lezak, 1995). However of the two, CPTs may be the purer measure of vigilance. Cancellation tasks require the subject to use a pencil, as well as to quickly and accurately scan rows of printed stimuli; thus, performance relies substantially on motor processing, visual-motor integration, and subject-driven visual scanning (Lezak, 1995; Wechsler, 1997b; Woodcock et al., 2001). Motor response requirements for CPTs, on the other hand, are typically limited to tapping a computer key. In addition, because the rate, duration, and number of stimuli presentations in CPTs are fixed, and because stimuli are presented one at a time, performance is not affected by visual scanning ability.

Rosvold, Mirsky, Sarason, Bransome, and Beck designed the very first CPT in 1956, the basic paradigm of which has endured to the present. Their testing apparatus was elaborate and unwieldy by today's standards, consisting of a large revolving drum stenciled with letters and enclosed in a box-like case with a response key mounted on one side. Individual letters, visible through a visor beside the response key, were presented in pre-designated sequences at fixed (.92 sec) ISIs, while a tester simultaneously recorded responses on counters mounted on the opposite side of the case. In their initial research, Rosvold et al. observed that the performance of brain-damaged individuals on the CPT was significantly impaired compared to the non-brain-damaged subjects (1956). Since then, this observation has been replicated numerous times in various populations, and using a variety of CPTs (Riccio & Reynolds, 2001). Owing in part to their apparent requirement for attentional control, CPTs have gradually become widely used in ADHD research and clinical assessment (Epstein et al., 2003).

Major CPT outcome measures include omission errors (missed targets) and commission errors (non-targets responded to as targets), average Reaction Time (RT), and RT

variability. Performance may reflect deficits described by the two contemporary explanations of ADHD presented above. In particular, according to Barkley, commission and omission errors "...seem to directly assess the core symptoms of ... (ADHD)... namely, impulsiveness and inattention" (Barkley, 1998, p.302) as a function of an impairment in behavioural inhibition. In addition, Douglas has identified the CPT as potentially useful for identifying "micro-level" control problems associated with a deficit in self-regulation in individuals with ADHD (Douglas, 1999).

Hence, CPTs appear to be an ideal candidate to supplement the methods currently available to assess DSM-IV ADHD symptoms. It is therefore not surprising that they are so widely used in research and clinical settings today (Barkley, 1998; Epstein et al., 2003; Riccio & Reynolds, 2001). Indeed, some prominent researchers (e.g. Barkley, 1998) consider them the most reliable objective tests for discriminating groups of ADHD from normal children. CPTs are popular also in part because they are readily available and affordable and come in a variety of computerized, easy-to-administer versions with built-in automatic scoring and report-producing tools (McGee at al., 2000).

The Conners' CPT. The most popular CPT used in contemporary clinical settings is the Conners' Continuous Performance Test (McGee et al., 2000). The Conners' Continuous Performance Test (CCPT; Conners, 1995, 2002) has recently been updated. The CCPT-II (Conners, 2002) has an expanded normative database; however, the task itself is identical to that of its immediate predecessor. It is a visual CPT consisting of six blocks of sixty trials each, with interstimulus intervals of one, two and four seconds randomly distributed within each block. A trial consists of a letter presented for 250 ms on the centre of a computer screen. Sixteen of the 26 alphabetic letters (12 consonants and four vowels) are used and presented repeatedly over the 360 trials. Each trial is either a target (any letter

besides X) or a non-target (an X). Subjects are instructed to press the spacebar as quickly as possible upon observing a target, but not for a non-target. Major dependent variables are RT, RT standard error, and two types of errors: commission errors (responding for an X) and omission errors (failing to respond for a non-X). In addition, an Overall Index is available, which is a weighted sum of all CCPT-II measures.

The underlying paradigm of the CCPT is unique among CPTs in two primary ways, which, according to its creator, enhances its potential for the detection of ADHD symptoms. Inter-stimulus intervals are variable, therefore "...it is possible to assess ability to adjust to changing tempo and task demand ...(which)... contributes to the sensitivity of the CPT to detect attention problems" (Conners, 2002, p.19). A second unique feature is a high target-to-non-target stimulus ratio, providing "...reliable and accurate collection of RT and variability in RT measures" and more information on "speed and consistency of processing, both of which are considered essential elements of attentional function" (Conners, 2002, p. 556). A high target-to-non-target stimulus ratio also facilitates the measurement of response inhibition, which, as mentioned above, may reflect behavioural inhibition or self-regulation. In particular, because more-or-less continuous target stimuli are interspersed with only rare nontarget stimuli, the subject must inhibit ongoing responses to avoid commission errors, and "...a greater demand is placed on response inhibition than the more passive responding of the conventional ...[X-type]... tasks" (Conners, 2002, p.556).

As mentioned above, the Conners CPT is the most widely used CPT in clinical ADHD assessment. It is described by its creator as a "...traditionally useful measure of attention and learning disorders..." (Conners, 2002, p.6). Because it is so popular in the clinical assessment of ADHD, it is crucial that it be truly useful in this context. An

examination of the evidence for the validity of the CCPT in the assessment of ADHD reveals mixed findings.

CCPT validity. Validity is probably the most crucial element of a useful test (Graham & Lilly, 1984). In essence, a valid test truly measures the variable (or variables) that it purports to measure, an obvious necessity in the context of ADHD assessment. While true test validity is a theoretical unknown, its existence is supported (in the current context) if there exists a clear and logical relationship between specific test outcome variables and ADHD symptoms (face validity). In addition, test outcome variables should converge with other measures (in this context, rating scales) commonly used to evaluate ADHD symptoms as defined by the DSM-IV: inattention, impulsivity, and hyperactivity, as well as with the DSM-IV symptom criteria themselves (criterion-related validity). Finally, researchers should demonstrate significant deficits in CCPT test performance in groups of individuals with ADHD compared to groups without ADHD.

It appears that the CCPT has high face validity in the form of clear and logical relationships between specific outcome variables and ADHD symptoms. As the test's creator puts it, an individual's unique pattern of CCPT scores "...may be conveniently grouped into indicators of inattention, impulsivity, and vigilance" (Conners, 2002, p.28). However, acceptance of these specific relationships was not supported by the results of a recent study. Epstein, Erkanli, Conners, Klaric, Costello, and Angold (2003) examined relationships between specific CCPT outcome variables and individual DSM-IV symptoms and symptom domains. They interviewed the parents of 817 children who had been administered the CCPT-II. Using interview data, they calculated totals for each of the 18 DSM-IV ADHD symptoms and two symptom domains (Inattention and Hyperactivity/ Impulsivity), which they then correlated with six CCPT-II outcome variables, including the

four major measures (omission and commission errors, average RT, and RT variability). Analysis revealed a significant positive relationship between the collective CCPT outcome measures and ADHD: taken together, the "CPT-derived measures predicted the presence of most all (*sic*) ADHD symptoms confirming a relationship between this neuropsychological task and ADHD" (Epstein et al., 2003, p.548). However, individual CCPT-II outcome variables were not related to any one DSM-IV ADHD symptom or symptom domain[3].

Other researchers have also investigated the criterion-related validity of the CCPT. Conners (2002) describes some such analyses, which in general have not greatly endorsed the test. For example, he compared CCPT Overall Index scores from a group of 50 children with subscale totals from parent/teacher ADHD-symptom rating scales, finding only "mild" correlations (Conners, 1997), and reports on Cohan (1995), who found only "...limited relationships between the Conners' CPT and parent/ teacher behaviour ratings" (Conners, 2002, p.76). In explanation for the poor criterion-related validity of his test in this body of research, Conners states "...the correlations are not higher because the CPT and rating scales assess different aspects of the complex construct of attention" (Conners, 2002, p.76). This explanation appears contradictory in that parent/teacher rating scales are commonly used to measure behaviours associated with the DSM-IV symptom domains of inattention and impulsivity, the very symptoms that, as mentioned above, are purportedly measured by the CCPT (Conners, 2002).

Two recent reviews reported on a number of studies using a variety of CPTs, including the CCPT, in subjects with and without ADHD. Overall, it appears that the ability of the CCPT to differentiate between groups with and without ADHD depends on

[3] The lone exception was average RT, which was significantly correlated with "runs about and climbs", one of the six DSM-IV symptoms of hyperactivity.

the characteristics of the comparison group (or groups), in particular, the presence or absence of other conditions.

Riccio and Reynolds (2001) discuss three recent studies investigating CCPT performance in adults with and without ADHD, two of which used normal (nonclinical) control groups. Both of these found significantly more omission and commission errors on the part of the ADHD group compared to a nonclinical control group. Results of the third study, however, were not so clear-cut. Roy-Byrne, Scheele, Brinkley, Ward, Wiatrak, Russo, Townes, and Varley (1997) tested a group of 143 adults who had presented for clinical assessment of ADHD. Through a thorough diagnostic process, clients were classified as "Definite ADHD", "Questionable ADHD" (met DSM-IV criteria for symptoms, but lacked clear evidence of a childhood history and/or had current severe psychiatric comorbidity), or "Definitely not ADHD" (did not meet DSM-IV criteria). Unlike the relatively pure samples used in the other two studies, there were other possible confounding variables present. The Definite Non-ADHD subjects were significantly older than those in the other two groups, while subjects in the Definite and Questionable ADHD groups reported significantly higher current alcohol intake compared to the non-ADHD individuals. Finally, among subjects classified as Definite ADHD, there was an "...apparent higher prevalence in learning disability as suggested by clinical report" (Roy-Byrne et al., 1997, p.137). Analysis of CCPT performance did not support CCPT validity: the Questionable ADHD group, but not the Definite ADHD group, had a lower Overall CCPT Index compared to non-ADHD peers, "...whereas the definite ADHD group was in between and not significantly different from either" (Roy-Byrne et al., 1997, p.136). In addition, no significant differences emerged on any of the four major CCPT outcome measures: commission and omission errors, average RT, and RT variability.

The reviewers concluded that the ability of CPTs to identify ADHD in adults depends on the nature of the comparison group, and that CPTs (including the CCPT) are sensitive, but not specific. In other words, they are useful for correctly differentiating individuals who truly have ADHD from individuals who have neither ADHD nor any other condition that may affect attention. However, they may incorrectly identify ADHD in individuals who do not have the disorder, particularly if it is necessary to differentiate from amongst clinical cases (Riccio & Reynolds, 2001).

