Visual and Auditory Habituation Processes: A Comparison of ADHD Adults and Controls

By

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Abstract

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Habituation has an important role in attention. By reducing our sensitivity to a constant source of stimulation, it frees up attention resources to process new distinct items. Individuals with damage to frontal lobe brain regions have difficulty habituating to irrelevant stimuli and their behavior is marked by distractibility, hyperactivity and impulsivity. Attention-Deficit/ Hyperactivity Disorder (ADHD) is thought to be caused by a disruption in frontal lobe functioning and is also marked by distractibility, hyperactivity and impulsivity. As there are similar attentional and behavioral disruptions in both disorders it is speculated that incomplete or slowed habituation may be an important factor in ADHD. Incomplete or slowed habituation may result in difficulties sustaining attention on tasks due to an inability to modulate the repeated intrusion of irrelevant stimuli. The present study attempted to test this hypothesis by examining visual and auditory habituation processes and correlating self-reports of sensory gating disturbances with response habituation. A total of 41 adults (21 ADHD and 20 Controls) participated in the study. Troxler fading, a phenomenon of visual perception, in which an object in peripheral vision fades if one fixates on a central point, was employed to measure visual habituation. A modified auditory Troxler fading task was created for this experiment to measure auditory habituation. Findings of the present study replicated previous research which found slowed visual habituation to stationary stimuli in children

with ADHD (see Jansiewicz et al., 2004). The present study also extended prior research and found that adults with ADHD were slower to habituate to auditory stationary stimuli. In addition, and unexpected, adults with ADHD were quicker to respond to novel auditory moving stimuli suggesting the presence of comorbid auditory processing difficulties. The self-reported ratings of sensory gating disturbances were significantly greater for individuals with ADHD. The SGI was also significantly correlated with visual and auditory habituation. These findings further support impaired auditory and visual habituation in ADHD. Further investigation of habituation processes should combine ERP and fMRI measures to explore the underlying neural components.

Dedication

This dissertation is dedicated to my parents, Marianne and Carl Massa, who made it possible for me to pursue my degree. It was from both of you that I inherited my own special brand of curiosity. You know the type that annoys most people - Why? Thanks so much for loving me and believing in my ability to contribute something to the world.

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Chapter I

Introduction

Attention-Deficit/Hyperactivity Disorder (ADHD) is one of the most common disorders of childhood (American Psychological Association (APA), 2000; Barkley, 1998). The disorder is characterized by a persistent pattern of inattention and/or hyperactivity-impulsivity that is more frequent and severe than observed in individuals of comparable levels of development (APA, 2000). ADHD is most commonly diagnosed in children and is believed to affect 3–7% of the population (APA, 2000). Recent clinical and empirical evidence indicates that 50-70% of children with ADHD display the cardinal signs of the disorder well into adulthood (Barkley, 1998). Even though ADHD is generally diagnosed in childhood a number of individuals are not diagnosed until they become adults. In fact, the alarming increase in the number of adults diagnosed with ADHD in the past decade has caused many critics to dispute the validity of an adult form of the disorder.

Much of the controversy over establishing an adult form of the disorder stems from the fact that diagnosing ADHD is not an easy task. ADHD is not viewed as a medical illness, as is cancer or diabetes, and thus there is no standard battery of tests to conclusively prove that it is present or absent. Rather, the diagnosis of ADHD is facilitated by standardized rating scales that are completed by the presenting patient, their parent(s), partner or boss. These rating scales are not only highly subjective but may be impacted by inaccurate or biased childhood memories (Barkley, 1998). The diagnosis of adult ADHD is further complicated by the overlap of ADHD symptoms with other medical and psychiatric disorders (Faraone, Biederman, Feighner, & Monuteaux, 2000).

Finally, and perhaps most important, is the fact that diagnosis of adult ADHD is based on the established childhood criteria. At present, it is unknown if adult ADHD is identical to the childhood form or if the subtypes of the disorder generalize to adults. Any or all of these complications can lead to misdiagnosis or overdiagnosis of ADHD in adult populations (Shaffer, 1994; McGough & Barkley, 2004).

