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COGNITIVE INHIBITION IN CHILDREN WITH ATTENTION DEFICIT HYPERACTIVITY DISORDER

by

Elizabeth Nadine McLaughlin

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The undersigned hereby certify that they have read and recommend to the Faculty of Graduate Studies for acceptance a thesis entitled "Cognitive Inhibition in Children with Attention Deficit Hyperactivity Disorder" by Elizabeth McLaughlin in partial fulfilment for the degree of Doctor of Philosophy.

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Research Supervisor:

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Abstract

The view that Attention Deficit Hyperactivity Disorder (ADHD) is a disorder of inhibitory control has recently gathered considerable support. Barkley (1997a) proposed a model of ADHD in which response inhibition is the primary deficit that sets the stage for all of the observed deficits in executive functioning and behaviour. Inhibition is a diverse construct, however, and it is unlikely that children who have deficits in one type of inhibition would necessarily have deficits on all types of inhibition. To assess the scope of the inhibitory control deficit in ADHD, children with (n = 16) and without (n = 24) ADHD were tested on seven different measures of inhibition. Participants ranged in age from 9 to 13 years, and the control and ADHD groups were matched for age and sex. The results were interpreted in the context of a recent taxonomy of inhibition proposed by Nigg (2001). Children with ADHD had longer Stop Signal Reaction Times than control children, and they made more exogenously triggered premature saccades on the Memory-Guided Saccade task. Each of these patterns reflects a deficit in the ability to inhibit prepotent responses or to stop ongoing responses; both are subsumed under Nigg's Executive Motor Inhibition. Children with ADHD showed more interference on the Stroop Colour Word Task, but not on the Simon and Flanker tasks. Kornblum's (1994) dimensional overlap model provides a framework for understanding the pattern across these three measures of Executive Interference Control. There were no group differences on the two measures of Automatic Inhibition: Negative Priming and Inhibition of Return. Taken together, these data limit the scope of the inhibitory control deficit in ADHD to response inhibition. The pattern of findings provides support for Nigg's taxonomy of inhibition, for Kornblum's dimensional overlap model of interference, and for Barkley's model of ADHD.

List of Abbreviations

ADD	Attention Deficit Disorder
ADHD	Attention Deficit Hyperactivity Disorder
ADHD-C	ADHD-Combined Type
ADHD-H	ADHD-Predominantly Hyperactive-Impulsive Type
ADHD-I	ADHD-Predominantly Inattentive Type
APA	American Psychiatric Association
CBCL	Child Behaviour Checklist
CD	conduct disorder
CPRS-PM	Pittsburgh Modified IOWA Conners Parent Rating Scale
CTRS-PM	Pittsburgh Modified IOWA Conners Teacher Rating Scale
DBD-P	Disruptive Behavior Disorder Parent Rating Scale
DBD	disruptive behaviour disorder
DBD-T	Disruptive Behavior Disorder Teacher Rating Scale
DSM-III	Diagnostic and Statistical Manual – third edition
DSM-III-R	Diagnostic and Statistical Manual – third edition – revised
DSM-IV	Diagnostic and Statistical Manual – fourth edition
FCE	Flanker Compatibility Effect
ICD-9	International Classification of Diseases – ninth edition
ICD-10	International Classification of Diseases – tenth edition
IO	Inattention/Overactivity score
IOR	inhibition of return
IRS-P	Pittsburgh Impairment Rating Scale: Parents
IRS-T	Pittsburgh Impairment Rating Scale: Teachers
LD	learning disorders
OD	Oppositional/Defiant score
OCD	obsessive-compulsive disorder
ODD	oppositional defiant disorder
RSI	response stimulus interval

RT	reaction time
S-R	stimulus-response
S-S	stimulus-stimulus
SOA	stimulus onset asynchrony
SS	standard score
SSRT	stop-signal reaction time
TRF	Teacher's Report Form
WISC-III	Wechsler Intelligence Scale for Children – third edition
WRAT-3	Wide Range Achievement Test – third edition

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Cognitive Inhibition in Children with Attention Deficit Hyperactivity Disorder

Overview

In this dissertation, Barkley's (1997a; 1997b) theory that inhibitory control is the primary deficit in attention deficit hyperactivity disorder (ADHD) is examined by comparing the performance of children with and without ADHD on a battery of tasks designed to measure different types of inhibition. In this chapter, the clinical features of ADHD are reviewed briefly, Barkley's inhibitory control model of ADHD is introduced, and the argument is made that the core assumption of this model needs to be examined more thoroughly. In Chapter 2, the study sample is described, and the overall methods and procedures are discussed. Five tasks were used to measure seven types of inhibition. Each task is presented as a separate study (with Introduction, Method, Results, and Discussion) in Chapters 3 to 7. In Chapter 8, the results from the seven measures of inhibition are interpreted together. The conclusions are discussed in terms of what these data tell us about the validity and scope of the inhibitory control model of ADHD, and about current taxonomies of inhibition as a cognitive construct. In this final chapter, areas for future research are also discussed.

Clinical Features of Attention Deficit Hyperactivity Disorder (ADHD)

ADHD is a neurodevelopmental disorder characterized by symptoms of inattention, hyperactivity, and impulsivity (DSM-IV, APA, 1994).[1] Recognized as a distinct constellation of symptoms for almost a century, this disorder has been given many labels, such as minimal brain dysfunction, hyperkinetic disorder, and attention deficit disorder (ADD). The diagnostic criteria have varied by classification system and across editions of each classification system (see Barkley, 1998 and Quay, 1999 for reviews of the history of the labelling and diagnosis of this disorder).[2]

The DSM-IV criteria (APA, 1994) are the current standard for the clinical diagnosis of ADHD in North America. Depending on the number of symptoms present for at least six months in each of two symptom clusters (Inattention and Hyperactivity-Impulsivity), a child may be considered to have one of three types of ADHD: Predominantly Inattentive (ADHD-I), Predominantly Hyperactive-Impulsive (ADHD-H), and Combined (ADHD-C).

To meet diagnostic criteria, the symptoms must have been present and significant at an early age (before age 7 years), must be developmentally

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[1] Diagnostic and Statistical Manual of Mental Disorders, 4th Edition, American Psychiatric Association, 1994.

[2] Although there are important differences between sets of diagnostic criteria, in reviewing the literature, the term ADHD will be used to refer to research participants diagnosed using criteria from the European International Classification of Diseases (ICD-9 or ICD-10, World Health Organization, 1978; 1993) or from the North American DSM system (DSM-III, DSM-III-R, or DSM-IV, APA, 1980; 1987; 1994).

inappropriate (in terms of the frequency or severity of symptoms in comparison to others of the same age and gender), must be present in more than one situation or context (e.g., home and school), and must cause clinically significant impairment in at least one area of functioning (e.g., academic, social). Finally, the symptoms must not be exclusively due to or better accounted for by another disorder.

The prevalence of ADHD is high, with estimates typically ranging from 3 to 5% in school-aged children, although this is considered to be a conservative estimate by some (APA, 1994; see Barkley, 1998 and Lahey, Miller, Gordon, & Riley, 1999 for reviews; see Szatmari, Boyle, & Offord, 1989 for an epidemiological study with Canadian elementary school children). Rates of diagnosis are higher in boys than in girls; male-female ratios range from 2:1 to 9:1, depending on whether samples are clinic-referred or population-based (APA, 1994; Gomez, Harvey, Quick, Scharer, & Harris, 1999; Lahey, et al. 1994; Szatmari et al., 1989).

ADHD frequently co-occurs with other disorders, most often with learning, oppositional defiant, conduct, mood, and anxiety disorders, but also with tic, elimination, sleep, and substance use disorders. Rates of comorbidity with at least one other disorder are high, with most estimates ranging from 30 to 80%, depending on the sample studied (Biederman, et al., 1999; Eiraldi,

Power & Nezu, 1997; Faraone, Biederman, Weber, & Russell, 1998; Jensen, Martina, & Cantwell, 1997; Pliszka, Carlson, & Swanson, 1999).

The impact of ADHD is significant. ADHD accounts for 33 to 50% of referrals to mental health clinics (Barkley, 1990). For affected children, it can be associated with poor peer relationships, poor parent-child relationships, increased family stress, poor academic performance, and low self-esteem (e.g., Biederman, et al., 1999; Faraone, et al., 1998; Lahey, et al., 1994; Szatmari, Offord, & Boyle, 1989; see Barkley, 1998 for a review).

ADHD is also a pervasive disorder that can cause longstanding difficulties into adulthood. Prospective follow-up studies have shown that up to 75% of children with ADHD continue to have clinically significant symptoms of ADHD in adulthood (e.g., Weiss, Hechtman, Milroy, & Perlman, 1985; Weiss and Hechtman, 1993). Mannuza, Klein, Bessler, Malloy, & La Padula (1993) followed 91 boys with ADHD for 13 to 19 years. They reported that although only 11% of probands met criteria for a diagnosis of ADHD in adulthood, irrespective of their current diagnosis, adults who had had childhood ADHD were more likely than control subjects to have lower socioeconomic status, educational achievement, and occupational status. Childhood ADHD has also been found to predict substance abuse, mood disorders, and anxiety disorders in adults (Mannuza et al., 1993; Weiss et al., 1985).

With regard to etiology, there is strong evidence for a genetic component to the disorder, and anatomical and physiological anomalies (see Castellanos, 2001, for a review of studies using neuroimaging and neuroelectrophysiological techniques, and see Castellanos, 1999, for a broader review of the psychobiology of ADHD). Also, psychosocial factors (such as socioeconomic status and parenting) probably interact with or contribute to the manifestation of symptoms (Lahey, et al., 1999; Waschbusch, 2002).

Both pharmacological and psychological treatments have been effective in managing the symptoms of ADHD (Barkley, 1998; Multimodal Treatment Study of Children with ADHD [MTA] Cooperative Group, 1999). Psychostimulant medication (e.g., methylphenidate or Ritalin) is the standard pharmacological treatment, but other drugs such as antidepressants have also proven to be effective for some children with ADHD (Schachar & Ickowicz, 1999; Solanto, Arnsten, & Castellanos, 2001). The primary psychological treatments for ADHD make use of behavioural principles, especially contingency management. Behavioural interventions on their own, and those combined with pharmacological interventions have received considerable empirical support (see Pelham & Waschbusch, 1999, for a review).

ADHD is a complex developmental disorder. Despite decades of research, many questions remain to be answered. In her recent review, Tannock (1998) noted that the two main scientific approaches currently used to explore the

nature of ADHD are studying neurobiological models of genetic, neuroanatomical, or neurochemical anomalies, and studying cognitive models of information processing anomalies. The present study is an example of the latter approach.

For decades, researchers have attempted to validate the existence of, and characterize the nature of, a primary cognitive deficit in children with ADHD. Cognitive constructs currently under investigation include: executive function, arousal, motivation, resource allocation, response to reinforcement, delay aversion, and cognitive control (these constructs are not mutually exclusive or unrelated; see Douglas, 1999, and Tannock, 1998 for descriptions of the prominent theories). As noted by Nigg (2001), common to many of these theories is some reference to an impairment in inhibitory control, although each differs in terms of how central or fundamental inhibition is to the theory, and in terms of how inhibition is defined.

One theory that has gained considerable attention over the past few years is that offered by Barkley (1997a, 1997b), who proposed that inhibitory control is the primary deficit in ADHD. With this comprehensive model, Barkley attempts to account for much of the existing data on the cognitive

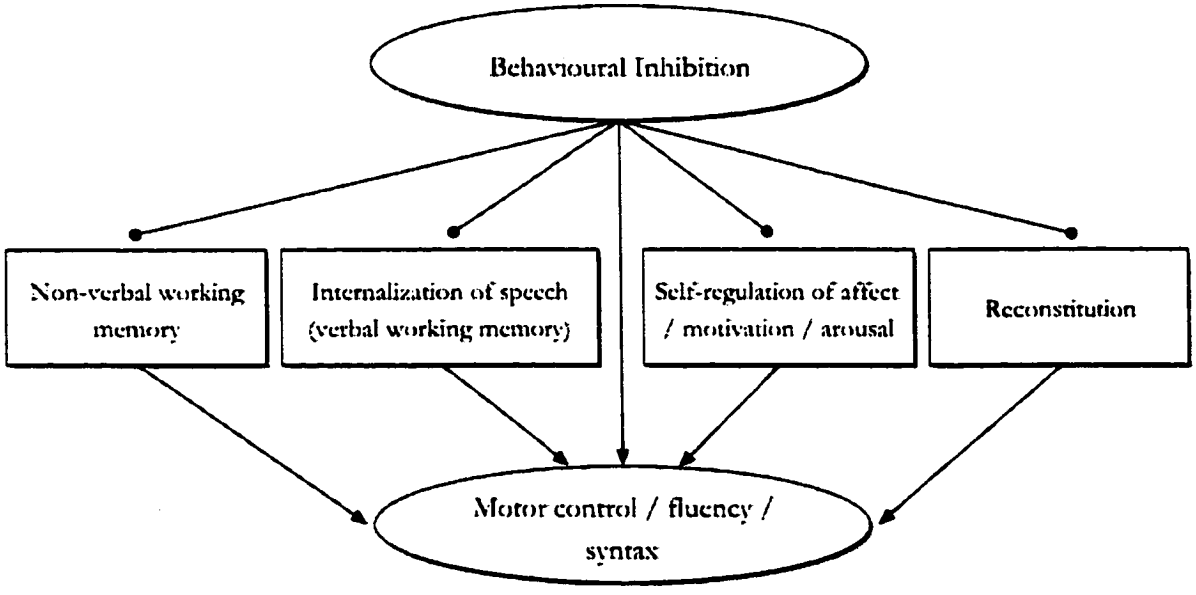
underpinnings of ADHD, to integrate what is currently known about the psychobiology of ADHD, and to explain how the underlying dysfunction relates to the behaviours and symptoms that characterize children with ADHD. Barkley's model is the focus of this study. It is important to note that this model applies only to children with ADHD-H and ADHD-C, and not to those with ADHD-I.

Barkley's Hybrid Model of Executive Functions

Figure 1.1 represents the key elements of Barkley's model (1997a, 1997b; see also Barkley, 1999, 2001). Barkley draws from four neuropsychological models of pre-frontal lobe function (those of Bronowski, Goldman-Rakic, Fuster, and Damasio) to construct what he calls a "Hybrid Model of Executive Functions". In this model, behavioural inhibition is an executive function that allows for the occurrence of four other classes of executive functions: non-verbal working memory, internalization of speech (verbal working memory), self-regulation (of affect, motivation, and arousal), and reconstitution. All five are necessary for motor control, fluency, and syntax. Within this model, a primary deficit in behavioural inhibition would set the stage for secondary impairments in each of the four 'intermediate' executive functions, which in turn, would lead to deficits in motor programming, response execution, and

goal-directed action. Primary deficits in behavioural inhibition could also lead directly to deficits in motor control.

Figure 1.1. Barkley's Hybrid Model of Executive Functions (Adapted from Barkley, 1997a).



Barkley provides supporting evidence for his classification of the executive functions from factor analytic and neuroimaging studies. To apply the model to ADHD, for each component (e.g., nonverbal working memory), he reviews the neuropsychological, cognitive, and behavioural evidence in support of a deficit among children with ADHD. He also links these deficits to the symptomatology of ADHD, and uses the model to make predictions about as yet untested deficits in children with ADHD.

The core element of the Hybrid Model's application to ADHD is the deficit in behavioural or response inhibition.[3] The primacy and importance of behavioural inhibition in this view of ADHD is reflected in the following statement by Barkley (1997a):

> The essential impairment in ADHD is a deficit involving response inhibition. One consequence is that improvement or amelioration of the inhibitory deficit in ADHD should result in improvement in the four executive functions that depend upon inhibitory capacity and also in motor control that those executive functions afford. (p. 65)

For Barkley, inhibition is composed of three different processes: inhibition of prepotent responses, cessation of ongoing responses, and interference control. In defining each, an example will illustrate how each sets the occasion for (but does not directly cause) deficits in the other executive functions.

A prepotent response is one that will receive immediate (positive or negative) reinforcement, or one that has been reinforced in the past. Included in this definition are reflexive and automatic responses. The inhibition of a prepotent response allows for a delay in responding so that other executive functions (e.g., problem solving) can take place. It is also necessary in situations of delayed gratification or in those requiring self-control.

Cessation of, or interruption of, ongoing responses refers to stopping responses that are not reinforced, or motor programs that are not effective.

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[3] Barkley (1997a, 1997b) restricts his theory to "response" or "behavioural" inhibition. He refers briefly to "cognitive inhibition" (which is not defined) only to say that his model does not pertain to this type of inhibition.

This permits, or sets the stage for, sensitivity to errors, response to feedback, and behavioural flexibility.

Interference control protects against internal or external distractions throughout periods of delay before responding. Many of the other executive functions take place during these delays. Accordingly, interference control supports these other executive functions.

Barkley (1997a) reviews two types of evidence to support the hypothesis that children with ADHD have a deficit in response inhibition. First are parent/teacher ratings and observational studies of hyperactivity and impulsivity, which could be behavioural manifestations of poor response inhibition. Barkley recognizes that this line of evidence involves a circular argument, since children are diagnosed with ADHD partly based on these behaviours. Also, hyperactivity and impulsivity could be the result of other factors, and do not necessarily imply an underlying deficit in inhibitory control. Second are laboratory measures of the three types of inhibition. Barkley's review will not be repeated here. Instead, a few examples of the kinds of evidence he offers for each type of inhibition will be highlighted.

For inhibition of prepotent responses, Barkley refers to tasks in which there is a conflict between prepotent responses (those that are reinforced during the task or that have a history of being reinforced), and instructions to inhibit those responses. For example, the observation that children with ADHD make

more errors than control children on "no-go" trials in a go/no-go task (e.g., Shue & Douglas, 1992) is taken as evidence of their inability to withhold a prepotent response. Also in this category are studies showing that children with ADHD make more errors of commission on the Continuous Performance Task (see Corkum & Siegel, 1993 and Losier, McGrath, & Klein, 1996 for reviews), and more impulsive errors on the Matching Familiar Figures Test (e.g., DuPaul, Anastopoulos, Shelton, Gueveremount, & Metevia, 1992).

Barkley (1997a) refers to two types of evidence to support the existence of a deficit in the cessation of ongoing responses. The first is the finding that children with ADHD have impaired performance on the stop-signal paradigm (to be discussed in detail in Chapter 3). This paradigm directly measures the ability to withhold a response and indirectly measures the time taken to stop. The second is the pattern of perseveration observed in children with ADHD on tasks such as the Wisconsin Card Sort Test (see Barkley, Grodzinsky, & DuPaul, 1992 for a review, although see the more recent review by Sergeant, Guertz, & Oosterlaan, 2002 for a different conclusion).

The main evidence offered in support of a deficit in interference control is with regard to ADHD children's impaired performance on the Stroop Colour Word task (to be discussed in more detail in Chapter 4). On this task, the participant is required to name the ink colour that a colour-word is printed in. For example, if the word "red" were presented in blue ink, the correct response

would be blue. This task requires interference control because the interfering or distracting written word must be ignored in order to give the correct response (although see MacLeod, 1991 for alternative theories). In addition to performance on this specific task, Barkley refers to studies demonstrating that children with ADHD have a harder time ignoring distractions embedded within a task (e.g., Leung & Connolly, 1996).

Assessing the scope of the inhibitory control deficit in ADHD

Barkley assembles a great deal of data to substantiate the primary assumption of his model that children with ADHD have deficits in inhibitory control. Proponents of competing models have challenged this assumption by arguing that the observed deficits could be maintained by, or are secondary to, mechanisms other than inhibitory control (see Douglas, 1999; Sergeant, Oosterlaan, & van der Meere, 1999). Another challenge stems from the fact that, based on what is known about inhibition from the point of view of cognitive psychology, the notion of an inhibitory control deficit in ADHD has not been fully evaluated.

Inhibition is a diverse construct with many different subtypes, each of which is probably subserved by a different neural mechanism (Klein & Taylor, 1994; Nigg, 2000; Rafal & Henik, 1994). It is unlikely, therefore, that an individual (or group of individuals with a common cognitive deficit) with difficulties on one type of inhibitory task would have difficulties on all types of

inhibitory tasks. A more thorough assessment of inhibition in children with ADHD, in which multiple types of inhibition are examined within the same group of children, will test the validity of the inhibitory control model, and assess the scope of the inhibitory control deficit in ADHD.

Klein and Taylor (1994) also remind us that cognitive inhibition[4] is a "hypothetical construct whose effects within the information processing stream are inferred to exist on the basis of observable human behaviour" (p. 113). Even within the cognitive literature, where the nuances of information processing are typically studied much more so than they are with clinical populations, and where techniques such as neuroimaging and computational modelling are frequently employed, it is difficult to determine with certainty whether an observed pattern of performance is due to one kind of inhibitory mechanism versus another, or due to some other mechanism. A parallel goal of this research, therefore, is to learn about the taxonomy of inhibition based on the patterns of deficits observed in children with ADHD. If children with ADHD are impaired on some inhibitory tasks and not on others, we may be able to identify tasks that share the same underlying mechanisms.

The tasks chosen for study were: a Stop Signal Task, a Stroop Colour Word Task with a Negative Priming manipulation, a Simon Task combined

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[4]For Klein and Taylor (1994), cognitive inhibition is distinct from neural inhibition but would include Barkley's 'response inhibition'.

with a Flanker Task, a test of Inhibition of Return, and a Memory-Guided Saccade Task. The specific rationale for using each task will be discussed when the task requirements are described (in Chapters 3 to 7). To introduce each measure, the main inhibitory process assessed in each is listed in Table 1.1, as are some of the unique features.

In the same table, five of the measures are labelled according to Barkley's definition of inhibition. The remaining two measures, Negative Priming (Chapter 4) and Inhibition of Return (Chapter 6), were selected for study because they do not fall under Barkley's definition. Barkley's model would not predict deficits on these measures in children with ADHD, although a model with a broader definition of inhibition might. These two measures, in particular, were chosen to assess the scope of the inhibitory deficit in children with ADHD.

Table 1.1. Measures selected to assess different types of inhibitory processes, their unique features, and their categorization according to Barkley's (1997a) definition of response inhibition and Nigg's (2001) taxonomy of inhibition.

Measure	Main process examined	Unique features	Barkley's definition	Nigg's taxonomy
Stop Signal	Stopping an ongoing response.	Yields a measure of time taken to stop.	Cessation of an ongoing response; Inhibition of a prepotent response	Executive Motor
Stroop Colour-Word	Interference or response competition due to automatic word reading.	A central feature of the target stimulus must be inhibited.	Interference control	Executive Interference Control or Response Conflict
Negative Priming	An indication of the amount of inhibition recruited on a previous (Stroop) task.	An automatic corollary of inhibition on another task.		Automatic Cognitive
Simon	Interference or response competition due to the location of the stimulus.	The location of the target stimulus must be inhibited.	(Interference control)	(Executive Interference Control or Response Conflict)
Flanker	Interference or response competition due to peripheral stimuli.	The identity of peripheral stimuli must be inhibited.	(Interference control)	Executive Interference Control or Response Conflict
Inhibition of Return	Inhibition of a previously attended location.	An automatic process following the orienting of attention.		Automatic Motor
Memory-Guided Saccade	Inhibition of (a) reflexive saccades and (b) to-be-executed saccades.	(a) Inhibiting a reflex. (b) Inhibiting information held in working memory and needed for an upcoming response.	(a) Inhibition of a prepotent response (b) (Interference control)	(a)Executive Motor (b) Executive Motor

Note. Items in parentheses refer to tasks that were not directly mentioned in Barkley's (1997a) or Nigg's (2001) reviews. In these cases, the label was assigned based on the definitions given for each type of inhibition and the categorization of similar tasks within each model.

Also in Table 1.1, each measure is labelled according to a recent, comprehensive taxonomy of inhibition offered by Nigg (2000; 2001). Based on a review of the broader cognitive and personality literatures, Nigg categorized inhibitory processes into three main types: Executive, Automatic, and Motivational.

Executive Inhibition refers to the effortful, controlled, or 'top-down' suppression of a response, in order to comply with task demands. It is comprised of three types: Motor (suppression of a primary or reflex response), Interference Control or Response Conflict (suppression of an interfering or competing response), and Cognitive (suppression of a thought, as required in a directed forgetting task). Together, the Executive Motor and Executive Interference Control categories constitute Barkley's definition of response inhibition.

In contrast, Automatic Inhibition refers to inhibition that 'automatically' takes place regardless of the specific intentions of the participant. Automatic Inhibition is categorized into two types: Motor and Cognitive. An example of the first type is Inhibition of Return, in which orientation toward a recently attended location are automatically suppressed (see Chapter 6). An example of the second type is Negative Priming, in which an item suppressed on one trial is the required response on the subsequent trial (see Chapter 4).

The first two categories in Nigg's taxonomy were taken from models in the cognitive literature. The third type, Motivational Inhibition, was taken from theories of personality. Motivational Inhibition refers to the withholding of a response or behaviour because of anxiety or the fear of punishment. In cognitive tasks, this type of inhibition would be elicited in tasks that implement reward and response cost for performance.

In an effort to categorize the nature of the inhibitory control deficit in ADHD, Nigg (2001) surveyed the existing literature on inhibitory tasks of each type in ADHD. He concluded that the majority of evidence pointed to a deficit in Executive Motor Inhibition in children with ADHD. The data were mixed with regard to deficits in Executive Interference Control, Executive Cognitive Inhibition, and Motivational Inhibition. There were not enough studies to be able to draw conclusions about Automatic Inhibition. Consistent with the decision to include two measures of Automatic Inhibition in this study, Nigg (2000) argued that although not often used in studies of developmental psychopathology, tests of Automatic Inhibition have the potential to add to our understanding of disorders of disinhibition. He hypothesizes, for example, that performance on measures of Automatic Inhibition may be able to distinguish between different populations with equivalent deficits on measures of Executive Inhibition. For example, Nigg (2000) hypothesizes that although difficulties on the Stroop task have been reported in both schizophrenia and

ADHD, the groups may differ in terms of their performance on measures of Automatic Inhibition.

The extensive reviews by Nigg (2001) and Barkley (1997a) both point to a deficit in Executive Motor or response inhibition in children with ADHD. Accordingly, in this study, children in the ADHD group are expected to show deficits on the Stop Signal task (Chapter 3) and the Memory-Guided Saccade task (Chapter 7). Both Nigg and Barkley refer to performance on the Stroop Colour Word Task as evidence for a deficit in interference control in ADHD, although both also caution that the findings with this task are mixed. As will be argued in Chapter 4, the Stroop task used in this study incorporated some methodological improvements designed to isolate the interference control aspect of the task. In addition, two other measures of interference control, the Simon and Flanker tasks (Chapter 5) were administered. Both are measures of interference control or response competition, but they differ from the Stroop in terms of the nature of the stimuli to be inhibited. Finally, two measures of Automatic Inhibition, Negative Priming (Chapter 4) and Inhibition of Return (Chapter 6) were studied. If children with ADHD have a deficit in the broadly defined construct of inhibition, they should show deficits on these tasks. If their deficit is constrained to executive or response inhibition, they should not.

Chapter 2. General Methods

General Participants

Overall approach to defining the main sample

As discussed in Chapter 1, a significant proportion (30 to 80%) of children with ADHD meet diagnostic criteria for a comorbid disorder. The inhibitory control theory of ADHD refers to the deficits specific to ADHD, rather than to the comorbid conditions (Barkley, 1997a). It could be argued, therefore, that in order to properly examine the inhibitory control theory, one should restrict the ADHD group to children with "pure" ADHD, or ADHD with no comorbid disorder. The epidemiological data suggests, however, that to do so would exclude up to 80% of children with the disorder, and would thus compromise the external validity and generalizability of the findings.

On the other hand, the risk of including children with comorbid conditions is that any difference identified between the clinical and control groups could be either partly or wholly due to the presence of the coexisting condition, or a related variable, severity of impairment. Pennington and Ozonoff (1996) reviewed studies using executive function tasks in children with ADHD and various comorbid disorders. They found that the executive function deficits were specific to the presence of ADHD, and not to comorbidity with learning disorders (LDs) or other disruptive behaviour

disorders (DBDs). Nigg, Hinshaw, Carte, and Treuting (1998) found the same pattern when they compared neuropsychological test performance in children with ADHD and comorbid oppositional defiant disorder (ODD), conduct disorder (CD), and reading problems. These findings support the position that including children with comorbid disorders in this study may allow for an increase of statistical power and generalizability without altering or confounding the relation between ADHD and the variable in question (inhibition).

We cannot ignore, however, the many examples of studies showing that comorbidity does affect performance on cognitive tasks. For example, Schachar, Tannock, and Logan (1993) reported that children with comorbid ADHD and CD did not differ from control children on the stop signal paradigm, a measure of inhibitory control, whereas children with ADHD alone did differ from controls. McLaren (1989) found the opposite, that children with ADHD and comorbid ODD were more severely impaired on several cognitive measures than those with ADHD alone.

The ideal approach is a compromise between the most exclusive (i.e., no comorbidity) and most inclusive (i.e., unlimited comorbidity), in which subgroups of children with pure ADHD and ADHD with specific comorbid disorders are compared. The main limitation of this strategy is the large sample size that it would require, and the feasibility of recruiting large groups of children of each subtype. Given the scope and goals of this study (to test the

specific hypothesis that children with ADHD have a deficit on a variety of inhibitory control tasks that have not been assessed in this population), it would be unreasonable to set out to create multiple subgroups at this preliminary stage.

Considering all of these competing factors, the following approach was chosen. Recruitment targeted children who had received a diagnosis of ADHD, regardless of the presence of comorbidity. In principle (i.e., assuming random sampling from the population), such a strategy should result in a mixed and representative sample of children with ADHD.[5] The decision of whether to form subgroups was based on the number of participants of a particular subtype in the total sample. The selection of the final group was made with the following considerations: power (i.e., whether the resulting sample would have enough power to detect group differences), internal validity (i.e., whether including certain participants would compromise the interpretation of the findings), and external validity (i.e., whether including certain participants would compromise the generalizability of the study).

Another related decision was whether to include children with ADHD-I (Predominantly Inattentive Type). The inhibitory control model does not apply to these children (Barkley, 1997a), even though they are part of the same broad

[5] Note that recruitment from non-clinical settings may yield lower comorbidity rates than recruitment from a clinical setting (see Jensen, et al., 1997, and Waschbusch, 2002, for discussions of this pattern known as Berkson's bias).

diagnostic category as those with the other types of ADHD, and even though there is a great deal of symptom overlap between this type and the Combined Type (to which the inhibitory control theory applies). The inhibitory control model predicts an intriguing dissociation which somewhat challenges the notion that the three types of ADHD are truly variants of the same disorder (see Barkley, 1998 and McBurnett, Pfiffner, & Frick, 2001 for discussions about the current DSM-IV nosology). It predicts that ADHD children of the Predominantly Hyperactive or Combined Types should show a deficit in inhibitory control, whereas those of the Predominantly Inattentive Type should not. While an interesting hypothesis to test, a comparison of subtypes of ADHD is beyond the scope of this study, especially because many of the tasks have not yet been tested in the primary population of interest. As a compromise, children with ADHD-I who volunteered to participate were tested. Given the lower prevalence of this diagnosis, a sizeable subgroup for equal comparison with the two main groups was not anticipated. Rather, it was hoped that some preliminary hypotheses might be generated based on the available sample.

In the remainder of this section, the process involved in determining the sample for the main analyses is described. In addition to comorbidity and diagnostic subtype, other inclusion and exclusion criteria related to medical history, medication use, and behaviour ratings made by parents and teachers.

Ratings from teachers were obtained to minimize the influences of source bias from having only parent ratings, and to confirm that the symptoms occur in more than one setting.

Recruitment

Children who had previously been diagnosed with ADHD were recruited from the general population. The approach to recruiting participants for the ADHD group might be considered to be somewhat between a clinic-based study and a population-based study, each of which has its own advantages and disadvantages. In a clinic-based study, only children recently referred for treatment are included. This may bias toward those with more severe impairment, more disruptive behaviour, or more access to treatment. At the other extreme, in a population-based study, all children in a population (e.g., a school or a community) are screened for symptoms of ADHD and those who surpass a threshold are automatically, or following further assessment, entered into the clinical sample. With this type of recruitment, children who might never have been identified by their parents or teachers as having difficulties significant enough to warrant formal assessment or treatment may become part of the clinical group. The approach chosen here includes only those who, at some point in the past, had been referred for an assessment of their ADHD symptoms, but who are not necessarily actively part of a treatment program. This approach is not without its drawbacks, because it relies more heavily on

parents to volunteer for the study, in contrast to the other two approaches, for which an entire sample (e.g., everyone referred for treatment during a certain period of time, or everyone in a particular school) is invited to participate.

Fifty two children participated in this study. They were recruited by one of four methods. Firstly, 20 (38.46%) of the children had previously participated in research conducted by Dr. D. P. Munoz, Queen's University, Kingston, Ontario. Following their participation, they and their parents had indicated that they would be willing to be contacted for any future studies. Secondly, 8 (15.38%) responded to an advertisement published in the newsletter for members of the Attention Deficit Association of Nova Scotia, Halifax, Nova Scotia (see Appendix A). Thirdly, 12 (23.08%) responded to a similar advertisement (see Appendix B) posted at a family fitness centre in Halifax, Nova Scotia. Finally, 12 (23.08%) of the participants learned of this study by word of mouth, via other participants or associates with either the Physiology Department at Queen's University or the Psychology Department at Dalhousie University.

Preliminary inclusion criteria

At this preliminary stage, children were invited to participate in the study if they were between the ages of 9 and 13 years, and either (in the case of the control group) had no known diagnosis under the DSM-IV (APA, 1994), or (in the case of the ADHD group) had been diagnosed with ADHD by a physician

or psychologist. Non-diagnosed children were not invited to be part of the control group if they had a first degree relative with ADHD (and consequently, siblings of the children in the ADHD group were not invited to participate). Children were also required to have normal or corrected-to-normal vision, and for English to be their first language.

The ultimate goal was to have two groups matched for age (within 6 months) and gender. As noted earlier, the prevalence of ADHD is higher in boys than in girls. Not surprisingly, there were more male volunteers in the ADHD group than female. To achieve a matched sample, some females without ADHD who volunteered near the end of the study were not tested.

Informed consent

Those who expressed an interest in the study were provided with a written description of the rationale, the basic methods and procedures, and information regarding the voluntary nature of the study (see Appendix C for information letters).[6] The letter indicated that children who took psychostimulant medication would be asked to refrain from taking it 24 hours prior to each testing session. The letter also indicated that participants would be compensated for their time and travel expenses with $15 per 90 minute session, and that they could withdraw from the study at any time. Parents and children were invited to

[6] The letter for participants in Kingston and Halifax were identical, with the exception of contact numbers and location information. For this and all remaining parent and teacher letters, only the Halifax letter will be presented in the Appendices.

ask any questions about the study at that time. They were then asked to provide written informed consent (see Appendix D). The parents of two children with ADHD declined to participate at this stage because they did not want to take their child off stimulant medication for three separate 24-hour periods.

Defining the sample

Characteristics of the sample were obtained through three methods: a questionnaire completed by parents, rating scales completed by parents and teachers, and four screening tests completed as part of the study protocol. In this section, each of these methods will be described, and the corresponding exclusion criteria will be outlined. A similar package of four rating scales was given to both parents and teachers. Each scale will be described separately, and the exclusion criteria based on these scales will be discussed together.

All of the inclusion and exclusion criteria are summarized initially in Table 2.1, and reasons for excluding specific participants are presented following the discussion of all of the measures, in Table 2.2.

Table 2.1. Inclusion (A) and Exclusion (B) criteria for the control and ADHD groups.

A. Preliminary Inclusion Criteria

Criterion	Controls included if:	ADHD included if:
Age	9-13 years	9-13 years
Diagnosis	No known DSM-IV diagnosis	Diagnosed with ADHD by a physician or psychologist
Family history	No first degree relatives with ADHD	
Vision	Normal or corrected-to-normal	Normal or corrected-to-normal
Language	First language English	First language English

B. Exclusion Criteria

Criterion	Controls excluded if:	ADHD excluded if:
Parent forms	Not returned	Not returned
Medical and educational history	History of neurological problems Evidence of significant learning or behaviour problems	History of neurological problems
Medication	On any kind of psychotropic medication	On any kind of psychotropic medication during testing
Diagnosis and Comorbidity	Any DSM-IV diagnosis	Diagnosis of Tourette's Disorder or ADHD-I
CBCL and TRF	Any T score > 65	
DBD-P and DBD-T	Endorsement of a clinically significant number of ADHD-I, ADHD-H, ADHD-C, ODD, or CD symptoms	Lack of endorsement of a clinically significant number of ADHD-H or ADHD-C symptoms
CPRS-PM and CTRS-PM	Above cut-offs on the IO or OD scale	
IRS-P and IRS-T	Any rating > 3	Fewer than two ratings > 3
WISC-III	Estimated IQ < 80	Estimated IQ < 80

Note. CBCL = Child Behaviour Checklist; TRF = Teacher's Report Form; DBD = Disruptive Behavior Disorders Rating Scale; P = Parent; T= Teacher; CPRS-PM = Pittsburgh Modified IOWA Conners Parent Rating Scale; CTRS-PM = Pittsburgh Modified IOWA Conners Teacher Rating Scale; IO = Inattention/ Overactivity subscale; OD = Oppositional Defiant Subscale; IRS = Impairment Rating Scale; WISC-III = Wechsler Intelligence Scale for Children-III.