Conners (2002) also reviewed a number of studies using his test for group comparisons. Using the CCPT-II standardization data from subjects under the age of 18 years, he made group mean comparisons using scores from ADHD and nonclinical groups. Consistent with conclusion of the other reviewers (Riccio and Reynolds, 2001), the test was sensitive in that it correctly differentiated individuals with ADHD from those with no clinical condition, in showing "a large and significant difference between the ADHD and nonclinical groups with the ADHD group performing worse on all of ...[the CCPT]...measures" (Conners, 2002, p.63). Conners made other comparisons using ADHD, neurologically impaired, and nonclinical adults. Here, results were less clear: While the ADHD and neurologically impaired groups performed significantly more poorly than the nonclinical group for every outcome measure, supporting test sensitivity for both clinical conditions, test specificity was supported only for the neurologically impaired individuals. Compared to the ADHD group, they made more omission errors, had longer average RT, and greater RT variability (Conners, 2002).

In describing the work of Epstein et al. (1998), Conners, like the other reviewers, notes the finding of significant group differences between ADHD and non-ADHD adults, in support of the validity of his test. Conners also describes the work of Effler (1997), in

which ADHD children displayed significantly slower and more variable RTs on the CCPT in comparison to two non-ADHD groups: a nonclinical group, and a group with LD (Conners, 2002). However, in his summary of the work of Roy-Byrne et al. (1997), he, in direct contradiction to Riccio and Reynolds (2001) and even the original authors, reports evidence *in support of* CCPT validity: "...the group with the ADHD diagnosis[4] had significantly poorer CPT scores than the group not meeting ADHD criteria[5]" (Conners, 2002, p.67).

An examination of six recent studies not included in the reviews reveals results that are similarly mixed, and, as is evident from the studies discussed in the two reviews, the likelihood of the CCPT differentiating between ADHD and non-ADHD groups apparently depends on the characteristics of the comparison group. The test is sensitive, but not specific. For example, two recent studies that compared CCPT performance between boys with ADHD and their nonclinical peers reported significant group differences.

Perugini, Harvey, Lovejoy, Sandstrom and Webb (2000) found a significantly higher CCPT Overall Index (and thus relatively impaired performance) on the part of boys with ADHD compared to age-matched nonclinical control subjects. They limited their ADHD group to boys who met DSM-IV criteria for either ADHD Combined Type (ADHD-C) or ADHD Hyperactive/ Impulsive Type (ADHD-H/I). Boys with Inattentive-type ADHD (and therefore without significant H/I symptoms) were excluded. In addition, approximately 75 percent of the boys in the ADHD group were also diagnosed with a Conduct Disorder or Oppositional Defiant Disorder. The non-ADHD group, on the other hand, was entirely nonclinical: subjects had no evidence of behavioural, emotional or

[4] This corresponds to the Definite ADHD group
[5] It is unclear whether he is referring to the Questionable ADHD group, the Definitely Not ADHD group, or both.

academic problems, past or present. Thus, in comparison to nonclinical controls, ADHD boys with significant symptoms of hyperactivity/impulsivity, (some with significant IA symptoms as well), and most of whom had a comorbid disruptive behaviour disorder, were impaired on the CCPT.

Similarly, DeShazo, Klinger, Lyman and Hawkins (2001) also found a significant group difference between boys with ADHD and their non-ADHD, nonclinical peers. In direct contrast to Perugini et al. (2000), all of the subjects in their ADHD group had significant symptoms of inattention and met the criteria for ADHD-IA or ADHD-C (none had ADHD-H/I). Also in contrast to Perugini et al. (2000), there were no clinical comorbidities reported for subjects in the ADHD group. Non-ADHD subjects were recruited from local schools and "did not meet any of the inclusion criteria for ADHD" (p. 195). Results indicated that boys with ADHD had significantly more omission errors and a higher Overall Index on the CCPT compared their non-ADHD peers. Thus, in comparison to non-ADHD, nonclinical peers, ADHD boys with significant IA symptoms (including some with significant H/I symptoms) but no clinical comorbidities were impaired on the CCPT.

In contrast, McGee, Clark and Symons (2000) found no significant difference in CCPT performance between children with and without ADHD. However, unlike the two studies described above, their design did not include a nonclinical control group. Instead, they compared the Overall CCPT Index of the following four groups: ADHD only (included all three subtypes), ADHD with comorbid reading disability (RD), RD only, and clinical controls. The clinical control group consisted of children without ADHD or RD, but who were identified as having one of the following: Disruptive Behaviour Disorder, family relational problems, a history of child sexual abuse", anxiety disorder, and

adjustment reaction disorder. Results revealed that RD was a better predictor of CCPT performance than was ADHD: in a three way comparison of group means, there was a main effect of RD but not of ADHD, and there was no significant interaction. In addition, the lowest CCPT Overall Index scores came from the ADHD/RD group, followed by the RD only group, with the best performance observed in the clinical control group. Hence, the CCPT-II did not demonstrate specificity: it did not differentiate between individuals with ADHD those with RD or another clinical condition.

Consistent with these three studies using children, as well as those discussed in the reviews of Riccio and Reynolds (2001) and Conners (2002), of three recent studies that assessed CCPT performance in adults with and without ADHD, only in comparison to a nonclinical control group were significant group differences reported. Walker, Shores, Troller, Lee and Sachdev (2000) compared the CCPT performance of adults with ADHD, adults with a psychiatric disorder, and normal adults. They controlled for other clinical conditions: individuals with ADHD plus a comorbid Axis I or Axis II disorder[6] were excluded from the study, as were individuals "...with a significant history of substance abuse, loss of consciousness of over 5 min., epilepsy or other neurological event or process" (p.117). Hence, the groups were relatively pure with respect to clinical comorbidities. Comparison of group performance on the CCPT revealed that the ADHD group was significantly impaired compared to the nonclinical group, as evidenced by significantly more omission and commission errors and greater RT variability, thereby supporting its sensitivity. However, once again, the test did not demonstrate specificity.

[6] Based on the DSM-IV, Axis I disorders include anxiety, mood, or substance abuse disorder; Axis II consists of anti-social or borderline personality disorder (American Psychological Association, 1994).

Adults with ADHD were not significantly impaired compared to adults with a psychiatric disorder.

In a study comparing CCPT performance in adults with ADHD versus a non-ADHD clinical control group, Kovner, Budman, Frank, Sison, Lesser and Halperin (1998) matched the two groups with respect to clinical conditions other than ADHD. That is, the two groups were equivalent with respect to "major co-morbid factors (i.e. learning disabilities, depression, anxiety)" (Kovner et al., 1998, p.233). They, too, reported no significant differences on the CCPT. Compared to their non-ADHD peers with equivalent other clinical factors, adults with ADHD were not impaired on the CCPT. Similarly, Katz, Wood, Goldstein, Auchenbach and Geckle (1998) found no significant differences in CCPT performance in adults with ADHD, most of who had a coexisting LD or mood disorder, and non-ADHD adults with major depression or dysthymia. Again, results did not endorse CCPT specificity.

In summary, evidence of the validity of the CCPT in the assessment of ADHD is mixed. Though its face validity is high, studies attempting to connect CCPT outcome variables to parent/teacher rating scales of DSM-IV symptoms and to the individual DSM-IV symptoms themselves do not support the criterion-related validity of the test. In addition, while group comparisons of individuals with and without ADHD report evidence of CCPT sensitivity, specificity is not endorsed. The CCPT is useful in differentiating groups of individuals with ADHD from groups with no clinical conditions, but not from clinical comparison groups, even if the groups are equivalent with respect to clinical conditions other than ADHD. Unfortunately, this kind of validity is of questionable utility in the context of ADHD assessment. It is a safe assumption that the vast majority of individuals seeking ADHD assessment are there because they are experiencing significant

clinical symptoms (Tombaugh, 2003). Hence, determining whether someone has ADHD as opposed to no clinical condition whatsoever is not generally a clinically relevant activity (Homack & Reynolds, 2005).

Models of Attention

Based on the above discussion, it appears that the CCPT is inadequate to completely address the need for an objective test to augment DSM-IV symptom criteria confirmable only through subjective, potentially-biased methods in order to identify ADHD in the clinic, particularly in adults. By their very nature, however, objective neuropsychological tests tend to examine cognitive functioning at a highly specific "micro-level" of analysis, as opposed to interviews and rating scales, which gather information concerning complex "macro-level" behaviours and experiences in real-life settings such as school, work, and home. In an attempt to shed light on specific underlying neuropsychological impairments associated with ADHD, two models of attention are relevant. Both provide a micro-level of analysis of attentional functioning consistent with the scope of neuropsychological tests, by dividing this broad construct into five distinct components.

Sohlberg and Mateer: A clinical model. Sohlberg and Mateer (1989) introduced a five-component model of attention in an attempt to enhance the understanding of cognitive impairment in individuals with traumatic brain injury (TBI), with reference to common clinical outcomes. "Focused attention" refers to the ability to respond discretely to specific stimuli, and is disrupted in only the earliest stages after head injury. "Selective attention" is defined as the ability to maintain a consistent behavioral or cognitive set despite the presence of distracting stimuli. "Examples of problems at this level include an inability to perform therapy tasks in a stimulating environment (e.g. an open treatment area) or to prepare a meal with the children playing in the background" (Sohlberg & Mateer, 1989,

p.121). "Sustained attention" is the ability to maintain a consistent behavioural response by virtue of the capacity to hold information in conscious awareness while manipulating it at the same time. "Alternating attention" is "the capacity for mental flexibility that allows individuals to shift their focus of attention and move between tasks having different cognitive requirements" (Sohlberg & Mateer, 1989, p.121), for example, in the case of a student who listens to a lecture and takes notes at the same time. "Divided attention", the final attentional factor in this model "involves the ability to respond simultaneously to multiple tasks or multiple task demands... [in which]...two or more behavioral responses may be required, or two or more kinds of stimuli need to be monitored" (p.121). Examples of common divided attention tasks are driving while listening to the radio, or having a conversation while preparing a meal.

Mirsky: The Restricted Taxonomy of Attentive Functions. As mentioned above, attentional impairment is a prominent symptom of many clinical disorders. Unlike the model just discussed, The Restricted Taxonomy of Attentive Functions (Mirsky et al., 1999; Mirsky & Duncan, 2001) does not confine itself to attentional impairment stemming from a single disorder. Instead, the model defines a number of etiological categories: Brain injury or infection, environmental (e.g. malnutrition, fetal alcohol syndrome), metabolic (e.g. phenylketonuria), and finally, familial/genetic (e.g. schizophrenia, epilepsy, and ADHD).