Even though great strides have been made in cognitive psychology and neuropsychology in understanding the cognitive and behavioral deficits associated with ADHD, we have been unable to uncover the specific cognitive deficits associated with the disorder. One of the most prominent changes in ADHD symptoms across the lifespan is seen in the outward display of behavior. ADHD adults no longer manifest the gross motor activity commonly displayed by their childhood counterparts. Instead, adults report feelings of restlessness, an inability to sit still or relax (Wender, 1995; Barkley, 1998). This reduction in motor hyperactivity suggests that inattention may be the most prominent symptom of adult ADHD. If this is true it is then possible to speculate that adult ADHD may be better explained by an inability to ignore or habituate to irrelevant stimuli, which in turn affects the ability to properly deploy attention.

Habituation is an automatic low-level learning process that plays an extremely critical role in attention (Cohen, Sparling-Cohen, & O'Donnell, 1993). By reducing our sensitivity to a constant source of stimulation, it accentuates new distinct items to attend to preferentially. Habituation plays an important role in filtering large amounts of information received from the environment. Without habituation we would be unable to distinguish meaningful information from our ever changing environment.

The primary goal of the present study is to investigate the visual and auditory habituation processes of adults diagnosed with ADHD. An additional goal of the present study is to assess self-reports of sensory gating disturbances to further examine habituation deficits in ADHD adults. In order to achieve these goals, the literature review (Chapter II) of this dissertation will be organized as follows: presentation of the primary characteristics of ADHD, epidemiology of ADHD, current theories of ADHD, impact of current research findings on theories of ADHD, and the present study rationale, which will include a discussion of theories and measures of habituation.

Chapter II

Literature Review

2.1 Attention-Deficit/Hyperactivity Disorder: Background and Overview

At the present time the primary characteristics of ADHD as set forth in the DSM-IV-TR (APA, 2000) for children with ADHD are generally used to diagnose adult ADHD. In other words, to be diagnosed with adult ADHD there must be credible evidence indicating that the presenting adult experienced the symptoms of ADHD in childhood (Barkley, 1998) because "ADHD does not develop de novo in adult life" (Wender, 1995, p. 765). As such, it is necessary to provide the childhood characteristics and diagnostic criteria of the disorder before discussing the primary characteristics of adult ADHD. It is also important to note that the DSM-IV-TR criteria only represent guidelines to follow when making a diagnosis of adult ADHD. Clinical judgment as well as common sense and flexibility are needed when applying these guidelines to individual cases (Barkley, 1998; Wender, 1998).

2. 2 Primary Characteristics and Diagnostic Criteria for Attention-Deficit/Hyperactivity Disorder

Children and Adolescents

ADHD is a common childhood psychiatric disorder that is characterized by a broad array of behavioral, cognitive, social, and emotional indicators. ADHD is usually diagnosed in early childhood or adolescence. The essential hallmarks of the disorder are a persistent pattern of inattention, impulsivity, and hyperactivity that is more frequent and severe than observed in individuals of comparable levels of development. The intensity and persistence of the symptoms characterize children with ADHD and separate them

from children without ADHD because intermittent attention, impulsive acts or hyperactivity are not necessarily abnormal behaviors in and of themselves (Wender, 2000).

Inattentive behavior is typically found to manifest in ADHD populations as failure to give close attention to details, careless mistakes on tasks and assignments, frequent shifting of attention from one uncompleted task to another, and distractibility or shortness of attention span. Individuals with ADHD often appear to be unfocused on the task at hand and appear as if they are not listening to or hearing what has just been stated. Inattention associated with ADHD can also be seen in social situations where individuals with ADHD often cannot follow conversations, detailed instructions on activities or the rules in game playing. Children with ADHD have difficulty sustaining attention on tasks and play activities. This makes it difficult for them to persist with activities (APA, 2000; Wender, 2000; Barkley, 1998).

The hyperactive behavior associated with ADHD is typically manifested by fidgeting and squirming when seated or the inability to remain seated, excessive climbing or running when not appropriate, restless moving of arms or legs while working or playing, difficulty playing quietly, and often acting as if "driven by a motor"(APA, 2000). The excessive movements often appear to have no purpose or goal because they are irrelevant to the present task or situation (Barkley, 1998). The hyperactive behavior is not just physical (i.e., unnecessary body movements); it may also be vocalized. Children with ADHD may talk excessively, talk to others out of turn, or make unusual vocal noises (i.e., humming or unusual sound effects) (Barkley, DuPaul & McMurray, 1990).