Participant information provided by parents

Parents were given a letter that provided the rationale for the questionnaire and rating scales (see Appendix E). Parents were asked to rate their children's behaviour when they were off psychostimulant medication if applicable. The rate of return of the parent packages was high (98%); the data is unavailable for one boy (#48) who was consequently excluded from the control group. The majority (92%, n = 47) of the remaining parent packages were rated by mothers, one (2%) was rated by both parents, and three (6%) were rated by fathers.

Participant Information Questionnaire

A participant information questionnaire was developed for the purpose of this study (see Appendix F). It was largely based on Barkley's (1990) ADHD Parent Interview. Each parent was asked to provide information pertaining to the child's educational, medical, treatment, and family history. Parents were contacted for further clarifying information as required. This questionnaire provided much of the information used to determine whether participants should be included in the main sample. In addition to the reasons for exclusion, Table 2.2 highlights other potentially relevant information pertaining to the participants' medical, academic, and treatment history.

Children in both groups were excluded if they had a history of neurological problems or seizures. One girl in the control group (#20) was

excluded based on this criterion. Participants were not excluded because of other medical problems (e.g., asthma).

Children in the control group were excluded if there was evidence of significant behavioural problems, as indicated by a report of multiple suspensions from school, enrolment in a special class for children with behaviour problems, or psychological treatment for behaviour problems. One boy (#26) in the control group, had received psychological treatment for behaviour problems, and was excluded, in part, on this basis. Two participants had previously received psychological treatment for other issues (separation of parents, death of a family member). They were not excluded.

Control children were excluded if they had a diagnosed LD, or if they were enrolled in a special class for children with learning problems. None met these criteria. They were not excluded if they had a history of resource help at school for specific learning needs (e.g., reading, math), recognizing that the referral patterns to these services vary widely (although receipt of these services is noted in Table 2.2).

Each parent also reported the medication that his or her child was taking. All medications are briefly noted in Table 2.2, and are described in more detail later. Children who took medications for a non-psychiatric medical condition (e.g., asthma) were not excluded. As mentioned above, children who usually took psychostimulant medication for ADHD symptoms refrained from taking it

for at least 24 hours prior to each testing session.[7] In some cases, however, these children took other medications (e.g., antidepressants) throughout the study. Because of longer half-lives and build-up in the system, in order to be medication-free at the time of testing, these medications would have to have been discontinued for days to weeks prior to the study. In some cases there would also be risks of going off the medications for a long period of time, outweighing the benefits of participating in this study.

There were four children in the ADHD group who were taking a psychotropic medication at the time of testing. One (#18) was taking a phenothiazine (chlorpromazine), one (#19) was taking an antipsychotic (haloperidol), one (#23) was taking a designer (noradrenergic-serotenergic) antidepressant (venlafaxine), and one was taking (#35) a tricyclic antidepressant (imipramine). All of these medications were prescribed, at least in part, to treat the symptoms of ADHD. Although it is not uncommon for children with ADHD to take medications other than stimulants, the generalizability of these four participants to the broader population of children with ADHD is low (Solanto, et al., 2001; see Goldstein & Turner, 2000, for a survey of the types of medications used by students with ADHD). Furthermore, even if this was a

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[7] A 24-hour stimulant-free period is standard in the literature examining cognitive performance in non-medicated children with ADHD. This period is longer than the "behavioural rebound effect", which can reportedly take place 5 to 15 hours after a dose of methylphenidate (Solanto, et al., 2001).

representative sample, these children were not medication-free at the time of testing. Each of these medications has the potential of impacting cognitive or motor functioning.[8] These four participants were, therefore, excluded from the analyses.[9]

Parents also reported their children's diagnostic history. Children in the control group were excluded if they had been diagnosed with any DSM-IV disorder. Parents of children in the ADHD group provided details such as when and by whom the child was diagnosed, the specific subtype of ADHD, and the presence of comorbid disorders. Three children with ADHD-I (#'s 18, 37, and 49) were excluded from the main analyses; one of these three (#18) was also excluded based on taking chlorpromazine. Of the remaining children in the ADHD group, there were three (#'s 9, 11, and 34) with a comorbid LD, one (#4) with comorbid CD, and one (#38) with comorbid Tourette's disorder. As discussed above, the competing demands of power, internal validity, and external validity were considered when making decisions based on these comorbid conditions.

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[8] Chlorpromazine (Thorazine) induces high sedation and moderate involuntary movements. Haloperidol (Haldol) induces low sedation and very high involuntary movements, and may impair performance on a sustained attention task. Venlafaxine (Effexor) has no sedative activity and can *improve* psychomotor and cognitive functions. Imipramine (Trofranil) is known to have a moderate sedative activity (Julien, 2001; Solanto et al., 2001).

[9] Two of the participants excluded because of medication taking (#18 and #19) also had strong family histories of bipolar disorder.

To exclude all of these participants (5 of the remaining 17, after excluding the children on psychotropic medication and those with ADHD-I) would seriously compromise the power of the study. The decision was made to retain those with LDs (n = 3) largely for reasons of external validity and generalizability, because a high percentage of children with ADHD have a comorbid LD (Faraone et al., 1998, recently reported that approximately 20% of children with ADHD in a clinically referred sample had a comorbid LD; other reported rates vary depending on defining criteria, Plizka et al., 1999). The participant with comorbid CD was also retained. CD is also highly comorbid with ADHD (reported rates range from 20 to 60%; see Barkley, 1998; Faraone, et al,. 1998). Pennington and Ozonoff's (1996) review, demonstrating that deficits in executive function are more closely linked to the presence of ADHD in those with comorbid CD, and are not present in children with CD alone, suggests that to include this participant should not seriously challenge the internal validity of the study. In contrast, the participant with comorbid, untreated, Tourette's Disorder was excluded because of motoric deficits in this disorder, because two of the tasks required subtle eye movement control, and because the existing literature on executive and inhibitory control deficits in this population is inconsistent (see Pennington & Ozonoff, 1996; Ozonoff, Strayer, McMahon, & Filloux, 1998).

Child Behaviour Checklist for Ages 4-18 (CBCL/4-18)

The CBCL/4-18 (Achenbach, 1991a) is a comprehensive child behaviour rating scale for parents, designed to screen for symptoms of the major dimensions of child psychopathology. This widely used scale has good test-retest reliability, good internal consistency, and good construct, concurrent, discriminant, and criterion-related validity (Achenbach, 1991a; Barkley, 1998).

On the main part of this measure, the Problem Scales, parents are asked to indicate on a three-point scale (Not True, Somewhat or Sometimes True, or Very True or Often True) to what extent their child displays behaviours or characteristics that are typical of children with a wide range of internalizing and externalizing disorders. Scores on the 118 items are grouped to yield eight empirically derived narrow-band behaviour problem scales (Withdrawn, Somatic Complaints, Anxious-Depressed, Social Problems, Thought Problems, Attention Problems, Delinquent Behaviour, and Aggressive Behaviour), and three composite broad-band scales (Internalizing, Externalizing, Total Problems). A T-score > 70 (which corresponds to > 98th percentile) on a given scale indicates clinical significance, relative to a normative sample of peers (there are separate norms for ages 4 to 6, 6 to 11, and 12 to 18, each split by gender).

To screen for the presence of clinically significant emotional and behavioural problems among children in the control group, a more conservative

cut-off of T>65 (93rd percentile) on any subscale was applied. There is disagreement about what cut-off is best for identifying children with ADHD using the Attention Problems subscale of the CBCL (see Eiraldi, Power, Karustis, & Goldstein, 2000). Other rating scales that are more specific to the DSM-IV criteria of ADHD will be used for confirmation of ADHD group status.

Disruptive Behavior Disorder Parent Rating Scale (DBD-P)

The DBD-P (Pelham, Gnagy, Greenslade, & Milich, 1992) consists of 45 items designed to assess children's symptoms of DSM-IV (APA, 1994) ADHD subtypes, ODD, and CD.[10] Parents are asked to indicate on a 4-point scale (scored 0 to 3) to what degree their child displays certain behaviours (Not at All, Just a Little, Pretty Much, Very Much). Both categorical and dimensional scores can be generated. The former will be used for exclusion criteria, the latter will be reported when describing the sample.

Using the categorical method, to meet criteria for ADHD-H or ADHD-I, six or more symptoms of hyperactivity-impulsivity or inattention, respectively, must be endorsed (i.e., rated as Pretty Much or Very Much on the parent scale, the teacher scale, or both scales combined). To meet criteria for ADHD-C, six

[10] The Disruptive Behavior Disorder Rating Scale was originally designed to assess teacher ratings of DSM-III-R (APA, 1987) symptoms. It has since been updated to assess DSM-IV symptoms of disruptive behaviour disorders. The updated version of the teacher scale has been found to possess good internal consistency and has been used to classify children into DSM-IV groups (Waschbusch, Willougby, & Pelham, 1998)

or more symptoms of inattention and six or more symptoms of hyperactivity-impulsivity must be endorsed. Endorsement of four symptoms of ODD or three symptoms of CD would indicate that the participant met criteria for these disorders on this scale.

Three factor or dimensional scores can also be generated by computing an average score from nine Inattention items, nine Impulsivity/Overactivity items, and eight Oppositional/Defiant items. Norms and cut-off scores are available only for teacher ratings of DSM-III-R (APA, 1987) symptoms (Pelham et al., 1992). Factor scores on the revised DBD scale will not be used to exclude participants, but they will be used as a continuous measure of DSM-IV symptoms to describe the sample.

Pittsburgh Modified IOWA Conners Parent Rating Scale (CPRS-PM)

The CPRS-PM (Pelham, Milich, Murphy, & Murphy, 1989) is a 23-item scale designed to measure behaviours that are typical of children with externalizing behaviour disorders. Parents are asked to rate each item on a 4-point scale (scored 0 to 3) referring to the extent to which the item describes their child (Not at All, Just a Little, Pretty Much, Very Much).

Scores from five of the items (e.g., "Fidgeting") are summed to yield an Inattention/Overactivity (IO) score. Scores from five other items (e.g., "Uncooperative") are summed to yield an Oppositional/Defiant (OD) score. Scores from seven items (e.g., "Bossy") are summed to yield a Peer Interaction

Score. Scores from 10 items taken directly from the Abbreviated Conners Rating Scale (Goyette, Conners, & Ulrich, 1978) can also be summed to yield an Abbreviated Conners score. The cut-off scores recommended by Pelham et al. (1989) are referred to, but used with caution, because norms are only available for teacher ratings up to grade 5.

Pittsburgh Impairment Rating Scale: Parents (IRS-P)

On the Pittsburgh IRS-P (Pelham, Gnagy, Waschbusch, et al., 1996), parents rate the extent to which a child's difficulties affect six specific areas of his or her functioning (peer relationships, sibling relationships, parent relationships, family relationships, academic performance, and self esteem), as well as a single rating regarding his or her overall functioning. Answers are rated on a continuous graphic rating scale ranging from "No problem/Definitely does not need treatment or special services" to "Extreme problem/Definitely needs treatment or special services". These ratings are converted to a number from 0 to 6.

A DSM-IV diagnosis of ADHD requires that a child's symptoms cause impairment in at least two areas of functioning. As such, this measure is a good complement to the ratings of symptom frequency. Norms for this measure are currently available only up to grade 5. As an alternative to norm-based cut- offs, absolute values were chosen to reflect the presence or absence of significant impairment. Participants in the control group were excluded if any single rating

exceeded 3 on the 7-point (0 to 6) scale, and those in the ADHD group were excluded if they did not have ratings of above 3 on at least two items. When describing the main sample, the single rating pertaining to overall functioning will be reported.

Participant information provided by teachers

Parents were asked to give a package of child behaviour rating scales to their child's teacher. As with all components of the study, this was voluntary. That is, parents chose whether they were willing to ask the child's teacher to be part of the study. If so, the parents signed a release of information form to provide to the child's teacher (see Appendix G). With the teacher package was a letter that described the study and provided a rationale for requesting the teacher's involvement (see Appendix H). The teacher's involvement was also voluntary. Each teacher was asked to complete four rating scales (each described below), and to rate the student's behaviour when he or she was off psychostimulant medication, if applicable.

The percentage of usable teacher questionnaires in the total sample was low (27 of 52 or 51.92% overall; 17 of 29 or 58.62% and 10 of 23 or 43.48% in the control and ADHD groups, respectively). Three completed packages were unusable because in each case, the teacher reported that since the child was always on stimulant medication when at school, he or she was only able to rate the child's behaviour when on medication. Twenty-seven packages were not

returned, or were not given to the teacher. Reasons for this included: the unavailability of a teacher familiar with the child's behaviour, the completion of the study during the summer with no access to a suitable respondent, the participant choosing not to ask teachers to be part of the study, and the teacher choosing not to complete the packages. With such a low rate of usable questionnaires, the pros and cons of using this information to exclude participants were considered extensively. A conservative approach was chosen: even though useable teacher ratings were only available for half of the participants in the total sample, teacher ratings could be used to exclude a participant.

Teacher's Report Form for Ages 5-18 (TRF)

The TRF (Achenbach, 1991b) is similar in use and in format as the CBCL, except that it is designed to be rated by teachers, and accordingly, the items differ slightly to assess classroom behaviour. Scores on the 118 items are grouped to yield scores on the same eight narrow-band and three broad-band scales as the CBCL. Also like the CBCL, a T-score of >70 (>98th percentile) on any scale indicates clinical significance, relative to a normative sample of similar age and same-sex peers. To screen for the presence of clinically significant emotional and behavioural problems among children in the control group, a cut-off of T>65 (93rd percentile) on any subscale was applied.

Disruptive Behavior Disorder Teacher Rating Scale (DBD-T)

Like the parent version of this scale, the DBD-T (Pelham et al., 1992) is a 45 item rating scale, which surveys teachers about the extent to which the child displays DSM-IV symptoms of ADHD, ODD, and CD (from "Not at all" to "Very Much"). As with the DBD-P, categorizations of ADHD-I, ADHD-H, ADHD-C, ODD, and CD are based on the number of endorsed symptoms, and three factor scores (Inattention, Impulsivity/Overactivity, Oppositional/Defiant) can be generated for a continuous rating. The former will be used for confirmation of group status, the latter will be used to describe the final sample.

Pittsburgh Modified IOWA Conners Teacher Rating Scale (CTRS-PM)

The items and format of the CTRS-PM (Pelham et al., 1989) are identical to those on the CPRS-PM described above. Four scores can be generated: Inattention / Overactivity (IO), Oppositional / Defiant (OD), Peer Interaction, and Abbreviated Conners. The cut-off scores recommended by Pelham et al. (1989) are be referred to, but used with caution because norms are only available for teacher ratings up to grade 5.

Pittsburgh Impairment Rating Scale: Teachers (IRS-T)

The IRS-T (Pelham et al., 1996) is identical in form and purpose to the IRS-P. Teachers rate the extent to which a child's difficulties affect five specific areas of his or her functioning (peer relationships, relationship with the teacher,

academic performance, functioning in the classroom, and self esteem), and his or her overall functioning on a single rating from "No problem/Definitely does not need treatment or special services" to "Extreme problem/Definitely needs treatment or special services".

As with the IRS-P, participants in the control group were excluded if any single rating exceeded 3 on the 7-point (0 to 6) scale, and those in the ADHD group were excluded if they did not have ratings of 3 or above on at least two items. When describing the main sample, the summary rating pertaining to overall functioning will be reported.

Confirmation of group status based on rating scale data

Children in the control group were excluded if they showed evidence of clinically significant behaviour problems based on the criteria detailed above and in Table 2.2. They were excluded if they had ratings greater than T = 65 on the CBCL and TRF, if a clinically significant number of symptoms of ADHD-I, ADHD-C, ADHD-H, ODD or CD were endorsed on the DBD-P or DBD-T, if their ratings exceeded recommended cut-offs (Pelham et al., 1989), on the IO or OD scales from the CPRS-PM or CTRS-PM, or if they had any impairment ratings greater than 3 on the IRS-P or IRS-T.

Based on these criteria, three children from the control group were excluded. Participant #26 had elevated ratings on the TRF, DBD-T (ADHD-H), and IRS-T. This participant was also excluded because he had received

psychological intervention for behavioural problems. Participant #28 was excluded based on elevations on the TRF, the DBD-P (ADHD-I), the DBD-T (ADHD-C), the IRS-P, and the IRS-T. Participant #47 was excluded based on elevations on the TRF, the CTRS-PM-IO, the DBD-T (ADHD-I), and the IRS-T.

ADHD group status was confirmed by categorical ratings on the DBD-P and DBD-T, requiring endorsement of a significant number of ADHD-H or ADHD-C symptoms by either parents or teachers, and at least two significant (greater than 3) ratings on the IRS-P or IRS-T. Five children from the ADHD group were rated on the DBD-P as being ADHD-I, and one had only sub-clinical ratings on the DBD-P. Each of these children was also excluded based on either medication taking (#'s 18, 19 and 35), ADHD-I (#'s 18, 37, 49), or comorbid Tourette's disorder (# 38). All remaining ADHD children had significant symptom and impairment ratings on the DBD-P and IRS-P, respectively, and when available, on the DBD-T and IRS-T.

The majority of the remaining participants in the ADHD group also had elevations on the CPRS-PM-IO and, when available, on the CTRS-PM-IO. This is with the exception of two (#'s 5 and 11) and three (#'s 10, 11, and 30) participants whose ratings were below the clinical cut-offs on the CPRS-PM-IO and CTRS-PM-IO, respectively. Since the DBD Rating Scales are more closely tied to the DSM-IV criteria for ADHD, and since the norms for the CPRS-PM

and CTRS-PM only go up to grade 5, more weight will be given to the confirmation of group status based on these measures.

Additional screening tests

Each participant completed four additional screening tests to measure their estimated IQ, handedness, colour vision, and word decoding ability.

Estimated IQ

All participants completed the Vocabulary and Block Design subtests of the Wechsler Intelligence Scale for Children – Third Edition (WISC-III, Wechsler, 1991). According to Sattler (1992), this is the most reliable two-subtest short form of the WISC-III for estimating Full Scale IQ. On the Vocabulary subtest, children are asked to define a series of words and their responses are scored from 0 to 2 based on accuracy. Block Design is a timed subtest on which children are asked to put together a set of red and white blocks to match the design on a picture card. Scores are given for both accuracy and speed. On both tests, raw scores are converted to standard scores based on age. Using the procedures described in Sattler's (1992) Appendix L, the standard scores on the two scales were used to create estimated Full Scale IQ scores, based on Canadian normative data (Wechsler, 1996). Participants with an estimated IQ < 80 were excluded. None of the 52 children tested met this criterion.

Modified Edinburgh Handedness Questionnaire

On the Modified Edinburgh Handedness Questionnaire (Oldfield, 1971) participants are asked to indicate which hand they prefer to use for each of 14 different tasks (e.g., drawing, throwing, brushing teeth). In order to minimize the likelihood that younger participants might confuse their verbal or written responses, the questionnaire was modified into a performance-based task. Participants were first asked to demonstrate or pretend they were doing each task, and the experimenter wrote down which hand they used in the demonstration. The experimenter then showed the participant the questionnaire, explained its purpose, and asked the child to validate that the answers were representative of their hand preference. The proportion of tasks done with each hand determines hand dominance.

Ishihara Test for Colour-Blindness

The Stroop Colour Word Test (Chapter 4) requires that the participants name the colour in which words are presented. Because young colour blind children often go undetected, it was important to assess colour vision in each participant. The Ishihara Test for Colour Blindness, 38 Plates Edition (Ishihara, 1980) consists of a series of 38 plates with differently-coloured dots on them, presented one at a time. The participant is required to identify what is seen within about 3 seconds of presentation. Individuals with and without colour deficiencies are able to identify different patterns among the dots. For the

majority of cards, those with normal colour vision see one digit whereas those with red-green colour deficiencies see a different digit, and those with total colour blindness see no digit. In other cases, those with normal colour vision see a digit when those with red-green deficiency cannot, and vice versa. To detect the presence of a colour-deficiency in individuals who can read numbers, only the first 21 plates are required, so only these were administered for the purposes of this study. Using this screening measure, none of the 52 participants in this study were identified as having colour deficiencies.

Wide Range Achievement Test-3 (WRAT3, Wilkinson, 1993)

The Stroop Colour Word Test also involves word reading. In order to be able to determine whether word-decoding ability is correlated with task performance, word decoding was assessed using the Reading subtest of the Wide Range Achievement Test – 3 (WRAT3; Wilkinson, 1993). On this test, a series of words with increasing difficulty is presented to the participant on a single card,[11] and the child is instructed to read each word aloud. The raw score is the number of items read correctly. Raw scores can then be converted into absolute scores (based on an interval scale regardless of age) and standard scores (based on age).

[11] There are two equivalent test forms available. The Blue test form was used for this study.

Summary of the application of exclusion criteria

The goal of this study is to determine whether there are differences on measures of inhibitory control between children with ADHD-C or ADHD-H (hereafter referred to as ADHD[12]) and a non-clinical control group. To achieve good internal validity, relatively strict criteria were applied to the formation of both groups. With sensitivity to both power and generalizability, however, children with ADHD and a comorbid LD (n = 3) or CD (n = 1) were retained.

Of the 52 children who participated in the study, 12 were excluded. The reasons for exclusion are highlighted in Table 2.2. Five of the 29 children (17.2%) initially in the control group were excluded: 3 for evidence of externalizing behaviour problems, one because of a history of seizures, and one because of the unavailability of behaviour ratings. Seven of the 23 children (30.4%) initially in the ADHD group were excluded: 2 because they had ADHD-I, 1 because of comorbid Tourette's Disorder, and the remaining 4 because they were taking psychotropic medication that they were not able to withhold for the duration of the study.

There are not enough participants of any one type (e.g., ADHD-I, Tourette's, or taking a particular type of medication) to properly explore

[12] The inhibitory control model applies to children with both ADHD-H and ADHD-C. For the purposes of this study, no distinction is made between these two subtypes.

patterns in subgroups. From this point forward, only the remaining 40 participants will be discussed.

Table 2.2. Characteristics of the total (n = 52) sample and reasons for exclusion split by control (A) and ADHD (B), and sorted by location, sex, and order of participation.

A. Control (n =29, 22 male and 7 female)

ID #	Loc.	Sex	Age	Teacher forms?	Reason for exclusion if applicable (in italics), and other potentially pertinent information
2	ON	M	10:5	Y	
3			12:3	Y	Aortic valve malfunction, 1 year reading and spelling resource
6			9:7	Y	
7			11:8	Y	2 months writing resource
8			11:4	N	
13			9:1	Y	Asthma
15			12:3	Y	
21			13:3	N	
24			12:3	Y	
25			13:1	Y	1 year speech therapy
1		F	11:1	Y	
14			9:8	Y	Med: Flonase for asthma
20*			10:9	Y	*Seizures ages 2-7 years,* 4 months speech therapy, congenital heart problem, pacemaker
26*	NS	M	10:11	Y	*Psychotherapy for behaviour problems, Elevated ratings on the TRF, DBD-T (ADHD-H), and IRS-T*
27			9:4	N	
28*			12:4	Y	*Elevated ratings on the DBD-P (ADHD-I), IRS-P, TRF, DBD-T (ADHD-C), and IRS-T*
32			10:11	N	1 year reading resource
33			13:0	Y	
36			11:1	N	
43			11:11	N	
47*			9:8	Y	*Elevated ratings on the TRF, CTRS-PM-IO, DBD-T (ADHD-I), and IRS-T*
48*			11:5	N	*Parent forms not returned*
50			11:8	N	
51			10:4	N	
52			12:7	N	
39		F	12:6	Y	
40			9:11	Y	
44			13:11	N	
46			12:3	N	

B. ADHD (n = 23, 17 male and 6 female)

ID #	Loc.	Sex	Age	Teacher forms?	Reason for exclusion, if applicable, and other potentially pertinent information
4	ON	M	9:0	Rated on meds	CD, special class for behaviour problems, speech therapy, S
5			12:1	N	S
9			9:7	Y	LD, 2 years special class for learning needs, 2 years resource, asthma, S
10			13:0	Y	S
11			12:5	Y	Nonverbal LD, resource for 3 years, psychotherapy, S
12			11:7	Y	Med: oxybutynin for renal reflux, S
17			12:2	N	S
22			10:11	N	S
23*			13:1	Y	*Med: venlafaxine*, special class for behaviour problems, history of OCD, psychotherapy
16		F	10:3	Y	3 years resource for organizational skills and math, psychotherapy, S
18*			10:7	Rated on meds	*Med: chlorpromazine, ADHD-I, No elevations on DBD-P, S*, Family history of bipolar disorder
19*			9:7	N	*Med: haloperidol, Rated ADHD-I on DBD-P*, S, Family history of bipolar disorder
29	NS	M	9:4	Y	S
31			10:8	Y	S
34			10:5	N	Med: Nasocort for asthma, LD, 3 years resource for reading and writing, psychotherapy, S
35*			12:9	N	*Med: imipramine, Rated ADHD-I on DBD-P*, psychotherapy
38*			11:6	Y	*Tourette's Disorder, Rated ADHD-I on DBD-P*, asthma
41			10:8	N	2 years reading resource, S
45			11:7	N	4 years resource all subjects
49*			12:9	N	*ADHD-I, Rated ADHD-I on DBD-P*, LD, 5 years resource all subjects
30		F	13:10	Y	S
37*			12:9	Rated on meds	*ADHD-I, Rated ADHD-I on DBD-P*, 2 years reading resource, S
42			11:2	N	Psychotherapy, S

Note. ID # refers to the participant identification number. "Loc." refers to the testing location. M refers to male and F to female. Age is presented as years:months. In "Teacher forms?", Y indicates teacher rating scales were returned, N indicates they were not, and "Rated on meds" means that the teacher rated the child's behaviour while the child was on a stimulant medication. "Med" refers to medication (other than a stimulant) that the child was taking. "S" indicates that the child usually takes a stimulant medication. "Psychotherapy" refers to therapy for behaviour problems. Participants excluded from the main analyses are identified with an asterisk, and the reasons for the exclusion are in italics.

Characteristics of the final sample

The final sample includes 24 control children and 16 children with ADHD. The majority of the data is analyzed with repeated measures designs. With two-tailed alpha = .05, the power (1-Beta) to detect differences with medium and large effect sizes using a repeated measures design would be .67 and .95, respectively. Some of the data is analyzed with unpaired t-tests on non-repeated measures. For these analyses, the power to detect differences with medium and large effect sizes would be .33 and .67, respectively.[13]

There are more males (n = 13; 81%) than females (n = 3; 19%) in the ADHD group, reflecting the greater prevalence of ADHD among males. The same pattern is true in the control group, with more males (n = 18; 75%) than females (n = 6; 25%), reflecting the attempt to match the groups for gender prior to application of the exclusion criteria. There is no difference in the proportion of males and females in each group, $\chi^2(1, N = 40) = 0.22, p = .64$. The average age of children in the control group is 11 years, 5 months (range: 9 years, 1 month to 13 years, 11 months). The average age in the ADHD group is 11 years, 2 months (ranging from 9 years, 0 months, to 13 years, 5 months). There is no difference in the age (in months) between the two groups, $t(38) =$

[13] The software program G Power (Buchner, Faul, & Erdfelder, 1997) was used to compute power.

-0.70, $p = .49$. The parent and teacher ratings and scores on the screening measures for each group are presented in Table 2.3.

Table 2.3. Behaviour ratings and screening measures for the final sample: Mean scores and group differences.

Measure	Control (n = 24)		ADHD (n = 16)		p
	Mean	SD	Mean	SD	
CBCL Attention Problems[a]	50.83	2.60	69.75	9.73	<.0001
CBCL Internalizing[a]	42.58	7.12	58.44	9.61	<.0001
CBCL Externalizing[a]	36.29	6.61	61.88	8.59	<.0001
DBD-P Inattention[b]	0.28	0.42	2.67	1.43	<.0001
DBD-P Impulsivity[b]	0.13	0.21	1.85	0.52	<.0001
DBD-P Oppositional[b]	0.15	0.29	1.45	0.54	<.0001
CPRS-PM Abbreviated[c]	1.88	2.03	18.44	4.50	<.0001
CPRS-PM IO[c]	1.54	1.77	10.94	2.27	<.0001
CPRS-PM OD[c]	0.88	1.30	9.31	3.67	<.0001
CPRS-PM Peer[c]	0.69	2.84	1.17	0.56	= .51
IRS-P[d]	0.04	0.20	4.19	1.76	<.0001
TRF Attention Problems[a]	52.15	4.14	66.25	12.41	< .01
TRF Internalizing[a]	41.23	5.93	52.13	8.03	< .01
TRF Externalizing[a]	45.46	5.84	62.13	8.48	< .001
DBD-T Inattention[b]	0.60	0.72	2.06	0.82	< .001
DBD-T Impulsivity[b]	0.41	0.49	1.62	0.74	< .001
DBD-T Oppositional[b]	0.08	0.13	0.66	0.62	< .01
CTRS-PM Abbreviated[c]	4.54	4.52	13.29	4.11	<.001
CTRS-PM IO[c]	3.54	3.15	9.14	2.85	< .01
CTRS-PM OD[c]	0.39	0.65	3.00	2.52	< .01
CTRS-PM Peer[c]	0.11	0.15	0.84	0.76	< .01
IRS-T[d]	0.23	0.44	3.14	1.77	< .0001
WISC-III Est. IQ (SS)	115.29	18.28	104.75	13.09	= .06
WRAT-3 Reading (SS)	107.58	12.56	103.50	15.23	= .36
WRAT-3 Reading (AS)	510.21	9.94	506.06	14.61	= .29

Note. Scores are expressed as [a]T scores, [b]Factor scores, [c]Total scores or [d]Overall ratings; WISC-III Est. IQ = Wechsler Intelligence Scale for Children-III IQ estimated from the Vocabulary and Block Design subtests; SS = Standard Scores; AS = Absolute Scores; Rating scales completed by parents were available for all participants. The TRF and DBD-T were available for 21 participants, and the CTRS-PM and IRS-T were available for 20 participants. The final column contains the p values from unpaired t-tests.

Not surprisingly, parent and teacher ratings were higher for children in the ADHD group (as compared to controls) on each of the rating scales and subscales, with the exception of one (Peer Interaction ratings on the CPRS-PM). This confirms that the parents and teachers of children in the ADHD group do, in fact, see these children as more hyperactive, inattentive, and impaired than do the parents and teachers of those in the control group. Children in the ADHD group are also rated as having more overall internalizing and externalizing behaviours. This is a common finding, reflecting both comorbidity, and the impact of ADHD on these broader domains of functioning (e.g., Faraone et al., 1998; Plizka et al., 1999).

The mean estimated IQ of the ADHD group was in the Average range (104.75, SD = 13.09), and that of the control group was in the High Average range (115.29, SD = 18.28). A t-test revealed a marginally significant group difference: $t(38) = -1.99, p = .06$. This difference should be interpreted with caution. Only two subtests of the WISC-III (Vocabulary and Block Design) were used as an approximate measure of IQ, to rule out participants with borderline or intellectually deficient functioning. Although these scores are significantly correlated with Full Scale IQ ($r = 0.91$, Sattler, 1992), they are not an exact representation of IQ. Also, though the ADHD children did score in the average range, they were not taking their stimulant medication during testing, and this may have adversely affected their performance. Furthermore, it

is not unusual to find a group difference of 7 to 15 IQ points between children with ADHD and controls when the full WISC-III is administered (see Barkley, 1998, for a brief review). This difference has been attributed to differences in test-taking style (e.g., impulsivity), to real differences in "g" (general factor or general ability), or to comorbid LDs. For this sample, the mean IQ of the three participants with ADHD and a comorbid LD was 100.00 (SD = 10.82), versus 105.85 (SD = 13.70) for the 13 without a comorbid LD. When the three participants with comorbid LDs are removed, the t-test comparing the groups is not significant.

Overall, the estimated intelligence of the sample (Mean =111.98, SD = 17.23) is higher than one would expect in the general population. Environmental variables may have contributed to this high estimated IQ. Thirty-eight of the forty (95%) reported the highest education level of one or both parents. Of the 37 fathers, only one (2.70%) had not completed high school, 16 (43.24%) had completed some or all of university or college, and 11 (29.73%) had completed a post-graduate degree (MSW, LLB, MBA, MD, or PhD). Of the 37 mothers, all had completed high school, 26 (70.27%) had completed some or all university or college, and 6 (16.22) had completed a post-graduate degree. There were no group (ADHD versus control) differences in the highest level of education of the fathers, $\chi^2(3, N = 37) = 4.98, p = .17$, or mothers, $\chi^2(2, N = 37) = 1.76, p = .42$.

This is obviously a highly educated sample, probably reflecting, in part, the fact that many of the participants were associated with Queen's and Dalhousie Universities, and perhaps also because of a self-selection bias: those who are interested in the process of scientific research may be more likely to volunteer to participate. While the high parental education of this sample can help to explain the high IQ estimates found here, Pennington and Ozonoff's (1996) review suggested that the executive function deficits present in ADHD (including deficits of inhibitory control) appear to be independent of socio-economic variables.[14]

The majority of the participants (27 or 67.5%) lived with both of their biological parents. Eight (20%) lived with their biological mother only, one (2.5%) lived with two adopted parents, and four (10%) lived with their biological mother and stepfather. There were no group (ADHD versus control) differences in family constellation, $\chi^2(3, N = 40) = 4.06, p = .25$.

The majority of the participants were right-handed; 2 in the ADHD group and none in the control group were left-handed.

[14] In addition to parental education, family income and type of parental employment were collected as indicators of socio-economic status. These variables were not included in the analyses because of the lack of an adequate measure of employment ratings for the current Canadian job market, and because the majority of parents chose not to report family income.

The WRAT-3 Reading subtest score was in the average range for both groups (see Table 2.3) and there was no difference in performance between the groups, $t(38) = -1.07, p = .29$.

The medication taken by the participants in the final sample is described in Table 2.4. At the beginning of each session, the parent accompanying the participant completed a short medication record (created for this study, see Appendix I) to indicate when the child had last taken his or her medication. Participants had been asked to refrain from taking psychostimulant medication for 24 hours prior to testing. The reported range was longer, between 23 and 123 hours (Mean = 44.07, SD = 24.71, Median = 41). In many cases, participants had been off their medication for weekends or holidays at the time of testing.

Table 2.4. Medications taken by participants in the final sample.

ID #	Group	Sex	Medication	Type	Reason prescribed	Dose and regimen
14	Control	F	Fluticasone propionate (Flonase)	intranasal steroid	Asthma	PRN
4	ADHD	M	Methylphenidate (Ritalin)	psychostimulant	ADHD	07:30: 20 mg RR 12:00: 20 mg RR
5	ADHD	M	Methylphenidate	psychostimulant	ADHD	07:30: 20 mg SR
9	ADHD	M	Methylphenidate	psychostimulant	ADHD	07:30: 15 mg RR 12:30: 15 mg RR 16:00: 10 mg RR
10	ADHD	M	Methylphenidate	psychostimulant	ADHD	07:30: 20 mg SR 07:30: 5 mg RR 16:00: 5 mg RR
11	ADHD	M	Methylphenidate	psychostimulant	ADHD	08:00: 30 mg RR 12:00: 30 mg RR
12	ADHD	M	Methylphenidate	psychostimulant	ADHD	07:00: 20 mg RR 12:00: 10 mg RR
			*Oxybutynin (Ditropan)	anticholinergic	Renal reflux	Once per day
17	ADHD	M	Methylphenidate	psychostimulant	ADHD	07:30: 60 mg SR
22	ADHD	M	Methylphenidate	psychostimulant	ADHD	08:00: 15 mg RR 12:00: 15 mg RR
29	ADHD	M	Methylphenidate	psychostimulant	ADHD	07:00: 10 mg RR 12:00: 10 mg RR
31	ADHD	M				07:15: 10 mg RR 12:00: 5 mg RR
34	ADHD	M	Methylphenidate	psychostimulant	ADHD	07:30: 5 ml 12:00: 5 ml
			*Triamcinalone (Nasocort)	intranasal steroid	Asthma	2 sprays per day
41	ADHD	M	Methylphenidate	psychostimulant	ADHD	07:15: 20 mg SR
16	ADHD	F	Methylphenidate	psychostimulant	ADHD	08:00 "1" SR 12:00 "1/2" SR
30	ADHD	F	Methylphenidate	psychostimulant	ADHD	07:00: 5 mg RR 12:00: 5 mg RR 16:00: 5 mg RR
42	ADHD	F	Methylphenidate	psychostimulant	ADHD	07:00: 20 mg SR 16:00: 5 mg RR

Notes: Participant order is the same as for Table 2.2 for consistency. RR = Regular Release; SR = Slow Release. PRN = as needed (pro re nata). Asterisks indicate medications that were taken on the day of testing.

General Procedures

Each one of the next five chapters (Chapters 3 to 7) will be dedicated to one of the five tasks completed. In the remainder of this chapter, the general methods of the study will be described. Each of the specific tasks will be detailed in the Procedures section of the corresponding chapter.