With just this exception, however, the Restricted Taxonomy of Attentive Functions is highly similar to the model of Sohlberg and Mateer (1989). It too divides attention into five components, and, though they are not directly analogous, there is a great deal of overlap among individual components from the two models. "Focus/execute" is the ability to attend to and respond to specific stimuli despite distractions, while "shift" is the ability

to distribute and direct one's focus among different stimuli or among relevant aspects of a complex stimulus. "Encode" is the capacity to hold information in mind in order to perform some mental operation on it, while "Sustain" refers to "the capacity to maintain an attentional focus--a vigilant attitude--for an appreciable interval of time" (Mirsky & Duncan, 2001, p.21). Finally, "stabilize" is that component of attention that enables an individual to respond consistently.

The Restricted Taxonomy of Attentive Functions further hypothesizes that a combination of impairment in one or more of these five attentional components results in attentional impairments characteristic of the various etiological categories. In the case of ADHD, there are thought to be impairments in at least three of the following four attentional components: Focus/execute, shift, sustain, and stabilize (Mirsky et al., 1999).

An alternative model. In attempting to reconcile the five-component models of attention, three general themes emerge: the ability to focus on specific stimuli in the presence of competing information, the ability to sustain such focus over time, and finally, the ability to manipulate information efficiently while maintaining it in conscious awareness. Hence, it may be useful to divide attention into three, rather than five, components: selective attention, sustained attention, and information processing capacity (Tombaugh, 2003). Consistent with the five-component models, selective attention refers to the ability to focus on relevant stimuli or aspects of complex stimuli in the presence of competing stimuli. It enables an individual to focus on relevant stimuli and ignore what is irrelevant during complex tasks. Sustained attention, also known as vigilance, is defined as the ability to maintain this selective attention and mental control over a prolonged period of time: what is commonly referred to as "attention-span". The third attentional component, information processing capacity (IP capacity), incorporates the other models' references to

the "mental control" of information: the amount of information and efficiency of control over that information as it is processed in conscious awareness. In addition, IP capacity subsumes an aspect of attentional functioning not referred to in the other models: information processing speed (Tombaugh, 2003).

As mentioned above, the majority of research into the nature of ADHD has always focused on children. In addition, "...in spite of the obvious importance of attention to the understanding of ADHD, the number of studies *directly* examining one or more aspects of attention in children with ADHD is limited" (Tsal et al., 2005, p.142). In part due to the emergence of the Processing Speed Index in the third editions of the most commonly used IQ tests in North America, the Wechsler Adult Intelligence Scale, 3rd ed., (WAIS-III; Wechsler, 1997) and the Wechsler Intelligence Scale for Children (WISC-III; Wechsler, 1991), IP speed has recently become a topic of great interest in LD assessment research (Kleinman et al., 2005). However, only a few studies have been completed investigating IP speed in reference to ADHD, and, those that are available have demonstrated only modest and inconsistent findings (Kleinman et al., 2005).

Two New Tests of IP Speed

Two recently developed tests of IP speed may be useful in the assessment of ADHD. They are the Adjusting Paced Auditory Serial Addition Tests (Adjusting-PASAT; Tombaugh, 1999) and the Computerized Tests of Information Processing (CTIP; Tombaugh & Rees, 1999; Tombaugh, Rees, & Royan, 2001; Tombaugh, Rees, Stormer, Harrison, & Smith, 2006).

The Adjusting –PASAT (A-PASAT), a computerized test of IP speed, is a recent modification of the Paced Serial Addition Test (PSAT; Sampson, 1956). Originally developed as a test of temporal integration (Sampson, 1956) the original PSAT came in

both a visual (PVSAT) and an auditory (PASAT) version[7]. The latter requires an individual to mentally add a lengthy series of pre-recorded auditory digits. The object is to state aloud the sum of each digit and the digit that immediately follows it (for example, the first plus the second, the second plus the third, the third plus the fourth, and so on). The outcome variable of the PSAT is the total number of correct responses. Fixed ISIs are pre-set by the administrator, with their duration depending on the expected performance of the individual. This is because in individuals with severe impairments, excessively short ISIs may make the test too difficult, resulting in a floor effect, as they are unable to respond correctly, or can respond correctly to only a few items (Tombaugh, 2003).

The Adjusting-PASAT (A-PASAT; Tombaugh, 1999) differs from the auditory version of its predecessor, the PASAT, in that rather than being fixed, the length of each ISI depends upon the correctness of the response that preceded it: ISIs increase by 20 ms after an incorrect response, and decrease by 20 ms after a correct response (Tombaugh, 1999). The incorporation of this "psychological stair-step/ titration procedure" has some major advantages. First, it enables the determination of single outcome measure, a temporal threshold, beyond which an individual is no longer able to accurately process information (i.e., the shortest ISI preceding a correct response). A second advantage is that the test is less likely to be insensitive to the level of functioning of a given patient (Tombaugh, 2003).

The Computerized Test of Information Processing (CTIP) is a second new test of IP speed. It is a computerized measure of reaction time (RT) over three progressively greater levels of cognitive complexity: Simple, choice, and semantic choice. The simple condition is a basic signal detection task for which subjects must press the space bar each time an "X" appears in the centre of the screen. The choice condition introduces a concrete decision

[7] For a comprehensive review of the PASAT, the reader is directed to Tombaugh (2006a).

component based on the physical appearance of the stimulus: a single word (either "Duck" or "Kite") that appears in the centre of the screen. Subjects are instructed to press one of two keys, depending on which word appears for each trial. The third, most cognitively complex condition, semantic choice, involves a decision based on the meaning of the words. The object is to determine if the target stimulus (a single word) belongs to a given semantic category. Subjects are instructed to press one key for "Yes", and a different key for "No".

Initial research (Tombaugh, Rees, & Royan, 2001; Tombaugh et al., 2006) has demonstrated that that the CTIP is a potentially useful measure for use in clinical evaluations. To illustrate, in cognitively-intact adults, reaction time progressively increased by approximately 200 ms across the progressive conditions (between Simple and Choice, and between Choice and Semantic Choice), making the CTIP a potentially useful baseline against which to compare the performance of individuals with potential impairments in attention. In addition, within each condition, there were no practice effects: "...performance is extremely stable across the five blocks of trials" (Tombaugh & Rees, 1999, p.3). Finally, based on an exploratory comparison of the CTIP performance of adult TBI patients with normative data obtained from normal adult subjects, a "progressive differential" was seen in RT across the three conditions: the increase in RT between the conditions, particularly between the Choice and Semantic Choice conditions was proportionally greater than the consistent 200 ms increase observed in normals. Thus, "...performance from a limited number of TBI patients clearly demonstrated the ability of the CTIP to detect impairment in a person's speed of information processing" (Tombaugh & Rees, 1999, p.5).

Purpose

In response to the need for an objective test to assist in the identification of ADHD, the current study explored the possibility that the two new tests of IP capacity, the A-PASAT and the CTIP, may be useful in this application by determining if they could differentiate between a sample of adults with and without ADHD. It was predicted that individuals with ADHD would perform more poorly on these tests compared to individuals without ADHD. In addition, the sensitivity of a more traditional measure, the CCPT-II, along with a number of other traditional neuropsychological tests used to measure attention was evaluated. These are the Controlled Oral Word Association Test (COWAT; Benton & Hamsher, 1989), the Trail-Making Test (TMT; Reitan & Wolfson, 1993), the Stroop Test (Stroop, 1935), the Digit Span and Digit Symbol Coding subtests of the Third Edition of the Wechsler Adult Intelligence Scale (WAIS-III; Wechsler, 1997), and a cancellation task: the Visual Matching subtest from the Woodcock Johnston tests of Cognitive Ability (Woodcock et al., 2001). It was predicted that individuals with ADHD would be impaired on these tests compared to individuals without ADHD.

Method

Participants

Forty-three adult subjects, 22 with ADHD (55% men and 45% women) and 21 without ADHD (29% men and 71% women) participated in the study. The mean age of participants in the ADHD group was 28.9 years and the mean age of the participants without ADHD was 23.5 years. Because of the high level of comorbidity between ADHD and LD, the ADHD group was further divided into a Pure ADHD group, comprised of 55% men and 45% women with a mean age of 32.1 years, and an ADHD/LD group, comprised of 54% men and 46% women, with a mean age of 26.6 years. Non-ADHD participants

were volunteers from the Introductory Psychology course at Carleton University, and received course credit for their participation. Participants with ADHD were recruited from among clients registered with Carleton University's Paul Menton Centre for Students with Disabilities (PMC). Dr. Nancy McIntyre, Learning Specialist at the PMC, screened the documentation of potential ADHD participants to confirm eligibility. To be included in the ADHD group, participants had been formally diagnosed, either through a Medical Report signed by a licensed physician, or a psychoeducational assessment report signed by a registered, licensed psychologist. Eligible participants' documentation included a clear statement diagnosing ADHD.

To corroborate group membership, participants completed two brief questionnaires: the Short Version of the Conners' Adult ADHD Rating Scale (CAARS; Conners et al., 1999) and a subsection of the Post Secondary Screening Inventory for Learning Disabilities (PSSI; Herriot, 1996). To assess medical history, use of medication, and academic history, all participants completed an Intake Questionnaire (see Appendix A).

Materials

A-PASAT. The A-PASAT was administered using a computer and headphones. A series of aural digits from 1 to 9 was presented in a male voice through a Windows PCM WAV file format. Prior to test administration, volume was adjusted for each subject through the presentation of individual aural digits. The ISI length was controlled by the 32-bit multimedia timer function of Windows 98. Each administration began with a practice session of 12 trials, during which the ISI length was held constant at 3.0 s, followed by 200 test trials, with the first two digits presented with a 2.4 s ISI. Subsequent ISIs were contingent upon the correctness of the preceding response. Incorrect responses were followed by a 20 ms ISI increase, and correct responses by a 20 ms ISI decrease.

Participants were instructed to continually sum the two most recent digits, and to state the response aloud. The experimenter typed each response into the computer using the numeric keypad. The dependent measure was the length in ms of the shortest ISI that preceded a correct response.

CTIP. The CTIP was administered using a computer. Participants completed three progressive conditions (Simple, Choice, and Semantic Choice), each comprised of 5 blocks of ten trials preceded by 10 practice trials, and were instructed to respond as quickly as possible during each condition. For the Simple condition, subjects were instructed to press the space bar each time an "X" appeared in the centre of the computer screen. For the Choice condition, subjects were instructed to press the "Z" key if the word "Kite" appeared in the centre of the computer screen, or the "?" key if the word "Duck" appeared in the centre of the computer screen. Finally, for the Semantic Choice condition, subjects were instructed to determine if each word that appeared in the centre of the computer screen belonged to the category name directly above, and to press the "Z" key for "Yes", and the "?" key for "No". Dependent measures varied over the three conditions. For the Simple condition, the mean and median RT were collected. For the Choice condition, mean and median RT for both incorrect and correct responses were collected, and for the Semantic Choice condition, the number of correct responses and the mean and median RT were collected for the "same" and "different" trials.