The impulsive behavior associated with ADHD can be seen as impatience, blurting out answers before questions have been completed, difficulty waiting for one's turn, and often interrupting others (APA, 2000). In addition, children who are impulsive are often not able to control their behavior. Children who are behaviorally impulsive are quick to respond to situations without waiting for adequate instructions. They also have little or no appreciation for what constitutes acceptable behavior in a given situation (e.g., running into the middle of the street to retrieve an errant ball without looking for oncoming traffic). Thus, they often fail to consider potentially dangerous consequences of their behaviors (Barkley, 1998). The inability to control behavior also impacts their relationships with peers because they may carelessly make socially inappropriate remarks, destroy others' property or become easily upset when playmates do not conform to their will. The impulsive behavior also results in poor planning and organization. Impulsive children are often sloppy dressers, have untidy rooms and unfinished school assignments (Wender, 2000).

The primary characteristics of ADHD and diagnostic criteria developed for clinical assessment are set forth in the Diagnostic and Statistical Manual of Mental Disorders-Fourth Edition, Text Revision (DSM-IV-TR; APA, 2000). ADHD has three recognized subtypes: Predominantly Inattentive Type, Predominantly Hyperactive-Impulsive Type, and Combined Type (exhibiting symptoms of Inattentive Type and Hyperactive-Impulsive Type). For a diagnosis of ADHD to be made, the behavioral manifestations must be in at least two settings: school, home, or social, and the onset of the disorder must occur by the age of 7 years old. The inattentive, hyperactive and impulsive symptoms listed in the DSM-IV-TR (APA, 2000) appear in Table 1.

Table 1. *DSM-IV-TR criteria for Attention-Deficit/Hyperactivity Disorder*

A. Either (1) or (2):

(1) six (or more) of the following symptoms of inattention have persisted for at least 6 months to a degree that is maladaptive and inconsistent with developmental level:

Inattention

(a) often fails to give close attention to details or makes careless mistakes in schoolwork, work, or other activities
(b) often has difficulty sustaining attention in tasks or play activities
(c) often does not seem to listen when spoken to directly
(d) often does not follow through on instructions and fails to finish schoolwork, chores, or duties in the workplace (not due to oppositional behavior or failure to understand instructions)
(e) often has difficulty organizing tasks and activities
(f) often avoids, dislikes, or is reluctant to engage in tasks that require sustained mental effort (such as schoolwork or homework)
(g) often loses things necessary for tasks or activities (e.g., toys, school assignments, pencils, books, or tools)
(h) is often easily distracted by extraneous stimuli
(i) is often forgetful in daily activities

(2) six (or more) of the following symptoms of hyperactivity-impulsivity have persisted for at least 6 months to a degree that is maladaptive and inconsistent with developmental level:

Hyperactivity

(a) often fidgets with hands or feet or squirms in seat
(b) often leaves seat in classroom or in other situations in which remaining seated is expected
(c) often runs about or climbs excessively in situations in which it is inappropriate (in adolescents or adults, may be limited to subjective feelings of restlessness)
(d) often had difficulty playing or engaging in leisure activities quietly
(e) is often "on the go" or often acts as if "driven by a motor"
(f) often talks excessively

Impulsivity

(g) often blurts out answers before questions have been completed
(h) often has difficulty awaiting turn
(i) often interrupts or intrudes on others (e.g., butts into conversations or games)

B. Some hyperactive-impulsive or inattentive symptoms that caused impairment were present before age 7 years.

C. Some impairment form the symptoms is present in two or more settings (e.g., at school [or work] and at home).

D. There must be clear evidence of clinically significant impairment in social, academic, or occupational functioning

E. The symptoms do not occur exclusively during the course of a Pervasive Developmental Disorder, Schizophrenia, or other Psychotic Disorder and are not better accounted for by another mental disorder (e.g., Mood Disorder, Anxiety Disorder, Dissociative Disorder, or a Personality Disorder).

Note: From American Psychiatric Association (2000, pp. 92-93). Reprinted by permission.

Interestingly, children with ADHD, regardless of subtype, can often sustain their attention when they find something in the environment of interest, for example, a video game, movie or a building project. The problem comes in controlling their attention; that is, directing and maintaining it when the object itself does not have the properties to maintain the child's interest (Wender, 2000; Barkley, 1998).