Testing locations. All participants were tested in one of two laboratories. Participants from Kingston, ON, were tested in the laboratory of Dr. D. P. Munoz in the Department of Physiology, Queen's University. Participants from Halifax, NS were tested in the laboratory of Dr. R. M. Klein in the Psychology Department, Dalhousie University. Twelve of the 24 participants in the control group and 9 of the 16 in the ADHD group were from Kingston.

In each location, testing took place in two different, quiet, well-lit rooms. One of the two rooms was used only for the tasks for which eye movements were monitored, and the other room was used for all other tasks. Parents either dropped their children off or waited in a nearby waiting area.

Time of testing. Testing took place in three separate sessions, each 90 minutes in length. The time between testing sessions was typically one week, but ranged between 3 days and 42 days (Mean = 10.35, SD = 7.60, Mode = 7, Median = 7). There was no group difference in the number of days between testing sessions, $t(38) = 0.58, p = .57$.

The time of day and day of the week were both largely determined by participant convenience. The majority of the appointments for all participants were scheduled on weekends and school holidays. Time of day varied from 08:30 to 18:30 (start time). Of the 120 testing sessions, 52.5% of the sessions started in the morning (08:30 to 11:30), 19.2% started in the afternoon (12:00 to 15:30), and 28.3% started in the late afternoon (16:00 to 18:30). It was fairly common for parents of children in the control group to schedule sessions immediately after school: 30.6% (22 of 72) of the sessions for this group started in the late afternoon (between 16:00 and 18:30). In contrast, parents of children with ADHD generally chose not to schedule appointments at this time, reportedly so that the child did not have to be medication-free on a school day. Of the 48 sessions with children with ADHD, only 1 (2.1%) occurred in the late afternoon, and this was on a holiday. The group difference in the timing is reflected in a significant Chi square test, $\chi^2(2, N = 120) = 17.42, p < .001$.

Order of administration. The tests were administered in a fixed order, which is presented in Table 2.5. Screening measures were administered first. The two tests requiring eye movement monitoring (Inhibition of Return and Memory-Guided Saccade) were scheduled on two different days to avoid fatigue. Because some attrition across sessions was expected (fortunately, none occurred), the Stop Signal task, for which there was already considerable data in the literature,

was scheduled in the final session. The remaining tasks were scheduled according to administration time.

Table 2.5. Tests administered in each testing session and approximate time (in minutes).

Session 1	Time (min)	Session 2	Time (min)	Session 3	Time (min)
Screening: WISC-III Vocabulary and Block Design, WRAT-3 Reading, Ishihara Test of Colour Blindness	20	Memory-Guided Saccade Task	40	Stop Signal Task	25
		Edinburgh Handedness Questionnaire	3	Inhibition of Return	25
Simon – Flanker Task	25	Break	12	Break	15
Break	15	Memory-Guided Saccade Task	35	Inhibition of Return	25
Stroop Colour Word Task with Negative Priming	30				

A formal break (leaving the testing area for juice and cookies) was scheduled approximately half way through each session. Shorter rests occurred periodically throughout the testing session (e.g., when changing tasks, between sets on a given task).

Experimenter presence. The experimenter (the author) was present for all of the testing, and sat beside the participant for the duration of each experiment. There is some evidence to suggest that children with ADHD perform better when an experimenter is present, but that control children are not differentially affected by experimenter presence (e.g., Gomez & Sanson, 1994). In order to determine whether there is a group difference in inhibitory control, both groups should be functioning at their best. This avoids the possible alternative

hypothesis that group differences in performance would be due to experimenter absence.

It is important to note that neither the experimenter nor the participants were blind to group status. To avoid any impact that their expectations could have on performance, participants were not told the hypotheses of the study. The experimenter was not blind largely because of practical reasons. The principle investigator (the author) recruited participants, confirmed that participants were medication-free prior to testing, and reviewed the Parent Information Questionnaire to determine whether further information was needed from the parents. For the first few participants, the goal had been for the principal investigator to remain blind, and a research assistant scheduled all of the appointments. Within the first few participants it became evident that even without the practical disadvantages, it would be difficult to stay blind. Children with ADHD and their parents frequently made reference to ADHD, particularly in the context of being medication-free. Also, many of the characteristics of a child with ADHD off medication are very noticeable to an observer.

Several steps were taken to ensure that the experimenter's knowledge of group status did not adversely bias the results. The instructions for each task were scripted so that they could be delivered in a similar manner to all participants. Participants were told that the experimenter would not give them

feedback on the quality or accuracy of their performance, except for when they were learning each task (i.e., during practice trials). Participants were also told that the experimenter would not respond to off task conversation during a test, but that she would answer clarification questions about the task. If the participant was off task, the experimenter did verbally (e.g., "keep going", "keep your fingers on the keyboard") or nonverbally (by pointing at the computer screen) cue them to return to the task. Given that one of the hallmark characteristics of ADHD is an inability to remain on task over a prolonged period of time, it was recognized that there was a risk of giving more cues to the children in the ADHD group, thereby treating them differently from children in the control group. Consistent with this hypothesis, Byrne, DeWolfe, and Bawden (1998) demonstrated that preschoolers with ADHD required more experimenter instructions to stay on task during the administration of a test of language abilities than control children. For this study, the decision was made to give all children as many cues as they needed to stay on task, even though this could lead to differential treatment. Again, the goal was not to assess the participants' ability to stay on task, but to measure their performance once on task. Not providing sufficient cues could bias against ADHD children, and lead to group differences that are not specific to inhibitory control.

Point and reward system. In order to motivate children to complete 4.5 hours of testing, a point system was implemented. At the beginning of the first

session, each participant was told that he or she had the opportunity to earn 10 points per session (for a total of 30 points) that could be traded in for small prizes (e.g., pencils, collector cards, stickers). The majority of the points were awarded for simply completing a task, but others were awarded for certain behaviours, such as not touching the eye movement monitoring equipment, or staying in one's seat. The point system was designed to be reinforcing and motivating, and to support the experimenter's instructions to stay on task. All participants earned all points. At the completion of the study, each participant was given a certificate denoting points earned, and hours worked.

Behavioural observations. For each session, the experimenter made note of the participant's level of cooperation, arousal (i.e., if they appeared fatigued), motivation, and off task behaviour. Other variables that may have contributed to performance were noted, such as whether the participant had been at a sleepover the night before, or whether the participant reported having a cold. Though these observations were recorded on a standardized (i.e., identical for all participants) form, the form was created for the purposes of this study only, and there are no data on its reliability or validity. Also, even though the rater was a graduate student in clinical psychology with training in objective assessments, she was not blind to diagnosis, and this could have influenced some ratings. The ratings on these forms, therefore, were not used for any statistical analyses. Also, it is recognized that multiple factors could contribute

to a child's performance on any given day. The analyses focus on the presence or absence of ADHD.

Participants were also asked as part of the consent form if they would be willing to be videotaped during each session (see Appendix D). Consent to participate in the study was independent from consent to be videotaped. All parents gave written consent for their child to be videotaped, and all except two children gave their assent for videotaping. In principle, videotapes could be used to code participant and experimenter behaviour, and to validate coding of verbal responses (e.g., on the Stroop Colour Word task, WISC-III Vocabulary), although this was not found to be necessary.

Performance-based feedback and reinforcement. In addition to the inhibitory control theory of ADHD, other prominent theories refer to differential sensitivity or response to reinforcement, and the related construct of motivation (see Douglas, 1999; Nigg, 2000, 2001; and Sergeant et al., 1999 for reviews). Ideally, a study of inhibitory control in ADHD should try to be independent of, or control for, the effects of reinforcement. Therefore, for the majority of tasks, performance-based feedback, reward, or response cost were not given. This is with the exception of the two tasks involving eye movement monitoring, for which feedback regarding the position of the eyes is essential for task performance (more will be said on this in the relevant chapters).

It must be recognized, however, that for some of the tasks (e.g., the Stroop), children were likely aware of their performance, even if external feedback was not given. Although the data supporting the reinforcement-based theories of ADHD has been obtained largely with studies manipulating external reinforcement (e.g., Iaboni, Douglas, & Baker, 1995; Oosterlaan & Sergeant, 1998; Slusarek, Velling, Bunk, & Eggers, 2001), it is conceivable that any group differences in reinforcement mechanisms (if they exist) could also apply to internal monitoring. Also, as Nigg (2001) points out, while executive and motivational inhibition processes are distinct, they are related, and while it is possible to isolate executive inhibition for study, it is not possible to completely remove the influence of motivational inhibition.

Analytic strategy. The principle analyses will compare the performance of children with ADHD and control children on each measure. This will typically require a t-test or a mixed analysis of variance (ANOVA). Whereas both t-tests and ANOVAs are fairly robust in the presence of unequal sample sizes and deviations from normality, the issue of homogeneity of variance becomes more potentially important as the size of the groups becomes more discrepant (cf., Keppel, 1991; Tabachnick & Fidell, 2001). Most agree that sample sizes within a ratio of 4:1 (largest to smallest) are considered relatively equal (Tabachnick & Fidell, 2001). Here, the ratio is 24:16, or 1.5:1. Nevertheless, the assumption of equality of variance between groups will be assessed using an F-test, and results

will be mentioned when this assumption has been violated. According to Keppel (1991), equality of variance is violated when the ratio of the largest to the smallest variance (F_{max}) is greater than 3, although Tabachnick and Fidell (2001) argue that when the sample size ratio is less than 4:1 (as is the case here), an F_{max} of 10 is acceptable. Both caution that a large variance ratio is more problematic when the larger variance is associated with the group with the smaller sample size (in this case, the ADHD group). Because this could be the case, the more stringent F_{max} of 3 will be used to detect violations in this assumption. To correct for this, when applicable, a more stringent significance level will be adopted (following Tabachnick & Fidell, 2001).

Additional steps will be taken for mixed ANOVAs. Keppel (1991) argues that most ANOVAs with mixed designs, especially those with unequal sample sizes, violate the sphericity assumption. The F distribution assumes homogeneity of variances across levels of the repeated measure and homogeneity of correlations between pairs of levels of the repeated measure. In the case of mixed ANOVAs, there is the additional assumption of homogeneity of variances and correlations across levels of the between groups factor. Violations can lead to increases in Type 1 error on tests involving the repeated factor (the main effect of the repeated factor and the interaction between the repeated and between groups factors) but not on tests involving the non-

repeated (between groups) factor. The Geisser-Greenhouse correction is a procedure that assumes maximum heterogeneity. It involves changing the degrees of freedom so that the observed F value is evaluated against a larger critical F value. Keppel argues that while the uncorrected statistic is probably too lenient, the Geisser-Greenhouse correction is probably too strict (i.e., it overcorrects because it assumes maximum heterogeneity).

A sequence of steps based on Keppel's recommendations will be followed for all tests involving repeated measures. For each, the uncorrected F test will be reported. If the null hypothesis is rejected, the assumptions of homogeneity of variance and covariance will be assessed. The results will only be reported if these assumptions are violated. If this is the case, the F will be re-evaluated with the Geisser-Greenhouse correction.

An alpha level of .05 will be used for all statistical tests, except where noted.

Chapter 3. Stop-Signal Paradigm

As reviewed in Chapter 1, the inhibition of a prepotent response and the cessation of an ongoing response are two of the main types of response inhibition in Barkley's model (1997a; 1997b). In Nigg's taxonomy (2000; 2001), they are both subsumed under the category of Executive Motor Inhibition. For many years, response inhibition in ADHD was measured primarily via impulsive errors on the Matching Familiar Figures Test (e.g., DuPaul et al., 1992) or errors of commission on the Continuous Performance Task (see Corkum & Siegel, 1993 and Losier et al., 1996, for reviews). Errors on these tasks, however, are not necessarily due only to deficits in inhibition (see Schachar & Logan, 1990, for a brief discussion of this issue), and even if they were, these tasks do little to elucidate the mechanisms underlying the inhibitory deficits. Furthermore, these measures produce only a categorical variable, whether or not impulsive responses were made; time taken to stop cannot be assessed.

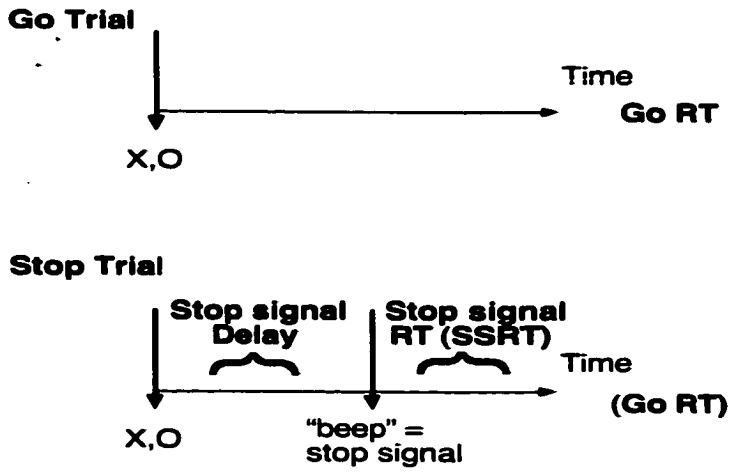
Logan and Cowan's (1984; see also Logan, 1994) mathematical race model of inhibition allows for the measurement of stopping time using a stop-signal paradigm, even though no overt response is made when inhibition occurs. In the typical stop-signal paradigm, participants are engaged in a choice reaction time task (e.g., discriminating between two visual stimuli and pressing one of two response keys as quickly as possible). On some proportion of trials

(typically 25%), they are given a signal (typically an auditory tone) to withhold their response. The "stop-signal delay", or the time between the onset of the visual stimulus and the onset of the stop-signal, varies. The ability to stop (rate of successful inhibition) is negatively correlated with stop-signal delay. If the stop-signal delay is short (i.e., if the stop-signal occurs immediately after the onset of the stimulus), it will be easier to inhibit a response. Conversely, if the stop-signal delay is long (i.e., the stop signal occurs just prior to the response, or just before the participant's typical reaction time on trials without a stop-signal), it will be much harder to stop a response. The race model presumes that on trials with a stop signal, a race ensues between processes related to "going" and to "stopping". The winner of this race determines the outcome (i.e., whether or not a response is made). Stop-signal reaction time (SSRT) can be estimated by measuring the reaction time (RT) on trials with no stop-signal ("go RT") and the probability of inhibiting a response on stop-signal trials with varying stop-signal delays.

The early stop-signal paradigms required considerable data and data analysis (e.g., plotting probabilities of inhibition). Later, Logan, Schachar, and Tannock (1997) validated a very straightforward method of determining SSRT, which requires fewer trials and calculations than the earlier stop-signal paradigms. They used a tracking algorithm, designed to lead to a 50% probability of inhibiting a response on stop-signal trials. With this algorithm, the

stop signal delay varies dynamically according to the performance on a given trial. If the response is successfully inhibited, the stop signal delay is shortened, and if an incorrect response is made, it is lengthened. According to the race model (see Logan, 1994), when the probability of inhibition is 50% (i.e., when the race is tied), the SSRT can be estimated by subtracting the average stop-signal delay from the average RT on "go" trials (those with no stop signal). See Figure 3.1 for a schematic representation of the relation between the three processes involved (go RT, stop signal delay, and SSRT).

Figure 3.1. A schematic representation of the elements used to calculate stop signal reaction time (SSRT) when the tracking algorithm is used to converge at a point of 50% probability of inhibition on stop trials.



Several studies outside of the ADHD literature have validated the stop-signal paradigm as a measure of response inhibition (see Logan, 1994). Performance on this task varies across the lifespan (e.g., Bedard, Nichols,

Barbosa, Schachar, Logan, & Tannock, 2002; Williams, Ponesse, Schachar, Logan, & Tannock, 1999) and is correlated with self-reported impulsive behaviour in young adults (Logan et al., 1997).

Studies using variants of the stop-signal paradigm have consistently shown that children with ADHD take longer to inhibit a response (i.e., have longer SSRTs) than control children (see Nigg, 2001 and Oosterlaan, Logan, & Sergeant, 1998 for reviews). Oosterlaan et al. (1998) conducted a meta-analysis of studies using the stop-signal paradigm in children with and without ADHD. Despite variations in methodology (e.g., number of trials, proportion of stop trials, type of primary task, sample characteristics) across studies, the pattern was homogeneous. Across seven studies, there was a medium combined effect size (Cohen's $d = .64$). SSRT was an average of 103 ms slower in children with ADHD as compared to controls. Both Nigg (1999) and Schachar, Mota, Logan, Tannock, and Klim (2000) replicated these earlier findings with a stop-signal paradigm using Logan et al.'s (1997) tracking algorithm in a population of children diagnosed with ADHD using DSM-IV (APA, 1994) criteria.

Because of the consistent pattern across studies, the simplicity of the task, and the specificity of this task to response inhibition, the stop-signal paradigm appears to be emerging as a standard for measuring inhibitory control in ADHD. It has been used to examine several issues relating to ADHD, including comorbidity (e.g., Oosterlaan et al., 1998; Schachar et al., 2000; Schachar,

Tannock, Marriott, & Logan, 1995), the effects of stimulant medication (e.g., Tannock, Schachar, & Logan, 1995), the delay aversion hypothesis (e.g., Solanto, Abikoff, et al., 2001), and comparisons with performance on other executive function tasks (e.g., Aman, Roberts, & Pennington, 1998).

The stop-signal paradigm was chosen for this study primarily to measure the time taken to inhibit a prepotent or ongoing response. Secondly, with such a strong body of literature showing that children with ADHD have slower SSRT's than control children, this paradigm was also used to compare this study (in particular, this sample) to the larger body of literature on ADHD. It was hypothesized that the children with ADHD would have longer SSRTs.

Method

Participants

The participants were the 40 children (24 control and 16 ADHD) described in Table 2.3.

Procedures

Task. The methods were adapted from Logan et al.'s (1997) stop-signal paradigm with tracking algorithm. Custom software was written by J. MacInnes for the purpose of this study to be run on a Macintosh laptop computer.

Children sat at a comfortable distance away from the computer screen and the viewing distance was not fixed. Based on the position of the laptop and external keyboard, they sat approximately 57 cm away from the screen. Two of

the keys ("z", located on the left side of the keyboard, and "/", located on the right side of the keyboard) were marked with stickers denoting them as "X" or "O" response keys. The coding was counterbalanced across participants so that half used their left hand for "X", and half used their right hand for "X".

All participants completed a practice block of 50 trials, followed by 8 experimental blocks of 50 trials, for a total of 400 experimental trials. The task took approximately 25 minutes to complete; the time varied slightly depending on the length of breaks between blocks.

Each block was initiated by pressing the computer mouse. After 500 ms, a brief fixation stimulus (a black dot) appeared in the centre of the screen, and it was followed by the target stimulus, which stayed on for 1000 ms. The inter-trial interval was 1500 ms, and the trials within a block ran continuously.

On each trial, one of two stimuli (a black uppercase letter "X" or "O" in Arial font) was presented in the centre of a computer screen with a light grey background. Each letter was approximately .8 by 1 visual degrees when viewed at a distance of 57 cm. The identity of the stimulus varied randomly, with the constraint that across the experiment, 50% were X's. The primary task was to press the response key corresponding to the stimulus (a choice reaction time task). The computer recorded RT and accuracy of the first response of each trial. No feedback was provided to the participant.

A 100 ms tone sounded on a random one quarter of the trials. The tone was the stop-signal that indicated to the participant that he or she was to withhold responding on that trial. The timing of the onset of the tone was relative to the onset of the stimulus. For the first trial, this "stop-signal delay" was set at 350 ms, meaning that the tone occurred 350 ms after the onset of the target stimulus. After this point, it varied dynamically depending on the participant's response on the previous trial. If the participant successfully inhibited on a stop-signal trial, the stop-signal delay was lengthened by 50 ms, making stopping more difficult on the subsequent stop-signal trial. If the participant was unable to inhibit his or her response on a stop-signal trial, the stop-signal delay was shortened by 50 ms, making it easier to stop on the subsequent trial. Neither accuracy of responses nor performance on trials with no stop signal affected the stop-signal delay. Over time, this "tracking algorithm" converges at a point at which the race between the stop and go processes are tied (Logan, 1994; Logan et al., 1997), or at a point where the probability of inhibition is approximately 50%.

Instructions to participants. Participants were told that they would be seeing one of two letters on the computer screen, and that they should press the corresponding key on the keyboard "as quickly as possible without making a lot of mistakes". They were instructed to keep their left hand near the left response key and their right hand near the right response key, and were given short

verbal cues during the task if they moved their hands away from the keyboard. They were told that on some of the trials they would hear a "beep", and that the "beep means stop", meaning that they should try to withhold responding on trials with a tone. To minimize the likelihood that they would use a strategy to avoid impulsive errors (such as waiting to ensure that there was no tone before initiating a response), they were told that they were not expected to successfully stop each time, and that they should not wait to see if the tone was going to happen. Performance was monitored closely during the practice block, and additional instructions were given if necessary.

Data Analysis. Trials with responses faster than 200 ms and slower than 2000 ms were excluded (this corresponded to 1.14% of the trials).

Following Nigg (1999) and Schachar et al. (2000), participants were excluded if their probability of inhibition was less than 20% or greater than 80%, if their accuracy on the primary task low (less than 66%), or if their SSRT was less than 50 ms. Data from one participant in the ADHD group (# 9) was excluded because his accuracy on the primary task was 42.04%. Data was unavailable for two other participants (one with ADHD and one from the control group, #'s 6 and 29).[15]

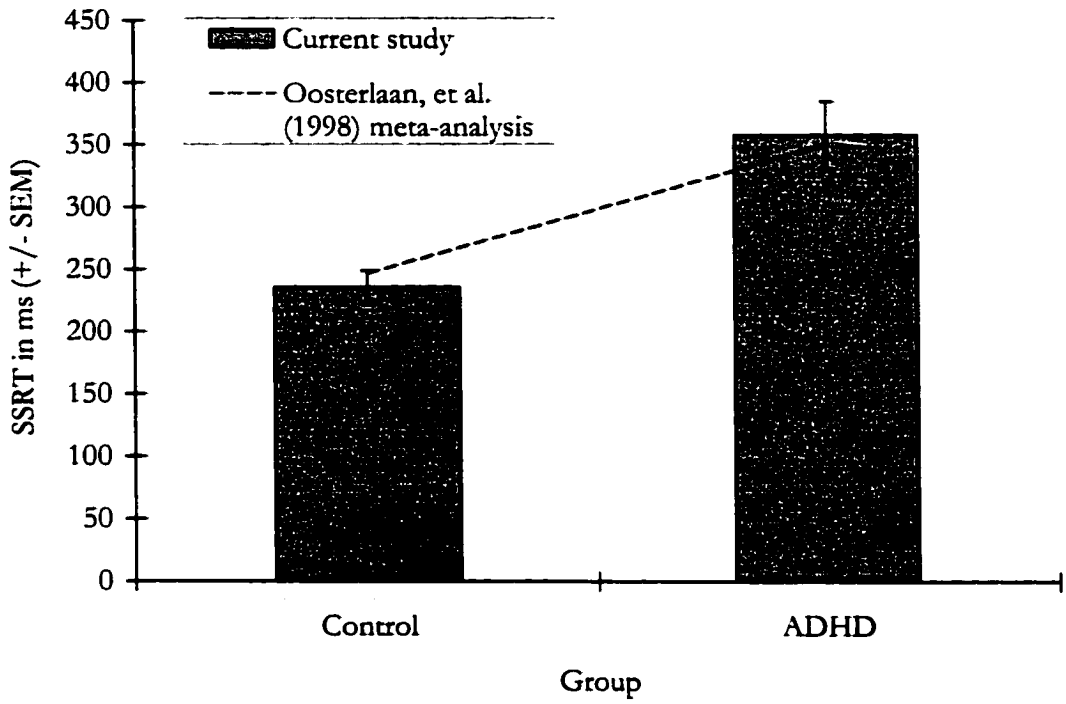
[15] Data from participant #6 was lost due to technical error. Data from participant #29 was lost because the laptop was stolen before the data was backed up.

When the probability of inhibition on stop trials is 50%, the SSRT for each participant can be obtained by subtracting the average stop-signal delay from the overall RT on no-tone trials (Logan, 1994; Logan et al., 1997). Across all trials for the 37 participants, the probability of inhibition was 50.18%, indicating that the tracking algorithm worked as expected. There was no difference in the probability of inhibition between groups (M = 49.51%, SD = 3.34, in the control group and M = 50.59%, SD = 2.50 in the ADHD group, $t(35) = 1.12, p = .27$).

Results

The main data from the stop signal paradigm is presented in Figure 3.2. There is a significant group difference on the primary variable of interest, SSRT, $t(35) = 4.54, p < .0001$. Children in the ADHD group have an average SSRT that is 121.79 ms longer than control children. The mean SSRT by group averaged across seven studies, as reported by Oosterlaan et al. (1998), is also represented on Figure 3.2 for comparison purposes.

Figure 3.2. Stop signal reaction time (SSRT) by Group.



The RT on the primary task was slower among children in the ADHD group, $t(35) = 2.58, p = .01$. The average "Go" RT was 641.53 ms (SD = 76.36) for children in the control group and 711.04 ms (SD = 84.83) for children in the ADHD group.

Overall mean accuracy on the primary task (on trials without a stop-signal) was 94.23% (SD = 4.44). Control children had higher accuracy (M = 95.61%, SD = 3.90) than ADHD children (M = 91.97%, SD = 4.45), $t(35) = 2.61, p = .01$.

Discussion

As expected, children in the ADHD group had longer mean SSRTs than those in the control group. This replicates previous findings with the stop-signal paradigm in general (e.g., Oosterlaan et al., 1998) and the tracking algorithm specifically (e.g., Schachar et al., 2000). To put this finding in context of the categories of inhibition defined in Table 1.1, children in the ADHD group showed a deficit in Barkley's (1997a) cessation of an ongoing response and inhibition of a prepotent response, and Nigg's (2001), Executive Motor inhibition.

The observation that children in the ADHD group had longer RTs on the primary task is a common finding (see Oosterlaan et al., 1998), and one that is consistent with the general pattern that children with ADHD tend to have longer and more variable RTs than controls (see Douglas, 1999, for a discussion of this issue). The majority of studies using the stop signal paradigm do not report accuracy on the primary task, because this is not the principle variable of interest. In one of the earlier studies, Schachar and Logan (1990) reported a nonsignificant trend toward more errors in the ADHD group. The higher error rate on the primary task in the ADHD group observed in this study may be an additional indication of impulsive responding.

Although the group difference in SSRT is not a new finding, it was important to establish in this study. By replicating a robust finding in the

literature, we can infer that the sample used in this study is comparable, at least in this one important way, to other samples of ADHD children.

Chapter 4. Stroop-Negative Priming Task

Another type of inhibition that is central to both Barkley's (1997a) response inhibition and Nigg's (2001) executive inhibition is interference control or response conflict. The majority of the evidence supporting deficiencies in interference control in children with ADHD comes from studies using the Stroop Colour Word Test (Stroop, 1935).

On the interference portion of this test, the participant is presented with a colour word written in coloured ink, and the task is to name the ink colour. For example, if the word "red" were written in blue ink, the correct response would be blue. To properly complete this task, the participant must ignore or inhibit the automatic reading of the interfering written word (although see MacLeod, 1991, for a discussion of other interpretations). The amount of interference is measured by comparing performance (RT and accuracy) on interference trials to trials with no interference (for example, naming the colour of non-words or reading colour words printed in black). Hundreds of studies have demonstrated the Stroop interference effect: longer RTs or more errors on interference trials (see MacLeod, 1991, for a review).

Children with ADHD are ubiquitously found to have increased reaction times in the interference condition relative to control children (for reviews, see Barkley, Grodinsky, & DuPaul, 1992; Nigg, 2001; Pennington & Ozonoff, 1996; and Sergeant, Geurts, & Oosterlaan, 2002). This is consistent with the

hypothesis that children with ADHD have more difficulty inhibiting the automatic reading of the written word than do controls. In cases for which reading ability is lower in the ADHD group, the interference effect is upheld, and the amount of interference is statistically unrelated to reading ability (e.g., Carter, Krener, Chaderjian, Northcutt, & Wolfe, 1995a).[16] There are a few findings, however, that suggest that more research is needed in this area.

Several studies (e.g., Barkley et al., 1992; Grodinsky & Diamond, 1992) have found that children with ADHD were also slower, relative to controls, on non-interference trials. These findings challenge the specificity of the interference control deficit on this task. They raise the question of whether the ADHD deficit consistently observed on this task is truly one of inhibition and interference, or whether it is due to a more global difficulty. As suggested by Barkley et al. (1992), "the poor performances on the Stroop test may be due to several factors... such as scanning, rapid naming, and general reading dysfluency" (p. 183). More research is needed to determine whether the observed deficits on the Stroop task are, in fact, in support of the inhibitory control theory of ADHD.

Much of the ambiguity in these results may be due to limitations inherent to the procedures. The majority of research conducted with children with

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[16] Note that preliterate children do not show a Stroop effect, and the development of the Stroop effect is correlated with the early development of reading ability (see MacLeod, 1991, for a review).

ADHD on the Stroop task has been with the presentation of different trial types in blocks or sets. That is, the child is asked to read a list of some number (e.g., 20) of colour words written in black on a single card. The time that it takes to complete this task is compared to the time taken to name the ink colour of the same number of non-word stimuli (e.g., sets of XXX's) on another card. The interference score is obtained by comparing these times with the time taken to name the ink colour of a set of incongruent colour words (i.e., 20 stimuli for which the written word is different from the ink colour).

There are many disadvantages to administering the Stroop task in this manner. First, it yields only one reaction time for each block or trial type. Therefore, if a child were distracted from the task for a few seconds, his or her performance on an entire condition would be affected. Secondly, Tzelgov, Henik, and Berger (1992) have shown that when participants are able to create an expectancy for a certain trial type, they can alter their response strategy accordingly. This kind of top-down influence would be more likely to occur when one trial type is presented per block. Given that children with ADHD may have problems with global executive function and maintaining a response set (see Pennington & Ozonoff, 1996), this in itself may contribute to group differences in performance. Thirdly, in a blocked presentation of stimuli, the rule ("say ink colour") must be held in working memory throughout the entire set. The ability to do this could also differ across groups. Fourthly, as

mentioned above, Barkley et al. (1992) noted that children may perform poorly on the Stroop task because of poor scanning ability or reading dysfluency. These variables would be most influential when the child is required to read long lists of words on a single card. Finally, individuals often react to making an error by increasing their reaction time on subsequent trials. Since errors are more likely on interference trials, the increased reaction time on a block of interference trials may be partly due to a slowing in response to making errors.

Changing the administration of the Stroop task to trial-by-trial, or "discrete trials" presentation could eliminate the influence of each of these factors. In this version of the Stroop task, colour words are presented one at a time on a computer screen. The reaction time to identify the "ink" (display) colour is calculated for each stimulus, rather than for a block of multiple stimuli. Interference or colour incongruent stimuli and non-interference stimuli are all mixed within a block. This procedure is clearly more sensitive than the blocked version. Firstly, the effects of off task behaviour or distractibility should be randomized across conditions. Secondly, the random presentation of stimuli should minimize the influence of top-down executive processes or response set on performance. Thirdly, the rule can be recalled into working memory prior to initiating each trial. Fourthly, because words are presented one at a time, the influence of any specific difficulties associated with scanning and reading lists of words should be minimal. Finally, while children would still be expected to slow

down after making errors, in the discrete trials methodology, the slowing should randomly affect all trial types equally, rather than being more likely in blocks with lower accuracy. Without a doubt, if the goal is to study interference control, the discrete trials version of the Stroop task is superior to the blocked administration.

In the present study, the stimuli are colour words or XX's presented in one of four colours, and the task is always to name the colour of the letters. If children with ADHD have a global problem with executive control over the task, if they have specific problems with written stimuli, if they do not obey task rules, or if they do not pay attention to the task overall, then they should show deficits (i.e., increased RT or more errors) relative to controls on both non-word (non-interference) and incongruent (interference) trials. On the other hand, if they have a specific deficit in inhibitory control, then increased RT and errors relative to controls should be observed only on interference trials. Or, thirdly, if they have a deficit in inhibitory control as well as more global deficits (or if, as suggested by Barkley, 1997a, the former leads to problems in the latter), there should be an interaction, such that children with ADHD are impaired relative to controls on all aspects of the task, and especially on interference trials.

For many of the same reasons outlined here, Carter et al. (1995a) administered a discrete trials version of the Stroop task to children with ADHD

and controls. The difference in RT between interference and non-interference trials was larger for children with ADHD than for controls. That is, children with ADHD showed more Stroop interference as measured by reaction time. There was no group difference in accuracy overall, or with each trial type.

As Carter et al. (1995a) cautioned, however, there are many potential explanations for performance deficits on the Stroop task (e.g., global frontal impairment, an early filtering deficit; see also MacLeod, 1991). As mentioned above, an inhibition-based account of the Stroop effect assumes that the written word must be suppressed in order to respond correctly with the ink colour. One way to validate this theory is to look for evidence of the suppression, by requiring the suppressed response on a subsequent trial (see Neill, 1977; Neill & Westberry, 1987). That is, in a two-trial pair, the written word on the first trial (the "prime") appears as the ink colour on the subsequent trial (the "probe"). For example, "blue" written in red ink (blue is to be suppressed) is followed by "green" written in blue ink (blue is the correct response). An inhibition-based account would predict that the response to the probe on a pair such as this one, should be slower than if the probe was unrelated to the prime (e.g., if the second stimulus was "green" written in yellow ink). The data support this prediction, and this pattern has been called "negative priming" (Houghton & Tipper, 1994; Tipper, 1985).

Negative priming is measured using a variety of types of tasks other than the Stroop task (e.g., picture naming, Tipper, 1985; see Fox, 1995, for a review). Regardless of the specific stimuli or task, negative priming occurs when a response that is to be ignored on one trial is the required response on a subsequent trial. A negative priming score is calculated by subtracting RT and accuracy on neutral trials (trials with no relation between the stimuli on the prime and probe) from that on ignored repetition trials (trials for which the ignored stimulus on the prime is the required response on the probe).

Many (e.g., Houghton & Tipper, 1994) argue that the slower RT on ignored repetition trials occurs because the distracting information in the prime is actively inhibited, and this inhibition causes delayed responding on the subsequent trial. Although there are alternative explanations (see Milliken & Joordens, 1996 and Neill & Valdes, 1992), negative priming is considered by most to be a measure of attentional or cognitive inhibition (see Klein & Taylor, 1994; Nigg, 2001; Tipper, 2001).

Note that if children with ADHD have problems with inhibition, and if attentional inhibition is what causes the cost on ignored repetition trials, then children with ADHD should actually show *better* performance or less of a cost on ignored repetition trials relative to controls. Consistent with this logic, reduced or no negative priming has been demonstrated in several groups of people who are thought to have reduced cognitive inhibition, such as adults

with OCD (Enright & Beech, 1993), elderly people (Hasher, Stoltzfus, Zacks, & Rypma, 1991), and young children (Tipper, Bourque, Anderson, & Brehaut, 1989, although see Tipper & McLaren, 1990). A reduction or absence of negative priming has also been demonstrated in children labelled by their teachers as being socially impulsive (Visser, Das-Smaal, & Kwakman, 1996), children with Tourette's syndrome and comorbid ADHD, OCD or both (Ozonoff, et al., 1998), and children with attention deficits (McLaren, 1989).

To summarize, the discrete trials Stroop Colour Word Task with a Negative Priming manipulation will address several issues with regard to the inhibitory control hypothesis of ADHD. Firstly, the discrete trials approach to studying the Stroop interference effect will improve upon previous studies that may have been measuring more global deficits; the design used here will target specific deficits in interference. Secondly, it will assess attentional/cognitive inhibition using a negative priming manipulation. Consistent with the inhibitory control hypothesis, it is expected that children with ADHD will have an increased level of interference on the prime trial, and less negative priming on probe trials, relative to control children.

Method

Participants

The participants were the 40 children (24 control and 16 ADHD) described in Table 2.3. None were colour blind, according to their performance

on the Ishihara Test for Colour Blindness (Ishihara, 1980; see Chapter 2). Reading ability was assessed using the Reading subtest of the WRAT-3 (Wilkinson, 1993; see Chapter 2).

Procedures

Task. Children sat at a comfortable distance away from the computer screen and the viewing distance was not fixed. Based on the position of the laptop and external keyboard, they sat approximately 57 cm away from the screen. Custom software was written by J. Chrisie for a use on a Macintosh laptop computer.

All participants completed a practice block of 33 trials, followed by 132 experimental trials. Each trial consisted of a discrete pair of stimuli, presented successively. Each pair was initiated by pressing the space bar on the computer keyboard. The task took approximately 30 minutes to complete; the time varied slightly depending on the length of pauses between discrete pairs.

Prime stimuli were either colour words (RED, GREEN, YELLOW, BLUE) or X's, displayed in red, green, yellow, or blue in the centre of a black screen. All letters were in uppercase, and were approximately .8 by 1 visual degrees when viewed at a distance of 57 cm. Of the 132 prime trials, 33, or 25%, were displayed in each colour. Approximately one quarter (27.27%) of the prime trials were non-interference trials (XX's presented in one of the four colours). The remaining 72.73% of the trials were interference trials (colour-

words presented in one of the four colours). Colour words were always incongruent with ink colour (i.e., the word RED was never presented in red).

Probe stimuli were the same as prime stimuli, except they were always colour words (i.e., they were not XX's). Probe stimuli were equally likely to be any of the colour words, and in any colour, with the exception that the words were always incongruent with the ink colour.