CCPT-II. The CCPT-II was administered by computer. Participants first completed a 70 s practice, which presented multiple items separated by each of the three ISIs. Three hundred and sixty test trials consisting of 16 alphabetic letters (12 consonants and 4 vowels) were then presented for 250 ms each in the centre of the screen, with ISIs of 1, 2, and 4 seconds randomly distributed within each of three 60-trial blocks. Participants were

instructed to press the spacebar as quickly as possible upon seeing a target (any letter but X) but not for a non-target (the letter X). Dependent measures were mean RT, RT standard error (a measure of RT variability), omission errors, and commission errors.

Procedure

The testing session lasted approximately one and a half hours. Participants were introduced to procedures with one of two versions of the informed consent form, one for non-ADHD controls (see Appendix B) and one for participants with ADHD (see Appendix C). Following this, they were administered the Intake Questionnaire (see Appendix A). Tests and questionnaires, using standardized administrative procedures, were then administered in the same order for all participants: TMT, WJ-III Visual Matching, CTIP, Stroop, CCPT-II, COWAT, WAIS-III Digit Symbol Coding, WAIS-III Digit Span, Adjusting PASAT, CAARS, and PSSI. At the end of the session, participants were thanked and given one of two versions of the debriefing form (see Appendix D and Appendix E).

Results

Age

There were significant age differences between participants with ADHD (M = 28.9 years, SD = 6.9) and without ADHD (M = 23.5 years, SD = 7.4) (t [41] = -2.45, $p < .05$), as well as among participants grouped by ADHD and LD status (Pure ADHD vs. ADHD/LD vs. Controls; F [2, 40] = 4.83, $p < .05$). The ADHD participants (mean age = 32.1 years, SD = 7.8) were significantly older than the Controls (mean age = 23.5 years, SD = 7.4). These age differences are not surprising. Control participants were registered in a first-year Psychology class, while those with ADHD were at various levels of their academic programs.

ADHD Screening Questionnaires

In order to confirm the assignment of subjects to groups, a one-way ANOVA comparing CAARS ADHD Index percentiles for the Pure ADHD, ADHD/LD, and Control groups showed a significant difference (F [2, 40] = 5.05, $p < .05$). Post hoc analysis (Tukey a) revealed that consistent with expectations, the Control participants reported the presence of significantly fewer ADHD symptoms and associated difficulties compared to those in the Pure ADHD and the ADHD/LD groups, which in turn were equivalent. An identical pattern was observed using the proportion of items endorsed on the PSSI excerpt (F [2, 40] = 31.75, $p < .001$).

Primary Tests[8]

CTIP. Because research in this area tends to compare the performance of individuals with and without ADHD, the initial analysis examined participants' CTIP performance as a function of the presence or absence of ADHD, irrespective of LD.

Medians were chosen to represent each individual's average score in order to minimize the effect of outliers. Statistical analysis was then based on the arithmetic means of these median scores, as shown in Figure 1.

A 2 (Control vs. ADHD) by 3 (simple vs. choice vs. semantic choice) Analysis of Variance (ANOVA) using CTIP correct RT as a dependent variable revealed a significant effect of CTIP (F [2,82] = 233.73, $p < .001$) and Group (F [1,41] = 6.77, $p <. 05$). There was a progressive increase in RT for all participants over the 3 CTIP tasks, with those with ADHD demonstrating longer RTs overall compared to Controls. A significant interaction (F [2, 82] = 6.57, $p < .05$) points to a disproportionate increase in RT for the ADHD group (See Figure 1).

Insert Figure 1 about here

A subsequent analysis was conducted to parse out the effect of LD status within the ADHD group. Figure 2 presents mean CTIP reaction times for the Control, Pure ADHD, and ADHD/LD groups. A 3 (Control vs. Pure ADHD vs. ADHD/LD) by 3 (simple vs. choice vs. semantic choice) ANOVA revealed a significant effect of CTIP (F [2, 80] = 241.31, $p < .001$), Group (F [2, 40] = 5.99, $p < .05$), and a Group x CTIP interaction (F [4, 80] = 5.915, $p < .001$). These analyses, corroborated by post hoc tests (Tukey a) showed that the previously reported difference between the Control and the ADHD Combined group was due to the performance of participants with ADHD plus LD. The Control and Pure ADHD groups did not differ significantly (See Figure 2). In light of this observation,

[8] For summary data from all tests, see Appendix F.

all subsequent analyses were based on the comparison of three groups: Control, Pure ADHD, and ADHD/LD.

Insert Figure 2 about here

In order to examine potential group differences in variability in CTIP RT, the coefficient of variation statistic (CV) was calculated for each subject for each CTIP test. The CV equals the standard deviation of the RTs divided by the mean, multiplied by 100. It was used instead of the variance because "positive skew[9] leads to a number of extreme values that have a disproportionate influence on the calculation of the response time mean and similarly, on the size of the variance measures" (Leth-Steensen et al., 2000, p.169). A series of one-way ANOVAs revealed no significant differences among the three groups in RT variability for the Simple, Choice, or Semantic Choice CTIP tests.

A-PASAT. A one-way ANOVA comparing mean A-PASAT threshold values among the three groups showed a significant effect (F [2, 40] = 3.61, $p < .05$). Post hoc analysis (Tukey *a*) revealed that consistent with the pattern observed on the CTIP, only the difference between the Control and the ADHD/LD group was statistically significant ($p <$ .05).

CCPT-II

Analysis of the major CCPT-II outcome measures (omission errors, commission errors, RT, and RT variability) revealed no statistically significant differences.

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[9] Reaction time (RT) distributions tend to be positively skewed. Exaggerated positive skew in RT distributions of ADHD children typically reflects "a substantially larger number of abnormally slow responses" (Leth-Steensen et al., 2000, p.168).

Traditional Neuropsychological Tests

WAIS-III Digit Symbol Coding. An ANOVA conducted on WAIS-III Digit Symbol Coding percentiles approached significance (F [2, 40] = 2.733, p = .077). As confirmed through post hoc analysis (Tukey *a*), the ADHD/LD group (M = 40.1, SD = 24.6) had significantly lower scores than both the Control group (M = 60.1, SD = 24.5) and the Pure ADHD group (M = 63.3, SD = 34.6).

WAIS-III Digit Span. A one-way ANOVA using WAIS-III Digit Span percentiles approached significance (F [2, 40] = 2.96, p = .063). There was a progressive decline in performance across the three groups. Post hoc analysis (Tukey *a*) indicated that, again consistent with the A-PASAT and the CTIP, only the ADHD/LD group (M = 30.2, SD = 22.8) performed significantly more poorly than the Control group (M = 52.1, SD = 28.7).

COWAT, Stroop, TMT, and WJ-III Visual Matching. Three separate one-way ANOVAs using COWAT percentiles, Stroop Interference percentiles, and TMT percentiles, respectively, revealed no significant differences among the three groups. However, analysis of WJ-III Visual Matching percentiles showed a significant effect of Group (F [2, 40] = 3.506, $p < .05$). Post hoc analysis (Tukey *a*) indicated that, once again, only the ADHD/LD group (M = 24.4, SD = 18.6) performed significantly more poorly than the Control group (M = 45.8, SD = 23.3).