Adults

ADHD is currently viewed as a chronic disorder that originates in childhood, but the symptoms may frequently persist well into adulthood and can continue to cause distress. Even though clinicians and researchers in the 1960's and 1970's began to find evidence for the continuation of hyperactivity in adults, adult ADHD was not formally recognized until the 1980's. Wender, Reimherr, and Wood (1981) were the first researchers to identify adult ADHD and its core symptoms: inattention, hyperactivity, and impulsiveness. The researchers indicate that the symptoms of hyperactivity diminish in adulthood, but adults with ADHD continue to experience difficulties managing distractions, maintaining goal directed behavior, and maintaining healthy social relationships because of their short temper and impulsive behavior.

More recently Wender (2000) has indicated that only a small fraction of adults with ADHD report attention difficulties. However, Wender states that attentional capacity has not improved and that adults with ADHD continue to have poor attentional focus just like their childhood counterparts. He believes that the major reason that they do not report attentional difficulties is that they are no longer in school settings. They also may choose occupations that do not require them to focus their attention for long periods of time. In contrast to children with ADHD, adults with ADHD have learned that they have

attentional problems and compensate by avoiding situations that require focus in order to reduce their stress. However, adults with ADHD who attend college or technical school may continue to have persistent attentional problems, such as the inability to keep their "mind on" tasks they are not interested in or find boring. Attentional deficits also manifest in social activities and personal relationships. For example, an adult with ADHD often cannot watch an entire movie or read a novel from beginning to end. They also may not be able to concentrate on a conversation that they are having with their friends or relatives.

Adults with ADHD report many of the same symptoms of inattention that are listed in the DSM-IV-TR (APA, 2000) criteria and are reported by parents of children with ADHD. Murphy and Barkley (1996) found that adults diagnosed with ADHD self-report difficulties with sustained attention, difficulties focusing on conversations, failure to complete tasks or activities, frequent shifts from one uncompleted task to another and constant distraction.

The hyperactivity experienced by adults who have ADHD does not manifest as the gross motor activity (i.e., excessive running around, climbing, or acting "as if driven by a motor") commonly found in children with ADHD. Adults with ADHD often report experiencing a sense of restlessness, difficulties sitting still for long periods of time, difficulties relaxing, and disliking inactivity (Wender, 1995; Barkley, 1998). Adults with ADHD often prefer to stand and will move from task to task (Wender, 1995). Fidgeting and foot movements (also referred to as "Wender signs") continue to affect adults with ADHD. Constant motion of the feet, for example, toe tapping or the crossed-knee foot jiggle, is one of the most common observable signs of adult ADHD. Murphy and

Barkley (1996) found that adults with ADHD often fidget with their hands or feet, have difficulty remaining seated and complain that they often talk excessively.

Adults with ADHD also display impulsive behavior. The impulsive behavior of ADHD adults is consistent with the impulsive behavior displayed by children with ADHD. Adults with ADHD continue to make decisions without reflecting on the possible consequences of their behavior. The major difference between children and adults with ADHD is that adults may be at greater risk for self-injurious behavior (Wender, 1995). In fact, Barkley and Murphy (1998) report that adults with ADHD have increased risk for motor vehicle accidents. Interestingly, Barkley (1998) reports that when adult patients with ADHD are placed on driving simulators they have a tendency to accelerate instead of brake when faced with a critical road incident.

Adults with ADHD often have a history of multiple personal relationships ending without reflection. They also often quit their jobs without thinking of the financial problems this action may cause them or their dependents. Adults with ADHD, like their child counterparts, are also very disorganized. They may be unable to keep work records organized, have messy desks, untidy homes, as well as untidy appearances (Wender, 1995). To provide an example, Wender (1995) describes a female client he diagnosed with ADHD as being a literate, absentminded professor who was unable to keep her white blouse clean and could not properly align the buttons. Her home life was equally disorganized. She was unable to keep her refrigerator in order and often had fungus growing on food that had not been properly wrapped or discarded.

2. 3 Epidemiology of Attention-Deficit/Hyperactivity Disorder

At one time it was assumed that ADHD was a developmental disorder that would abate in adolescence or young adulthood with the maturation of the frontal lobes, which have the most protracted period of growth in human development (see Grodzinsky & Diamond, 1992; Barkley, 1998). The frontal lobes have been characterized as the seat of executive functioning. Executive processes include the ability to switch attention, inhibit prepotent responses, maintain task set and monitor conflict.