The task on both the prime and probe was the same: to name the colour of the stimulus aloud.

An important variable was the relation between the stimuli on the prime and those on the probe. An 'ignored repetition' trial was one for which the word (distracter) on the prime 'became' the colour (correct response) on the probe. A 'neutral' trial was one for which neither the colour nor the word on the prime became the colour nor the word on the probe. Although many studies of negative priming include only these two types of trials, there is a risk that participants will learn to anticipate the negative priming pattern (i.e., learn to expect the prime word to become the required response on the probe), and that this expectation will lead to an attenuation of the negative priming cost (Christie & Klein, 2001). To make it difficult for participants to predict the identity of the probe based on that of the prime, two other types of trials were mixed in. On 'colour repetition' trials, the colour on the prime became the

colour on the probe. On 'word repetition' trials the word on the prime became the word on the probe.

In order to ensure the correct proportion of different trial types and stimuli, all 132 trial pairs were entered into the computer program as a stimulus set. The 132 pairs were presented in random order.

Before the start of the experiment, the computer microphone was adjusted for each participant so that small noises (e.g., movement in the chair) would not be detected, but that the participant's normal speaking voice would be detected. The computer recorded the RT of the first sound above that threshold that occurred after the onset of each stimulus. The experimenter sat beside the participant throughout the duration of the experiment to record the accuracy of the response, as well as to code when a non-voice sound triggered the microphone or when a voice-response was undetected by the microphone.

The timing of events was as follows. The prime was presented until a response was made, or for 2000 ms. The response stimulus interval (RSI), or the time between the response to the prime and the onset of the probe was 900 ms.[17] The probe was presented until a response was made, or for 2000 ms. Following the offset of the probe, feedback was provided to tell the participant (and signal to the examiner) whether a sound response had been recorded. If

[17] This value was chosen because it is within a range that is likely to produce negative priming (see Fox, 1995).

the computer recorded any sound during the 2000 ms presentation of a stimulus, the message "got it" was presented after the two-trial pair. No feedback regarding RT or accuracy was provided.

Instructions to participants. Prior to starting the experiment, children were asked to name the colours of a series of XX's printed on a sheet of paper in different coloured markers, and to read a set of colour words written in black ink. Next, they were asked to name the marker colours of colour-words written on the paper (akin to interference trials in the computer-administered test). These steps ensured that participants understood the task requirements prior to learning how to run the program on the computer, that they could read the words 'red, green, blue, and yellow', and that they could discriminate among the four colours.

Participants were told that, as with the examples on paper, they would be seeing either "RED", "GREEN", "BLUE", "YELLOW" or "XXXXX" displayed in one of four colours in the centre of the computer screen. They were told to try to ignore the letters and to say the colour of the letters out loud, as quickly as they could without making too many mistakes. They were told that the computer microphone would be keeping track of when they answered, by "hearing" the first sound that they made, so that they should try not to make other noises (such as tapping on the table, or saying "um"). In the point system (described in Chapter 2), points were awarded for "staying quiet except for

colour words". They were told that after two words, a message would appear that would signal whether the computer "heard" their answer. They were also told that the experimenter would be keeping track of what they answered, by writing their answers down on her paper. Finally, they were told that they could proceed at their own pace, because they could decide when to initiate each two-trial pair by pressing the space bar.

Performance was monitored closely during the practice block, and additional instructions were given if necessary. Throughout the experiment, participants were cued by the experimenter to speak louder if the computer microphone was not picking up on responses, or reminded to try not to make extraneous noises if non-voice sounds were frequently triggering a response.

Data Analysis. The two dependent variables of interest were RT to say the correct colour word, and accuracy (the rate of saying the wrong colour name). Trials for which a non-voice sound triggered the microphone were discarded from the RT analyses; this was the case for 7.33% and 7.54% of the prime and probe trials, respectively. On approximately one third of these trials (29.97% and 35.18% of the discarded prime and probe trials, respectively), a verbal response was made. Even though these trials were excluded from RT analyses, they were retained for analyses of accuracy.

Trials with responses faster than 300 ms were also excluded. This was the case for 1.21% of the prime trials and 1.36% of the probe trials.

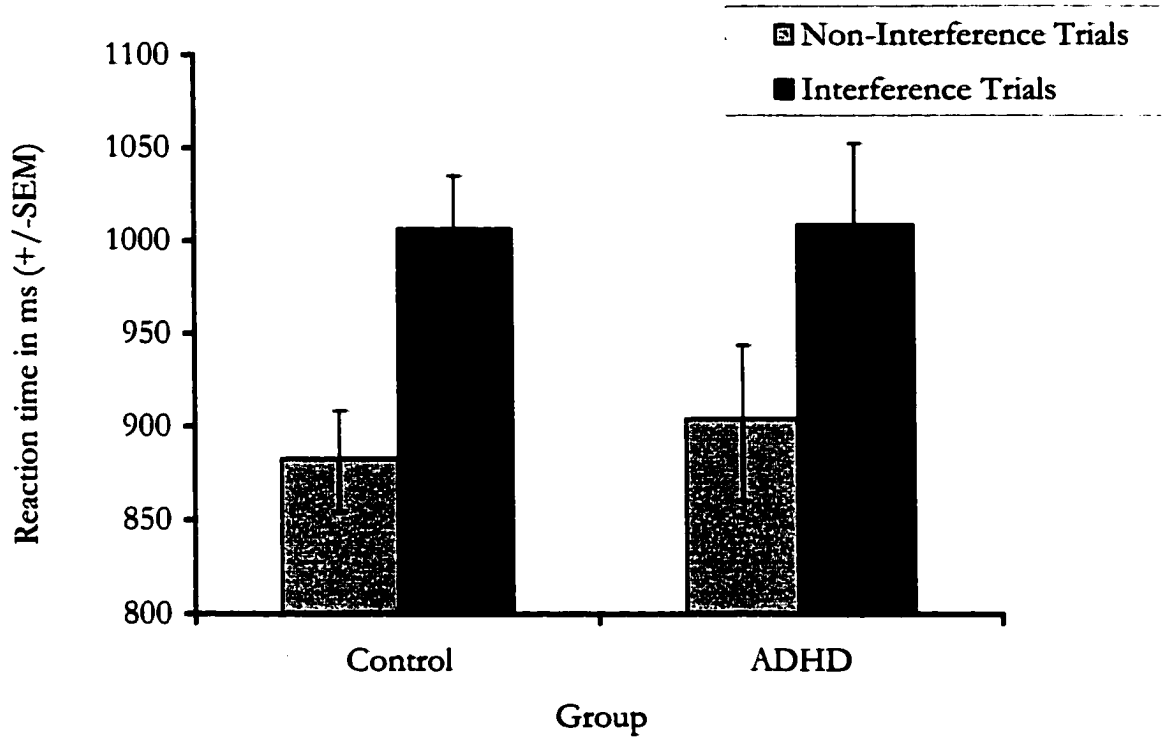
Results

Stroop Interference

The Stroop interference effect was measured on the prime trials only (i.e., the first trial in a two-trial pair), to avoid any influences that the prime might have on performance on the probe (such as negative priming). The data was analyzed with two mixed ANOVAs, one for RT (correct trials only) and one for accuracy (percent error). Each analysis had one between subjects variable (Group) and one repeated measure: Trial Type (Interference and Non-interference).

Reaction time. Figure 4.1 illustrates the RT to prime trials by Trial Type and Group. There was a significant main effect of Trial Type, $F(1, 38) = 141.68$, $p < .0001$, no main effect of Group $F(1, 38) = 0.06$, $p = .80$, and no interaction between Group and Trial Type, $F(1, 38) = 0.99$, $p = .33$. As would be expected, all children took longer to respond on Interference trials than on Non-interference trials, but unlike previously reported findings, children with ADHD did not differ in their RT (overall or by Trial Type) from controls.

Figure 4.1. Reaction Time by Prime Trial Type and Group.



The main variable of interest is the Stroop Interference Effect. A single score was computed for each participant by subtracting RT on Non-Interference trials from that on Interference Trials. The average Stroop Interference: RT score for children in each group is presented in Table 4.1. Positive difference scores in both groups indicate that children had a RT cost on Interference trials (this is comparable to the main effect of Trial Type in the ANOVA). Table 4.1 also presents the results of an unpaired t-test comparing the magnitude of the difference scores between groups. Note that this is statistically comparable to the interaction term in the mixed ANOVA.

Table 4.1. Derived difference scores representing the magnitude of Stroop Interference and Negative Priming for each group, and t-tests comparing the size of each effect between groups.

Derived Difference Score	Control (n = 24)	ADHD (n = 16)	Group comparison
Stroop Interference: RT	123.21 (57.59)	104.25 (61.61)	$t(38) = -0.99, p = .33$
Stroop Interference: Accuracy	8.10 (5.62)	13.59 (9.50)	$t(38) = 2.30, p = .03$
Negative Priming: RT	13.14 (77.18)	55.77 (91.12)	$t(38) = 1.59, p = .12$
Negative Priming: Accuracy	-0.92 (11.34)	1.98 (19.13)	$t(38) = 0.61, p = .55$

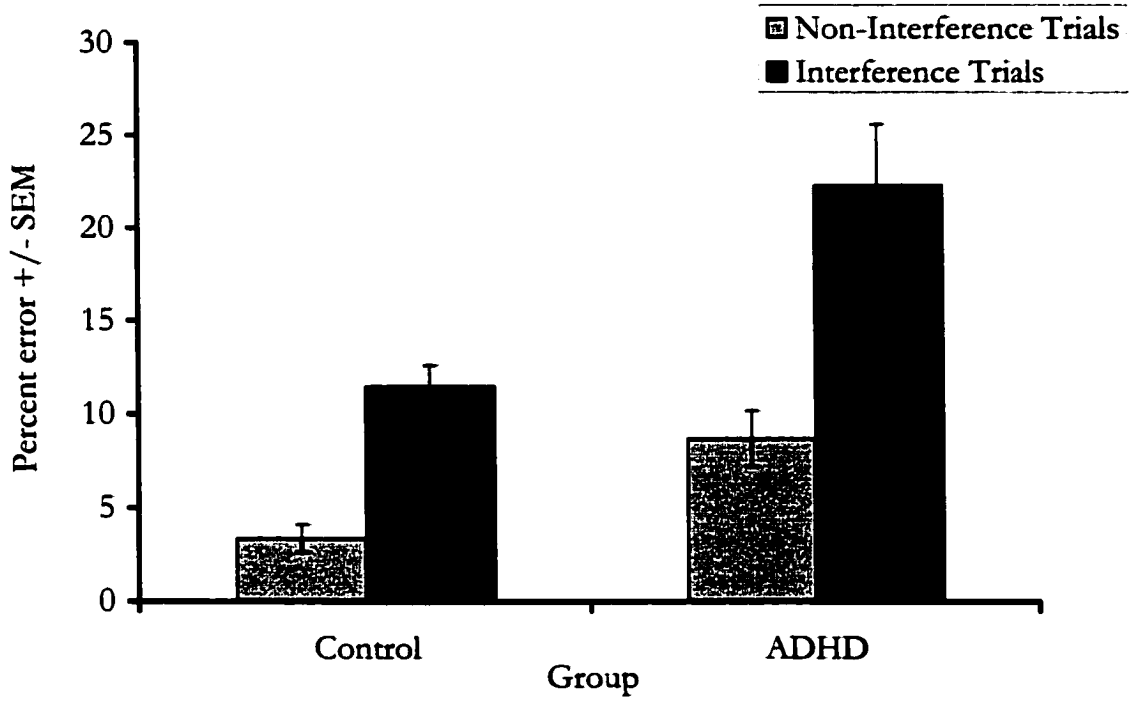
Note. Mean difference scores are presented with standard deviations in parentheses. Stroop Interference scores were computed by subtracting performance (RT or percent error) on Non-Interference trials from that on Interference Trials. Negative Priming scores were computed by subtracting performance on Neutral trials from that on Ignored Repetition trials. Note that t-tests comparing the difference scores are comparable to the interaction term in each of the respective mixed ANOVAs.

Accuracy. With accuracy as the measure of performance, there was a significant main effect of Trial Type, $F(1, 38) = 82.43, p < .0001$, a significant main effect of Group, $F(1, 38) = 15.10, p < .001$, and a significant interaction between Group and Trial Type, $F(1, 38) = 5.27, p = .03$.[18] As portrayed in Figure 4.2, all children made more errors on Interference Trials, children with

[18] The correlation between percent error on Interference and Non-Interference trials is .76 for the ADHD group and .33 for the control group. Although both correlations are positive, this violates the assumption of homogeneity of correlations required for a mixed ANOVA. The assumption of homogeneity of variances is also violated; Fmax = 2.60 and Fmax = 5.50 for the Non-Interference and Interference trials, respectively. For both levels of the repeated measure, the within-group variance is smaller in the control group than in the ADHD group. This is probably due, in part, to a floor effect in the control group. When Geisser-Greenhouse corrected F's are used, the pattern of results remains the same. In this case, the reader should pay particular attention to the significant difference in Stroop Interference: Accuracy difference scores as reported in Table 4.1. The t-test used for this comparison is more robust in the presence of heterogeneity of correlations and of variances than is the mixed ANOVA.

ADHD made more errors overall than control children, and children with ADHD made disproportionately more errors on Interference Trials.

Figure 4.2. Percent Error by Prime Trial Type and Group.



Stroop Interference: Accuracy scores, computed for each participant by subtracting percent error on Non-Interference trials from that on Interference trials, are presented in Table 4.1. The main effect of Trial Type is reflected in positive difference scores in both groups, and the interaction between Group and Trial type is reflected in a significant t-test comparing the magnitude of the interference.

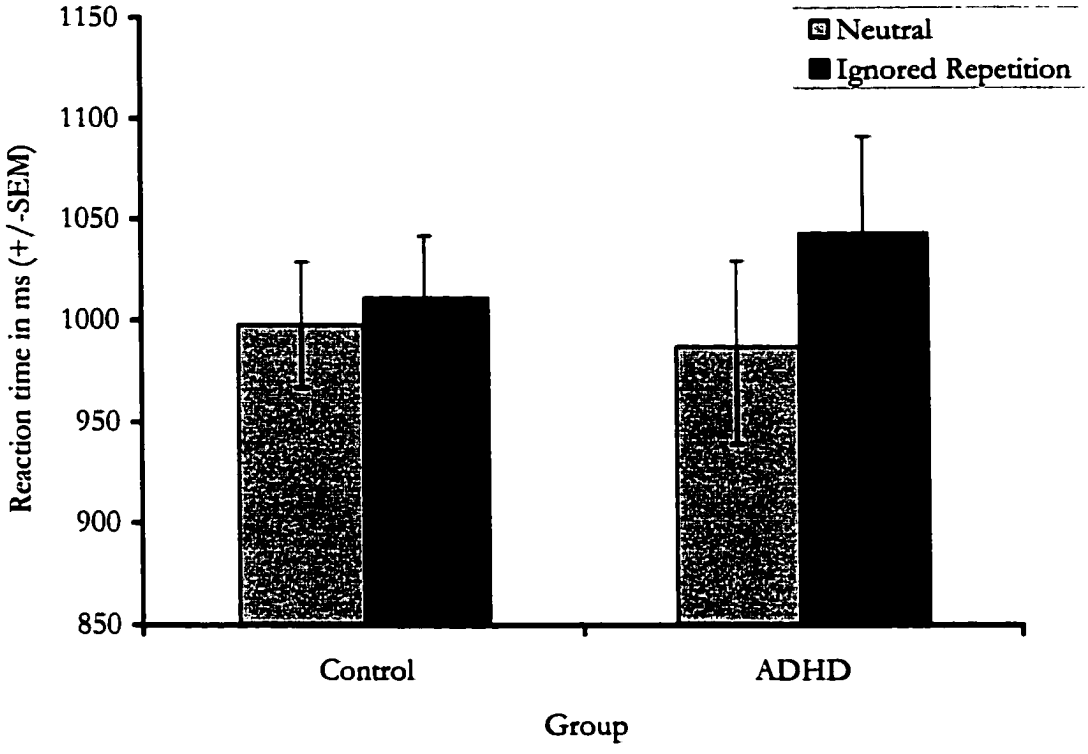
Negative Priming

Negative priming was examined only on the 87.03% of trials for which the prime response was correct, since it is only then that we can assume that the written word on the prime was successfully inhibited. It was analyzed with two mixed ANOVAs, each with one between subjects variable (Group) and one repeated measure: Prime-Probe Relationship (Ignored Repetition and Neutral). Separate analyses were conducted on the two dependent measures of performance on the probe: RT (on correct trials only) and accuracy (percent error).

Reaction time. For RT on the probe, there was a main effect of Prime-Probe Relationship, $F(1, 38) = 6.62, p = .01$, no main effect of Group $F(1, 38) = 0.04$, $p = .84$, and no interaction between Group and Prime-Probe Relationship, $F(1, 38) = 2.54, p = .12$. As reflected in Figure 4.3, the main effect of Prime-Probe Relationship is characterized by slower RTs on Ignored Repetition trials as compared to Neutral trials.

A single derived score representing the Negative Priming Effect: RT was computed for each participant by subtracting RT on Neutral trials from that on Ignored Repetition Trials (see Table 4.1). Positive values reflect the cost (slower RTs) on Ignored Repetition trials. The nonsignificant t-test indicates that the magnitude of this cost did not differ between groups.

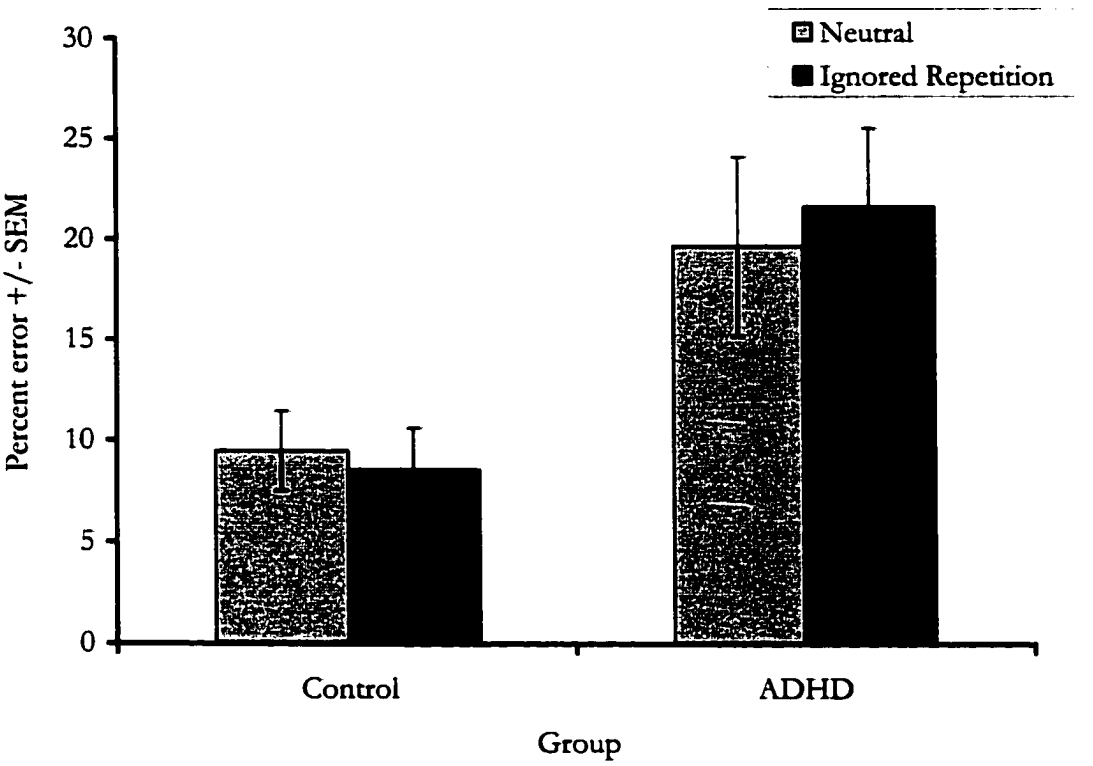
Figure 4.3. Reaction Time by Probe Trial Type and Group.



Accuracy. For accuracy on the probe, there was a significant main effect of Group, $F(1, 38) = 11.36, p = .002$, no main effect of Prime-Probe Relationship, $F(1, 38) = 0.05, p = .83$, and no interaction between Group and Prime-Probe Relationship, $F(1, 38) = 0.36, p = .55$. Children with ADHD made more errors overall than control children (see Figure 4.4), but there was no negative priming cost in accuracy, and no group difference associated with negative priming. This pattern is also reflected in the Negative Priming: Accuracy scores (see Table

4.1), which were computed by subtracting percent error on Neutral trials from that on Ignored Repetition trials.

Figure 4.4. Percent Error by Probe Trial Type and Group.



Correlations with reading ability

Since the interfering stimulus in this task is a written word, it is conceivable that good readers could experience more interference than poor readers. All participants demonstrated that they could read the four colour words used as interfering stimuli, but there was a range in word decoding ability as assessed by the WRAT-3 Reading subtest. As reported in Chapter 2, there

was no difference between groups in Reading subtest scores, $t(38) = -1.07, p = .29$. To determine whether there is a relationship between word decoding ability and amount of interference, the WRAT-3 Reading Absolute score (an interval scaled score representing performance regardless of age) was correlated with the Stroop Interference and Negative Priming scores (both RT and Accuracy). Consistent with observations by Carter et al (1995a), all correlations were nonsignificant (correlations ranged from $r = -.15$ to $r = .04$), all $ps > .35$).

Discussion

Stroop Interference

As expected based on decades of research using the Stroop paradigm, all participants were affected by the Stroop manipulation. That is, they responded more slowly and made more errors when they were to report the colour of a colour-word, than when they were to report the colour of a non-word.

For the purposes of this study, the main analysis was a comparison of the degree to which children with ADHD and controls were affected by the Stroop manipulation. As has been reported on several occasions (see Barkley et al., 1992; Nigg, 2001; Pennington & Ozonoff, 1996; and Sergeant et al., 2002, for reviews), children with ADHD in this study were more adversely affected by the Stroop manipulation than control children.

Unlike that reported in previous research, however, the difference in cost was evident in a *disproportionately high error rate* on Interference trials relative to Non-Interference trials among children with ADHD. In previous studies, the group difference has been evident in a *disproportionate increase in reaction time* on Interference trials. Since reaction time and accuracy are simply two different ways of measuring performance, one could argue that these two patterns are parallels of each other, and that we have simply sampled from a different point along the speed-accuracy trade-off function. That is, children with ADHD are impaired to the same degree in this and in previous research, and the difference is simply that here they were *as fast but less accurate* as compared to controls, and in previous research they were *as accurate but slower* as compared to controls. It may be that when participants initiate each trial at their own pace, this biases them toward aiming for fast responses, or vice versa, when they have a list of words in front of them, they are more focused on making fewer mistakes.

Alternatively, this difference could be a meaningful one based on the fact that a discrete-trials design was used in this study. As was previously discussed, this approach is better at measuring the differences in interference control than a blocked design, because the latter also measures several other global abilities. Specifically, a blocked design is more likely to be affected by off task behaviour and distractibility, expectancies and response set, an inability to maintain a rule in working memory, reading or scanning dysfluency, and reactions to errors. It

is difficult to say which of these, if any, might have contributed to the unique pattern observed here. The direct influence of most of these variables would be to increase reaction time, although as has been said, accuracy could still be affected because of a speed-accuracy trade-off (accuracy may decrease in an effort to keep reaction times fast).

Carter et al. (1995a) also used a discrete-trials methodology for their Stroop task. As with the studies using a blocked design, they reported group differences in interference as measured by reaction time and not by accuracy. A key difference in that study was that stimuli remained on the screen until a verbal response was made, whereas in this study there was a time limit of 2000 ms. Again, Carter et al. may have been sampling from a different point along the same speed-accuracy trade-off function. There is some evidence for this, as the participants in Carter et al.'s study had much lower error rates than those in this study. Their reported error rates were all below 3.3% per group and condition, whereas in this study error rates ranged between 3.7% and 22.3%. It should be noted that a direct comparison between these data should be done only with caution because of differences in task design.

To summarize, the group difference in Stroop Interference observed here in terms of accuracy could be the same difference measured by others in reaction time, simply sampled at a different point along the same speed-accuracy trade-off function. Alternatively, the difference in the nature of the

effect could be due to the improved methodology used here. The only way to know for certain would be to compare directly a discrete-trials design to a blocked design within the same sample. Either way, the conclusion here is strong: children with ADHD show increased Stroop Interference; they make disproportionately more errors on Interference trials relative to control children. This indicates that children with ADHD have an impaired ability to inhibit the competing response associated with the interfering colour-word relative to controls. That is, they have a deficit in the type of inhibition classified as "interference control" or "response competition".

Another finding that should be mentioned is the lack of a relation between word decoding ability and performance on the Stroop Colour Word task. This suggests that children in this sample had sufficient reading ability to experience interference from the written word, and that differences across individuals in their ability to read more complex words on the WRAT-3 (Wilkinson, 1993) was not associated with differences in performance on this task. Nevertheless, future research should continue to explore the use of Stroop tasks without a reading component, to isolate further the interference control demands of this task. For example, see Archibald and Kerns (1999) for a developmental study with two modifications of the traditional Stroop task that do not involve word reading. Instead, suns and moons or coloured fruit were used as stimuli. Performance on these measures was correlated with that on the traditional

Stroop Colour Word task, suggesting that the newly developed measures assess the same underlying ability in interference control.

Negative Priming

Across all participants, there was a cost associated with ignored repetition trials. Overall, children were slower to respond to a probe when the required response was the distracting colour word on the previous (prime) trial, than when there was no relation between the prime and probe. There was no cost in accuracy associated with ignored repetition trials.

The main comparison of interest was one of the size of the negative priming cost between groups. Recall that if a negative priming cost on the probe is due to lasting inhibition from the prime stimulus, and if children with ADHD have deficits in inhibition, then children with ADHD should have shown *reduced or no* negative priming. Non-significant interactions between Group and Prime-Probe Relationship with both RT and accuracy as the dependent measures indicate that there were no group differences in negative priming. Positive Negative Priming: RT scores (see Table 4.1) indicate that, as a group, children in ADHD did show a negative priming effect.

Relatively little research has been conducted on negative priming in children with ADHD. The existing studies (McLaren, 1989 with children with attention problems; Ozonoff et al., 1998 with a mixed group of children with Tourette's syndrome and comorbid ADHD or OCD; and Visser et al., 1996,

with non-diagnosed socially impulsive children), each showed a reduction in negative priming in the "ADHD-like" group. Each of these studies employed a blocked administration, comparing sets of trials with no relation between the prime and probe to sets for which the distracter on the prime becomes the correct response on the probe. In addition to the concerns raised regarding using a blocked design to measure the Stroop effect, there is an additional concern when this design is used to measure negative priming. With a blocked design, accuracy on the prime is not considered. An inhibition-based theory of negative priming presumes that the cost on the probe occurs because the distracter on the prime had been suppressed. Only when the correct answer is given on the prime can one be certain that the prime's distracter was inhibited. With a discrete-trials design, probe trials following incorrect responses on the prime can be excluded individually. In this study, 12.97% of the trials were excluded because of an incorrect response or no response on the prime.

Recall that in Nigg's (2001) taxonomy, Negative Priming is in a category called Automatic Cognitive Inhibition, which is distinct from Executive Inhibition. Inhibition of the word names activated by the conflicting material in the Stroop task leave a 'trace' that is picked up on the probe trial as Negative Priming. This presumably happens automatically, without top-down, or executive control from the participant. These data suggest that children with ADHD are not impaired in this type of inhibition.

Chapter 5. Simon-Flanker Paradigm

The Simon Task and the Flanker Task are two other paradigms that, like the Stroop Task, require interference control or involve response competition. In this study, these two tasks were combined into one. Each task and its predictions will be discussed in turn.

Simon Task

In the Simon task, an incorrect response is in competition with a correct response (see Lu & Proctor, 1995, for a review).[19] Whereas in the Stroop task, a written word must be suppressed in order to respond correctly, in the Simon task, the *location* of the stimulus must be suppressed. Various methods can be used to elicit this effect. In the version used in this study, the participant is required to make a speeded forced choice response to a visual stimulus that appears either to the left or right of fixation (centre). The participant must indicate by pressing a left or right key which of two potential stimuli is presented. The Simon Effect is evident when performance is better on trials for which the location of the stimulus is compatible with the location of the response key corresponding to that stimulus. For example, if the response key corresponding to a particular stimulus is the leftmost of the two keys, performance will be better when that stimulus is presented on the left of the

[19] The namesake for this task was J.R. Simon, who published the first demonstration of this effect with Small in 1969 (see review by Lu & Proctor, 1995).

screen, than when it is presented on the right. By comparison, performance is not as good if the location of the stimulus is incompatible with the response key (if the response key for a given stimulus is on the left and the stimulus is presented on the right).[20]

In the Simon task, the location information is irrelevant to the task; it is not needed and should be ignored. The Simon Effect is presumed to occur because of the influence that the irrelevant spatial information has on the performance of the correct response (interfering when the location of the target and correct response do not correspond; facilitating when they do correspond). Considerable research has been conducted to determine whether the influence takes place at the stimulus identification stage, the response selection stage, or the response execution stage. The majority of the data support the theory that the interference takes place at the response selection stage (Lu & Proctor, 1995). That is, a spatial code is generated for the location of the stimulus (left or right), and this information competes with the response key location (left or right) at the time when the response is being selected.

Tagliabue, Zorzi, Umilta, and Bassignani (2000) were the first to publish a study on the Simon Effect in young children (ages 5-8 years). Their design was different from a typical Simon task because they added a between-subjects

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[20] Note that, although they share some features, the Simon task is not the same as a stimulus-response compatibility task. The differences between these two types of tasks will be discussed later in this chapter.

manipulation intended to alter the strength of the association between stimulus locations and response locations. Twenty-four hours prior to completing the Simon task, half of the children completed a Same-side[21] Preceding Task for which they were instructed to respond according to the location of a single stimulus (to respond with the right key when the stimulus was presented on the right). The other half completed a Different-side Preceding Task, for which their task was to respond to the opposite location of a single stimulus (to respond with the right key when the stimulus was presented on the left). For our comparison purposes, the data for the group that completed the Same-side Preceding Task is most applicable. In every day life, actions are frequently compatible with stimulus locations (we respond in the direction of objects), so additional practice designed to strengthen the association between stimulus and response locations is unlikely to alter the nature of the Simon Effect that they observed. Conversely, data from the group that completed the Different-side Preceding Task should be seen as under the influence of the novel experimental manipulation designed to strengthen responses to stimuli on the opposite side.

For the Same-side group, there was a significant Simon Effect in reaction time. That is, children were faster to respond on compatible trials (to stimuli

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[21] Tagliabue, et al. (2000) referred to their two Preceding Tasks as Compatible and Incompatible tasks. Here, these two tasks are referred to as Same-side and Different-side, to avoid confusion with the use of the terms compatible and incompatible as they pertain to the two types of trials in the Simon task.

presented on the same side as the response key) than to incompatible trials (to stimuli presented on the opposite side from the response key). The magnitude of the Simon Effect (the difference between reaction time on compatible and incompatible trials) was 16 ms. For the Different-side group, the Simon Effect was reversed (-40.5 ms), indicating that, consistent with the experimental manipulation, these children responded more quickly to incompatible trials. Accuracy data are difficult to interpret because they were presented only in the context of an interaction with the Preceding Task (Same-side or Different-side). For our purposes, Tagliabue, et al.'s (2000) data provide evidence that, at least following practice of responses compatible with location, children as young as ages 5 to 8 years show a Simon Effect of approximately 16 ms.

To date, there have been no published studies on the Simon Effect in children with ADHD. Based on the inhibitory control theory of ADHD, which predicts increased interference or competition from distracting information, it is expected that children with ADHD will show more interference from the irrelevant location, and will therefore have a stronger Simon Effect relative to control children.

Before turning to the Flanker task, another task will be discussed because it shares some features with the Simon task. The typical Stimulus-Response (S-R) compatibility task is similar to Tagliabue et al.'s (2000) Same-Side and Different-Side Preceding Tasks described above. On some trials, children are

instructed to respond on the same side as the stimulus, and on others, they are instructed to respond on the opposite side from the stimulus (i.e., press a left key to a target presented on the right). Performance is typically worse (higher reaction times and more errors) on Different-Side (incompatible) trials. Both Sergeant et al. (1999) and Douglas (1999) reviewed the small body of literature using a version of this task with children with ADHD. Both concluded that although there was some evidence that children with ADHD were disproportionately impaired on the Different-Side trials (i.e., there was an interaction between the S-R compatibility manipulation and group), there were an equal number of studies that reported no such interaction. While this task is similar to the Simon task in some ways, there are some important differences. In an S-R compatibility task, the instruction is to respond either on the same side as, or on the opposite side from, the location of the stimulus. The location is the stimulus feature that tells the participant what key to press. In a Simon task, the location of the stimulus is irrelevant (with respect to the task instructions). The instruction is to respond according to the *identity* of the stimulus. On a random half of the trials, the response key for that stimulus is on the same side as the stimulus, and on the other half, it is on the opposite side. Although similar to an S-R compatibility task, the Simon task assesses a distinct type of response interference (Hasbroucq & Guiard, 1991).

Flanker Task

The Flanker task allows for a measure of filtering or selective attention (see Eriksen & Eriksen, 1974 and Miller, 1991). The typical Flanker task is a forced-choice reaction time task in which the target (usually a letter or shape) is presented in the centre of the display and on some trials, distracting information (other letters or shapes) surrounds or 'flanks' the target. As with the Simon and Stroop tasks, this task requires the suppression of irrelevant information (in this case, the flankers), and the amount of interference is measured by costs in reaction time and accuracy relative to trials with no interfering information. Both children's and adults' performance with respect to the target is affected by flankers (e.g., Enns & Akhtar, 1989; Eriksen & Eriksen, 1974; Miller, 1991). Features of the flankers can also modulate their impact, or the degree to which they interfere with the processing of the target. Examples of such features include spatial proximity to the target (nearby flankers produce more interference) and the relation of the flankers to the response set (flankers that could be targets on other trials produce more interference). Enns and Akhtar (1989) demonstrated how developmental changes in the relative influence of these features could point to differences in processing ability with age. A similar approach will be used here to study the nature of interference control in children with ADHD.

Two recent studies have compared performance of children with ADHD to that of control children on a Flanker task. Firstly, in Jonkman, Kemner, Verbaten et al. (1999), the target stimuli were arrows presented at fixation that signalled the correct response (left or right). They tested four flanker conditions: no-flankers, neutral flankers (plus signs), congruent flankers (arrows pointing in the same direction as the target) and incongruent flankers (arrows pointing in the opposite direction). All children responded more slowly to targets with incongruent flankers and faster to targets with no flankers, and there were no between group differences (ADHD versus control) in reaction time. With accuracy, all children made more errors on the incongruent trials than on the other types of trials, and children with ADHD made more errors overall than control children. The primary question of interest was whether there were group differences in the magnitude of the various types of interference studied. The flanker congruence effect (calculated as the difference between the error rate on neutral and incongruent trials) was larger in the ADHD group than in the control group. The flanker presence effect (calculated as the difference between the error rate on no-flanker and congruent trials) was not significantly different between the two groups. Putting these two latter findings together, Jonkman et al., (1999) concluded that there was a group difference in response interference (flanker congruence effect), but no group difference in perceptual interference (flanker presence effect).

In Brodeur and Pond's (2001) study, the targets were pictures of clothing (a shirt or a tie), and the distracters were either flanking pictures of clothing, or auditory words presented with headphones. Both visual and auditory distracters could either be "meaningful", i.e., come from the same response set as the targets (shirt or tie) or "irrelevant" (purse, which was never a target). Distracters could be in one modality (visual or auditory) or both modalities (visual and auditory). Participants were instructed to press one of two response keys to indicate whether they saw a shirt or a tie. Interference scores were derived by comparing performance in each condition against a single baseline score (performance on trials with no flankers). Brodeur and Pond (2001) found that children with ADHD had a larger reaction time cost in the presence of distracters (regardless of distracter type) than did control children. This observation is consistent with Jonkman et al.'s (1999) finding with accuracy as the dependent measure of performance. Additionally, whereas control children were differentially affected by distracter meaning (they showed more interference to meaningful distracters than to irrelevant distracters), children with ADHD were equally affected by both kinds of distracters. Brodeur and Pond (2001) hypothesized that children with ADHD "did not make a strong association between target and response, and therefore were not differentially affected by the presence of distracters that may elicit an incorrect response" (p. 237). Error rates in this study were low, and group differences, which only

emerged in the context of complex interactions with modality (visual, auditory, and both) and age (young and old), will not be reviewed here.

Both Jonkman et al. (1999) and Brodeur and Pond (2001) found greater interference related to flankers among children with ADHD. This is predicted by the inhibitory control theory of ADHD and is expected here.

Simon-Flanker Task

Because both the Simon and Flanker tasks involve a forced-choice speeded reaction time task, they were combined into one task for the present study. The main advantage of doing this was practical: measures from two different paradigms are obtained while the child learns and performs only one task. In addition, the tasks were combined in such a way that different levels of one measure (Flanker) were presented at different levels of the other measure (Simon). For example, different types of Flanker trials could be presented at centre, as in the straightforward Flanker task, or to the left or right of centre, corresponding to Incompatible and Compatible Simon trials). If performance deficits in these two tasks take place at different stages of processing, it would be theoretically possible to see additive effects when they are manipulated orthogonally; conversely, interactive effects would be expected if they operate at the same stages of processing.