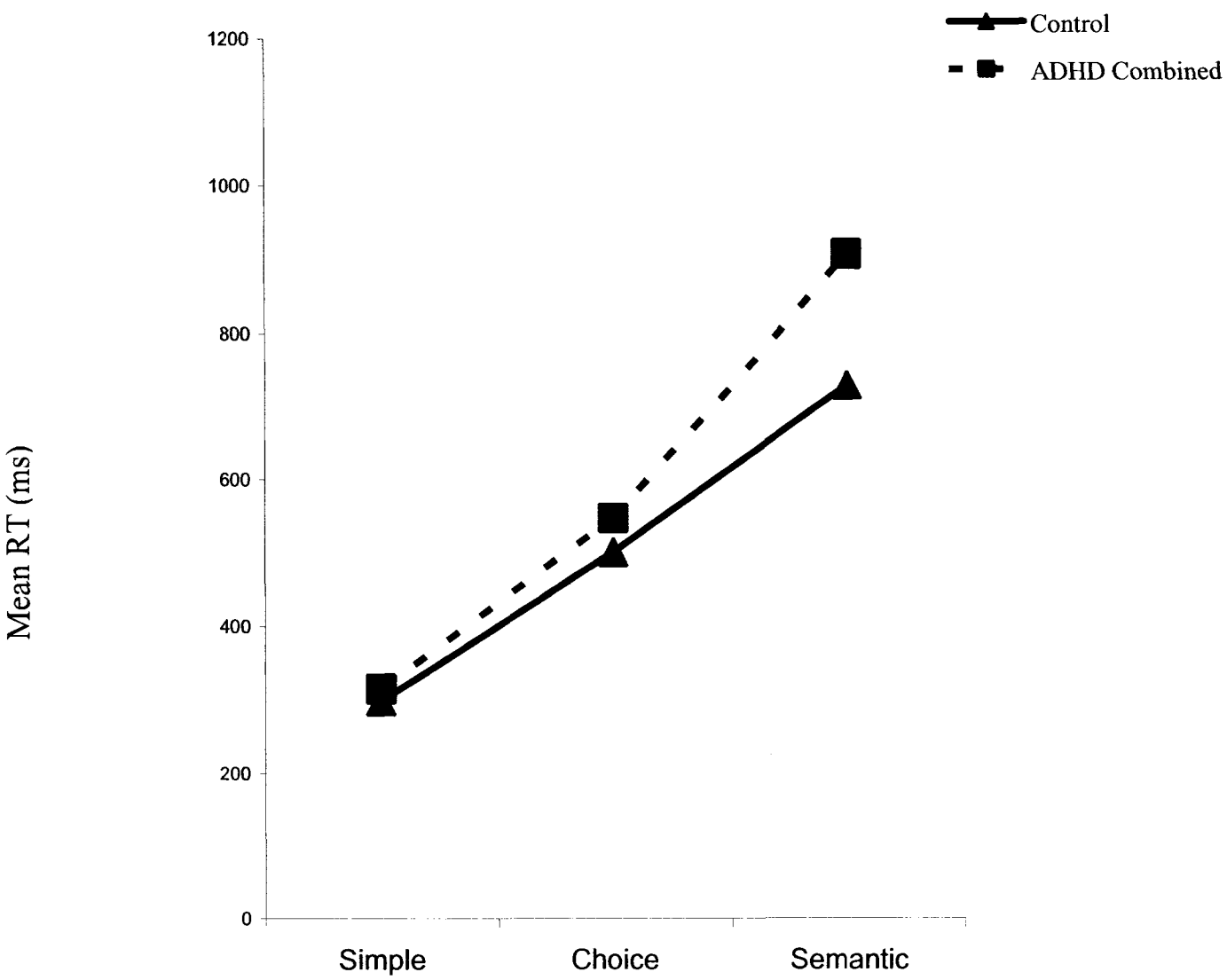

Figure 1. Mean CTIP Reaction Time for the Control and ADHD Combined Groups

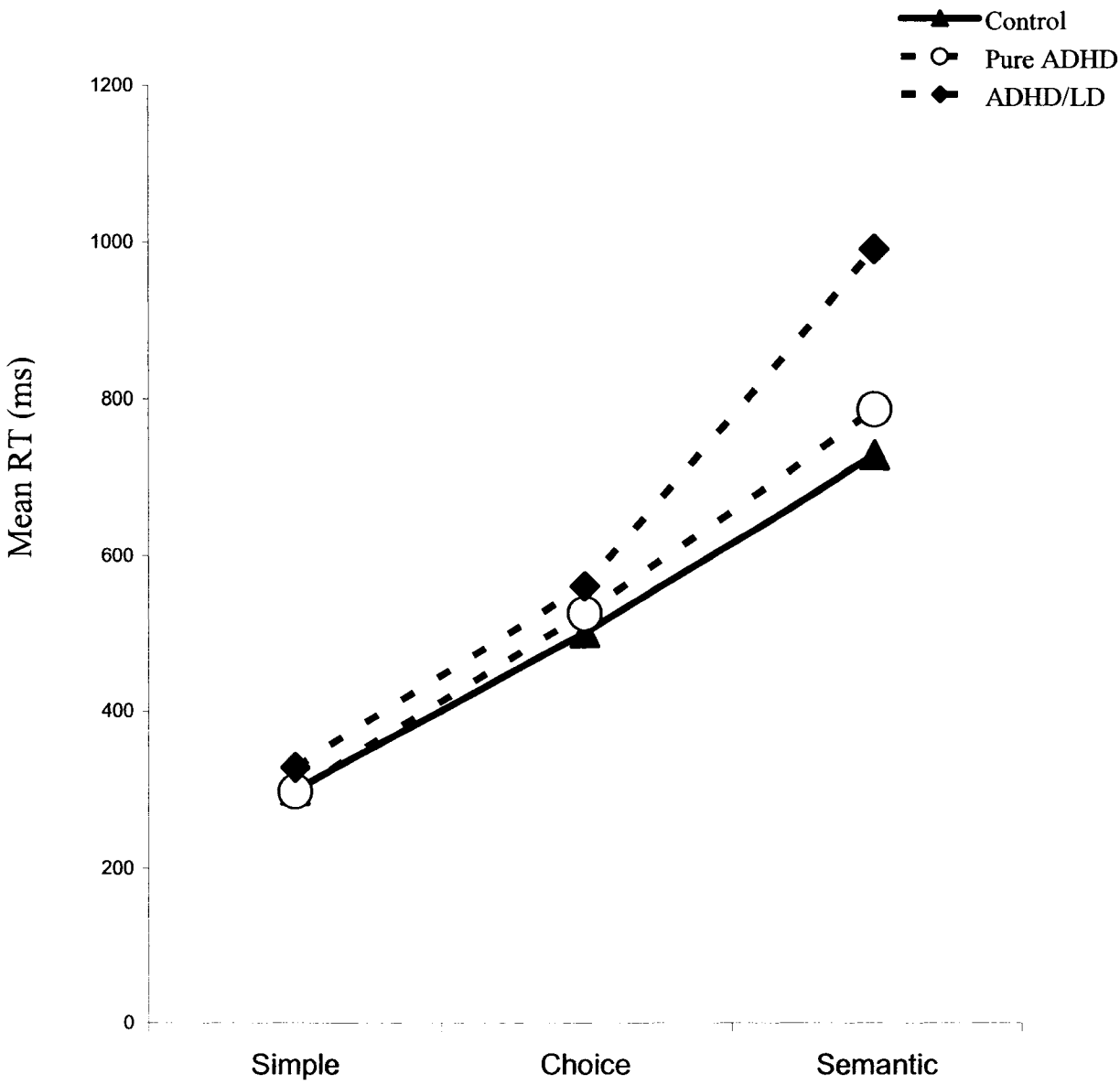

Figure 2. Mean CTIP Reaction Time for Control, Pure ADHD, and ADHD/LD Groups

Discussion

The CTIP and A-PASAT

The main purpose of this study was to evaluate the ability of two newly developed neuropsychological tests, the CTIP and A-PASAT, to detect deficits in adult students with documented ADHD. Reaction time scores from the simple, choice, and semantic choice conditions of the CTIP (Figure 1) showed that the performance of ADHD students consistently fell below the scores of those in the normal control group. Subsequent statistical analyses revealed a significant interaction, due to a progressively greater increase in RT for the ADHD group across the three CTIP conditions. Thus, when comparing the students' performance solely on the basis of the presence or absence of ADHD, those with ADHD took longer to make decisions and showed a disproportional decrement in decision-making speed as a function of increasing task complexity. Consequently, it would appear that ADHD produced a progressive decline in speed of information processing, implying that the CTIP would be useful in the identification of adult ADHD, potentially helping to fulfill a major clinical need.

More detailed analyses, however, revealed this conclusion to be premature. In particular, it was shown that these initial results were due to the heterogeneous composition of the ADHD group: some of the students had ADHD only (Pure ADHD) while some had ADHD with a coexisting LD (ADHD/LD). When the ADHD group was subdivided into two more homogeneous groups according to the presence or absence of LD, a different picture emerged. Figure 2 shows clearly that that the Pure ADHD group and the Control group had virtually identical performance on each of the three CTIP tests. However, the ADHD group with a coexisting LD progressively diverged from the other two groups as the complexity of the task increased on the CTIP, indicating that a deficit in information

processing speed was present. These more detailed analyses revealed that the group differences originally reported using the heterogeneous ADHD group were due solely to the performance of ADHD participants with a coexisting LD – the group with ADHD alone performed equivalently to the non-ADHD, non-LD control group. Hence, results obtained when the groups were subdivided into Pure ADHD and ADHD/LD are in marked contrast to the original analyses performed on the more heterogeneous group and clearly do not support the contention that the students with Pure ADHD are characterized by a deficit in IP speed.

Similarly, the ADHD/LD group displayed higher threshold values on the A-PASAT. Participants with ADHD plus a coexisting LD needed more time to add the numbers they had heard, compared to participants with ADHD alone, and to Controls.

The Traditional Neuropsychological Tests

The critical importance of separating the clinical participants into a pure ADHD and ADHD/LD group is further demonstrated by the results obtained from three of the neuropsychological tests traditionally used to assess attention. Significant differences which mirrored observations for the two new tests of IP speed were found on the WAIS-III Digit Symbol Coding and Digit Span tests, as well as on the WJ-III Visual Matching test. For these three tests, group differences were due solely to the relative impairment of ADHD/LD participants, while those with ADHD alone performed equivalently to the non-ADHD, non-LD controls.

No significant differences emerged on the Stroop, TMT and COWAT; however, this was not unexpected, as these tests have shown mixed results in comparisons of adults with and without ADHD. While a full review of the relevant literature is beyond the scope of this paper, a few studies serve to illustrate this point. Taylor and Miller (1997) observed

significant impairment on the Stroop interference score on the part of adults with ADHD compared to their peers with no diagnosis; however, no Stroop differences were observed between adults with and without ADHD by Seidman et al. (1998). Similarly, Taylor and Miller (1997), and Johnson et al. (2001), both reported significant differences in Trails B performance between adults with and without ADHD, while Walker et al. (2000) could not differentiate among nonclinical controls, psychiatric controls, and adults with ADHD using the TMT. With respect to the COWAT, Walker et al. (2000) found it capable of differentiating adults with ADHD from nonclinical controls, but not from controls with another psychiatric condition.

In summary, on those traditional neuropsychological tests for which significant differences were observed, consistent with the CTIP and A-PASAT, all observed group differences were a consequence of the performance of participants with ADHD plus LD, but not those with ADHD alone. Participants with ADHD plus a coexisting LD were significantly impaired compared to participants with ADHD alone, as well as to non-ADHD, non-LD Controls, a pattern identical to that observed on the two new tests of IP speed.

The CCPT-II

What remains to be explained is the overall lack of sensitivity of the Conners CPT to the effects of ADHD. As already discussed, CPTs, especially the Conners CPT, are widely used among clinicians and researchers seeking to identify ADHD. However, current results do not support its popularity in this application, as there was no significant effect of group on the four major outcome measures (with corresponding ADHD symptoms): omission errors (inattention), commission errors (impulsivity), average RT (impulsivity versus inattention) and RT variability (inattention).

Thus, the CCPT-II did not demonstrate sensitivity to ADHD, as it failed to differentiate individuals with ADHD from normal, nonclinical controls. This finding is inconsistent with previous results reported by DeShazo et al. (2001), Perugini et al. (2000), and Walker et al. (2000). However, these results are congruent with findings that have shown that the CCPT is not sensitive to the effects of ADHD when the comparison group was a clinical control group (Katz et al., 1998; Kovner et al., 1998; McGee et al., 2000). This suggests the possibility that the current control group was not truly "nonclinical". That is, despite having been recruited from a nonclinical population, it is possible the current Control group included some individuals characterized by significant clinical or other factors that influenced CCPT-II performance.

One such factor is age, as IP speed has been noted to decline with age (Bugg et al., 2006). However, the range of ages was not sufficiently large to view this variable as a serious contributing factor. In addition, Control participants were significantly *younger* than those with ADHD, a difference that would be expected to *increase* the likelihood of significant differences in CCPT-II performance in the expected direction. Hence, age could not have been a factor in the absence of differences in CCPT-II performance between individuals with and without ADHD.

Furthermore, analysis of responses to the questionnaires designed to assess symptoms of ADHD, the CAARS, and the PSSI, confirmed the assignment of individuals to clinical versus nonclinical groups. In contrast to the two ADHD groups, there was no evidence of significant ADHD symptoms on the part of Controls. While based on the Intake Questionnaire, participants with and without ADHD differed systematically with respect to a number of other clinical factors that could reasonably be assumed to influence CCPT-II results, a lesser greater proportion of individuals *with* ADHD reported such factors, again,

increasing the likelihood of differences in CCPT-II performance in the expected direction. These are (with approximate proportions in the ADHD vs. Control groups, respectively): past incidence of an accident or fall with a resulting loss of consciousness (50% vs. 24%), previous diagnosis of depression (50% vs. 5%), current diagnosed depression (18% vs. 5%), previous diagnosis of anxiety disorder (18% vs. 5%), and lastly, current diagnosed anxiety disorder (18% vs. 5%).