Recent clinical and empirical evidence, however, indicates that 50-70% of children with ADHD display the cardinal signs of the disorder well into adulthood (Barkley, 1998). Some studies report that approximately one half of children diagnosed with ADHD will continue to experience symptoms of inattention, restlessness, poor impulse control, and motor hyperactivity (Spencer, Biederman, Wilens, & Faraone, 1998). Thus, based on childhood prevalence, the percentage of adults with ADHD is estimated at approximately 2% (Wender, 1998). A more recent report indicates that the figure is the same as described for childhood populations, which is 3-7% (Lamberg, 2003).

It must be noted that the exact prevalence of adult ADHD is not known. A number of problems contribute to the lack of concrete prevalence rates for adults diagnosed with ADHD. First, the application of child-based criteria and the established subtypes of the disorder in childhood populations impact on the prevalence rate of adult ADHD. ADHD in adults may not manifest in the same way it does in childhood populations. Heiligenstein, Conyers, Berns, and Smith (1998) argue that the current DSM-IV-TR criteria are too stringent for use in adult diagnosis.

Second, the number of males diagnosed with ADHD outnumbers females by a ratio of 4 to 1 (APA, 2000; Barkley, 1998). The higher ratio of males in clinic samples may be due to selective referral rather than actual incidence. Furthermore, females may be more likely to exhibit internalizing symptoms that involve mood, affect and emotion, whereas males usually display more externalizing symptoms such as aggressive and antisocial behaviors. Thus, the ratio of males to females with ADHD may be due to the fact that the external behavioral symptoms displayed by males are more easily recognized, especially in the classroom setting. As a result of the disruptive classroom behaviors more referrals for males would also occur from school administrators.

According to recent research conducted by Hinshaw (2002) females experience significant struggles with ADHD symptoms that are often overlooked because their ADHD symptoms do not resemble the symptoms found in males. The reason for the discrepancy between male and female diagnosis is blamed on using male ADHD symptoms as the criteria marker against which females are measured. Adelizzi (1998) contends that researchers have neglected females who have ADHD because they do not generally display hyperactive behavior. Nadeau and Quinn (2002) stress that young women are at an increased risk for chronic low self-esteem, anxiety, depression, underachievement, and teen pregnancy. Undiagnosed adult women with ADHD are at an increased risk for divorce, financial crisis, single-parenting, eating disorders, substance abuse, and constant stress due to difficulty managing the demands of their daily life.

Unfortunately, the diagnosis of ADHD in males and females is complicated because there are no known biological causes of the disorder. As there are no medical tests that can be used to identify the presence or absence of the disorder, clinicians are over-reliant

on the behavioral criteria of the disorder. Additional research on the manifestation of ADHD in both genders will lead to a better understanding of the expression of ADHD symptoms and may lead to uncovering biomarkers of the disorder.

Finally, as adults age they are also more likely than children to suffer from a wide range of comorbid disorders (i.e., Antisocial Personality Disorder, anxiety disorders, affective disorders etc.) and are more prone to suffer medical conditions (i.e., high blood pressure, diabetes, hypo or hyperthyroidism etc.). It is difficult enough to separate factors associated with health complications from the normal aging process (Newman & Newman, 2003), let alone from those associated with ADHD. For example, differences in attentional performance may be attributed to taking prescription medication for a health related problem rather than symptoms of ADHD.

As can be seen, a number of issues affect the prevalence rate of adult ADHD and the figures provided above are only an estimate based on the prevalence of childhood ADHD. As it is extremely difficult to separate adult ADHD from co-occurring psychiatric and medical disorders it is likely that the disorder is even more heterogeneous in adults than children. The exact prevalence rate of adult ADHD will not be known until efforts are made to increase detection through the use of better assessment and diagnostic methods.

2. 4 Theories of Attention-Deficit/Hyperactivity Disorder

There has been abundant research conducted on ADHD, but there are very few theoretical models that explain both the cognitive and behavioral difficulties exhibited by individuals with ADHD. The main problem of not having useful models to explain ADHD is that research is mainly exploratory and descriptive (Barkley, 1997, 1998). The

goal of the present section is not to discuss every published theory on the emergence of ADHD, but to provide insight into the theoretical models that address both the underlying brain mechanisms involved in the disorder and how they may act to produce the cognitive deficits and observed behavioral symptoms of the disorder. To achieve this goal the following theoretical models will be discussed: Wender (1995), Barkley (1998), Sergeant (1996; 2000) and Halperin and Schulz (2006). Each theory's explanatory value in conceptualizing the current research findings on ADHD will be assessed in a latter section.