The target stimuli were the numbers 1, 2, 3, and 4. Participants were to press the left key in response to a 1 or 2, and the right key in response to a 3 or

4. The number stimuli were chosen so that the mapping of two stimuli to one response key would be relatively straightforward. Target stimuli were either presented alone (No Flankers, e.g., 2), with Identical Flankers (e.g., 222), with flankers requiring the same or Congruent Response as the target (e.g., 121), and with flankers requiring a different or Incongruent Response from the target (e.g., 323 or 424).

Following Enns and Aktar (1989), three different types of flanker interference will be assessed with the four different flanker conditions. Feature Number Interference will be assessed by comparing performance on No Flanker trials (e.g., 2) to that on Identical Flanker trials (e.g., 222). This measures the impact of an increased number of features with no competing information. Feature Type Interference, will be assessed by comparing performance on Identical Flanker trials (e.g., 222) to that on Congruent Response trials (e.g., 121). In this case, the stimulus includes competing information, but there is no competition at the response level because both stimuli yield the same response. The main type of flanker interference is assessed by comparing performance on Congruent Response trials (e.g., 121) to that on Incongruent Response trials (e.g., 323 or 424). This measure most closely corresponds to the Flanker Compatibility Effect (FCE; cf., Miller, 1991). Incongruent Response trials should have a similar amount of encoding interference as Congruent Response trials (because the both types of flankers

are physically different from the target), but there should also be additional competing response information (because the flankers correspond to a different response from the target).

As indicated in Figure 5.1, trials from each of the four Flanker conditions were presented at the centre, left, and right of the screen. The "pure" Flanker Effect was measured at the centre only. The Simon Effect was measured on trials in the periphery, and the "pure" Simon Effect was measured on trials with no flankers only. Each effect could also be examined at different levels of or at an average of the other effect.

Figure 5.1. All possible stimuli (when 2 is the correct response) and comparisons made to measure the Flanker and Simon Effects.

		Stimuli and Location			Effect Measured	Comparison
		Left	Centre	Right		
Flanker Type	No Flanker	2	2	2	Feature Number Interference	No Flanker versus Identical
	Identical	222	222	222	Feature Type Interference	Identical versus Congruent Response
	Congruent Response	121	121	121	FCE	Congruent Response versus Incongruent Response
	Incongruent Response	424 323	424 323	424 323		
Response Keys		1 or 2		3 or 4		
Simon condition		Comp		Incomp	Simon Effect	Comp. versus Incomp.

Note. FCE = Flanker Compatibility Effect; Comp = Compatible; Incomp = Incompatible.

The pure Flanker Effects are examined only on trials presented in the centre.

The pure Simon Effect is examined only on trials with no flankers, and those in the periphery.

Method

Participants

The participants were the 40 children (24 control and 16 ADHD) described in Table 2.3.

Procedures

Task. A program was developed by the author for the purposes of this experiment using VScope, version 1.2.5., (Rensink, 1994), a software package designed to assist in creating experiments on the Macintosh.

The target stimuli (see Figure 5.1 for examples with 2 as the correct response) were presented in black Arial font on a grey background. When viewed at a distance of 57 cm, single digits ranged from .32 to .64 visual degrees in width, and sets of three digits ranged from 1.75 to 2.22 visual degrees in width. All stimuli subtended .95 visual degrees in height. To be able to measure the Simon Effect, the stimuli could either appear at the centre of the screen, 5.5 degrees to the left of centre, or 5.5 degrees to the right of centre.

Two of the keys ("z", located on the left side of the keyboard, and "/", located on the right side of the keyboard) were marked with stickers denoting them as response keys. The left key was designated for responses to 1 or 2, and the right key was designated for responses to 3 or 4.

The identity of the stimuli varied randomly from a set of all possible stimuli. Of the 240 trials, there were 60 of each target stimulus (1, 2, 3, or 4), and 20 of these were presented in each location (left, right, centre).

Children sat at a comfortable distance away from the computer screen and the viewing distance was not fixed. Based on the position of the laptop and external keyboard, they sat approximately 57 cm away from the screen.

All participants completed a practice block of 30 trials, followed by 3 experimental blocks of 80 trials, for a total of 240 experimental trials. The task took approximately 25 minutes to complete; the time varied slightly depending on the length of breaks between blocks.

Each block was initiated by pressing the space bar. The timing of each trial followed Enns and Akhtar (1989). After 500 ms, a brief fixation stimulus (a black dot) appeared in the centre of the screen. The fixation stimulus stayed on for 500 ms; it was followed by the target stimulus 500 ms later, which stayed on until a response was made (or for 10 seconds). Trials within a block ran continuously.

The computer recorded RT and accuracy of the first response made on each trial. No feedback was provided to the participant.

Instructions to participants: Participants were told that they would be seeing one of four numbers on the computer screen, and that they should press the corresponding key on the keyboard "as quickly as possible without making a lot of mistakes". They were told that the numbers could appear in the centre, on the left, or on the right of the computer screen, and that sometimes the numbers would appear alone and sometimes they would appear with other numbers beside them. They were told to respond to the middle number only and to ignore the surrounding numbers. They were instructed to keep their left hand near the left response key and their right hand near the right response key,

and were given short verbal cues during the task if they moved their hands away from the keyboard. Performance was monitored closely during the practice block, and additional instructions were given if necessary.

Data Analysis: Trials with responses faster than 250 ms and slower than 3000 ms were excluded. Using these criteria, 1.43% of the trials were excluded. Reaction time was measured on correct trials only. Accuracy was measured as percent error.

Results

Simon-Flanker Task Overall

Performance on all levels of both tasks in both groups was measured with two mixed ANOVAs (one on RT and one on percent error), each with one between subjects variable (Group) and two repeated measures, Simon Condition/Location (Compatible, Incompatible, and Centre) and Flanker Type (No Flanker, Identical Flankers, Congruent Response Flankers, Incongruent Response Flankers).[22] The means in each condition are presented in the top portions of Tables 5.1 (RT) and 5.2 (accuracy).

[22] It is important to note that the centre location is not included in the typical Simon task. In the Simon-Flanker task, the centre location was necessary in order to obtain a measure of the Flanker Effect at fixation. The Centre condition will not be included in specific examinations of the Simon Effect, but it is included in the overall analysis of Simon Condition/Location.

Table 5.1. Mean reaction time (and SD) by Simon/ Location and Flanker condition for children in the control and ADHD groups, and between groups analyses of derived Simon and Flanker Effect scores.

Trial Type		**Control**	**ADHD**
Simon Condition /Location	**Flanker Type**	Mean RT in ms (and SD)	Mean RT in ms (and SD)
Incompatible	No Flanker (a)	**828.01 (169.56)**	**1004.08 (222.69)**
	Identical (b)	843.18 (161.52)	1001.73) (220.88)
	Congruent Response (c)	907.65 (205.74)	1063.33 (235.68)
	Incongruent Response (d)	933.93 (160.54)	1095.63 (253.05)
Compatible	No Flanker (e)	**788.43 (178.74)**	**982.77 (237.59)**
	Identical (f)	809.49 (158.77)	982.07 (232.71)
	Congruent Response (g)	892.37 (156.35)	1048.15 (281.55)
	Incongruent Response (h)	921.17 (175.68)	1071.48 (241.94)
Centre	No Flanker (i)	**779.15 (151.93)**	**943.05 (226.21)**
	Identical (j)	**779.42 (148.11)**	**931.38 (236.36)**
	Congruent Response (k)	**843.32 (186.10)**	**1021.32 (231.43)**
	Incongruent Response (l)	**885.28 (214.59)**	**1061.88 (298.93)**
Derived Scores	**Calculation**	**Control**	**ADHD**
Simon Effect	No Flanker: Incompatible – Compatible (a – e)	39.58 (83.04)	21.31 (111.20)
	Between groups analysis	$t(38) = 0.60, p = .56$	
Feature Number Interference	Centre: Identical – No Flanker (j - i)	0.27 (79.83)	-11.67 (68.98)
	Between groups analysis	$t(38) = -0.49, p = .63$	
Feature Type Interference	Centre: Congruent Response – Identical (k - j)	63.90 (100.64)	89.94 (111.36)
	Between groups analysis	$t(38) = 0.77, p = .45$	
Flanker Compatibility Effect	Centre: Incongruent Response – Congruent Response (l - k)	41.97 (70.57)	40.56 (106.32)
	Between groups analysis	$t(38) = 0.05, p = .96$	

Note. Reaction time (RT) was calculated on correct trials only. Values used in the calculation of derived scores are highlighted in bold.

Table 5.2. Mean percent error (and SD) by Simon/ Location and Flanker condition for children in the control and ADHD groups, and between groups analyses of derived Simon and Flanker Effect scores.

Trial Type		**Control**	**ADHD**
Simon Condition/ Location	**Flanker Type**	Mean % Error (and SD)	Mean % Error (and SD)
Incompatible	No Flanker	**5.21 (6.65)**	**6.64 (6.29)**
	Identical	2.46 (4.63)	6.30 (6.47)
	Congruent Response	2.78 (7.23)	6.00 (7.41)
	Incongruent Response	5.90 (5.34)	10.19 (7.99)
Compatible	No Flanker	**1.93 (3.04)**	**5.05 (5.72)**
	Identical	2.81 (4.75)	4.74 (10.98)
	Congruent Response	1.74 (3.46)	6.35 (7.79)
	Incongruent Response	3.65 (3.95)	5.43 (6.98)
Centre	No Flanker	**3.30 (3.88)**	**5.99 (4.88)**
	Identical	**2.62 (2.41)**	**2.15 (2.78)**
	Congruent Response	**3.47 (4.86)**	**5.96 (7.99)**
	Incongruent Response	**4.53 (4.92)**	**6.23 (7.98)**
Derived Scores	**Calculation**	**Control**	**ADHD**
Simon Effect	No Flanker: Incompatible – Compatible (a – e)	3.28 (5.71)	1.59 (5.62)
	Between groups analysis	$t(38) = 1.70, p = .36$	
Feature Number Interference	Centre: Identical – No Flanker (j - i)	-0.68 (4.72)	-3.84 (4.84)
	Between groups analysis	$t(38) = 2.06, p = .047$	
Feature Type Interference	Centre: Congruent Response – Identical (k - j)	0.85 (5.21)	3.81 (6.75)
	Between groups analysis	$t(38) = 1.56, p = .13$	
Flanker Compatibility Effect	Centre: Incongruent Response – Congruent Response (l - k)	1.06 (6.77)	0.27 (5.87)
	Between groups analysis	$t(38) = 0.38, p = .71$	

Note. Values used in the calculation of derived scores are highlighted in bold.

Reaction Time: With RT as the dependent measure, there was a significant main effect of Group, $F(1, 228) = 7.37, p = .01$, a significant main effect of Simon Condition/Location, $F(2, 228) = 22.32, p < .0001$, and a significant main effect of Flanker Condition, $F(3, 228) = 40.04, p < .0001$. There were no significant two-way or three-way interactions.

The main effect of Group is characterized by longer RTs in the ADHD group than in the control group. Collapsed across all levels of Simon/Location and Flanker, the mean RT was 1017.24 ms (SD = 228.22) in the ADHD group and 850.95 ms (SD = 159.93) in the control group.

The other two main effects will be explored in more detail below.

Accuracy: A parallel mixed ANOVA was performed on percent error. There was a significant main effect of Group, $F(1, 228) = 5.73, p = .02$, and a significant main effect of Flanker Condition, $F(3, 228) = 5.05, p = .003$. There was also a marginally significant main effect of Simon Condition/Location, $F(2, 228) = 2.85, p = .06$. There were no significant two-way or three-way interactions.

The main effect of Group is characterized by a higher error rate overall in the ADHD group than in the control group. Collapsed across all levels of Simon/Location and Flanker, the mean percent error rate was 5.92 (SD = 4.42) in the ADHD group and 3.37 (SD = 2.30) in the control group.

The other two main effects will be explored in more detail below.

Specific Effects

Because there were no interactions between different levels of Simon Condition/Location and Flanker Type, it would be statistically permissible to collapse across the levels of one task to examine the effects in the other task. Since there is very little research on the Simon Effect and the Flanker Effect in children with ADHD, however, the remaining focus will be on the "pure" measures of each of these effects. That is, the Simon Effect will be examined on trials with no flankers (single digits in the periphery), and the Flanker Effect will be examined on trials presented at centre. Figure 5.1 highlights the trial types to be used in the next series of analyses.

Simon Effect

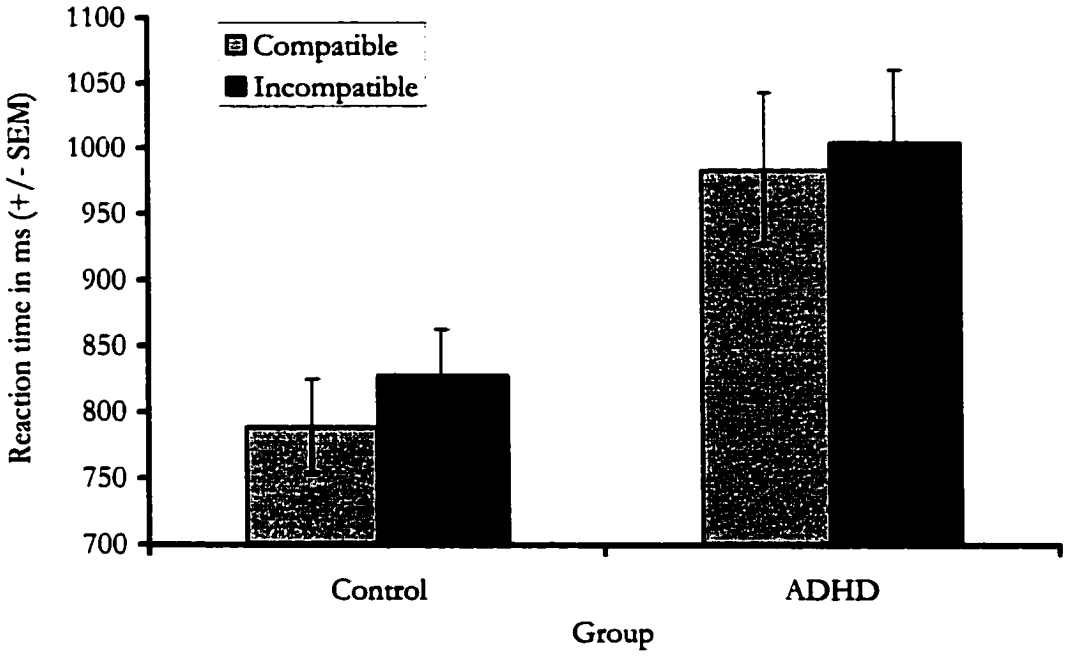
The pure Simon Effect was measured on trials with no flankers, with two mixed ANOVAs (one for RT and one for accuracy), each with one between subjects variable (Group) and one repeated measure (Simon Compatibility: Compatible and Incompatible).

Reaction Time: With RT as the dependent measure, there was a marginally significant main effect of Simon Compatibility, $F(1, 38) = 3.93, p = .0547$. [23] As portrayed in Figure 5.2, this effect is characterized by slower reaction times (M = 898.44, SD = 208.95) on Incompatible trials than on Compatible trials (M =

[23] The assumptions of homogeneity of variance and of correlations are both met, so no corrections need to be applied. When all four Flanker types are included in the analysis, the main effect of Simon Compatibility is significant, $F(1, 38) = 5.83, p = .02$.

866.17, SD = 223.27). Collapsed across Group, there is a Simon Effect of 32.27 ms.

Figure 5.2. Reaction Time by Simon Condition and Group (No Flanker Trials Only).



There was also a main effect of Group $F(1, 38) = 8.89, p = .005$. Collapsed across Simon Conditions, children with ADHD had longer reaction times (M = 993.43, SD = 223.45) than control children (M = 808.22, SD = 169.19).

The principle analysis of interest, the interaction between Group and Simon Compatibility, was not significant, $F(1, 38) = 0.35, p = .56$.[24] The lack of a significant interaction indicates that there is no group difference in the magnitude of the Simon Effect observed between children in the ADHD and control groups.

The main variable of interest is the magnitude of the Simon Effect. A single derived score was calculated for each participant, by subtracting RT on Compatible trials from that on Incompatible trials. The mean Simon Effect: RT for each group is presented in the bottom half of Table 5.1, along with the results of an unpaired t-test, which is statistically comparable to the nonsignificant interaction term in the ANOVA.

Accuracy: With accuracy as the dependent measure, there was a significant main effect of Simon Compatibility, $F(1, 38) = 7.07, p = .01$. More errors were made on Incompatible trials (M = 5.78, SD = 6.46) than on Compatible trials (M = 3.18, SD = 4.52). Collapsed across Group, there was a cost of 2.61% errors on Incompatible trials. There was no main effect of Group, $F(1, 38) = 2.24, p = .14$, and no interaction between Group and Simon Compatibility, $F(1, 38) = 0.86, p = .36$ (see Figure 5.3).

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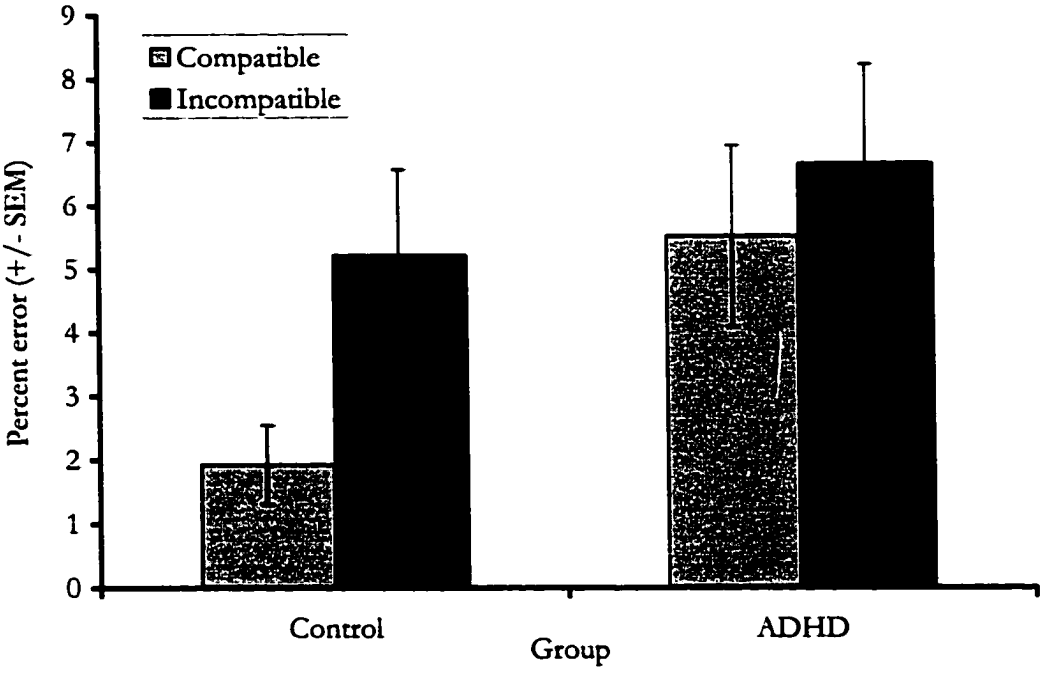
[24] This pattern is upheld when all four Flanker types are included in the analysis, $F(1, 38) = 0.08, p = .78$.

Figure 5.3. Percent Error by Simon Condition and Group (No Flanker Trials Only).



Simon Effect: Accuracy difference scores were computed for each participant by subtracting percent error on Compatible trials from that on Incompatible trials. The mean Simon Effect: Accuracy score in each group is presented in the bottom half of Table 5.2. The nonsignificant t-test comparing the difference scores between groups is comparable to the nonsignificant interaction between Group and Simon Compatibility in the ANOVA.

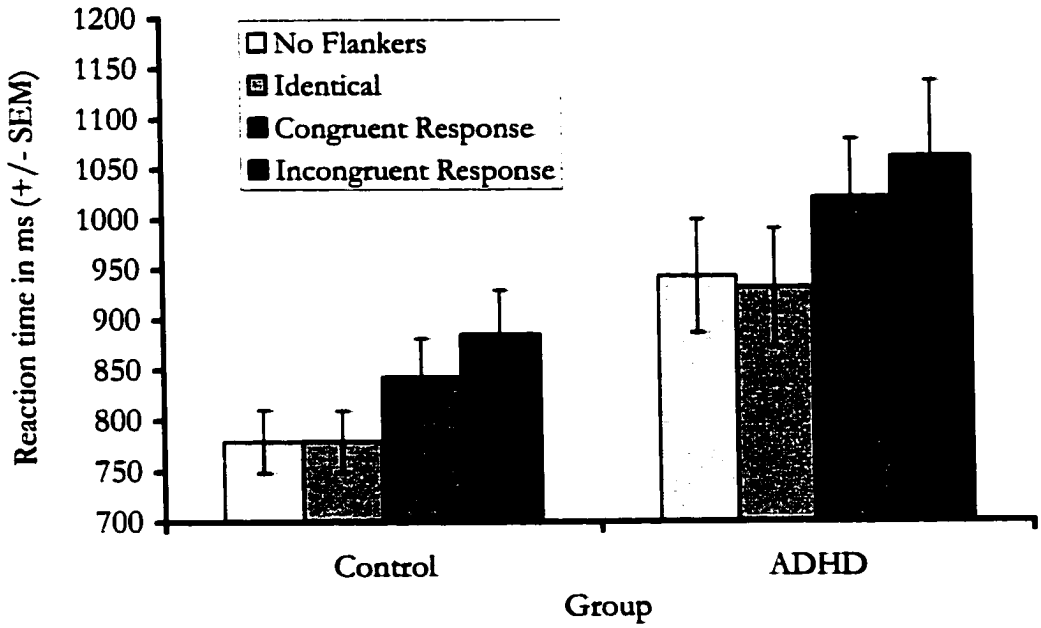
Flanker Effect

The Flanker Effect was measured on trials presented at fixation, with two mixed ANOVAs, each with one between subjects variable (Group) and one repeated measure (Flanker Type: No Flanker, Identical, Congruent Response,

and Incongruent Response). Separate analyses were conducted on RT and percent error.

Reaction Time: With RT as the dependent measure, there was a significant main effect of Group $F(1, 114) = 6.76, p = .01$, and a significant main effect of Flanker Type, $F(3, 114) = 25.09, p < .0001$. The main analysis, the interaction between Group and Flanker Type, was not significant, $F(3, 114) = 0.29, p = .84$. The lack of a significant interaction indicates that there is no group difference in the magnitude of the Flanker Interference Effects (see Figure 5.4).

Figure 5.4. Reaction Time by Flanker Type and Group (Centre Trials Only).



Across all Flanker Types, children with ADHD had longer RTs (M = 989.14, SD = 250.01) than children in the control group (M = 821.79, SD = 180.23).

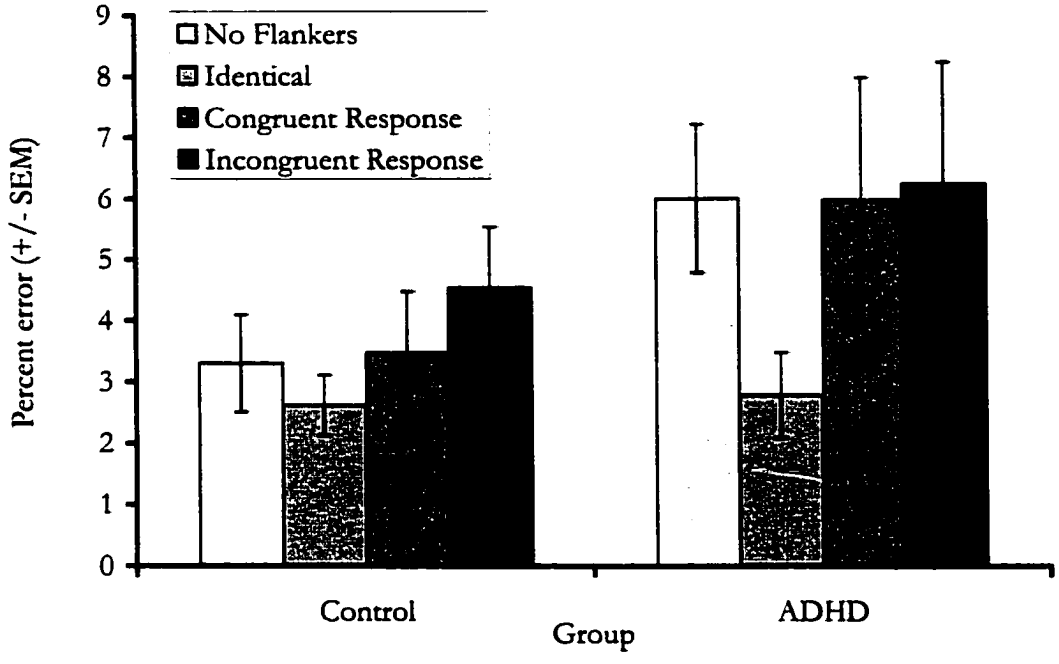
The main effect of Flanker Type indicates that RT differed according to whether and what type of flankers were presented. The Tukey-Kramer modification of the HSD test was chosen for post hoc analyses because all pairwise comparisons were made (Kirk, 1982; Shavelson, 1981). All comparisons were significant (alpha < .05) with the exception of the comparison between RT on No Flanker and Identical Flanker trials. Responses were faster on No Flanker trials (M = 844.71, SD = 199.77) than on Congruent Response (M = 914.52, SD = 220.96) and Incongruent Response (M = 955.92, SD = 263.07) trials, faster on Identical Flanker trials (M = 840.21, SD = 200.27) than on Congruent Response or Incongruent Response trials, and faster on Congruent Response trials than on Incongruent Response trials.

Three difference scores were calculated for each participant to quantify the different types of Flanker interference (see Figure 5.1 for examples of stimuli in each calculation). Feature Number Interference was computed by subtracting RT on No Flanker trials from that on Identical Flanker trials. Feature Type Interference was computed by subtracting RT on Identical trials from that on Congruent Response trials. The Flanker Compatibility Effect (FCE) was computed by subtracting RT on Congruent Response trials from that on

Incongruent Response trials. The mean of each difference score for each group is summarized in Table 5.1. It is important to note that because two of the difference scores are computed with the same value (RT on Congruent Response trials), these scores are non-orthogonal. T-tests were performed on each difference score to examine group differences (ADHD versus control) in the magnitude of each type of the interference. None approached significance (see Table 5.1).

Accuracy: With percent error as the dependent measure, there was a significant main effect of Flanker Type, $F(3, 114) = 3.48, p = .02$, no main effect of Group, $F(1, 114) = 2.07, p = .16$, and no interaction between Group and Flanker Type, $F(3, 114) = 1.07, p = .37$ (see Figure 5.5). Collapsed across Group, percent error rates on each of the Flanker Types were as follows: No Flankers, M = 4.38, SD = 4.45; Identical Flankers, M = 2.43, SD = 2.54; Congruent Response Flankers, M = 4.47, SD = 6.33; Incongruent Response Flankers, M = 5.21, SD = 6.28. Post hoc Tukey-Kramer HSD tests yielded only one significant pairwise comparison. There were significantly fewer errors on Identical Flanker trials than on Incongruent Response trials. For the overall mixed ANOVA, the assumption of homogeneity of variance was met, but the assumption of homogeneity of correlations was not. Unpaired t-tests comparing the magnitude of difference scores between groups are more robust in the presence of this type of violation.

Figure 5.5. Percent Error by Flanker Type and Group (Centre Trials Only).



Flanker Effect: Accuracy difference scores were computed in the same manner as the RT difference scores. The mean scores for each group are presented in Table 5.2, along with between groups comparisons. There was one statistically significant result. Children in the ADHD group showed an advantage on trials with Identical Flankers (as compared to trials with No Flankers) whereas control children did not. Figure 5.5 shows that children in the ADHD group had fewer errors on the Identical Flanker trials than on any of the other trial types. This pattern was re-examined at all three levels of Simon Condition/Location. Collapsed across location, children with ADHD do not

show a facilitation associated with Identical Flankers (Feature Number Interference = -1.50 %, SD = 3.10), and there is no group difference in the amount of Feature Number Interference, $t(38) = 0.62, p = .54$.

Summary of Simon and Flanker Analyses

Collapsed across all Simon and Flanker trial types, there was a main effect of Group in both RT and accuracy. Overall, children with ADHD responded 166.29 ms more slowly and made 2.55% more errors than children in the control group. There was also a main effect of Simon Compatibility/ Location (including the Centre condition) in RT, and a significant main effect of Flanker Type in both RT and accuracy. There were no significant two-way or three-way interactions in either RT or accuracy.

The nature of the Simon Effect was explored in detail on trials with no flankers and in analyses without the Centre condition (see Figures 5.2 and 5.3). Across all participants, there was a significant cost on Incompatible trials of 32.27 ms and 2.61% errors relative to Compatible trials. There was a main effect of Group in RT but not in accuracy. There was no difference in the magnitude of the Simon Effect (39.58 ms and 3.28 % errors in the control group, and 21.31 ms and 1.59% errors in the ADHD group) between groups.

The nature of the Flanker effect was explored in detail on trials presented at fixation (see Figures 5.4 and 5.5). There was a main effect of Flanker Type in both RT and accuracy. Across both groups, responses were faster on No

Flanker trials than on the Congruent Response or Incongruent Response trials, faster on Identical Flanker trials than on Congruent Response or Incongruent Response trials, and faster on Congruent Response trials than on Incongruent Response trials. More errors were made on the Incongruent Response trials than on the Identical Flanker trials. There was a main effect of Group in RT but not accuracy. There was a difference in the magnitude of facilitation associated with Identical Flankers when accuracy was the dependent measure. There was no difference in the magnitude of Feature Type Interference or the Flanker Compatibility Effect between groups in either RT or accuracy.

Discussion

The first thing to note is that overall, participants showed the expected interference effects (costs in RT or accuracy) as a result of the irrelevant location of stimuli (Simon Effect) and the irrelevant flanking stimuli (Flanker Effect).

Across all participants, there was a significant cost associated with Incompatible Simon trials in both speed and accuracy. This is consistent with the broad literature on the Simon Effect in adults (Lu & Proctor, 1995), and with Tagliabue et al.'s (2000) demonstration of a Simon Effect in young children (ages 5 to 8 years). These results of this study extend the finding to older children (ages 9 to 13 years) with and without ADHD.

Three different types of Flanker interference were measured. There were RT costs related to Feature Type Interference (Congruent Response versus Identical Flankers) and the Flanker Compatibility Effect (Incongruent versus Congruent Response Flankers). Both of these findings are replications of patterns reported by Enns and Akhtar (1989) and Jonkman et al. (1999). There was no cost related to Feature Number Interference (Identical versus No Flankers). In fact, children in the ADHD group showed an advantage (made fewer errors) on Identical Flanker trials. Enns and Akhtar (1989) reported a cost in reaction time associated with the presence of identical flankers in their sample of young children (ages 4 to 7 years) and young adults. Jonkman et al. (1999) showed no difference associated with identical flankers in their sample of older children (ages 7 to 13 years) with and without ADHD. Jonkman et al.'s (1999) data show no trend toward an advantage in reaction time or accuracy in either group. In this study, the children in the ADHD group made fewer errors when all three numbers were the same (e.g., "222"). One might infer that if children with ADHD have problems inhibiting distracters, on trials of this type, the distracters could have helped their performance. Children with ADHD did not, however, have the same advantage when the flankers corresponded with the same response as the target (e.g., "121"). This is an interesting finding and one worthy of further exploration, but it is not clear that it relates directly to interference control. Furthermore, there are a number of issues that suggest

more research is needed before any strong conclusions can be drawn. This pattern is not consistent with earlier studies using the Flanker task, and it occurred only at the centre location, not at the peripheral locations.

Over all trial types, children with ADHD responded more slowly and made more errors than children in the control group. Some may argue that these findings should be taken as evidence of impairment in the ADHD group. To a certain extent, this is true. At a global level, children with ADHD did not perform as well on the Simon and Flanker tasks. These tasks, however, were designed to assess more than global performance on a choice reaction time task. In fact, the pattern that children with ADHD have longer mean RTs on information processing tasks than control children has been demonstrated many times before, and could relate to a variety of factors (see Douglas, 1999, for a discussion of this issue). These tasks were designed in such a way to measure well-known information processing effects as differences between specific conditions in order to determine the efficiency with which irrelevant, interfering information could be inhibited.

Group differences in the amount of influence that the irrelevant information had on performance were assessed by examining the interaction effects in the mixed ANOVAs and between groups t-tests of derived difference scores. There were no significant findings of this nature, either in the overall

analysis (with all Simon and Flanker conditions) or in the analyses of the pure Simon and Flanker Effects.

To summarize, children with and without ADHD showed interference related to irrelevant locations and irrelevant flanking stimuli. Overall, children with ADHD responded more slowly and made more errors than control children. On centre trials only, they also showed a reduction in errors associated with the presence of Identical Flankers. Other than this, there were no group differences in the degree of impact that the irrelevant locations or flankers had on performance. That is, there were no group differences in the Simon Effect or the Flanker Effect, two measures of the interference control or response competition.

Chapter 6. Inhibition of Return

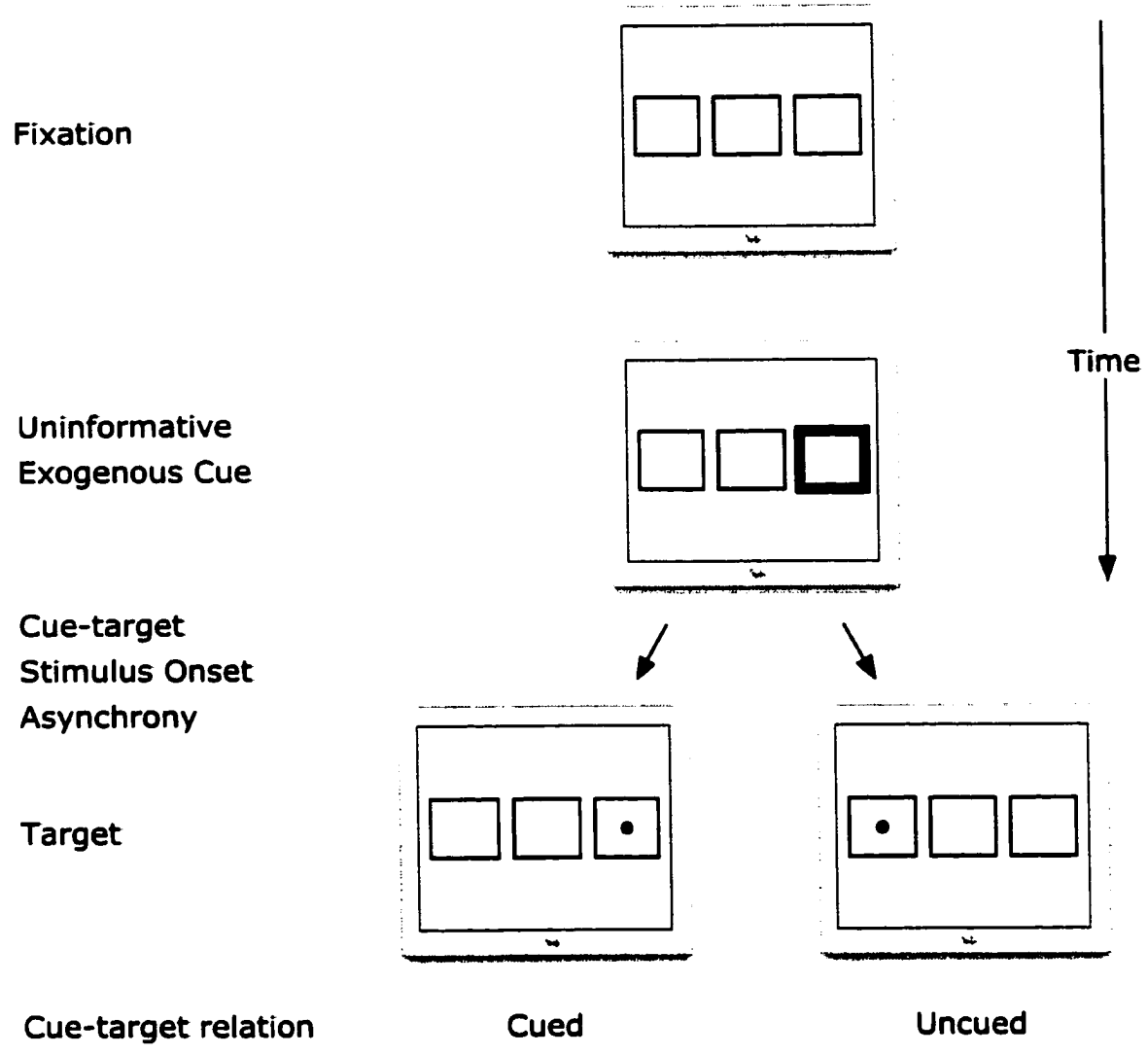
In studies of attentional orienting, inhibition of return (IOR) is the finding that, under certain conditions, participants are slower to respond to items in locations that were previously attended (see Klein, 2000, for a review). Because IOR is observed following exogenously generated, but not endogenously generated shifts of attention, the concept of exogenous covert orienting will be defined first, and some critical elements of visuospatial orienting tasks will be described. As well, previous studies using these tasks as a means of understanding attention in ADHD will be put into this context.