In summary, it appears that the current results provide evidence that fails to endorse the validity of the CCPT-II in the assessment of ADHD. Moreover, based on three recent studies (Katz et al., 1998; Kovner et al., 1998; McGee et al., 2000), the CCPT does not demonstrate specificity: it cannot distinguish individuals with ADHD from those with other clinical conditions.

Investigation of Systematic Differences between the Clinical Groups

As discussed above, evidence from the CAARS, PSSI, and Intake Questionnaire confirmed assignment of participants to the ADHD groups and the Control group, and supported the characterization of Controls as nonclinical. However, it is possible that besides LD status, some factor that differs systematically between the ADHD/LD group and the Pure ADHD group is responsible for significant differences on the two new tests and the three traditional tests. As previously mentioned, there are numerous other clinical conditions that tend to be more common in the ADHD population (Downey et al., 1997), and many other conditions may mimic the symptoms of ADHD (Taylor & Miller, 1997). By chance alone, the two current clinical groups may differ systematically in terms of these or some other potential confounds, which in turn may have led to significant differences on the tests of IP speed.

In order to investigate this possibility, clinical participants' responses to the Intake Questionnaire were compared. Approximately half of the participants within each clinical group reported having experienced a past accident or fall with a resulting loss of consciousness, thereby ruling out a past head injury as a differential contributor to IP speed differences between the Pure ADHD and ADHD/LD groups. Similarly, approximately half of each clinical group reported having experienced significant depression in the past, approximately one third of each group reported having used medication to treat ADHD symptoms as a child, and approximately half of each group reported having displayed behavioural difficulties (such as high distractibility and aggression) during childhood. On the other hand, the Pure ADHD and ADHD/LD groups differed in terms of the proportion of individuals who were current users of medication to treat symptoms of ADHD: just one of nine Pure ADHD participants (approximately 10%) versus four of 13 ADHD/LD participants (approximately 30%). However, because all participants had refrained from using ADHD medication for at least 24 hours prior to the testing, it is unlikely that the effects of medication contributed to observed differences in IP speed between the two groups.

In another effort to isolate potential systematic differences between the Pure ADHD and ADHD/LD groups, participants' documentation was examined[10]. To illustrate, one concern was the type of ADHD between the two groups, as some consider ADHD-Inattentive (ADHD-IA) to be a separate disorder from the two other types, ADHD-Hyperactive/Impulsive (ADHD-H/I) and ADHD-Combined (ADHD-C) (e.g. Barkley, 1997, 1998). From examination of diagnostic letters and psychoeducational assessment

[10] Recall that participants in the Pure ADHD and ADHD/LD groups were Carleton University students who had registered with that institution's service centre for students with disabilities (PMC) and had originally

reports, it was determined that within the Pure ADHD group and ADHD/LD group, respectively, the proportion of individuals with ADHD-IA was roughly equivalent: (approximately 55% and 50%, respectively), as was the case for ADHD-IA (approximately 22% and 33%, respectively). Unfortunately, these results are based on a limited number of participants; which was further reduced by the fact that the diagnosing clinician did not specify type of ADHD for two individuals.

Psychoeducational assessment reports were also examined. For the purposes of the current discussion, a psychoeducational assessment report is defined according to the following specifications that were adapted from those set out in 2001 by the Learning Disabilities Association of Ontario for the identification of LD. Briefly, relevant scores are reported from a comprehensive, standardized measure of IQ (e.g., Wechsler Adult Intelligence Scales; Wechsler, 1997b), as well as standardized tests of achievement in reading, writing, and math. In addition, established tests of memory and other types of information processing[11] are expressly investigated and reported on (for example, through the use of a comprehensive memory battery such as the Wechsler Memory Scale (Wechsler, 1997a) or the Learning and Memory Battery (Tombaugh & Schmidt, 1992), along with specific indicators of other types of information processing[12].

Unfortunately, this exercise also failed to yield any consistent results. While all of the ADHD/LD participants had submitted a psychoeducational assessment report, only some of the Pure ADHD participants had done so. Moreover, there were inconsistencies among those psychoeducational assessment reports that were available. For example, one of the

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submitted their documentation, including psychoeducational assessment reports and/or diagnostic letters, in order to receive appropriate accommodations and supports.

[4] Examples of other types of information processing commonly reported are processing speed and visual-motor integration.

[12] For example, the WAIS-III Working Memory Index and the Processing Speed Index

reports submitted by the ADHD/LD participants and one submitted by the Pure ADHD participants did not report actual scores or percentiles; rather, results are described in terms of ranges, such as "average" and "below average". In addition, only some of the psychoeducational assessment reports from each group contained specific data for measures of IP speed. There were also discrepancies in the particular aspects of reading, writing, and math that were reported. For example, in their evaluation of reading, some psychologists reported separate measures of comprehension *and* decoding, others used measures of comprehension *or* decoding, and still others lumped the two together in a single score. Finally, among the ADHD/LD participants, there were inconsistencies in the type of evidence presented to substantiate ADHD. Most evidence was obtained from standardized questionnaires and ratings scales; however, different instruments were used. Thus, the attempt to compare psychoeducational assessment reports for the ADHD/LD and the Pure ADHD groups in order to isolate potential confounding variables was abandoned.

In summary, there is no evidence at present to suggest that some systematic difference (besides LD) existed between the students with Pure ADHD and those with ADHD/LD that could be responsible for differences in performance on the two new tests and the traditional neuropsychological tests of IP speed. Unfortunately, such differences may exist, as current documentation is insufficient to rule out this possibility. That being said, ADHD/LD, but not ADHD alone, was associated with a deficit in IP speed in the current study.

Current Literature on ADHD with Coexisting LD

A review of some relevant studies demonstrates that the current finding is not unique. As has been noted numerous times in this paper, the traditional empirical approach in the area of ADHD assessment has been to compare the performance of individuals with and without ADHD on a variety of neuropsychological tests. However some researchers

have also specifically investigated the impact of coexisting LD on measures of IP speed. Unfortunately, none used adult participants, and, not surprisingly, none used the A-PASAT or the CTIP[13]. However, they did employ tests that putatively reflect IP speed, including the CCPT and a selection of other traditional tests used in the current study.

Besides the fact that these studies used children and did not use the CTIP or A-PASAT, integration of their results with those obtained in the study is clouded by other factors. Not all employed a nonclinical control group and there were variations in how the LD and ADHD/LD groups were composed. Despite such methodological inconsistencies, overall conclusions are largely consistent with the possibility that individuals with ADHD plus LD, but not with ADHD alone, are impaired on measures of IP speed/ capacity in comparison to nonclinical controls.

Three-Group Comparisons: ADHD, ADHD/LD, and Controls. Wu, Anderson and Castiello (2002) investigated what they termed "lower level abilities" (defined as underlying components of executive functioning) in boys with ADHD, with and without LD. Of particular relevance to the current discussion, they identified "processing speed" as one such lower level ability and measured it using a cancellation test[14] and the Stroop Color Naming score[15]. While there was no significant difference among the groups' performance on the cancellation test, a pattern identical to that observed in the current study was revealed on the Stroop. The boys with ADHD plus LD took significantly longer to name the colours than did those with ADHD alone and the nonclinical controls, which in turn performed equivalently.

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[13] The current study is the first to have used the research versions that are currently available.
[14] The Sky Search subtest from the test of Everyday Attention for Children (TEACH; Manly et al., 1999)
[15] Because they tested children, a ceiling effect was not likely.

In a second example, Seidman, Biederman, Monuteaux, Doyle and Faraone (2001) investigated the "executive functioning" of three groups of young males: a nonclinical control group, a pure ADHD group, and an ADHD/LD group. They divided their ADHD/LD group into three separate subgroups according to area(s) of achievement affected by their LD: (1) reading disability, (2) arithmetic disability, and (3) both. As they did not set out specifically to examine IP speed, only one of their reported measures, the Stroop, is relevant to the current discussion. Like Wu et al. (2002), they observed a pattern that mirrors current observations. The group of boys with ADHD alone was not significantly slower on the Stroop compared to nonclinical controls, whereas each of the three ADHD/LD groups was. These authors took things a step further by pointing to a possible relationship between IP speed impairment and severity of LD, as defined by the number of achievement areas impacted. In particular, the degree of Stroop impairment was associated with severity of LD: the group of ADHD/LD boys who had a reading LD *plus* an arithmetic LD was significantly impaired compared to all four other groups. ADHD boys with an LD affecting one achievement area only were significantly impaired in comparison to the group with ADHD alone, and to nonclinical controls.

Hence, results from two studies that, like the current study, subdivided ADHD participants according to LD and employed a nonclinical control group are consistent with current findings. Individuals with ADHD plus LD were impaired in measures of IP speed while individuals with ADHD alone were not. Like the current study, neither included a pure LD group; hence, the question of whether the impairment is a function of LD or the combined effect of ADHD and LD is left unanswered. To address this question, three studies are reviewed below that include a pure LD group in addition to ADHD alone, ADHD/LD, and Controls. Unlike the current study, however, in which individuals with

different types of LD participated, the three groups of researchers included only participants with Reading Disability (RD).

Four-group Comparisons: Pure ADHD, ADHD/LD, Pure LD, and Controls. McGee et al. (2000) compared the performance of four groups: ADHD alone, Reading Disability alone (RD alone), ADHD/RD, and a clinical control group comprised of boys identified as having Disruptive Behaviour Disorder, family relational problems, a past experience of sexual abuse, Anxiety Disorder, or Adjustment Reaction Disorder. They administered a number of tests, including an earlier version of the cancellation task used in the current study, the Visual Matching subtest of the Woodcock-Johnson Psychoeducational Battery–Revised (WJ-R; McGrew, Werder & Woodcock, 1991). However, no significant differences in IP speed were observed; hence, there is no evidence to address the question as to whether IP speed impairments observed in three-group comparisons (the current study, Wu et al., 2002; Seidman et al., 2001) were due to the combined effects of ADHD/LD or LD alone.

It may be that the combined group was not significantly impaired on the cancellation task because the researchers limited their participants to individuals with RD. Had individuals with a variety of types of LD been included, this observation may have been noted, along with any impairment that may be associated with LD alone. An alternative explanation concerns the composition of their control group. Compared to a nonclinical control group, relative impairment on the part of the ADHD plus LD group would likely have been observed, as this would be consistent with the current study and the other three-group comparisons reviewed above (Wu et al., 2002; Seidman et al., 2001). And, should a relationship between IP speed and RD alone exist, this group too, may have been significantly impaired compared to nonclinical controls. Unfortunately, possible

explanations for the absence of significant differences between the ADHD only and the other two clinical groups are not forthcoming.