A. Wender's Theory of ADHD

In the early 1970's Wender developed a theory of ADHD based on the characteristics of children with Minimal Brain Dysfunction (MBD; a much broader and earlier conceptualization of ADHD; Wender, 1995). Wender (1995) argued that MBD may be genetic in origin and produced by decreased catecholaminergic functioning. The genetic basis of ADHD was postulated based on the following findings: (1) increased frequency of MBD among the siblings of individuals with MBD, (2) an increase of psychopathology (depression, alcoholism, and sociopathology) among the parents of MBD patients and an absence of psychopathology in the adoptive parents of MBD patients, and (3) the prevalence of MBD in foster children whose biological parents had psychiatric illnesses.

Wender hypothesized decreased catecholaminergic activity for the following reasons: (1) MBD-like behavior was exhibited in children recovering from Von Economo's encephalitis, (2) adults who recovered from symptoms of acute Von Economo's encephalitis manifested Parkinson's syndrome, (3) adults who died from Von Economo's

encephalitis had lesions of the basal ganglia (common in Parkinson's syndrome), (4) Parkinson's syndrome is associated with degeneration of dopaminergic neurons, (5) some drugs were effective in reducing or eliminating MBD symptoms indicating that the medication was remediating a biological deficit, (6) the most effective drugs were dopaminergic, and (7) dopaminergic drugs also decreased MBD symptoms in animals. Wender (as cited in Barkley, 1998) also described the following behavioral/psychological symptoms of children with MBD: short attention span, poor concentration, distractibility, hyperactivity, poor motor control, poor impulse control, mood instability, aggression, temper outbursts, emotional difficulties, social difficulties, and learning difficulties.

Wender (1995) theorized that the dysfunctions found in MBD children could be accounted for by three primary deficits: (1) decreased experience of pleasure and pain, which leads to less sensitivity to reward and punishment, (2) a high level of activation that is poorly modulated which leads to hyperactive behavior, poor impulse control, and emotional disturbances, and (3) extroversion. Wender further indicated that hyperactivity could lead to secondary poor concentration. Overall, Wender (1995, 2000) contends that ADHD is transmitted genetically and is associated with neurotransmitter deficiency.

Many researchers currently believe that noradrenergic and dopaminergic neurotransmitter deficiencies produce both the behavioral and cognitive deficits associated with ADHD (for a review see Pliszka, McCracken, & Maas, 1996; Solanto, 2002). In fact, both the cognitive and behavioral symptoms associated with ADHD are improved when individuals with ADHD take prescription stimulant medication. The improved cognitive functioning associated with the use of stimulant medications suggests that ADHD may result from a dysfunction of the prefrontal cortex as stimulants improve

the functioning of the prefrontal cortex (Solanto, 2002). Stimulant medications used to treat ADHD, however, have been shown to have a much greater beneficial effect on improving behavioral than cognitive performance (Swanson, Sergeant, Taylor, Sonuga-Barke, Jensen, & Cantwell, 1998). It has also been shown that the improved behavioral control and inattention associated with stimulant medications are not specific to ADHD, as normal children and adults have also shown improved attention and behavioral control (Rapoport, Buchsbaum, Weingartner, Zahn, Ludlow, & Mikkelsen, 1980). Overall, this indicates that deficiencies in dopamine and noradrenaline are not conclusive at this time and suggests that additional non-catecholaminergic neurotransmitter systems, such as serotonin may act with the catecholamines to produce the cognitive symptoms found in ADHD populations (Durston, 2003).

It is also highly likely that dysfunctional neurotransmitter systems have a profound effect on fundamental learning and memory processes, such as habituation. Alterations of neurotransmitters in ADHD can affect how the individual adapts to novel information in their environment, which in turn will affect long-term changes in neuronal structures. These alterations are brought about by neuromodulatory processes. Habituation is a neuromodulatory process that results in a reduction in post synaptic responses accompanied by reduced neurotransmitter release (Delcomyn, 1997; Thompson, 1993). A dysfunctional habituation mechanism may explain why individuals with ADHD have reduced neural transmitter release to specific brain regions. In fact, studies that have examined neurotransmitter influences on habituation in animals and humans have implicated roles for serotonin, dopamine, acetylcholine and glutamate (for review see Gu, 2002; Leussis & Bolivar, 2006).