Covert orienting is the directing of attention independently of gaze direction. For example, if you were told that a bear was creeping up upon you from the left, you could focus your attention toward the left, without having to turn either your head or eyes in that direction. You should be faster to fight off the bear if it appeared to your left after the warning, since you had directed your attention there, as compared to if it unexpectedly appeared to your right. Taking this example into the laboratory, in the visuospatial orienting task developed by Posner (1980), covert orienting is inferred by participants' faster reaction times to stimuli which appear in locations to which they had just previously directed their attention, in comparison to reaction times to stimuli in other locations.

Participants typically view a computer screen with a central fixation point and two boxes in the periphery (to the left and right of fixation). They are instructed to focus their attention on the central fixation point and to respond (typically with a button press) as quickly as they can to the onset of a "target" in the periphery (e.g., an asterisk inside one of the boxes).

On most trials, targets are preceded by a "cue", which may indicate the location in space containing the target (such trials are usually referred to as Valid or Cued) or may indicate the location in space that does not contain the target (Invalid or Uncued). Cues can appear in the periphery (e.g., the brightening of one of the peripheral boxes) or at fixation (e.g., an arrow pointing to one side). See Figure 6.1 for a representation of the sequence of events in a typical visuospatial orienting task with peripheral cues.

Figure 6.1. Sequence of events in a visuospatial orienting task with exogenous cues.



Cues can exert two different types of control on orienting (Posner, 1980; Klein & Shore, 2000). Orienting is considered under exogenous control when a localizable stimulus (such as a flash in the periphery) pulls attention towards a given location. In its pure form, exogenous orienting is elicited when the

peripheral event carries no information about the upcoming target's location (that is, a flash on the right is equally likely to be followed by a target on the left or right). Exogenous orienting is rapid and is generally considered involuntary and automatic (though this characterization now seems to be context dependent; cf. Folk, Remington, & Johnston, 1992).

Orienting is considered under endogenous control when information that is presented to, or picked up by, the observer leads to the strategy of pushing attention toward a given location. For example, an arrow at fixation informs the observer where the target is likely to appear, by correctly pointing to the location of the target on the majority (e.g., 80%) of trials. Endogenous orienting is voluntary and requires "top-down" control.

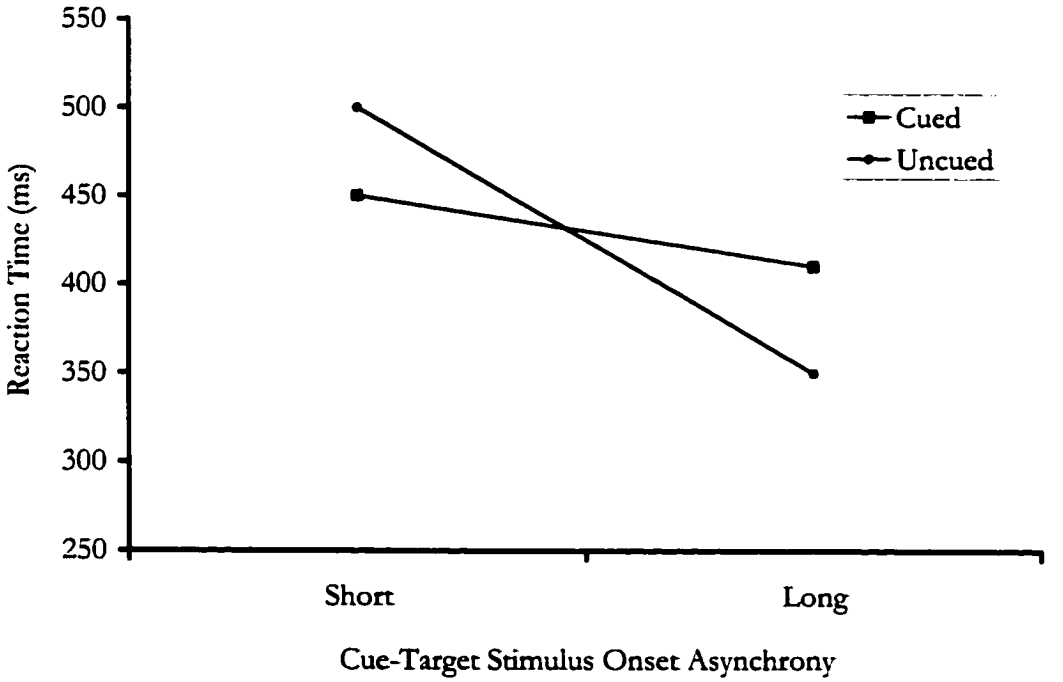
Many studies of covert orienting have assessed a hybrid of these two forms of control, by using peripheral cues which are informative about the upcoming target's location. Suppose, for example, that a flash in the periphery signals the location of the target on the majority of trials. Such a cue may reflexively pull attention to its location, but in addition, the observer may decide not to try to resist such a cue's effect and will likely leave attention at the cue's

location because the probability manipulation provides incentive to do so. Here, this type of cue (that is, a peripheral cue that is informative about the target's upcoming location) is called a mixed (endogenous and exogenous) cue. Pure exogenous cues, on the other hand, are uninformative with regard to the location of the upcoming target.

A critical variable in the visuospatial orienting task is the stimulus onset asynchrony between the cue and the target (cue-target SOA). This refers to the gap in time between the onset of the two stimuli. At short cue-target SOA's, RT to cued targets is faster than RT to those preceded by uncued targets (like the bear example, above). This general pattern holds whether the cues are exogenous, endogenous, or mixed. At long cue-target SOA's, and when exogenous cues are used, the pattern reverses: responses to uncued targets are faster than to cued targets. This pattern, called Inhibition of Return (IOR) is generally assumed to reflect a bias against "returning" to the cued location from which attention has just been withdrawn (Posner & Cohen, 1984; Rafal, Calabresi, Brennan, & Sciolo, 1989; see Klein, 2000 and Taylor & Klein, 1998, for reviews). See Figure 6.1 and 6.2 for a representation of the procedures and

typical findings in studies of IOR (idealized findings from the classic study by Posner & Cohen, 1984, are presented in Figure 6.2).

Figure 6.2. Idealized reaction time pattern on a visuospatial orienting task with exogenous cues and differing stimulus onset asynchronies.



By definition, covert orienting occurs independently of eye movements. In contrast, overt orienting is the orienting of attention with a shift in eye gaze (with or without orienting of the head). There are two ways to control for eye movements in order to study only covert orienting. The first is to use very short

(i.e., less than 200 ms) cue-target SOA's, so that the measured orienting occurs prior to the time necessary to initiate an eye movement. Secondly, at longer cue-target SOA's (>200 ms), eye movements should be monitored to ensure that no overt orienting is taking place. Furthermore, it has been argued that overt orienting could also play a role even at short cue-target SOA's (see Rafal & Henik, 1994). If an eye movement is made after the target disappears but before the response to the target has been initiated (as might be likely in the trials with short cue-target SOA's and no eye movement monitoring), the neural mechanisms underlying overt orienting can still be activated, and can potentially change the nature of the target processing which continues after the target is removed, eventuating in a response. If the goal is to study covert orienting of attention, therefore, eye movements should be monitored, participants should be given feedback when they make eye movements and trials with eye movements should be excluded from analyses. Otherwise, not only would it be unclear which type of orienting is being studied, but also, overt orienting could interfere directly with RT on a covert orienting task. For example, looking

towards a flash in the periphery could slow the RT to a target on the opposite side.

Monitoring eye movements is probably even more important when studying group differences between children with ADHD and control children. Both Munoz, Hampton, Moore, and Goldring (1999) and Ross, Hommer, Brieger, Varley, and Radant (1994) have demonstrated that children with ADHD are less able to inhibit eye movements than control children. A study of group differences on an orienting task with no eye movement monitoring may, therefore, be studying disproportionately more overt orienting (versus covert orienting) in the ADHD group than in controls.

To date, there have been seven published studies comparing children with ADHD to control children on versions of Posner's visuospatial orienting task. Four of the studies, those by Aman, Roberts, and Pennington (1998), Nigg, Swanson, and Hinshaw (1997), Swanson, Posner, Potkin, Bonforte, et al. (1991), and Wood, Maruff, Levy, Farrow, & Hay (1999), used an orienting task with mixed cues. Pearson, Yaffee, Loveland, and Norton (1995) examined both mixed and endogenous cues. McDonald, Bennett, Chambers, and Castiello

(1999) studied only endogenous cues. Carter, Krener, Chaderjian, Northcutt, and Wolfe (1995b) used exogenous and endogenous cues. To summarize a review by McLaughin and Klein (2002), while each study reported group differences on some aspect of visuospatial orienting, there was no identifiable pattern across studies. The inconsistent and, at times, contradictory findings were probably due in part to differences in design (e.g., type of cue, cue-target SOA's) and procedure (e.g., failure to control for eye movements). The only consistent finding in this review was that children with ADHD showed a general pattern of slowing. In all but one study (Aman et al, 1998), children with ADHD had higher RTs overall (regardless of cue condition or cue-target SOA). As mentioned previously, this is a common pattern found with groups of children with ADHD.

The only study of these seven that permits an assessment of IOR is that of Carter et al. (1995b). Targets were preceded by either peripheral flashes (exogenous cues) or arrows at fixation (endogenous cues). On trials with peripheral cues, cues were uninformative with regard to the location of the target (a cue on the left was equally likely to be followed by a target on the left

or right). One third of the targets were preceded by valid cues, one third by invalid cues, and one third by neutral cues (both peripheral boxes flashed). Endogenous arrow cues correctly predicted the location of the target 80% of the time. The cue-target SOA was either 150 or 800 ms.

In this study, eye movements were monitored by the experimenter, who was sitting behind the computer monitor. While probably reliable, this methodology should be seen as being inferior to automated eye tracking technology. Carter et al. (1995b) reported that in the endogenous (arrows) condition, eye movements occurred on 14% and 17% of trials in the control and ADHD groups, respectively. In the exogenous (peripheral flash) condition, eye movements occurred on 21% and 27% of trials in the control and ADHD groups, respectively. The higher percentage of eye movements following a peripheral flash versus a central arrow is to be expected, given the involuntary and reflexive nature of eye movements to exogenous cues. Although the percentage of eye movements was not statistically different across groups, they are suggestive of a trend toward more eye movements in the ADHD group.

Despite the high proportion of overt orienting taking place, Carter et al. (1995b) chose to retain trials with eye movements in their analyses.

With simple detection, it is important to use a small proportion of trials on which the target never appears. "Catch" trials are intended to prevent participants from responding prior to actually seeing the target, and to measure the frequency with which such a strategy is employed. Carter et al. did not include catch trials, even though their target task was a simple detection task (to press the button when the target appears). To deal with anticipations, they used the common approach of rejecting trials with abnormally fast RTs (less than 150 ms). Even with these trials excluded, however, these data could still include unknown numbers of responses initiated prior to the onset of the target and reaction time effects compromized by unassessable response biases (e.g., the tendency to respond prior to the onset of the target). Further, Carter et al. reported that children with ADHD made more anticipation errors than controls, at least following endogenous cues, suggesting that the adoption of this strategy may be related to diagnostic status. Despite these procedural limitations (retention of trials with eye movements, failure to include catch

trials), Carter et al. (1995b) is, methodologically speaking, one of the stronger studies of visuospatial orienting in ADHD (McLaughlin & Klein, 2002).

Recall that with Carter et al.'s design, IOR (reflected as slower responses to cued targets) would be expected when exogenous cues preceded the target by 800 ms. This pattern was observed in both the ADHD and control groups. Carter et al. reported no group differences in RT to cued targets, uncued targets or to targets which followed neutral cues, when the cue-target SOA was 800 ms. There did, however, appear to be a non-significant trend, that children with ADHD showed approximately 30 ms less IOR than control children (when IOR is calculated as RT on uncued trials minus that on cued trials).

An intriguing theory proposed originally by Klein (1988; see also Klein & MacInnes, 1999 for recent supporting data) is that IOR is a process that facilitates visual search by biasing attention or motor movements away from locations that have already been searched, and towards new locations. Barkley's (1997) inhibitory control model of ADHD would predict less IOR in children with ADHD, or less of a cost at cued locations at long cue-target SOAs. This is consistent with the trend toward reduced IOR observed by Carter et al. (1995). The following is an attempt to determine whether this suggestive pattern will be maintained in the face of the following methodological improvements: the

inclusion of catch trials, and the exclusion of trials with eye movements identified with eye tracking technology.

Method

Participants

The forty children described in Table 2.3 participated. Five participants in the ADHD group (#'s 4, 9, 16, 22 and 34 from Table 2.2) were excluded from the main analyses because of insufficient or statistically outlying data (see Data Analysis section for details). The final sample was made up of 24 control children and 11 children with ADHD. Participants were able to wear their prescription lenses during testing.

Procedures

Eye tracking system. Participants' eye position and eye movements were monitored using the video-based EyeLink eye tracking system (S.R. Research Ltd., Toronto, Canada). Each participant wore a head apparatus that allowed a small digital camera to monitor the position of the right pupil every 4 ms. The EyeLink system factors out head position and head movement with an infrared tracking system that measures the position of the apparatus worn by the participant in relation to the computer monitor. Neither the distance from the screen nor the head position needs to be fixed. Using this system, the speed and direction of eye movements can be monitored in relation to the location and

timing of stimuli presented on the IBM compatible computer, to which the EyeLink is connected.

Calibration of eye tracking equipment. Prior to the start of the experiment, the equipment must be calibrated for each participant. Once the apparatus is positioned squarely on the participant's head and the camera is pointed at the eye, the participant is instructed to follow a small white circle (.5 visual degrees in diameter) as it is presented in nine different positions on the computer screen. In the calibration phase, the Eyelink calculates the difference between the coordinates of the target on the screen and the landing points of the saccades made by the participant. If there are only small differences (less than one visual degree for any one of the targets), then the steps are repeated in the validation phase. In this second phase, the participants' own landing points during the calibration phase are compared to those in the validation phase. The experiment is only initiated if the two sets of landing points (calibration and validation) for all targets are less than one visual degree apart.

For most participants, calibration and validation is straightforward. Because it depends on a variety of factors, however, including good positioning of the camera, good fixation stability, and accurate, consistent saccades, it sometimes takes several attempts before validation is established.

Anecdotal findings (non-blind experimenter observations) in this experiment were that younger children and children with ADHD had a harder

time completing the calibration and validation phases of this task. This was not always the case, as some older control subjects had difficulty, and some younger children with ADHD did not. In other experiments conducted in the same laboratory with the same equipment, there is anecdotally understood to be a proportion of university undergraduate volunteers who also have a hard time calibrating as well. An association between calibration difficulty and age or diagnostic status would be consistent with the findings of Munoz and colleagues and Ross and colleagues, who have shown that saccadic control improves with age (Munoz, Broughton, Goldring, & Armstrong, 1998; Ross, Radant, & Hommer, 1993) and is not as good in children with ADHD as in controls (Munoz, et al., 1999; Ross et al., 1994). Unfortunately, the EyeLink system does not collect data on eye positioning during the calibration and validation phases. We can hypothesize that those who took a long time to achieve validation had difficulty making accurate saccades to small the targets or had difficulties with fixation stability.

One indirect finding that supports this hypothesis is that, even though all participants had roughly the same amount of time available to complete this task (approximately 50 minutes), there was a wide range in the number of trials completed (50 to 300, including practice trials). The number of trials completed is positively correlated with age ($r = 0.31, p = .0495$) and is lower in the ADHD group $t(37) = 4.75, p < .0001$. Of course, in addition to time taken to calibrate

and validate the EyeLink system, the number of trials completed could be influenced by many other factors, including time taken to learn the task, time taken to explore the equipment, length of breaks (a snack break was scheduled in the middle of two 25 minute segments of the IOR task and some participants requested additional brief rests during the task), whether more than 50 minutes was available, and, as will be discussed later, time taken to initiate each trial (the latter of which is also related to saccadic control).

Stimuli and Task. Custom software was written by J. MacInnes for the purposes of this experiment to be run with the EyeLink eye tracking system and an IBM compatible computer. Each participant was seated approximately 71 cm away from the computer monitor. Head position was not fixed, although some participants chose to use a chin rest for comfort. Throughout the experiment, there were three white boxes presented on a black computer screen. Each box measured 1.4 by 1.4 visual degrees. One was positioned in the centre of the screen, the other two were 4 visual degrees to the left and right of centre. The box was drawn with a thin line measuring 1 pixel (22 pixels = 1 visual degree). Box "brightening" (cuing) was achieved by increasing the width of the line by 1 pixel. The fixation point was a small, white circle measuring .5 visual degrees with a .1 visual degree black circle in its centre. The target stimulus was identical to the fixation stimulus, except that it did not have a black circle in the centre.

Prior to the start of each trial, the participant was required to focus on the fixation point in the centre of the screen. When ready, he or she was to press a button (on a button pad similar to those used for computer games) to initiate the trial. The trial only began if the participant's eyes were stable and focused on the fixation point at the time of the button press. This ensured that any eye movement measurements would be calculated accurately from fixation.

After 500 ms, one of the two peripheral boxes "brightened" (see above) for 300 ms. The left box brightened on a random 50% of the trials, the right on the other 50%. Targets could appear either 150 ms (Short SOA) or 1000 ms (Long SOA) after the onset of the cue (with a 50% probability of each SOA). Ten percent of the trials at each SOA were Catch trials, meaning that no target appeared. On trials that a target did appear, it was equally likely to appear on the same side as the cue (Cued) as the opposite side (Uncued).

Note that in the Short SOA condition, the cue appeared 150 ms after the onset of the box brightening, the latter of which had a total duration of 300 ms, meaning that the cue and target overlapped by 150 ms. On Long SOA trials, 200 ms after the offset of the cue, the centre box brightened for 300 ms, in order to draw the participant's attention back to centre.

Each participant's task was to press the response button on the button box as quickly as possible when he or she noticed the onset of the target. If the

button was pressed on a catch trial, or if the participant failed to press the button on a trial with a target, a feedback tone would sound.

Each participant was instructed to maintain his or her gaze at centre fixation for the duration of the trial. A saccade was defined as a movement in eye position greater than 1 visual degree, with a velocity of 30 degrees per second or greater. If an eye movement was detected, if eye position drifted outside of a 2 degree radius, or if the participant blinked, a short feedback tone sounded. As discussed in Chapter 2, for the most part, feedback regarding task performance was deliberately not given. Feedback about eye position was essential in order to help participants to be aware of when they were making eye movement errors, because this is not something of which observers (adult and child alike) are always conscious. Also, the main focus was on trials with no eye movements, so feedback regarding eye movements should not interfere directly with the dependent variable of interest. In order to minimize the likelihood that participants would get frustrated by the feedback tones, participants were told in advance that it was normal to have some "beeps", and trials with eye movements were not recycled. Eye position was also monitored on a separate computer monitor that was out of view of the participant, and specific feedback regarding eye movements was provided when necessary.

Participants completed a block of 50 practice trials, followed by 50, 100, 150, 200, or 250 experimental trials. All participants had the same amount of

time available for this task (approximately 50 minutes). The number of trials completed depended on the time taken for calibration and validation (discussed above) and the time taken to initiate each trial, as well as on other factors such as the time taken to learn the task and length of breaks or pauses between trials.

Instructions to Participants. Participants were told that they would be seeing a small circle appear in one of the two peripheral boxes, and that their job was to press the button on the button pad as quickly as possible when they saw it. They were told that on some trials, no target would appear, so they needed to wait until they saw the target before responding. They were instructed to keep their eyes on the centre throughout the duration of the task, and were told that if their eyes moved or if they blinked, they would hear a "beep". They were told that it was normal to have some "beeps", but that they were to try to keep their eyes on the centre. Performance was monitored closely during the practice block, and additional instructions were given if necessary.

Data Analysis. The computer recorded whether one or more eye movements or blinks were made on a given trial, and trials with eye movements or blinks were excluded. Button press reaction time on non-catch trials was recorded relative to the onset of the target and trials with responses faster than 150 ms and slower than 1000 ms were excluded.

Four participants (#'s 4, 16, 22 and 34; one girl and three boys with ADHD) with no useable trials in at least one of the four conditions (Short SOA

and Long SOA by Cued and Uncued) were excluded from all analyses. Each of these participants had completed 50 or fewer experimental trials, many of which were excluded. One of the main dependent measures was a single IOR score (defined below). Participant #9 (from the ADHD group) was excluded because his IOR score was greater than 4.5 SD's from the overall mean.

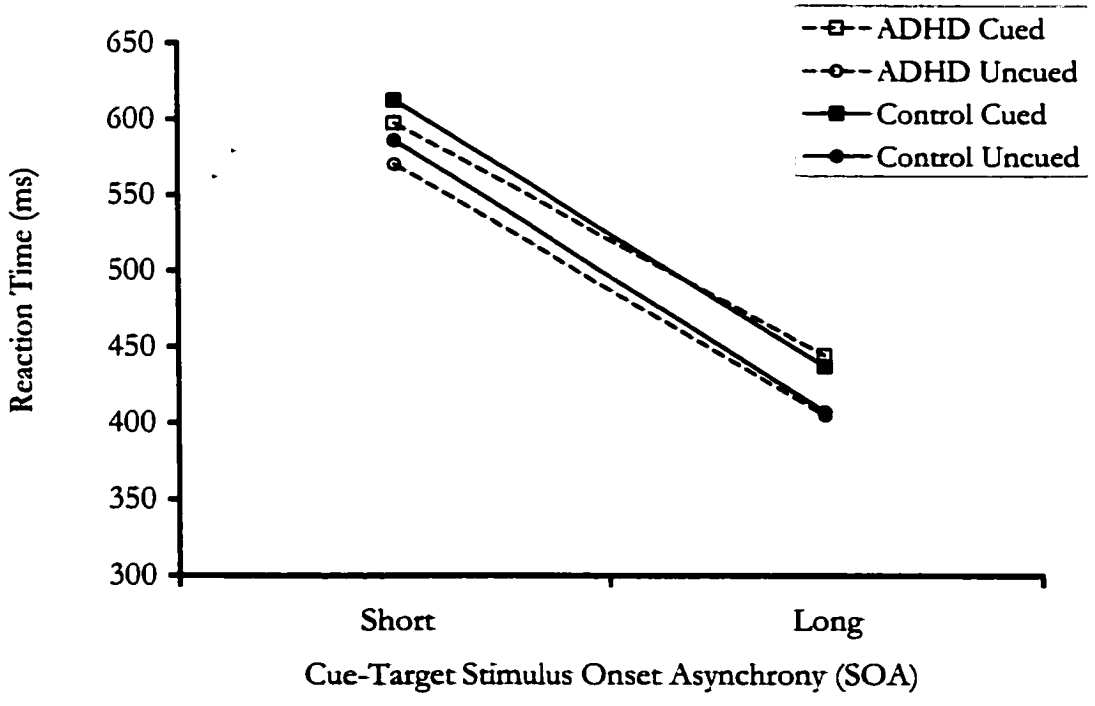
Eye movements were made on a high percentage of trials (42.94%, SD = 26.92 and 37.78%, SD = 20.04 of the trials in the ADHD and control groups, respectively). There was no group difference in the percentage of trials with eye movements, $t(33) = 0.63, p = .53$. Less than 1% of trials in each group were excluded because of blinks, and there was no group difference in the number of trials with blinks between groups, $t(33) = 0.73, p = .47$. There was also no group difference in the percentage of responses faster than 150 ms, $t(33) = -0.38, p = .70$ (M = 1.67%, SD = 2.50 of trials in the ADHD group and M = 2.05%, SD = 2.83% of trials in the control group). Finally, there was no group difference in the percentage of responses slower than 1000 ms, $t(33) = -1.81, p = .08$ (M = 3.16%, SD = 2.90 of trials in the ADHD group and M = 5.56%, SD = 3.93 of trials in the control group).

Results

Reaction time to Cued and Uncued targets at each cue-target SOA by Group is presented in Figure 6.3. The data were analyzed with a mixed ANOVA, with one between subjects variable, Group (ADHD and control) and

two repeated measures, SOA (Short versus Long cue-target SOA) and Cuing (Cued versus Uncued). There was no main effect of Group, $F(1, 33) = 0.05, p = .82$. There was a main effect of SOA, $F(1, 33) = 98.25, p < .0001$. As portrayed in Figure 6.3, RTs were faster at the Long SOA. There was a main effect of Cuing, $F(1, 33) = 14.95, p < 0.001$. Collapsed across Group and SOA, RTs to Cued targets were 29.67 ms slower than RTs to Uncued targets. There were no significant two-way or three-way interactions between Group, SOA, and Cuing.

Figure 6.3. Reaction time to Cued and Uncued Targets at Short and Long SOAs by Group.



Difference scores reflecting IOR, or the cost associated with cuing at the Long SOA, were calculated for each participant, by subtracting RT on Cued

trials from that on Uncued trials. The mean IOR score for children in the ADHD group was 39.32 ms (SD = 59.65), and that for children in the control group was 29.92 ms (SD = 48.11). There was no difference in the size of the IOR effect between groups, $t(33) = -0.50, p = .62$.

Performance on Catch trials (trials with no target) was analyzed separately. There was no group difference in the percentage of Catch trials with button presses between ADHD children (M = 22.16, SD = 12.77) and control children (M = 16.20, SD = 13.97), $t(33) = 1.20, p = .24$.

Discussion

The principle analysis of interest in the visuospatial cuing task was a comparison of the amount of inhibition of return (IOR) between groups. There was no difference in the RT cost associated with Cued trials at the Long SOA (relative to RT on Uncued trials) between groups. That is, there was no group difference in IOR. The non-significant trend toward reduced IOR in ADHD observed by Carter et al. (1995b) was not replicated.

In addition to the main result, there are many other performance measures on this task that will be discussed. Firstly, collapsed by Group and Cuing, responses were faster at the long cue-target SOA. This is a common and expected pattern (see Figure 6.2), which is due to the increased preparedness at the longer interval.

Collapsed by Cuing and SOA, there was no difference in RT between groups on this simple detection task. This is in contrast to the common observation that children with ADHD have longer RTs than control children (see Douglas, 1999), a pattern observed on the Simon and Flanker choice reaction time tasks in this study (Chapter 4).

One unexpected finding was the lack of facilitation (shorter responses to Cued targets) at the short interval in both groups. Recall that at short cue-target SOA's, responses to Cued targets are typically faster than those to Uncued targets (see Figure 6.2). The short cue-target SOA in this study was 150 ms, which is within the range of intervals at which facilitation is usually observed (see Klein, 2000). McLaughlin and Klein (2001) tested undergraduate students using the same task parameters and equipment, and found the same pattern, suggesting that this observation is not simply because of the age of the participants. Looking more closely at the parameters of this task, it is possible that the reason for the lack of facilitation at the short interval is because of the temporal overlap between the cue and target. Recall that the peripheral box brightened for a total of 300 ms, and the target appeared 150 ms after its onset. It may be that the luminance change associated with target onset was not substantial enough to exogenously attract attention in the presence of the cue. In contrast, a target with a sudden onset appearing on side opposite to the cue may have had a larger impact.

The more general performance of participants on this version of the visuospatial orienting task will inform our interpretation of other studies of covert orienting in children. Children made at least one eye movement greater than one visual degree on a high percentage of trials (42.94% and 37.78% of trials in the ADHD and control groups, respectively). There was no group difference in the percentage of eye movements when a maximum of one eye movement was counted per trial. These percentages are almost twice as high than those reported by Carter et al. (1995b), who reported eye movements on 27% and 21% of trials for the ADHD and control groups (and no group difference). The lower rate observed by Carter et al. is likely due to the fact that they identified eye movements through experimenter observation, and may not have identified all saccades, especially those with small amplitudes. Ross et al. (1994) measured horizontal eye movements to a peripheral stimulus with an infrared eye-tracking device and reported more "premature saccades" among their ADHD group than among their control group (29.3% and 15.1% respectively). In Ross et al.'s study, only saccades made in the direction of the target, and at least half of the distance to the target were counted as premature saccades. This difference in the operational definition of an eye movement error probably accounts for the lower rate observed by Ross et al. (1994). The percentages observed in this study are most comparable to the rate of "intrusive saccades" reported by Munoz et al. (1999), who monitored eye movements with

electrooculography. Munoz et al. counted the number of eye movements greater than 2 visual degrees during periods when the participants were to be focused on the centre (either with or without a fixation point illuminated), and found that children with ADHD made intrusive saccades at a higher rate (0.52 saccades per second) than controls (0.23 saccades per second). Taken together, these data suggest that previous studies of covert orienting in children without eye movement monitoring were probably studying overt orienting on a high proportion of trials, and probably disproportionately more so in children with ADHD.

A related finding was that children in the ADHD group were not able to complete as many trials on this task as the control children. This result could have been due to a variety of factors, including time taken to learn the task, length of breaks and pauses between trials, but could also be related to difficulties calibrating the eye tracking equipment (which requires reliable saccades and good fixation stability) and ability to initiate trials easily (which requires good fixation stability). The latter interpretation would be consistent with Munoz, et al.'s (1999) reports that children with ADHD do not have as good fixation stability as those without ADHD. It is also worth noting that the five participants excluded for insufficient data (numbers 4, 16, 22, and 34) or outlying data (number 9) on this task were among the youngest in the ADHD group. Of the eight children younger than 11 years in the ADHD group, five

were excluded from these analyses. In contrast, no children from the control group, whether young or old, were excluded.

Finally, there was a high rate of false alarms on Catch trials (22% and 16% in the ADHD and control groups, respectively). Future studies using the Posner paradigm with simple detection of targets should include Catch trials. Otherwise, reaction time data will probably include a significant proportion of trials that are initiated prior to the onset of the target, and again, potentially more so in children with ADHD.

To summarize, with the proper controls in place (such as excluding trials with overt orienting and including Catch trials), no difference was observed in the amount of IOR in children with ADHD and controls. On a broader level, younger children with ADHD had more difficulty on this task than did controls, as reflected in the high percentage of participants excluded from ADHD group (all younger than 11 years) because of a lack of reliable data (31% of participants in the ADHD group versus none in the control group). It is most probable that poor saccadic control underlies this pattern, because the total number of trials completed and the number of trials retained for analyses are positively related to good saccadic control. Although the task was not designed specifically to test the theory that children with ADHD have impaired saccadic control, there is other evidence in the literature to support this conclusion (e.g., Munoz et al., 1999).

Chapter 7. Memory-Guided Saccades

The final task administered was a memory-guided saccade task. One of the main advantages of studying eye movements to understand a group hypothesized to have deficits in cognitive processes is that much is known about the neurophysiology and neuroanatomy of eye movements, largely from single cell recording studies in primates and lesion studies in adults. Eye movements involve a complex interaction of cortical and noncortical structures, primarily the prefrontal cortex, basal ganglia, thalamus, and superior colliculus (Kandel, Schwartz, & Jessell, 2000).

Studies of children with ADHD have identified anomalies in some types of eye movements and on some types of eye movement tasks, but not others. Castellanos, Marvasti, Ducharme, et al., (2000) found no differences in smooth pursuit eye movements between girls with ADHD and controls. As mentioned previously, Munoz et al. (1999) demonstrated that children with ADHD made more intrusive saccades during a test of fixation stability.

Saccadic eye movements are shifts of the fovea to a visual target in another location. Munoz et al. (1999) reported no differences in saccadic reaction time (the latency to make an eye movement to a visual target), but found that, compared to controls, children with ADHD had a higher coefficient of variation of saccadic reaction time (a measure of intra-subject variability). Note that this latter finding is not specific to saccadic responses; the increased

variability in response times in ADHD is ubiquitous (see Douglas, 1999; Sergeant et al., 1999).

On the anti-saccade task, the participant is required to look in the opposite direction of an exogenous stimulus, requiring both suppression of the reflexive response (looking at the stimulus) and generation of the alternative response (looking away from the stimulus). Using this paradigm, Munoz et al. (1999) found that children with ADHD made more direction errors (looking toward the stimulus) than did controls. In contrast, Aman et al. (1998) reported no group differences in direction errors on their anti-saccade task. Aman et al. attributed their null result to the specific parameters of their task. The fixation point extinguished before the imperative stimulus was presented and all participants, regardless of group, had significant difficulty inhibiting reflexive saccades in the direction of the stimulus. Aman et al. did, however, report a significant interaction between group and session in direction errors: control children showed a decrease in errors over two sessions, whereas children with ADHD did not.

Castellanos et al. (2000) studied girls with ADHD and controls on a go-no go task with 10 trials. The task was to look at boxes cued by a surrounding green box and not at those cued with a surrounding red box. Compared to controls, girls with ADHD made more than twice as many commission errors (looking at boxes with red cues) and three times as many intrusion errors

(looking at uncued boxes). There was no difference in the latency of visually guided saccades.

Another eye movement paradigm that has been used to study inhibitory control deficits in ADHD is the delayed memory-guided saccade task. On this task, the participant is required to withhold making an eye movement during the presentation of a visual stimulus and during a subsequent delay period when the stimulus is no longer displayed. Following a signal (typically the offset of the fixation point), the participant is to make an eye movement to the remembered location. One advantage of this paradigm is that it may be able to distinguish inhibition from other components of executive function, such as working memory. Problems with visuospatial working memory may cause direction errors as the to-be-remembered location is lost from working memory, whereas premature responses would reflect inhibitory failure.

The first study using this task in children with ADHD was by Ross et al. (1994). Participants (13 with ADHD, 10 controls) were required to hold their gaze on a fixation point until, during, and following the presentation of a brief (100 ms) stimulus (a dot) in the periphery. The presentation of the stimulus was followed by a delay period of 800 ms, during which the child was still required to maintain fixation. The disappearance of the fixation point acted as the signal to look where the dot had previously appeared. Participants were given 600 ms after fixation offset to make a memory-guided saccade, after which a new

fixation point would appear in the location where the stimulus had previously been (that is, the dot's position on trial n became the position of the fixation point for trial n+1). Horizontal eye movements were measured using an infrared eye tracking device. All peripheral stimuli were presented on a horizontal plane. Ross et al. defined a visually-triggered saccade as any eye movement in the same direction as the stimulus, that was at least 50% of the distance between the fixation point and the stimulus. They also restricted their analyses to eye movements that began at least 80 ms following the onset of the stimulus, referring to literature suggesting that it takes this long for the brain to initiate a visually-triggered saccade.

Ross et al. measured three distinct processes during this task. Firstly, the ability to inhibit eye movements was measured by counting eye movements made after the onset of the stimulus but prior to the offset of the fixation point. These anticipation errors were further classified a posteriori by their latencies as either visually triggered saccades or as saccades resulting from an inability to hold a motor plan in working memory without executing it. Secondly, the ability to prepare the motor system to make an eye movement was assessed by measuring the latency of saccades following fixation offset (shorter latencies imply increased preparedness). And thirdly, visuospatial working memory was assessed by studying the accuracy of the memory-guided saccades.

Ross et al. reported that children with ADHD were no different from control children in the latency of their saccades following fixation offset or in the accuracy of their memory guided saccades. In contrast, children with ADHD (whether or not they were on a stimulant medication at the time of testing) were found to be impaired in their ability to withhold premature saccades during the 800 ms delay. Within both groups, approximately half of the premature saccades had latencies of less than 530 ms (and were considered to be exogenously triggered by the presentation of the stimulus in the periphery) and half had latencies greater than 530 ms (and were considered to be failures to inhibit information stored in working memory). According to Ross et al. (1994), taken together, these findings suggest a specific inhibitory control deficit in ADHD, and not a broader deficit in working memory.

Ross et al. (1994) hypothesized that one possible reason for not finding group differences in visuospatial working memory and response preparation was that their delay period of 800 ms was not sufficiently long. Later, Ross, Harris, Olincy, and Radant (2000), comparing adults with ADHD to controls (aged 25-50 years), replicated the pattern they found with children using a similar paradigm but with longer delay periods (1000 and 3000 ms).

Castellanos et al. (2000) also used a memory-guided saccade task as part of their battery of eye movement tasks with girls with ADHD. In their task, girls were required to fixate on a white square in the centre of the screen, as a green

square was presented for 50 ms to the left or right of centre at one of two different eccentricities. There was a delay period of 1200 ms, after which the white square at fixation disappeared, cuing a memory-guided saccade. If no memory-guided saccade was made during the delay period, the green square reappeared 750 ms after fixation offset. Saccades greater than 2 visual degrees were counted. Each participant completed 13 trials. (Ross et al., 1994, did not report the number of trials in their task.) Castellanos et al. reported that girls with ADHD made more saccades during the delay period than controls. Similar to Ross et al.'s (1994) finding with boys, the latencies of memory-guided saccades were similar between groups.

The memory-guided saccade used in the present study was a modification of Ross et al.'s paradigm. Each trial began with fixation on a point at the centre of the computer screen. The stimuli were squares presented in one of the 12 clock positions, to include non-horizontal eye movements. Also, in order to minimize the adoption of a strategy to wait for a fixed amount of time before initiating an eye movement, which one might do if the delay period were fixed, two different delay periods (700 ms and 1300 ms) were randomly intermixed.