Weiler, Bernstein, Bellinger and Waber (2000) also compared the performance of four groups of children: ADHD alone, RD alone, ADHD plus RD, and controls. It is not clear if children in the control group could be classified as nonclinical, as although the authors do not specify any disorders or symptoms present in this group, all participants had originally presented to a pediatric clinic to investigate "school-related problems". The researchers administered a number of tests of IP speed, including the WISC-III Digit Symbol Coding (the child version of the DS Coding used in the current study; Wechsler, 1991) and Symbol Search (a cancellation task using abstract geometric symbols; Wechsler, 1991), and found a main effect of ADHD. The children with ADHD performed poorly on these two tests of IP speed compared to those without ADHD, irrespective of RD. However, when examining the interaction of ADHD and RD on the cancellation task, the ADHD plus RD group was significantly slower than both the RD alone and control groups, but *not* the group with ADHD alone.

The absence of a significant difference between the ADHD/LD group and the Pure ADHD group appears incompatible with the current study; as well as the other three-group comparisons discussed above (Seidman et al., 2001; Wu et al., 2002). However, it should be noted that, unlike the current study in which subjects with all three types of ADHD participated, they included only participants with Predominantly Inattentive ADHD (ADHD-IA). In addition, and as noted above, they limited their LD participants to children with reading disability.

In summary, due to methodological inconsistencies, results from studies that included a pure LD group in four-group comparisons are mixed. In addition, because the

LD participants in these studies had a specific type of LD (reading disability) interpretation is somewhat limited. As to whether the current observed impairment in IP speed on the part of individuals with combined ADHD/LD, but not ADHD alone is due to the combined effect of comorbid ADHD and LD or due solely to the impact of LD, the jury is still out. Regardless, a major question needs to be addressed. In the current study, what aspect of LD could have made the ADHD/LD group slower on IP speed measures?

What is LD?

A full discussion of current scientific conceptualizations of LD is beyond the scope of this paper. Instead, in an attempt to qualify later speculation as to what aspect of LD made the ADHD/LD group slower on measures of IP speed, a contemporary definition is introduced and relevant elements discussed.

The Learning Disabilities Association of Canada (LDAC) is a national organization "dedicated to a level playing field for individuals with learning disabilities to enable them to function as citizens with equitable opportunities and to develop to their chosen potential" (LDAC, 2006). In 1997, dissatisfied with their then-dated definition of LD, they launched "Project Think Tank", commissioning a group of prominent LD researchers, educators, and theorists to update it. Taking into account "recent findings in neuropsychology, genetics, brain development, neuroanatomy, imaging techniques, electrophysiology, as well as a range of other related fields", as well as input from provincial/territorial Learning Disabilities Associations, the LDAC Professional Advisory Committee, the National Legal Advisory Committee, and experts and practitioners throughout North America the LDAC recently adopted the Official Definition of Learning Disabilities (LDAC, 2002).

In its simplest terms, this definition proposes that LD is manifested as a significant discrepancy between (at least normal-range) intellectual ability and achievement, or in the

absence of such a discrepancy, achievement consistent with intellectual ability maintained only by abnormally high effort and support. LD results from "impairments in one or more processes related to perceiving, thinking, remembering or learning". Such impairments, in turn, are attributed to a specific impairment in one or more of the following underlying "processing categories": language processing, phonological processing, visual spatial processing, processing speed, memory and attention, and executive functions (LDAC, 2002).

Hence, LD is identified by both the suspected underlying information processing impairment(s) and the affected achievement area(s). While it contains many elements worthy of discussion, for current purposes the most relevant is the categories of underlying information processing deficits, in particular, "processing speed". Accepting the proposition that IP speed is impaired in individuals with LD initially appears to answer the question as to what aspect of LD made the ADHD/LD group slower on the IP speed measures in the current study. Recall that this group demonstrated significant deficits on the CTIP, A-PASAT, WJ-III Visual Matching, and WAIS-III DS Coding and Digit Span tests, all measures of IP speed.

Why were the IP Speed Tests Not Sensitive to Pure ADHD?

One final question must be addressed. Why was the Pure ADHD group not impaired on the tests of IP speed? One possibility is that IP speed *is* impaired in ADHD without coexisting LD, but the tests used in the current study do not detect it. While a review of literature connecting the traditional tests to IP speed is beyond the scope of this paper, it appears that the CTIP and A-PASAT are sensitive to IP speed impairments. As already mentioned, they have demonstrated this sensitivity in populations for which this symptom is known to be pervasive, including individuals with traumatic brain injury (Tombaugh,

Rees, Stormer, Harrison, & Smith, 2006), and multiple sclerosis (Reicker, Tombaugh, Freedman, & Walker, 2006).

A second possibility is that, in addition to being measures of IP speed, those tests that successfully differentiated ADHD/LD (but not Pure ADHD) from nonclinical controls in the current study also tap another, related information processing mechanism, one that is not impaired in ADHD but that is impaired in ADHD/LD (and possibly also LD alone). Baddeley and Hitch first introduced the concept of working memory in 1974 as a refinement of then-prominent views of human memory as being composed of long-term memory and short-term memory, two structurally distinct stores (Hulme & Mackenzie, 1992). According to this view, the two types of memory differ only with respect to capacity (how much information could be held) and duration (for how long), and function primarily as passive information repositories (Hulme & Mackenzie, 1992). Working memory, on the other hand, is defined as a complex, active system that integrates information from both stores during the course of everyday cognitive tasks: "It comprises those functional components of cognition that allow humans to comprehend and mentally represent their immediate environment, to retain information about their immediate past experience, to support the acquisition of new knowledge, to solve problems, and to formulate relate, and act on new goals" (Baddeley & Logie, 1999, p.28).

Working memory may be characterized as a mental blackboard, "in which new and old information is constantly being transformed, combined, and transformed" (Solso, 2001, p.200). It has three components: two "slave systems" which actively maintain verbal and visual information (the phonological loop and the visuo-spatial sketchpad, respectively), under the control of the Central Executive (Baddeley & Logie, 1999). This third component is an active processor with no inherent storage

space that coordinates the two slave systems, focuses, divides, and switches the orientation of the individual, and interacts with long-term memory by retrieving relevant information and/or encoding it for permanent storage. The Central Executive thus essentially performs the "mental manipulation of material held in the slave systems" (Baddeley & Logie, 1999, p.30).

The Central Executive, as defined in this model of working memory, is clearly similar to the mental control aspect that together with IP speed comprises "IP capacity" as defined by the three-component model of attention proposed in the introduction to this paper (Tombaugh, 2003). In addition, there is considerable overlap between the Central Executive and some elements of the five-component models of attention also discussed earlier. In particular, Sohlberg and Mateer's (1989) "sustained attention" includes an element of mental control, analogous to the major role of the working memory's Central Executive, as does Mirsky and Duncan's (2001) "encode". Furthermore, "Focus/execute" and "shift" (Mirsky & Duncan, 2001), "selective attention", "alternating attention", and "divided attention" (Sohlberg & Mateer, 1989), could all be logically conceived of as subsystems integral to the Central Executive's role in focusing and switching attention. In addition to providing correspondence with clinical models of attention, working memory may also qualify as "memory and attention", one of the seven categories of information processing potentially impaired in LD (LDAC, 2002). Indeed, while a thorough discussion is beyond the scope of this paper, some prominent researchers postulate a relationship between working memory impairment and LD (e.g. Swanson, 1994).

Finally, if taken into consideration along with IP speed, working memory may be useful in attempting to understand current observations, particularly if one accepts the premise that the two are inversely related (Homack & Reynolds, 2005). That is, the greater the size

and efficiency (capacity) of working memory, the shorter the time required to process a given quantity of information. Conversely, the faster a given quantity of information is processed, the greater the capacity of working memory. Highly complex tasks put a greater load on working memory capacity (Baddely & Logie, 1999); hence, their performance also benefits from fast processing.

On their face, most of the tests on which the current ADHD/LD group (but not the Pure ADHD group) was significantly impaired compared to Controls appear vulnerable to working memory impairment. In particular, skillful performance on the A-PASAT would require the ability to hold auditory verbal information (numbers), integrate it with relevant knowledge retrieved from long-term memory (addition rules) and manipulate it (add the numbers). Furthermore, its predecessor, the PASAT, is considered to be a measure of many components of attentional processes, including auditory-verbal working memory, in addition to processing speed (Tombaugh, 2006a). Performance on the WAIS-III Digit Symbol and the WJ-III Visual Matching would also appear to benefit from good working memory, in particular, the visuo-spatial sketchpad. Finally, the WAIS-III Digit Span would seem to require both an intact phonological loop and Central Executive for optimum performance.

A critical exception, however, is the CTIP, for which a working memory component is not apparent. The CTIP is primarily a measure of IP speed (Tombaugh, 2006b). This is most evident in the case of the first, "simple" subtest, described as a "pure speed of information processing measure" (Tombaugh et al., 2006, p. 6). Recall that the subject is required merely to respond quickly (by pressing the spacebar on a keyboard) to a single, simple stimulus (an X that appears in the centre of a computer screen). There is no need to hold, manipulate, integrate, and/or retrieve information for optimal performance: the faster

an individual responds, the better their performance. For the second (choice) subtest, a decisional component based on the concrete/literal "form" of the stimulus is added, and the third (semantic) subtest further increases the information processing load "by adding a conceptual component to the decision process" (Tombaugh et al., 2006, p.6). However, even with these increases in task complexity, a working memory requirement for good performance is not evident. In particular, there is no apparent need to store and maintain information, either phonological or visuo-spatial, in order to respond quickly and accurately, and the need to direct processing resources is minimal. Again, the faster an individual responds, the better their performance. Hence, attributing observed group differences to a working memory impairment associated with LD or to the combined effects of ADHD/LD is not adequate to explain all of the current data, in particular, data from the CTIP. Hence it cannot effectively address the question: Why was the Pure ADHD group not impaired on the tests of IP speed?

A second and more parsimonious possibility is that IP speed is not impaired in ADHD. As is evident from the introduction to this paper, there is a lack of agreement as to the specific impairments underlying ADHD, as well as how attention should be defined. While there may be an impairment in attention associated with ADHD, based on the current results, this attentional impairment does not include an impairment in IP speed. A third and final possibility is that there may be an IP speed impairment associated with ADHD; however, the current ADHD participants are representative of a relatively high functioning population, adult university students, who may have developed effective compensatory strategies and overcome this aspect of their attentional impairment.

Conclusions

The approach in the current study was unique in a number of ways, compared to the bulk of the literature, leading to substantially different conclusions. First, subdividing the ADHD group according to the presence or absence of a comorbid LD (a practice employed by only a handful of other researchers) enabled the discovery of a significant impairment in performance on the tests on the part of individuals with ADHD and a coexisting LD, but not ADHD alone. Second, an attempt was made to focus on a single, specific, potential impairment in ADHD: IP speed. This was operationally defined using two new tests, as well as appropriate measures from a number of traditional neuropsychological tests. Initially, results seemed to demonstrate a significant difference in this construct as measured by the two new tests between individuals with and without ADHD. However, it now appears that the presence of a coexisting LD in individuals with ADHD is a key determinant of their performance on these tests.

It appears that along with the three traditional tests, the CTIP and A-PASAT may be useful in the ADHD clinic; however, not as originally expected. While they are apparently unable to distinguish between individuals with and without ADHD, they may be useful in distinguishing individuals with ADHD and a co-existing LD from normals and from individuals with ADHD alone. Unfortunately, due to inconsistencies in participants' documentation, this conclusion is somewhat tenuous. It may be that some other systematic difference or differences, besides LD, between the two clinical groups was responsible for results. It is therefore critical that future research in this area be conducted using individuals with comprehensive, consistent documentation, or even administer to all participants identical psychoeducational assessments and ADHD diagnostic interviews/questionnaires, to ensure that alternative explanations for observed results may

be ruled out. Control over documentation would also enable the proportion of individuals with ADHD-IA, ADHD-H/I, and ADHD-C to be held constant across the clinical groups, thereby eliminating a potential source of between-group variability. In addition, with consistency of documentation, type of LD could be restricted to those affecting reading, in order to enhance comparability to the literature.

Finally, as mentioned repeatedly, the absence of a fourth Pure LD group limited the interpretation of the current data. Was the observed impairment on the tests of IP speed a consequence of LD or of the combined effect of LD and co-existing ADHD? The answer to this question would require the addition of a fourth, Pure LD group.

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Appendix A

Intake Questionnaire

SUBJECT ID _____ Gender: M F HANDEDNESS: R L AGE: _____

MEDICAL HISTORY: _____

(1) Check off all that apply:

Seizures/epilepsy		Visual Impairment	
Depression		Accident/ fall with loss of consciousness	
Anxiety disorder		Head injury	
Hearing Impairment		Hand or arm injury	

(2) Age ADD first diagnosed _____

(3) If you also have a learning disability, age it was first identified _____

(4) Please list your current medication(s) and daily dose(s):

(5) Have you ever used medication for ADD in the past? Y N

If yes, how old were you?

(6) If you currently take medication for ADD, when did you last take it?

(7) Have you ever had counselling or therapy to cope with ADD? **Y N**

If yes, for how long? _____ Was it helpful? **Y N**

Academic History: _____

(8) Do you have a previous degree from: University? Y N

College? Y N

(9) Did you have academic difficulties in primary school? If yes, briefly explain.

(10) Did you have academic difficulties in high school? If yes, briefly explain.

(11) Are you currently experiencing academic difficulties? **Y N**

If yes, briefly explain.

(12) Please indicate any other methods you have used to cope with ADD (e.g. study skills training, reduced course load, accommodations). Use the back of this page if needed.

Appendix B

Informed Consent: Controls

The purpose of an informed consent is to ensure that you understand the purpose of the study and the nature of your involvement. The informed consent must provide sufficient information such that you have the opportunity to determine whether you wish to participate in the study.

Present Study: Assessing Information Processing in Adults with ADHD

Research Personnel:
If you have any questions, please contact Laura Brawn, Secondary Investigator (520-2600, ext.2241). If you have any ethical concerns about this study, please contact the chair of the Carleton University Research Ethics Committee for Psychological Research (Dr. M. Gick, 520-2600, ext. 2664) or the Chair, Department of Psychology (Dr. J. Logan, 520-2600 ext 2690).

Purpose:
The purpose of this study is to better understand attention by exploring the use of tests to detect attention problems in adults. Some of these tests are new and experimental, and we hope they may be useful in identifying and treating people with attention difficulties. Results will be part of a thesis for a potential M.A. Candidate.

Task Requirements:
This study requires that you complete a series of computerized and non-computerized tests of attention.

Duration:
If you participate in the testing, it will require approximately two hours of your time. You will receive no payment, but may receive two experimental credits for PSYC 1001 or PSYC 1002.

Potential Risk/ Discomfort:
There are no physical or psychological risks in this study.

Anonymity/ Confidentiality:
All of your identifying information and test scores will be kept in a locked file cabinet, with access restricted to primary research personnel. At no time will your name appear in any report, publication, or presentation based on this study.

Right to Withdraw:
Your agreement to participate in this study is completely voluntary and independent of your status as a Carleton University student.

You have the right to refuse to answer any specific questions or participate in any specific task. You also have the right to withdraw your consent and terminate your participation at any point. Exercising any or all of these rights will have no consequence on your rights and status as a Carleton University Student, now or in the future.

I have read the above description of "Assessing Information Processing in Adults with ADD" and understand the conditions of my participation. My signature indicates that I agree to participate in this study.

Name: _____ Signature: _____ Date: _____

Researcher: _____ Signature: _____ Date: _____

Appendix C

Informed Consent: ADHD

The purpose of an informed consent is to ensure that you understand the purpose of the study and the nature of your involvement. The informed consent must provide sufficient information such that you have the opportunity to determine whether you wish to participate in the study.

Present Study: Assessing Information Processing in Adults with ADHD

Research Personnel:
If you have any questions, please contact Laura Brawn, Secondary Investigator (520-2600, ext.2241). If you have any ethical concerns about this study, please contact the chair of the Carleton University Research Ethics Committee for Psychological Research (Dr. M. Gick, 520-2600, ext. 2664) or the Chair, Department of Psychology (Dr. K. Matheson, 520-2600, ext. 2648).

Purpose:
The purpose of this study is to better understand attention by exploring the use of tests to detect attention problems in adults. Some of these tests are new and experimental, and we hope they may be useful in identifying and treating ADD in the future. Results will be part of a thesis for a potential M.A. Candidate.

Task Requirements:
This study requires that you allow a qualified Learning Specialist, Dr. Nancy McIntyre, to evaluate your documentation. If you have the specific kinds of attention difficulties that we are investigating, you will be contacted by phone to make a testing appointment. Testing involves completing a series of computerized and non-computerized tests of attention. If you do *not* have the specific kinds of attention difficulties we are investigating, you will be contacted by phone to arrange for the confidential return of your documentation.

Duration:
If you participate in the testing, it will require approximately one-and-a-half hours of your time. You will receive no payment or credit for your participation.

Potential Risk/ Discomfort:
There are no physical or psychological risks in this study.

Anonymity/ Confidentiality:
Your documentation will be kept in a locked file cabinet, with access restricted to the Dr. McIntyre and primary research personnel. In addition, all of your identifying information and test scores will be stored in locked files accessible only to primary research personnel, and *not* to Dr. McIntyre or any other Paul Menton Centre staff. At no time will your name appear in any report, publication, or presentation based on this study.

Right to Withdraw:
Your agreement to participate in this study is completely voluntary and independent of your status as a Carleton University student and/ or as a client of the Paul Menton Centre.

You have the right to refuse to answer any specific questions or participate in any specific task. You also have the right to withdraw your consent and terminate your participation at any point. Exercising any or all of these rights will have no consequence on your rights and status as a Carleton University Student or as a Paul Menton Centre client, now or in the future.

I have read the above description of "Assessing Information Processing in Adults with ADD" and understand the conditions of my participation. My signature indicates that I agree to participate in this study.

Name: _____ Signature: _____ Date: _____

Researcher: _____ Signature: _____ Date: _____

Appendix D

Debriefing: Controls

Thank-you for helping us to learn! Improved methods to detect and treat attention deficit disorder in adults are greatly needed. We are hopeful that by giving newly developed computerized tests of attention to adults with and without attention deficit disorder we will be able to determine if they may address this need in the future.

The generous participation of individuals like you is essential to our research.
Once again, thank-you for helping us to learn!

If you have any questions, please contact Laura Brawn, Secondary Investigator (520-2600, ext.2241). If you have any ethical concerns about this study, please contact the chair of the Carleton University Research Ethics Committee for Psychological Research (Dr. M. Gick, 520-2600, ext. 2664) or the Chair, Department of Psychology (Dr. J.Logan, 520-2600, ext. 2690).

Appendix E

Debriefing: ADHD

Thank-you for helping us to learn! Improved methods to detect and treat attention deficit disorder in adults are greatly needed. We are hopeful that by giving newly developed computerized tests of attention to adults with attention disorder we will be able to determine if they may address this need in the future.

The generous participation of individuals like you is essential to our research.
Once again, thank-you for helping us to learn!

If you have any questions, please contact Laura Brawn, Secondary Investigator (520-2600, ext.2241). If you have any ethical concerns about this study, please contact the chair of the Carleton University Research Ethics Committee for Psychological Research (Dr. M. Gick, 520-2600, ext. 2664) or the Chair, Department of Psychology (Dr. K. Matheson, 520-2600, ext. 2648).

If you have any questions or concerns about your ADHD or your documentation, we advise you to contact your current medical or psychological practitioner. Or, you may contact Carleton's Paul Menton Centre for Students with Disabilities at 520-6608, and explain that you wish to meet with a coordinator.

Appendix F

Table 1

Summary Data for Pure ADHD, ADHD/LD, and Control Groups

Dependent Variable	Group					
	Control		Pure ADHD		ADHD/LD	
	M	*SD*	*M*	*SD*	*M*	*SD*
CTIP RT (msec):						
Simple	300	42	298	86	329	86
Choice	501	68	526	78	561	94
Semantic Choice	730	120	787	178	991	299
A-PASAT Thresholds (msec)	1572	336	1840	445	1902	386
CCPT-II percentiles:						
Omissions	44	22	51	25	40	16
Commissions	66	22	75	26	74	19
Average RT	22	18	21	17	25	18
Average RT SD	38	25	45	22	54	25
WAIS-III percentiles:						
DS Coding	60	25	63	35	40	25
Digit Span	52	29	40	22	30	23
COWAT percentiles:	34	18	30	21	29	27
STROOP Interference percentiles	49	30	54	35	52	30
TMT						
Trails A	40	28	39	33	37	24
Trails B	42	28	29	24	30	24
WJ-III Vis. Matching percentiles	46	23	37	27	24	19