Consistent with the inhibitory control model, it is predicted that children with ADHD will make more exogenously controlled saccades, and more saccades during the waiting period, because these types of errors are directly related to inhibition of prepotent responses and to interference control. Barkley

(1997a) argues that poor inhibition leads to deficits in nonverbal working memory, but it is not clear whether this means that children with ADHD should show reduced accuracy in memory-guided saccades, or reduced preparedness (i.e., longer saccade latencies following fixation offset).

Method

Participants

The forty children described in Table 2.3 participated. One boy from the ADHD group (number 29) was excluded from the main analyses because of insufficient data (see Data Analysis section for details). Data from one girl in the ADHD group (number 42) was lost due to technical error (a computer crash). The final sample was comprised of 24 control children and 14 children with ADHD. Participants were able to wear their prescription lenses during testing.

Procedures

Eye tracking system. The video-based EyeLink eye tracking system described in Chapter 6 was used to monitor eye movements. Recall the hypothesis put forth earlier that overall performance using the eye tracking equipment is related to diagnostic status and age. Indirect support for this theory was drawn from the observation that children with ADHD, especially younger children within this group, did not complete as many IOR trials as controls (the number of trials completed relates partly to time taken to calibrate the equipment and to initiate each trial). Calibration and validation of the equipment for the memory-

guided saccade task was identical to that described in Chapter 6 for the IOR task. Because the memory-guided saccade task was the only task to be completed in one 90-minute session (in contrast to the IOR task, which was scheduled for 50 minutes of a 90 minute session), most participants were able to complete an adequate number of trials. Only one participant from the ADHD group (number 29; age 9 years, 4 months) was excluded because of insufficient data. (This participant was not excluded from the IOR analyses.) As with the IOR task, the number of experimental trials completed (which ranged between 0 and 192, following 48 practice trials) was moderately positively correlated with age ($r = .28, p = .09$), and was lower in the ADHD group as compared to the control group, $t(37) = 2.78, p = .009$. Furthermore, the number of trials completed in the memory-guided saccade task was positively correlated with the number of trials completed in the IOR task, $r = .52, p < .001$. This correlation is consistent with the hypothesis that the number of trials completed is related to an individual characteristic.

In Chapter 6, it was suggested that one such characteristic could be fixation control (this theory has support from the literature, see Castellanos et al., 2000 and Munoz et al., 1999). Recall that the participant must make accurate and consistent saccades to calibrate the equipment quickly, and must have fixation stability to initiate each trial. There are, however, many other individual characteristics related to diagnostic status and age that could be associated with

completing fewer trials, such as the tendency to take long breaks, or to take longer pauses between trials. Also, the significant correlation between the numbers of trials completed on both tasks may be due in part to the fact that difficulty with the Eyelink on Session 2 (memory-guided task) may have led to frustration or low expectations with the same equipment on Session 3 (IOR task).

Stimuli and Task. Custom software was written for the purposes of this experiment by J. MacInnes to be run with the EyeLink eye tracking system and an IBM compatible computer. Each participant was seated approximately 71 cm away from the computer monitor. Head position was not fixed, although some participants chose to use a chin rest for comfort.

At the start of each trial, the participant was required to fixate on a small, white circle (measuring 0.5 visual degrees in diameter, with a 0.1 visual degree black point in its centre) presented in the centre of the screen. When ready, he or she was to press the space bar on a computer keyboard to initiate the trial. The trial only began if the participant's eyes were stable on centre at the time of the button press. This ensured that any eye movement measurements would be calculated accurately from fixation.

After 800 ms, a square drawn in white, measuring 1.4 by 1.4 visual degrees, appeared in one of twelve clock positions, each 4 visual degrees from centre. This stimulus stayed on for 100 ms, and was followed by a delay period of

either 700 ms or 1300 ms. Each participant was instructed to fixate on the centre point during both the presentation of the stimulus and the delay. After the delay period, the fixation point disappeared, and this was the signal for the participant to move his or her eyes to where the square had been displayed. If the landing point of the saccade were within a 1 visual degree radius of the white square, a green square would appear where it had been. Of a block of 48 trials, the square was presented in each of the 12 clock positions 4 times in a random order, and was followed by the 700 ms and 1300 ms delay periods on 2 of these 4 trials.

As with the IOR task, feedback regarding eye movements was considered to be essential to the participant's awareness of his or her saccades. If the Eyelink system detected an eye movement (a saccade greater than 1 visual degree, and with a velocity of 30 degrees per second or higher), eye drift (greater than 2 visual degrees from centre) or a blink prior to the offset of the fixation stimulus, a tone would sound, and the trial was aborted. To minimize frustration, the experimenter told the participants in advance that some eye movements were normal and expected, but that they should try their best not to make them) and the trials with eye movements were not recycled. Eye position was also monitored on a separate computer monitor that was out of the participant's view, and specific feedback regarding eye movements was provided by the experimenter when necessary. Each trial usually ended following an eye

movement. If no eye movement was made, the trial ended 1500 ms after fixation offset.

Participants completed a block of 48 practice trials, followed by 48, 96, 144, or 192 experimental trials.[25] All participants had the same amount of time available for this task (approximately 75 minutes). The number of trials completed depended on the time it took for calibration and validation (see procedures outlined in Chapter 6), the time taken to initiate each trial (because successful initiation requires stability on fixation), and on other factors such as time taken to learn the task and length of breaks or pauses between trials.

Instructions to Participants. Participants were instructed to keep their eyes on the centre of the fixation point (specifically, the black dot inside the white circle) and to press the space bar on the computer keyboard when they were ready to begin. They were told to keep their eyes on the fixation point, and that if the computer detected even a small eye movement, they would hear a "beep". They were told that a white square would appear in one of the 12 clock positions, and that they were to try to keep looking at the point in the centre of the screen until it disappeared, at which point, they were to look "where the square was". Finally, participants were told that if they looked in exactly the

[25] This may seem like a lot of practice trials, especially in comparison to the 13 experimental trials in the study by Castellanos et al. (2000). A large number of practice trials were administered to allow children time to learn how to use the eye tracking equipment and to learn the sequence of events in the task.

right place (the computer was very "picky"), a green square would appear where the white square had been. They were told that it was normal to have some "beeps", because it is not always possible to stop moving ones eyes, but that they were to try to keep their eyes on the centre. Each participant was asked to explain the procedures to the experimenter to ensure understanding. Performance was monitored closely during the practice block, and additional instructions were given if necessary.

Data Analysis. One participant (number 29, a boy with ADHD) with no useable trials (he completed only one practice block of 48 trials) was excluded from the analyses. Trials with blinks (M = 4.07%, SD = 4.87) in the ADHD group and M = 2.33%, SD = 4.76 in the control group) were excluded. There was no group difference in the percentage of trials with blinks, $t(36) = 1.08$, $p = .29$.[26]

The computer recorded the latency, amplitude and landing coordinates of the first saccade made following the onset of the stimulus (the square) on each trial. Each trial was coded as being one of three types. "Anticipation Errors" were trials on which the participant made a saccade greater than 1.5 degrees in amplitude, at least 80 ms after stimulus onset (cf., Ross et al., 1994), and prior to the offset of the fixation point. "Correct Trials" were those on which the

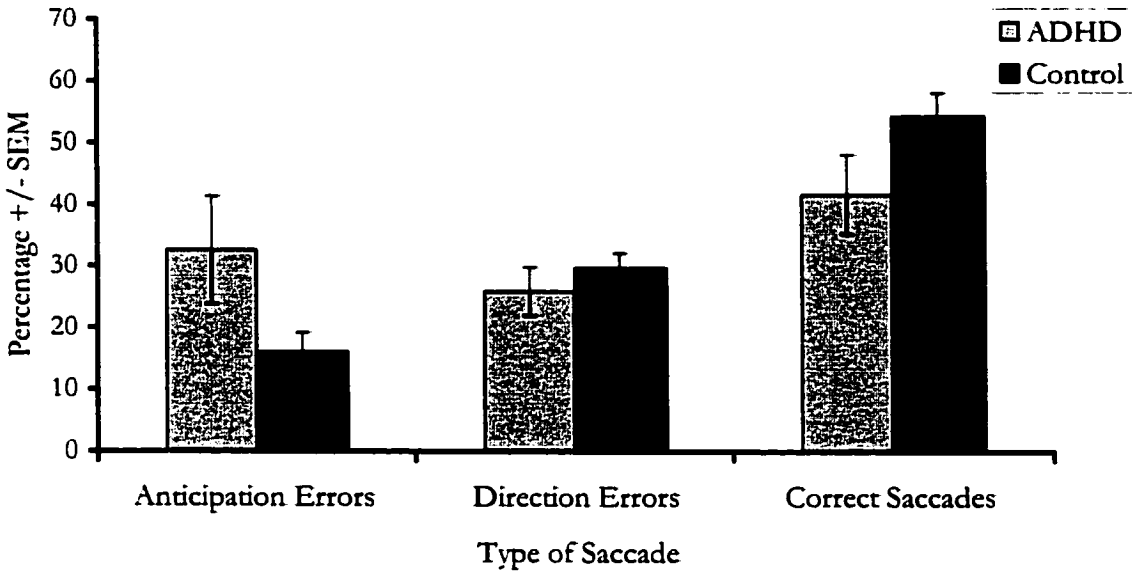
[26] Note that these rates are higher than in the IOR task (less than 1%) because the total duration of sampling time is longer in this task (3600 ms) than in the IOR task (1500 ms).

participant waited until the fixation point was turned off and then made a saccade to the target location. Trials with "Direction Errors" were those on which the participant waited until the fixation point was turned off before making a saccade but did not land on the correct location (the criterion for success was a landing point within a 1 visual degree radius of the target). The timing of saccades was also examined relative to the stimulus onset and fixation offset.

Results

The proportion of trials in each of the aforementioned categories is presented in Figure 7.1. The group differences in the two types of errors were analyzed with two non-orthogonal unpaired t-tests. In terms of the principle analysis, children with ADHD made about twice as many anticipation errors (M = 32.55%, SD = 32.74) as control children (M = 16.06%, SD = 15.30). This corresponds to a significant group difference in anticipation errors, $t(36) = 2.12$, $p = .04$.

Figure 7.1. Percentage of Anticipation Errors, Direction Errors and Correct Memory-Guided Saccades by Group.



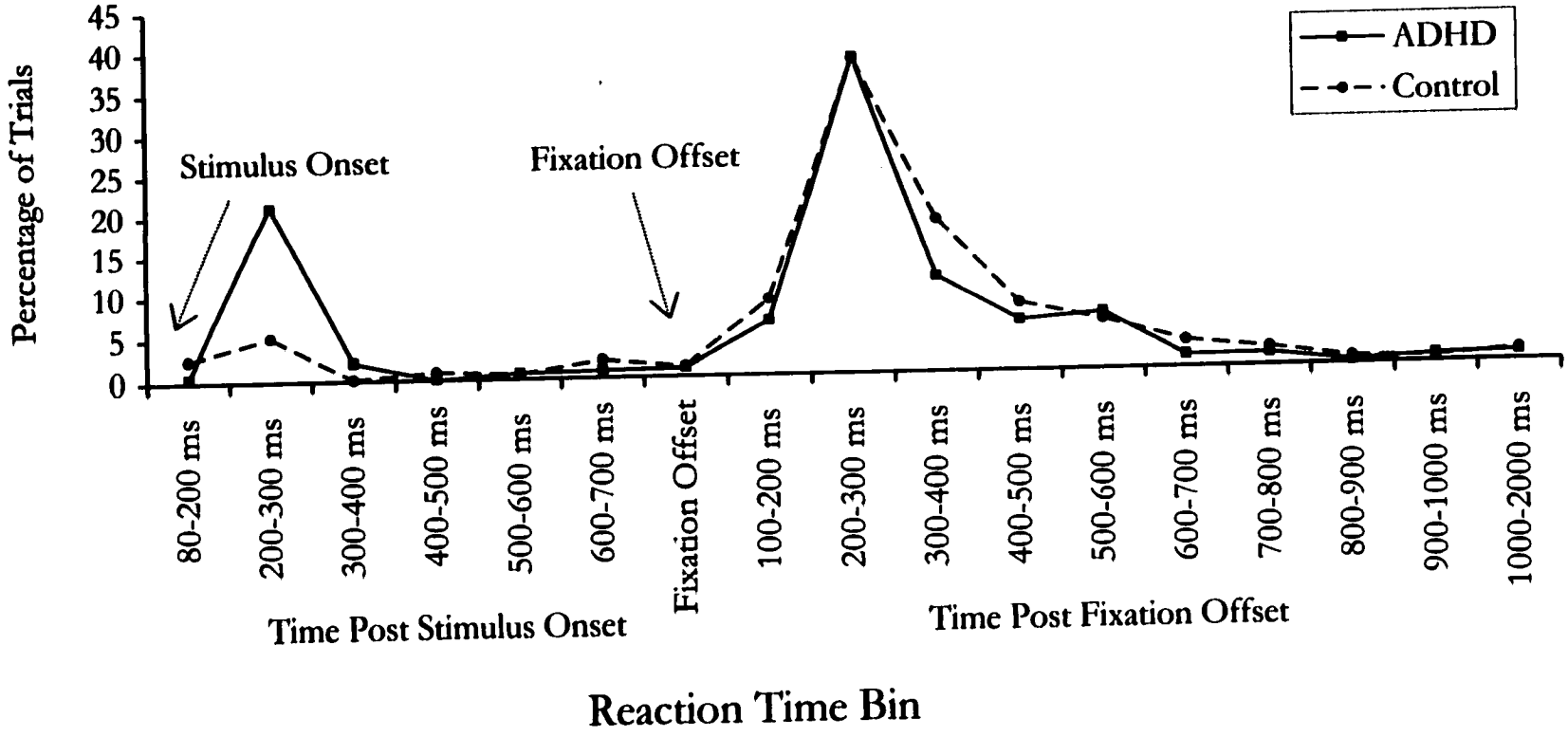
Note. Because each trial was categorized as one of three types, for each group, adding across types would yield 100%.

There was no difference in the percentage of Direction Errors (M = 25.83%, SD = 14.62 and M = 29.64%, SD = 11.96, in children in the ADHD and control groups, respectively; $t(36) = 0.87, p = .39$), or in the magnitude of those errors when they occurred (M = 1.67 visual degrees, SD = 0.24 and M = 1.70 visual degrees, SD = 0.20, in children in the ADHD and control groups, respectively; $t(34) = 0.38, p = .71$). The criterion for a correct landing point was relatively precise: within a 1 visual degree radius of the target. Looking more closely at the magnitude of the direction errors, 47.75% were within a 1.5 visual degree radius of the target, and an additional 32.03% were within a 2 visual degree radius of the target. There were no group differences in the percentage

of direction errors that were within 1.5, 2, or greater than 2 visual degrees of the target (all *ps* > .6) strongly suggesting that memory for the location of the target did not differ between groups.

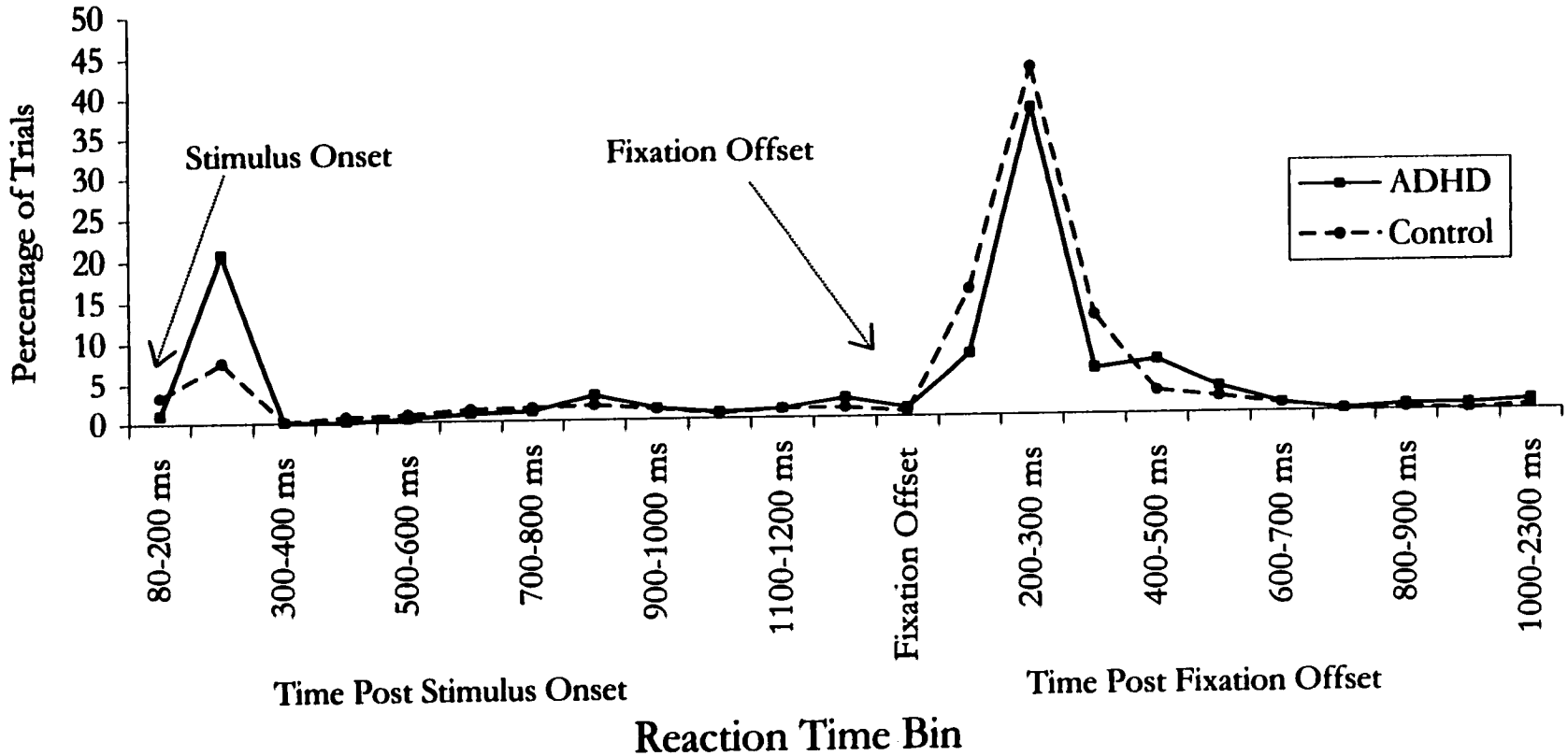
For the purposes of looking at the distribution of reaction times, saccades that were initiated after the fixation offset and that landed within 2 visual degrees of the target were included in the analyses. The data are presented separately for the 2 delay periods in Figures 7.2 and 7.3 (700 ms and 1300 ms delay periods, respectively). Saccadic latencies were divided into 100 ms bins. The percentage of trials that occurred in each bin is presented in relation to the stimulus onset and fixation offset. There are three patterns common to both figures. Firstly, the group difference in Anticipation Errors reported above is primarily due to a greater percentage of saccades occurring in the 200-300 ms Post-Stimulus bin in the ADHD group. Secondly, for both groups and for both delays, there are relatively few saccades that occur in any one bin in the remainder of the delay period. And thirdly, for both groups and for both delays, the majority of saccades occur in the 100 to 400 ms Post-Fixation Offset bins. Each of these effects will now be explored in more detail.

Figure 7.2. Distribution of latencies of saccades made in relation to the Stimulus Onset and Fixation Offset by Group: Trials with a 700 ms delay period



Note. Saccades made 80 ms after Stimulus Onset, throughout the 700 ms delay period, until Fixation Offset are Anticipation Errors. Saccades made after the Fixation Offset are Memory-guided saccades. Memory-guided saccades that landed within 2 visual degrees of the target are represented. Saccades greater than 1.5 visual degrees in amplitude are represented.

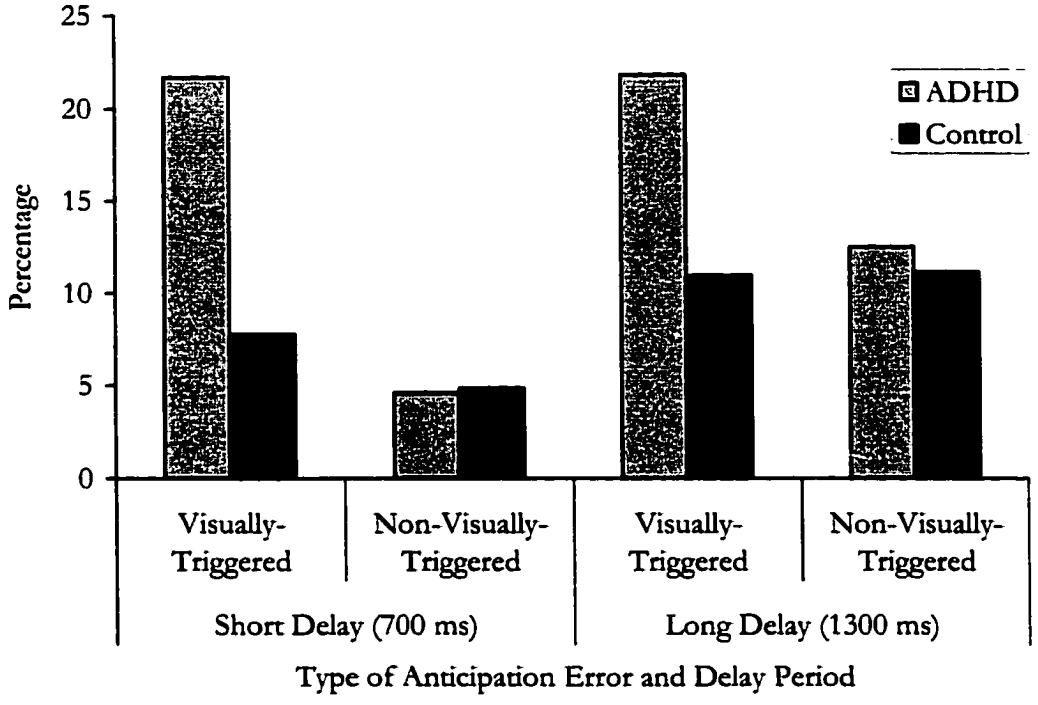
Figure 7.3. Distribution of latencies of saccades made in relation to the Stimulus Onset and Fixation Offset by Group: Trials with a 1300 ms delay period.



Note. Saccades made 80 ms after Stimulus Onset, throughout the 1300 ms delay period, until Fixation Offset are Anticipation Errors. Saccades made after Fixation Offset are Memory-guided saccades. Memory-guided saccades that landed within 2 visual degrees of the target are represented. Saccades greater than 1.5 visual degrees in amplitude are represented.

As suggested by Ross et al. (1994), Anticipation Errors on the memory- guided saccade task can be classified into two categories: those that are triggered by the onset of the stimulus and those that represent a failure to suppress a to-be-executed saccade held in working memory. For both groups and at both delays, the majority of the Anticipation Errors occurred within 300 ms of the onset of the stimulus. These latencies correspond closely to children's saccadic reaction times to visual targets as reported by Castellanos et al. (2000) and Munoz et al. (1998; 1999). It is likely, then, that saccades in the first two bins of Figures 7.2 and 7.3 represent those that are visually triggered by the onset of the stimulus. Saccades in the bins corresponding to the remaining 400 ms and 1000 ms of each delay period (in the 700 ms and 1300 ms delay conditions, respectively) correspond to non-visually triggered saccades. The same data are re-plotted in Figure 7.4 with this operational definition of what constitutes a visually-triggered and a non-visually-triggered saccade. Non-visually-triggered saccades occur more frequently on trials with a longer (1300 ms) delay. This would be expected because there is 600 ms more time with which to make this kind of error on the long delay trials. With the data plotted in this way, it is clear that there is no group difference in the proportion of non-visually-triggered saccades.

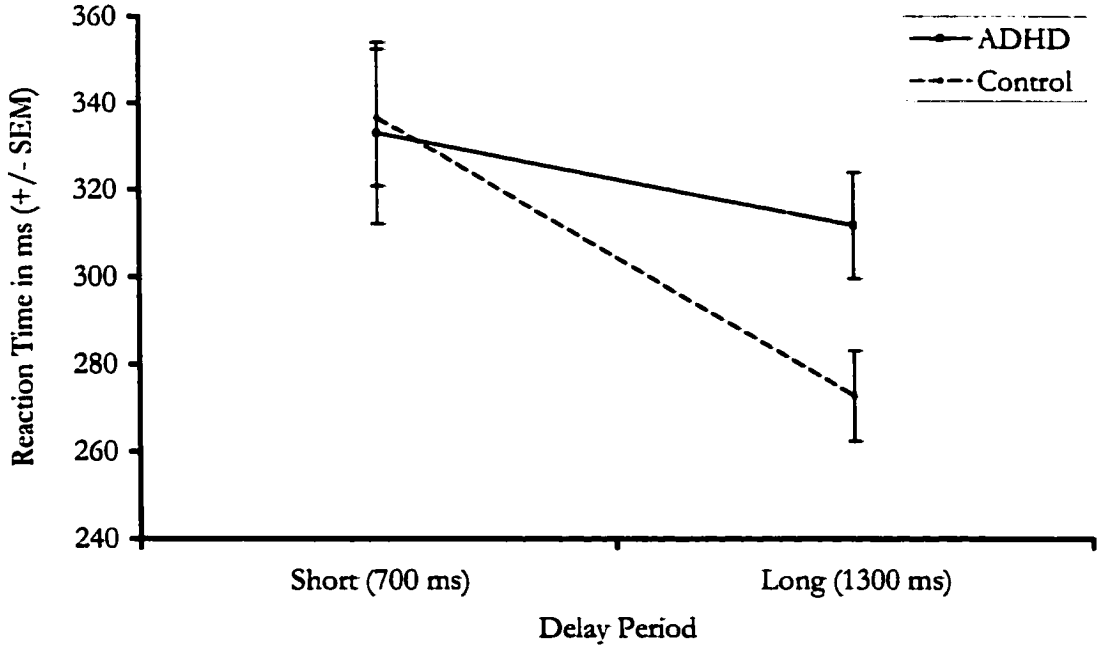
Figure 7.4. Percentage of visually-triggered and non-visually-triggered anticipation errors by Group and Delay.



The latencies of the memory-guided saccades depicted in Figures 7.2 and 7.3 were analyzed using a repeated measures ANOVA, with one between subjects variable, Group (ADHD versus control) and one repeated measure, Delay (Short, 700 ms versus Long, 1300 ms). There was a main effect of Delay, $F(1, 34) = 12.82, p = .001$, but no main effect of Group, $F(1, 34) = 0.874, p = .36$, and no interaction between Group and Delay, $F(1, 34) = 3.24, p = .08$. The mean latency for each Group at each Delay is presented in Figure 7.5. The main effect of Delay is characterized by shorter latencies on trials with a 1300 ms

delay. Despite the non-significant interaction in the overall ANOVA, the data, as depicted in Figure 7.5 were suggestive of a trend toward a group difference at the longer delay. Post-hoc t-tests confirmed that there was a group difference in latencies on trials with a 1300 ms delay (slower in the ADHD group; $t(34) = 2.29, p = .03$) and no group difference in latencies on trials with a 700 ms delay, $t(34) = 0.13, p = .90$.

Figure 7.5. Latencies of memory-guided saccades for trials with Short and Long Delays by Group.



Discussion

In this memory-guided saccade task, children were instructed to keep their eyes on fixation while a stimulus (a square) was presented in the periphery and

during a subsequent delay period. Children with ADHD made about twice as many anticipation errors as did children in the control group. An analysis of the distribution of latencies of these errors revealed that this group difference was due to a greater percentage of visually-triggered saccades (those that occurred within 300 ms of the onset of the stimulus) in the ADHD group, and not to a difference in the percentage of errors that took place in the remainder of the delay period.

Both Castellanos et al. (2000) and Ross et al. (1994) reported more anticipation errors among children with ADHD on a memory-guided saccade task. Castellanos et al. (2000) did not distinguish between the two types of anticipation errors (visually-and non-visually-triggered saccades). Ross et al. (1994) divided the latencies into two halves: greater than and less than 530 ms. They reported that the proportion of saccades with latencies less than 530 ms was similar for both groups, implying that children with ADHD made more of both kinds of anticipation errors.

The reaction time analysis conducted in this study was more sensitive than that used in previous studies: on average 148 latencies per subject were examined in bins of 100 ms. This allowed for the direct observation that children with ADHD made more visually-triggered anticipation errors than control children. The finding that children with ADHD have a harder time inhibiting a saccade to a visual stimulus is consistent with the findings of Munoz

et al. (1999) using an anti-saccade task and Castellanos et al. (2000) using a go-no go task and with the inhibitory control model: children with ADHD have a harder time inhibiting the prepotent response to look towards a visual stimulus.

Non-visually-triggered anticipation errors were defined as saccades with latencies greater than 300 ms, throughout the remaining 400 ms or 1000 ms of the 700 ms or 1300 ms delay periods, respectively. During these delays, participants needed to fixate on the centre, hold the location of the stimulus in working memory, and prepare to make a saccade to that location once the fixation point disappeared. Accordingly, saccades made during the delay period could be due to fixation instability or to an inability to suppress a motor program held in working memory. Collapsed across group and across reaction time bin, such non-visually-triggered saccades took place on approximately 5% of the trials with a 700 ms delay, and approximately 12% of the trials with a 1300 ms delay (reflecting the greater time possible to make these kinds of errors on trials with a longer delay). Importantly, there was no group difference in the percentage of anticipation errors of this type (see Figure 7.4).

This suggests that once the initial impulse to look toward the stimulus was successfully inhibited, children with ADHD did not have a harder time maintaining fixation, or maintaining a to-be-executed saccade in working memory without executing it prematurely. It is important to note, however, that only the first saccade made after the stimulus onset was recorded, and the trial

terminated after any saccade was made. This means that on a trial in which the participant was unable to inhibit a saccade to the stimulus (i.e., a saccade was made in the first 300 ms after stimulus onset), there would have been no "opportunity" to make a non-visually-triggered saccade later in the delay period. If, as the inhibitory control theory would suggest, the inability to inhibit prepotent responses is correlated with tendency towards impulsive responding and poor interference control, the lack of a group difference in the percentage of saccades that take place later in the delay period may be due, in part, to the fact that the trial terminated after the first saccade.

According to Ross et al. (1994), the latency of memory-guided saccades reflects the degree of preparedness to make a saccade once the fixation point is extinguished. Neither Ross et al. (1994), nor Castellanos et al. (2000) reported group differences in the latency of memory-guided saccades. In this study, there was no group difference in the latency of memory-guided saccades following a 700 ms delay period, but children with ADHD were slower than controls following a 1300 ms delay period. Note that the interaction term for the overall mixed ANOVA was not significant ($p = .08$), but a post-hoc t-test examining only the 1300 ms delay period was significant ($p = .03$). One explanation for this trend could be reduced preparedness in the ADHD group on trials with a longer delay. Recall that on the IOR task, however, children with ADHD showed the same decrease in reaction time as controls across two delay periods;

that is, they showed the same amount of preparedness at both delays (see Figure 6.3). Combined with the results from the IOR task, on which no information was being actively maintained in working memory and simultaneously inhibited, the pattern here suggests that children with ADHD may have difficulty maintaining alertness or preparation when capacity is allocated to other functions.

The accuracy of the memory-guided saccades was similar for both groups, as was the magnitude of errors when they occurred. These findings are consistent with those of Castellanos et al. (2000) and Ross et al. (1994). As suggested by Ross et al. (1994), the accuracy of a memory-guided saccade reflects the accuracy of the target location or motor program held in working memory. It also reflects the ability to execute saccades to a location with no stimulus. Ross et al. (1994) argued that group differences in the accuracy of memory-guided saccades would point to differences in working memory, not in inhibition. As such, the lack of a group difference in these variables is consistent with the hypothesis that children with ADHD have a specific deficit in inhibitory control.

A final observation on this task was that children in the ADHD group were not able to complete as many trials as control children. This replicates the similar finding with the IOR task. As discussed in detail earlier in this chapter

and in Chapter 6, reasons for this could be related to saccadic control, or to other features of ADHD such as inattention and distractibility.

To summarize, children with ADHD made more anticipation errors on the memory-guided saccade task. An analysis of the saccadic latencies indicated that, as compared to controls, children with ADHD made more premature saccades that were visually-triggered by the stimulus, and comparable percentages of premature saccades that were not visually-triggered. Children with ADHD were slower than controls to make memory-guided saccades following long delays, and there was no group difference in the accuracy of memory-guided saccades. Taken together, these findings are consistent with the inhibitory control theory: children with ADHD have a reduced ability to inhibit prepotent responses (eye movements to the onset of a visual stimulus in the periphery). There was no evidence for a specific deficit in visuospatial working memory in children with ADHD, but the reduced preparedness at the long delay in this group may be due to a cost associated with holding a motor program in working memory.

Chapter 8. General Discussion

Synthesis of results from the seven measures of inhibition

To this point, each measure has been discussed individually in terms of its consistency with the hypothesis that children with ADHD have a primary deficit in inhibitory control. Recall from Chapter 1 that inhibition is not a unitary construct, and that children who are impaired on some measures of inhibition should not necessarily be impaired on all measures of inhibition. The main goal of this study was to examine patterns of group differences across measures of inhibition in order to assess the scope of the inhibitory control deficit in ADHD. In Table 1.1, each task was categorized according to Barkley's (1997a) definition of response inhibition and Nigg's (2001) more comprehensive taxonomy of inhibition. The patterns of group differences from each task are summarized in Table 8.1, along with these categorizations. The patterns will first be discussed according to the two types of cognitive inhibition in Nigg's taxonomy, Executive and Automatic Inhibition.

Table 8.1. Pattern of group differences on each measure of inhibition, unique features of each task, and categorization of each according to Barkley (1997a) and Nigg (2001).

Measure	Unique features	Barkley's definition	Nigg's taxonomy	Group difference in inhibition?
Stop Signal	Yields a measure of the time taken to stop.	Cessation of an ongoing response; Inhibition of a prepotent response	Executive Motor	Yes. Longer SSRT in ADHD group.
Stroop Colour-Word	A central feature of the target stimulus must be inhibited.	Interference control	Executive Interference Control or Response Conflict	Yes. Larger Stroop Interference: Accuracy in ADHD group.
Negative Priming	An automatic corollary of inhibition on another task.		Automatic Cognitive	No.
Simon	The location of the target stimulus must be inhibited.	(Interference control)	(Executive Interference Control or Response Conflict)	No.
Flanker	The identity of peripheral stimuli must be inhibited.	(Interference control)	Executive Interference Control or Response Conflict	No.
Inhibition of Return	An automatic process following the orienting of attention.		Automatic Motor	No.
Memory-Guided Saccade	(a) Inhibiting a reflex. (b) Inhibiting information in working memory.	(a) Inhibition of a prepotent response (b) (Interference control)	(a)Executive Motor (b) Executive Motor	(a) Yes. More visually-triggered errors in ADHD group. (b) No.

Note. Items in parentheses refer to tasks that were not directly mentioned in Barkley's (1997a) or Nigg's (2001) reviews. In these cases, the label was assigned based on the definitions given for each type of inhibition and the categorization of similar tasks within each model.

Executive Inhibition. Nigg (2001) proposed that there are three distinct types of Executive Inhibition: Motor, Interference Control, and Cognitive. This study included measures of the first two types.

Executive Motor Inhibition. A group difference between children with ADHD and controls was found on both measures of Executive Motor Inhibition studied. Firstly, on the stop-signal task, children with ADHD had a longer mean SSRT than controls. As noted in Table 8.1, longer SSRTs could be due to both the inability to inhibit a prepotent response and to the inability to stop an ongoing response. Secondly, children with ADHD, as compared to controls, made more visually triggered premature saccades on the memory-guided saccade task. This is another example of the inability to inhibit a prepotent response (exogenously triggered saccades). The inability to inhibit saccades was also reflected in a greater difficulty with the eye tracking equipment (on both the IOR and memory-guided saccade tasks), especially among young children with ADHD. There was no group difference in the percentage of premature saccades made during the delay period, indicating that once the initial response was inhibited, children with ADHD did not have a harder time holding the upcoming saccade in working memory.

Executive Interference Control or Response Conflict. Three different types of interference control or response conflict were assessed. There were group differences in the magnitude of interference on the Stroop Colour Word Task:

children with ADHD made disproportionately more errors than controls on interference trials. In contrast, there were no group differences in the magnitude of the Simon or Flanker Effects.

In each of these three measures, some type of interfering information must be suppressed in order to respond correctly. What differs between each is the nature of the interfering information in relation to the required response. In the Flanker task, the interfering information is on either side of the target stimulus, and the interfering information comes from the same response set as the correct response (digits). In the Simon task, the interfering information (location) is a feature of the target stimulus, and although it comes from the same response set as the required response (left or right), it is not central to the task (the correct response is based on identity, and location is irrelevant). In the Stroop Colour Word Task, the interfering information (written word) is a central feature of the target stimulus (colour word), and it comes from the same response set as the correct response (ink colour).

In order to understand why children with ADHD may have shown deficits on one of the measures of interference but not on the other two, we will turn to a recent taxonomy of interference tasks from the cognitive literature. Kornblum and colleagues (Kornblum, 1994; Kornblum, Stevens, Whipple, & Requin, 1999; Zhang, Zhang, & Kornblum, 1999) have developed a model, called the Dimensional Overlap model, to distinguish between different measures of

interference (including the Stroop, Simon, and Flanker effects). They developed this model based on analyses of the time course of each effect, on manipulations of the strength of each effect, on experimental and computational (parallel distributed processing) approaches, and on reviews of the existing literature. Dimensional overlap pertains to "the degree to which relevant and/or irrelevant stimulus sets are perceptually, conceptually, or structurally similar to the response set in the task and/or to each other" (Kornblum et al., 1999, p. 688). Under this model, there are three main types of dimensional overlap: Stimulus-Response (S-R) Relevant, S-R Irrelevant, and Stimulus-Stimulus (S-S) Irrelevant. Factorial combinations of the different types of overlap can produce eight different types of interference tasks. These are portrayed in Table 8.2, along with the prototypical example of each type of task from the literature. The S-R compatibility task is a measure of S-R Relevant overlap. Recall that on this task, participants are instructed to respond with a key on the same side (compatible) or opposite side (incompatible) of a stimulus. There is overlap between the location of the stimulus (left or right) and the location of the response (left or right), and the location of the response is relevant to the task (i.e., it is central to the task instructions). The Simon task is a measure of S-R Irrelevant overlap. As with the previous type, there is overlap between the location of the stimulus and the location of the response, but in this case, the location is irrelevant to the task instructions. An example of S-S

Irrelevant overlap is the Flanker task: there is overlap or similarity between the relevant and irrelevant stimuli. The Stroop task is unique in that it combines all three types of overlap. The relevant response set (colour names) overlaps with the relevant stimulus dimension (ink colour) and the irrelevant stimulus dimension (colour word).

Table 8.2. Kornblum's taxonomy of interference tasks.

Type	Example task	Dimensional Overlap		
		S-R Relevant	S-R Irrelevant	S-S Irrelevant
1	Neutral RT tasks	No	No	No
2	S-R compatibility	Yes	No	No
3	Simon	No	**Yes**	No
4	Flanker	No	No	**Yes**
5	Rarely studied	Yes	Yes	No
6	Never studied	Yes	No	Yes
7	Never studied	No	Yes	Yes
8	Stroop	**Yes**	**Yes**	**Yes**

Note. This table is adapted from Kornblum (1994), Kornblum, et al. (1999), and Zhang et al., (1999). S = Stimulus; R = Response; RT = reaction time. Yes and No refer to whether each kind of dimensional overlap is present. The 8 task types represent all possible combinations of dimensional overlap. The types of dimensional overlap measured in this study are highlighted in bold.

Kornblum's model provides a framework for understanding the pattern of results across the different interference tasks assessed in this study. The only type of dimensional overlap that is unique to the Stroop task (the task for which group differences were identified) is S-R Relevant overlap. The data from this study suggest, therefore, that children with ADHD are impaired on tasks that

include this type of overlap, and not on those that include only the other two types of overlap. Of course, the Stroop task is also unique in that combines all three types of overlap. An alternative conclusion would be that children with ADHD are impaired on tasks with multiple types of dimensional overlap. One way to reconcile these two alternatives would be to study measures of pure S-R Relevant overlap, for example, S-R compatibility. Recall from Chapter 5 that there has been a small body of literature using the S-R compatibility task in children with ADHD, and that the results were equivocal. Further research using this task should be able to determine whether the critical feature of the Stroop task is the S-R Relevant overlap.

Another way to determine whether the group difference in Stroop Interference is related to its unique type of overlap (S-R Relevant), is to look for conceptual similarity between the S-R Relevant component and other tasks on which children with ADHD show a deficit. For example, it may be that tasks with S-R Relevant overlap require the inhibition of prepotent responses more so than the other two types of dimensional overlap. Because the interfering information is part of the same response set as the target, the interfering information is, in some sense, prepotent. Viewed in this way, S-R Relevant interference may be subsumed under Barkley's (1997a) definition of response inhibition and Nigg's Executive Motor Control. One way to explore this hypothesis would be to manipulate the degree of prepotency in interference

control tasks, much like Tagliabue et al. (2000) did (see Chapter 5) by having children practise either compatible or incompatible responses.

Note that Sergeant et al. (1999) suggested that one possible moderating variable in the S-R compatibility task is event rate. In an earlier study, van der Meere, Vreeling, and Sergeant (1992) had demonstrated an interaction between event rate and S-R compatibility: children with ADHD showed a disproportionate increase in reaction time on incompatible trials when the event rate was slow, but not when it was fast. Consistent with this finding, Sergeant et al. (1999) have put forth a "cognitive-energetic" model of ADHD, whereby one of the main factors is arousal or activation, which can be influenced by event rate. They hypothesize that children with ADHD perform more poorly when a slow event rate is used because of underarousal. In the present study, event rate was slower on the Simon-Flanker task than it was on the Stroop task, and yet the deficits were observed on the Stroop task. Of course, Sergeant, van der Meere, and their colleagues could argue legitimately that for the effect of event rate to be properly assessed, it should be manipulated within a task, rather than compared across tasks. Nevertheless, the patterns of results in the present study do not seem to be accounted for by differing event rates.

One other feature of the Dimensional Overlap model is worth noting here. Kornblum et al. (1999) demonstrated that the Simon and Flanker effects follow a different time course. Accordingly, when the two tasks are combined they

should interact. In this study, there was no interaction between Simon Task conditions and Flanker Task conditions. There are at least three possible reasons for the discrepancy. Firstly, the differences reported by Kornblum et al. (1999) were small in magnitude. Small additive or interactive patterns may not have been manifested in this study in the presence of large within and between group variability. Alternatively, there may be developmental differences in the nature of one or both effects. Finally, it may be that this is an aspect of the dimensional overlap model that would need to be modified based on these data.

Automatic Inhibition. In Nigg's (2001) taxonomy, there are two types of Automatic Inhibition. Nigg identified IOR as an example of Automatic Motor Inhibition and negative priming as an example of Automatic Cognitive Inhibition. In this study, there were no group differences on either of these measures.

Summary of Inhibitory Deficits. The pattern of results using Nigg's (2001) taxonomy of inhibition and Kornblum et al.'s (1999) Dimensional Overlap model of interference is clear. Children with ADHD showed deficits on measures of Executive Motor Inhibition, and Executive Interference Control with S-R Relevant overlap. In contrast, they did not show deficits on measures of Executive Interference Control with S-R Irrelevant or S-S Irrelevant overlap or on measures of Automatic (Motor or Cognitive) Inhibition. It was suggested

that Interference Control with S-R Relevant overlap could require a special form of Motor Inhibition, or inhibition of prepotent responses.

The pattern of findings is consistent with Barkley's (1997a) hypothesis that children with ADHD have a deficit in response inhibition, which is defined as the inhibition of prepotent responses, the cessation of ongoing responses, and interference control. In Barkley's definition, interference control is comprised of interference as measured in the Stroop task, as well as the ability to avoid responding or distractions during a delay. There was no deficit of the latter sort observed on the memory-guided saccade task. There was support for ADHD deficits in the other components of Barkley's (1997a) definition. Furthermore, the present study identified no ADHD deficits in measures that are not subsumed under this definition of inhibitory control.

In addition to defining the scope of the inhibitory deficit in ADHD, these data provide support for Nigg's (2001) taxonomy of inhibition, because patterns of deficits are grouped according to different types of inhibition. They also validate Kornblum et al.'s (1999) assertion that different interference tasks measure different processes. Note that the dimensional overlap model was developed based on experimental cognitive psychology, and that this data provides converging evidence from a clinical population.

Future Directions

The approach taken in this study could be applied to a variety of clinical and non-clinical samples to answer many questions about inhibition in general, and inhibition in ADHD specifically. With a large, non-clinical, adult sample, a factor analytic approach could be used to validate Nigg's (2000, 2001) taxonomy of inhibition (including Motivational Inhibition and Executive Cognitive Inhibition, which were not assessed in this study) and to identify measures of inhibition that cluster together. Other properties of these measures, such as their test-retest reliability, and their relation to state and trait characteristics could also be investigated. With a large, non-clinical, child sample, the development of each type of inhibition could be assessed, as could relations between behavioural characteristics and performance measures.

In Chapter 2, there was much discussion regarding the sampling, inclusion, and exclusion of participants in both the control and clinical groups. Choices were made to maximize the internal validity, the external validity, and the power of this study. Of the 52 children tested, 12 were excluded from the main analyses, and the final sample was comprised of 24 control children and 16 with ADHD. This sample is comparable in size to many samples in the literature, and in some important ways, it is less heterogeneous than other samples. For example, children with ADHD-I and comorbid Tourette's Disorder were excluded, as were children in the control group who were rated by their parents

or teachers as exhibiting symptoms of ADHD in at least one context. Furthermore, because most of the analyses were conducted on repeated measures, there was adequate statistical power to detect differences with medium to large effect sizes. Nevertheless, some (e.g., Barkley, 1997a) argue that research examining group differences between children with ADHD and control children (using statistical tests with non-repeated measures) should include much larger samples. In this study, the possibility of Type II error was considered when interpreting each analysis. In the majority of cases, the presence or absence of group differences was clear, both in terms of the statistical analyses and in the comparison of raw means, and it was difficult to see how adding more subjects would change the nature of the findings. In the few cases for which there were differences between group means that were not statistically significant, the differences were not in the predicted direction. For example, in Figure 5.3 (Percent Error by Simon Condition and Group) it appears as though there may be a greater cost in accuracy associated with Incompatible Simon trials in the Control group, as compared to the ADHD group. This difference was not statistically significant ($p = .36$), and it was opposite to the hypothesis that children with ADHD should show more interference because of their deficit in inhibitory control. To summarize, although the sample tested in this study was relatively small, the use of a repeated measures design for most analyses led to adequate power, and the

possibility of Type II error was considered when interpreting each analysis. Nevertheless, it would be useful if these measures were collected with a larger sample.

In addition, future research should be conducted with both clinic-based and population-based samples, which may include children who are more and less impaired, respectively, than the participants in this study. Also, the specificity of these data to ADHD remains to be established. Children with LDs, other DBDs, and other disorders of executive function have been shown to have deficits on some of the tasks used here (see Pennington & Ozonoff, 1996 and Sergeant et al., 2002, for reviews). The pattern of deficits across the different types of inhibition should be explored in children with these other disorders, and in children with ADHD and comorbid disorders to determine to what extent the deficits observed here are specific to ADHD. Furthermore, Nigg (2000) hypothesized that measures of different types of inhibition may be able to differentiate between groups of individuals with similar deficits on other measures of executive functioning. In addition, Barkley (1997a) argues that his model does not apply to children with ADHD-I, and for that reason, children with ADHD-I were excluded from this study. These children may show a different pattern of inhibitory deficits. For example, they may have more difficulty on measures of interference with S-S Irrelevant overlap (such as the Flanker task) or on measures of Automatic Inhibition.

There are also many theoretical avenues that remain to be explored. This study was designed to assess the scope of the primary assumption in Barkley's (1997a) model. Researchers have begun to test some of the predictions made based on the model, and to assess other important elements of the model, such as the relation between the inhibitory deficits and secondary deficits in executive functioning. For example, Barkley predicted that deficits in inhibitory control should lead to deficits in working memory, and that these in turn, should lead to difficulties with the subjective sense of time. Kerns, McInerney, and Wilde (2001) demonstrated that children with ADHD have deficits in time reproduction. Interestingly, these deficits were correlated with some inhibitory deficits, but neither time reproduction nor inhibition was correlated with performance on measures of working memory, on which children with ADHD performed as well as controls.

Proponents of competing models of ADHD have questioned the notion that inhibition is the primary deficit in ADHD, and have argued that inhibitory deficits are secondary to other factors such as motivation, arousal, and response to reinforcement (see Douglas, 1999 and Sergeant et al., 1999 for reviews). Sonuga-Barke (2002) has recently argued that delay aversion (motivational style) and inhibitory control are "independent co-existing characteristics of ADHD", p. 29, and that the two processes may be associated with two distinct subtypes

of ADHD. With the nature of the inhibitory control deficit defined more clearly, the relation between it and other deficits should be easier to assess.

Another worthwhile avenue of research would be to translate these measures into tasks that are more naturalistic or game-like. Other researchers have begun to do this with other laboratory measures of cognitive processes (e.g., Archibald & Kerns, 2002; Sparkes & Klein, 2002). Recognizing that these modifications may represent manipulations of motivation and arousal that could differentially affect the performance of children with ADHD, Sparkes and Klein (2002) are comparing performance on measures of cognitive processes assessed with these two different approaches.

Other suggestions have also been made regarding future directions with the specific measures of inhibition. For example, the blocked version of the Stroop Colour Word task should be compared directly to the discrete trials approach, both in terms of how each relates to ADHD and how each relates to other measures of inhibition and interference. Also, the S-R compatibility task should be studied in children with ADHD as a means of assessing S-R Relevant overlap, and the amount of prepotency should be experimentally manipulated to determine whether this is the shared element of all of the inhibitory deficits in ADHD.

Perhaps the strongest element of this study is the fact that it borrowed heavily from the cognitive literature, in order to select a diverse array of

measures of inhibition, to design each task in order to be able to isolate the inhibitory process as much as possible, and to be able to interpret the findings in the context of decades of research on each task. Indeed, one of the strengths of Barkley's (1997a) model is that it was built upon broader (i.e., not ADHD specific) neuropsychological theories. Further research aimed at understanding the nature of the deficits in ADHD should continue to take advantage of the progress made in other related areas, and conversely, should strive to inform these other literatures based on patterns in a clinical sample.

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Appendix A

Recruitment notice for the Attention Deficit Association of Nova Scotia newsletter

Study on Inhibitory Control

Dear Parents and Guardians,

Some of you may have attended Dr. Russell Barkley's workshop in Halifax in September, 1998. If you recall, Dr. Barkley proposed that the primary deficit in children with ADHD is with inhibiting responses. In the classroom, problems with inhibition might be seen as an inability to refrain from getting out of one's seat, or talking out of turn. In laboratory tests, inhibition could be seen as an inability to filter out distracting information, or to stop an action once it has already been started. While Dr. Barkley's model is very promising, more research is needed in order to determine whether, in fact, children with ADHD have difficulties with inhibitory control.

I am a PhD student in clinical psychology at Dalhousie University, working under the supervision of Dr. Ray Klein. The goal of my dissertation is to determine whether children with ADHD have less inhibitory control than other children. To accomplish this goal, I have been administering several measures of inhibition to children with and without ADHD.

I am looking for children to participate in this study. I am looking for boys and girls, aged 9-13 years, who have received a diagnosis of attention deficit hyperactivity disorder from a physician. Children will be asked to participate in three 90-minute sessions at Dalhousie University, and will be given $10 for each hour of participation (totalling $45).

Interested parents who contact me at the number below will be sent a letter detailing the study, including a description of the tasks, and important ethical considerations (for example, that the study is entirely voluntary and confidential). If you would like to be sent further information, or would like to speak to me, or my supervisor, Dr. Ray Klein, about our study, please contact us at 494-6551.

Thank you,

Elizabeth McLaughlin

Appendix B

Community Recruitment Notice

Child Psychology Study

Are you a parent or guardian of a 9-13 year old boy or girl?

I am looking for children to participate in our computer tasks (for example, naming the colours of words appearing on a computer screen).

Children will be asked to participate in three 90-minute sessions at Dalhousie University, and will be given $45 for their participation.

I am a PhD student in clinical psychology at Dalhousie University, under the supervision of Dr. Ray Klein.

If you would like to be sent further information, or would like to speak to me or to Dr. Klein, about our study, please contact us at 494-6551.

Thank you

Elizabeth McLaughlin

Appendix C

Information letter for parents or guardians

Dear Parent or Guardian,

I am a PhD student in clinical psychology at Dalhousie University. I am working under the supervision of Dr. Ray Klein, on a study of inhibitory control in children with attention deficit hyperactivity disorder (ADHD). I have received approval for this study from Dalhousie University. This letter is to invite you to give consent for your child to participate in this study.

Recently, leading ADHD expert, Dr. Russell Barkley, proposed that the primary deficit in children with ADHD is with inhibiting responses (and not with attention, as was previously thought). In the classroom, problems with inhibition might be seen as an inability to refrain from getting out of one's seat, or talking out of turn. In our laboratory tests, inhibition could be seen as an inability to filter out distracting information, or to stop an action once it has already been started. While Dr. Barkley's model is very promising, more research is needed in order to determine whether, in fact, children with ADHD have problems with inhibition. The goal of this project, therefore, is to determine whether children with ADHD have less inhibitory control than other children. To accomplish this goal, we will be administering several tests of inhibition to children with and without ADHD.

An example of one of these tests is the "stop signal" task. For this task, the child is asked to look at a computer screen and to press one of two buttons to indicate which letter appeared. On some of the trials, a "beep" noise will occur, which tells the child not to press any buttons on that trial. We can measure inhibitory control by measuring how well each child can withhold responding when the beep sounds.

Two of the tasks require that we monitor eye movements. When we do this, participants sit comfortably in front of a computer screen, wearing a headset with two small cameras attached to it. They then either look at, or make key board responses to items that appear on the computer screen. This procedure allows us to measure where the child is looking.

On one of our eye movement tasks, the child is required to look at the centre of the screen while an object (for example, a star) quickly appears and disappears somewhere on the screen. The child is asked to wait until a signal occurs which lets him or her know to look at the location that the star had previously appeared. On this task, we are predicting that children with poor inhibitory control will make an eye movement before they are given the signal to do so.

Procedures: If you give your consent to have your child participate, three 90 minute appointments will be scheduled for you, at your convenience (usually after school or on weekends), at the Life Sciences Centre, Dalhousie University. If your child is currently taking stimulant medication to help with his or her symptoms of ADHD, we would ask that your

child does not take his or her medication for 24 hours prior to each session. This is so that we can measure your child's performance without the use of medication.

Over the three sessions we will administer three types of tasks. The first are measures of general ability level (reading, defining words, putting together blocks). These measures will help us to ensure that our participants with and without ADHD all have the same level of cognitive abilities. The second type will require participants to sit in front of a computer and to make responses to items on the computer screen (such as in the stop signal paradigm, described above). The third type involves eye movement monitoring. This is a non-invasive system. We have already administered these tasks to 26 children; children tend to enjoy participating in these tasks.

All of our measures of inhibition are designed to be more like games than tests. Children will be told that they should respond naturally, and that performance will not be judged as being good or bad. Elizabeth McLaughlin will be present with your child at all times through each of the tasks. Your child will also be invited to take breaks at regular intervals. Participants will be given the opportunity to earn tokens for following the rules of the laboratory (for example, not touching the computer or eye movement monitoring equipment unless invited to do so). At the end of each session, children will be able to exchange their tokens for small prizes.

At some point (either during one of your child's sessions, or at your convenience) we would also ask that you fill out some questionnaires. It is important for us to know whether the children in our ADHD and non-ADHD groups differ in any ways other than the presence of ADHD (for example, learning disabilities, seizure disorders, etc). These scales ask about your child's educational, medical, and family history, as well as your child's current behaviour. It is also very important for us to know if your child's strengths and weaknesses are the same across different situations (such as home and school). Therefore, we are also requesting that you ask your child's teacher to complete two rating scales.

Important ethical considerations: Participation in this study is entirely voluntary. You are under no obligation to volunteer. Even if you give your written consent, your child will still be told on the day of the study that his or her participation is entirely voluntary. The nature of the study will be explained to your child and he or she will be asked if he or she would like to participate. Your child can refuse to participate, or can stop participating at any time during the experiment.

Your identity, and that of your child, will be kept confidential at all times. Your child's data, and the information that you provide on the questionnaires, will be identified with an alphanumeric code. If the results of this study are published, only group or anonymous data will be presented, making it impossible to identify you or your child. All of the data will be locked safely in our laboratory until it is destroyed.

There are no known physical or psychological risks associated with participation in this study. In the unlikely event that your child expresses any discomfort while completing the tasks, it will be made clear to him or her that he or she can take breaks from or withdraw from the study at any time.

Some children (and their families) may find it difficult for the child to go off their stimulant medication for three 24-hour periods. Such children may become hyperactive and distractible during that period. While we know of no adverse physiological consequences of stopping and starting stimulant medication, we encourage you to discuss any specific concerns that you may have with your prescribing physician. We ask that our participants come to the sessions free of stimulant medication because medication might significantly alter the ADHD child's performance on our measures. Our study's goal is to understand the deficits associated with unmedicated ADHD. However, in the event that you feel that the cost of staying off medication for 24 hours outweighs the benefits of participating in this study, we would encourage you not to participate. If you are particularly concerned about how being off medication would affect your child's performance at school, we could schedule your appointments on weekends or holidays.

There are no direct benefits for participation in this study. Your participation, however, will help us to learn more about ADHD, and may benefit others in the future. As well, you will be given $10 for each hour of participation (totaling $45), to compensate for your and your child's time, and for the cost of parking and transportation. Finally, your child will be given the opportunity to earn small gifts for following laboratory rules.

If you are willing to have your child participate, please sign the consent form (next page), and call us at 494-6551 to set up an appointment. You may keep this letter for your information, but please bring the "Parent/ Guardian Informed Consent Form" to your first session. Please call or write to us if you would like to know more about this study, or if you would like a copy of the results. You can contact Dr. Ray Klein or myself by calling 494-6551 or by writing to us at Department of Psychology, Dalhousie University, Halifax, NS, B3H 4J1.

Sincerely,

Elizabeth McLaughlin

Appendix D

Informed Consent Form

Parent/Guardian Informed Consent Form

I, _____ give consent for _____ to take
(your name) (child's name)
part in the study on inhibitory control in ADHD. By signing my name below, I am indicating that I have read the description of the study and that Elizabeth McLaughlin has answered related questions to my satisfaction.

I understand that I can refuse consent or withdraw my consent at any time and for any reason. My child will also be told that his or her participation is entirely voluntary and that s/he can withdraw from the study at any time.

I also understand that my child's data will be identified with an alphanumeric code, and that therefore, his or her data will be completely confidential.

If I have any general questions about this study, I may feel free to contact Elizabeth McLaughlin or Dr. Ray Klein at 494-6551. This study has been reviewed by the Dalhousie Psychology Ethics Committee. If I have any specific ethical concerns, I can contact a member of the Dalhousie University Psychology Ethics Committee.

Signed,

_____ _____ _____
(signature) (relationship to child) (date)

Consent to be videotaped

I have given consent for my child to participate in the study on inhibitory control in ADHD. I also give consent for my child's testing sessions to be videotaped, so that important information about my child's behaviour during testing (for example, getting out of seat, talking during tasks) can be measured.

I understand that the videotapes will be identified by an alphanumeric code, will be viewed only by those directly involved with the study, and will be kept in a locked cabinet until they are destroyed. I also understand that my child will be aware that he or she is being videotaped and that he or she will also be asked for his or her permission to have the session videotaped. I have been told that I can withhold my consent for my child to be videotaped, and still have my child participate in the rest of the study.

Signed,

_____ _____ _____
(signature) (relationship to child) (date)

Appendix E

Letter accompanying parent questionnaires and rating scales

To Parents and/or Guardians of study participants:

Thank you for giving your consent to have your child participate in our study on inhibition in attention deficit hyperactivity disorder (ADHD). Our goal is to obtain further understanding of the underlying deficits of this pervasive and often debilitating disorder.

As you know, for this study, we are trying to identify differences in inhibitory control in children with and without ADHD. It is important for us to know whether the children in our ADHD and non-ADHD groups differ in any ways other than the presence of ADHD (for example, learning disabilities, seizure disorders, etc). Therefore, we would like to ask you to complete the following questionnaires, which ask about your child's educational, medical, and family history, as well as your child's current behaviour.

Any identifying information (such as your child's name) will only be seen by Elizabeth McLaughlin (the student in charge of this project) and/or Dr. Klein's research assistant. From that point forward, the information contained in this questionnaire will be referred to with an anonymous alphanumeric code, which can be later matched to your child's test performance. We ask that you try to answer each question to the best of your ability. If, for any reason, you choose not to answer some of these questions, we ask that you proceed to the ones that you are willing to answer, and we still invite your child to participate in the remainder of the study.

In some cases, we may require further elaboration of your responses. For example, if you indicate that your child has had a head injury, we may wish to contact you to obtain further details about the nature and severity of the injury. Do you give permission to Ms. McLaughlin to contact you by phone, if necessary? If so, please tell us how you would like us to contact you.

Name of person to contact: _____
Relationship to child: _____
Phone number (home or work): _____
Best time to call: _____

Please note that this information will be used for research purposes only (and not for diagnostic or treatment purposes). However, in rare cases, responses here may indicate a potential problem that could warrant medical attention. For your child's protection, if your responses suggest clear evidence of a significant reason for concern, Ms. McLaughlin will draw this to your attention. (Ms. McLaughlin will make this decision by consulting child psychologist, Dr. D. Waschbusch.) Please note that your responses are completely confidential, and (with the exception of the things that we are ethically bound to report: child abuse, or evidence that your child is at risk of harming himself or herself, or others), Ms. McLaughlin will not divulge your responses to your child's teacher, physician, or anyone else.

Also included in this package are two forms that we would like your child's teacher to complete. It is very important for us to know if your child's strengths and weaknesses are the same across different situations (such as home and school). We are asking for your assistance in obtaining this information. Please give the designated envelope to your child's teacher. (Note: If your child has more than one teacher, choose one based on how well you think each teacher knows your child. In most cases, this would be the child's home room teacher.)

We invite you to look at the letter and forms that are to go to the teacher. If, for any reason, you are not comfortable in giving these forms to your child's teacher, feel free not to. Your child may still participate in the remainder of the study, even if you decide not to pass the teacher forms along. It is important to be aware that if you do give the forms to the teacher, your child's participation in this study will no longer be entirely confidential (because the teacher will be aware of his or her participation).

If you do give your consent to have the child's teacher fill out the forms, please fill out the attached permission sheet. This authorizes your child's teacher to complete the forms, and asks that he or she keep your child's participation confidential.

If you have any questions or concerns about the items in these questionnaires, please contact Elizabeth McLaughlin or Dr. Ray Klein at 494-6551.

Thank you again for your participation. We know that these forms can take a long time to complete. Please know that the information that they give us is essential for making a proper interpretation of the results of our study.

Sincerely,

Elizabeth McLaughlin

Appendix F

Participant Information Questionnaire

Participant Information

- Please answer all of the questions that you can.
- Feel free to ask for clarification of any items.
- If you are completing this form on behalf of a participant in the study, please answer the questions with respect to the participant.
- If, for any reason, you choose not to answer some of these questions, please skip ahead to the next question that you are willing to answer.

Thank you for helping with our study.

A. General

1. Name of child: _____

2. Birth date: _____

3. Age of child: _____

4. Sex of child: _____

5. Ethnicity of child: _____

6. Handedness of child: (check one) Right__ Left__ Both __ Don't know ___

7. Rater's name: _____

8. Rater's relationship to child: _____

- Please answer all of the questions that you can.
- Feel free to ask for clarification of any items.
- If you are completing this form on behalf of a participant in the study, please answer the questions with respect to the participant.
- If, for any reason, you choose not to answer some of these questions, please skip ahead to the next question that you are willing to answer.

B. Educational history

1. Current grade: _____

2. Has child ever been in a special education program? No Yes (Circle answer)

(If No, please move on to question #3)

2a) If yes, please describe the class (for example, was it for children with learning disabilities? with behavior or emotional disorders?)

_____

_____

2b) Number of months/years in special program? _____

2c) Currently in special program? No Yes

3. Has child ever received resource help? No Yes

3a) If yes, for what subject(s)? _____
3b) For how many months/years?_____

4. Has child ever received a diagnosis of a learning disability? No Yes

4a) If yes, when? _____
4b) What type? Verbal____ Nonverbal____ Don't know____

4c) Has he or she received any treatment for the learning disability? No Yes
4d) If yes, what type of treatment? _____
4e) For how long? _____

5. Has child ever been suspended from school? No Yes

5a) If yes, how many times? ___

6. Has child ever been expelled from school? No Yes

6a) If yes, how many times? ___

C. Medical history

1. How would you describe child's health? (Circle one)
   Very Good Good Fair Poor Very Poor

2. How is his or her hearing?
   Good Fair Poor

3. Does child wear a hearing aid? No Yes

4. Has his or her hearing ever been tested? No Yes
   4a) If yes, what were the results? _____

5. If the child's hearing has never been tested, do you have any reason to suspect any difficulties? No Yes
   5a) If yes, please explain: _____

6. How is his or her vision?
   Good Fair Poor

7. Does child wear glasses? No Yes

8. Has his or her vision ever been tested? No Yes
   8a) If yes, what were the results? _____

9. If the child's vision has never been tested, do you have any reason to suspect any difficulties? No Yes
   9a) If yes, please explain: _____

10. Is child colour blind? No Yes Don't know

11. How is his or her gross motor coordination (running, jumping, bike riding)?
   Good Fair Poor

12. How is his or her fine motor coordination (writing, doing up buttons)?
   Good Fair Poor

13. How is his or her speech articulation?
   Good Fair Poor

14. Has he or she had any chronic health problems (for example, diabetes, asthma, heart condition)? No Yes
   14a) If yes, please specify: _____

   14b) If yes, when was the onset of the chronic illness? _____

15. Has child had any accidents resulting in the following? (Check those that apply)

Broken bones ___ Eye injury ___
Severe lacerations ___ Lost teeth ___
Head injury ___ Sutures ___
Severe bruises ___ Other (please specify) _____
Stomach pump ___

16. How many accidents has the child had? (Circle answer)

One 2-3 4-7 8-12 12+

17. Does child have any problems sleeping? No Yes

17a) If yes, please specify by checking one:

Difficulty falling asleep? ___ Difficulty staying asleep? ___
Early morning awakening? ___ Other? _____

17b) Is child a restless sleeper? No Yes Don't know

18. Does child have tics? No Yes

18a) If yes, are the tics Motor?___ Verbal?____ Both?____

19. Has child ever received a diagnosis of Tourette's disorder? No Yes

19a) If yes, is she or he being treated for this? No Yes

19b) In what way? _____

20. Has child ever had a seizure? No Yes

20a) If yes, has child been diagnosed with a seizure disorder or epilepsy? No Yes

20b) If child has had a seizure, but has not received a diagnosis, please indicate why. _____

20c) Is child receiving treatment for a seizure disorder? No Yes

20d) What treatment? _____

21. Has child ever received a diagnosis of ADHD? No Yes

21a) If yes, when? _____

21b) By whom? (profession only - check one)

Family doctor___ School Psychologist___
Other Psychologist____ Other? (please specify)_____

21c) Has there been any change in the diagnosis since the child was first diagnosed with ADHD? No Yes

21d) If yes, what was the change, and why? _____
_____

21e) How old was the child when you first started noticing symptoms of ADHD? _____

22. Has child ever been diagnosed with any of the following? Check those that apply. If yes, indicate when the child was first diagnosed, and whether, to your knowledge, the child currently meets criteria for the diagnosis.

An anxiety disorder?___
Age when diagnosed:_____ Current? No Yes

Depression?___
Age when diagnosed:_____ Current? No Yes

Bipolar mood disorder?___
Age when diagnosed:_____ Current? No Yes

Oppositional defiant disorder?___
Age when diagnosed:_____ Current? No Yes

Conduct disorder?___
Age when diagnosed:_____ Current? No Yes

Autism?___
Age when diagnosed:_____ Current? No Yes

Asperger's disorder?___
Age when diagnosed:_____ Current? No Yes

Psychosis?___
Age when diagnosed:_____ Current? No Yes

Schizophrenia?___
Age when diagnosed:_____ Current? No Yes

Neurological disorder? (specify) _____
Age when diagnosed:_____ Current? No Yes

Other? (specify) _____
Age when diagnosed:_____ Current? No Yes

D. Treatment history

1. Has the child ever been prescribed any of the following prescription medications?
No Yes

If yes, check those that apply. For each, indicate how long the child took the medication for by stating how old the child was when he or she started and stopped taking the drug. If less than one year, indicate how many months the child took the drug.

Ritalin ___	Age started: ___	Age stopped: ___ Months?__
Dexedrine ___	Age started: ___	Age stopped: ___ Months?__
Cylert ___	Age started: ___	Age stopped: ___ Months?__
Tranquilizers ___	Age started: ___	Age stopped: ___ Months?__
Anti convulsants ___	Age started: ___	Age stopped: ___ Months?__
Antihistamines ___	Age started: ___	Age stopped: ___ Months?__
Other (specify) _____	Age started: ___	Age stopped: ___ Months?__
Other (specify) _____	Age started: ___	Age stopped: ___ Months?__

2. Is child currently taking any medication? No Yes

2a) If yes, please fill out the following:

Drug _____
Regimen (Dose/day, time)_____ Date started: _____
Drug _____
Regimen (Dose/day, time)_____ Date started: _____
Drug _____
Regimen (Dose/day, time)_____ Date started: _____

3. If child is currently taking medication to reduce the symptoms of ADHD, please indicate how effective you think it is at doing so. Circle one.

Not effective Somewhat effective Effective Very Effective

4. Has the child ever had any of the following forms of psychological treatment?
4a) If so, for what disorder or problem? _____

4b) Check those that apply and indicate how long treatment lasted for (in months).

Individual psychotherapy	___	Duration of treatment _____
Group psychotherapy ___		Duration of treatment _____
Family therapy with child ___		Duration of treatment _____
Inpatient evaluation ___		Duration of treatment _____
Residential treatment ___		Duration of treatment _____
Other (specify) _____		Duration of treatment _____

E. Family history

1. Who does child live with? (Please include brothers and sisters and indicate their age and grade.)

2. If child does not live with both biological mother and biological father, please indicate reason. (If parents are divorced or separated, please give year of divorce/separation.)

3. Is the child adopted? If yes, at what age was he or she adopted?

4. Please indicate child's mother's:

Type of employment: _____
Education (highest grade or degree): _____

5. Please indicate child's father's:

Type of employment: _____
Education (highest grade or degree): _____

6. What is the family's approximate annual salary? _____

6. Please indicate if there is a family history of any of the following.

When yes, place a check next to the item and indicate the relationship of the family member to the child (for example, paternal grandmother = grandmother on father's side, maternal uncle = uncle on mother's side) Include relatives by marriage and siblings.

Problems with aggressiveness, defiance, oppositional behaviour as a child:

Problems with attention, activity, and impulse control as a child:

A diagnosis of ADHD:

Learning disabilities:

Failed to graduate from high school:

Mentally challenged:

Psychosis or schizophrenia:

Depression for greater than 2 weeks:

Bipolar mood disorder:

Anxiety disorder (specify if possible):

Tics or Tourette's disorder:

Alcohol abuse:

Substance abuse:

Antisocial behaviour (assaults, thefts, etc):

Arrests:

Physical abuse:

Sexual abuse:

Other (please specify): _____

Thank you very much!

Appendix G

Release of information form for teachers

Date: _____

To: _____
(child's teacher)

I have given consent for _____ to participate in the following
(child's name)
study on inhibitory control.

I also give consent for you to fill out the attached forms and to return them to Ms. McLaughlin.

Signed,

_____
(signature)

_____
(relationship to child)

Appendix H

Information letter for teachers

Dear Teacher,

I am a PhD student in clinical psychology at Dalhousie University. I am working under the supervision of Dr. Ray Klein, Dalhousie University.

One of your students is a participant in our research project on inhibitory control in children with and without attention deficit hyperactivity disorder (ADHD). Recently, leading ADHD expert, Dr. Russell Barkley, proposed that the primary deficit in children with ADHD is with inhibiting responses (and not with attention, as was previously thought). In the classroom, problems with inhibition might be seen as an inability to refrain from getting out of one's seat, or talking out of turn. In our laboratory tests, inhibition could be seen as an inability to filter out distracting information, or to stop an action once it has already been started. While Dr. Barkley's model is very promising, more research is needed to determine whether, in fact, children with ADHD have problems with inhibition. The goal of this project, therefore, is to determine whether children with ADHD have less inhibitory control than other children. We will be administering several different tests of inhibition to children both with and without ADHD.

It is important for us to know whether children in our ADHD and non-ADHD groups differ in ways other than the presence of ADHD. It is also very important for us to know if each child's strengths and weaknesses are the same across different situations (such as home and school). Therefore, we are requesting that you take the time to complete the attached rating forms. We have included a self addressed, stamped envelope so that you can mail the forms back to us.

You should find a letter signed by the child's parent indicating that they give you permission to complete these forms. Please note that this letter also asks you to keep your responses, as well as the child's participation in this study, completely confidential.

We know that these forms can take a long time to complete. Please know that the information that they give us is essential for making a proper interpretation of the results of our study. If you would like to know more about this study, please feel free to contact Dr. Ray Klein, or myself at 494-6551.

Thank you for your participation.

Sincerely,

Elizabeth McLaughlin

Appendix I

Medication questionnaire

Session (1, 2 or 3): ____

Date: _____

Time: _____

Child's name: _____

Relationship to child: _____

Does your child usually take any prescription medications for ADHD/ADD?
(circle one) No Yes

If no, you do not need to complete the rest of this form.

If yes, please complete the following chart.

Drug	Regimen (Dose/day, time) Please note if the drug is "slow release" or "regular release"	When did your child take his or her most recent dose of this medication? (Date and Time)
1.		
2.		
3.		

Note: If your child has taken his or her medication in the last 24 hours, please let me know immediately.

Thank you.

Elizabeth