B. Barkley's Hybrid Model of ADHD

Barkley (1997, 1998) developed a theoretical model of ADHD to address problems in the diagnosis of ADHD and the inconsistencies found in ADHD research. When he developed his model, he argued that it was needed for several reasons. First, the majority of research on ADHD was atheoretical. Second, because the current clinical view of ADHD was based on two purely descriptive behavioral deficits (inattention and hyperactivity-impulsivity), a model was needed to account for the many cognitive and behavioral deficits associated with ADHD. Finally, a model was needed to attempt to differentiate between ADHD subtypes. Before describing Barkley's model it must be mentioned that it is based on research findings that have noted the similar pattern of distractible, impulsive, and hyperactive behavior between individuals with ADHD and frontal lobe damage (Barkley, 1998, Benton, 1991). As Barkley's model is quite complex in comparison to other models on the causes of ADHD, the whole model is presented in Figure 1.

Barkley's (1998) theory of ADHD has been termed the Hybrid Model of ADHD because it seeks to explain the overlap between executive functions and their relationship to the behavioral disinhibition commonly seen in ADHD populations. Barkley believes that behavioral inhibition is the underlying mechanism of the executive system and it supersedes executive functions. In other words, Barkley believes that inhibiting a response is the first action that must occur before executive functions can perform their duties. According to Barkley, this should not be taken to mean that inhibition directly causes executive functions to occur, rather it "sets the occasion for their performance" (Barkley, 1997, p. 68). Thus according to Barkley, behavioral inhibition aids the

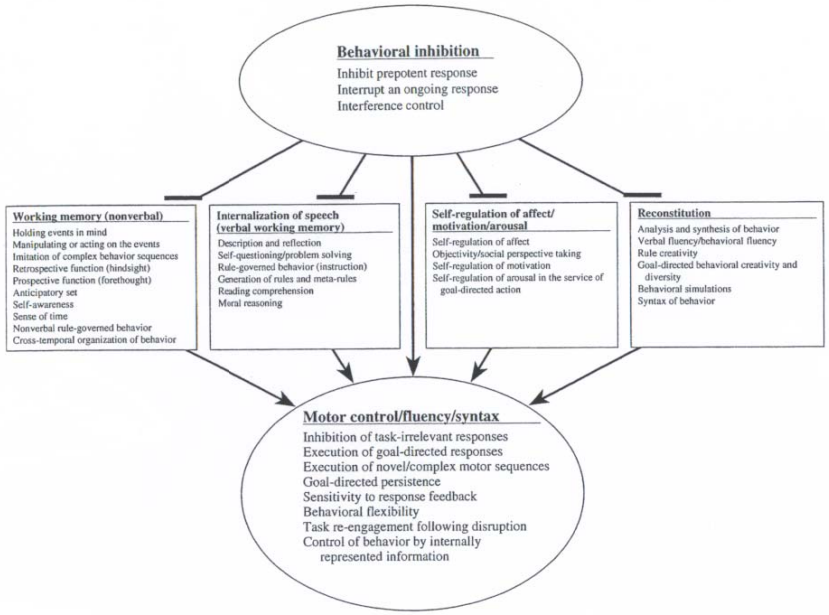

Figure 1. Diagram illustrating the complete hybrid model of executive functions (boxes) and the relationship of these four functions to the behavioral inhibition and motor control systems. From Barkley (1997b). Copyright 1997 by The Guilford Press. Reprinted by permission.

performance and support of the organism by inhibiting prepotent responses to an event, stopping an ongoing response or pattern of responding, protecting ongoing responses from interference by distractions, and maintaining goal-directed behavior.

Inhibition may be the core mechanism in Barkley's model, but he also recognizes the importance of four executive functions essential to the development of self-directed action: non-verbal working memory; internalization of speech (or verbal working memory); self-regulation of affect, motivation, and arousal; and reconstitution (or motor control).

The construct of working memory is defined as the ability to hold both verbal and nonverbal information (either separately or combined) in memory for immediate use in controlling an action (Goldman-Rakic, 1995). In Barkley's model of ADHD, poor nonverbal working memory can disrupt behavioral actions. This disruption can be seen in a child's inability to imitate behavioral sequences and this inability can lead to problems in temporal continuity. If a child has a disruption in verbal working memory he/she will be unable to develop the skills necessary to internalize speech. Difficulties internalizing speech will in turn cause problems following instructions.

In Barkley's model, self-regulation is not well developed in children with ADHD. He believes that self-regulation is linked to emotion, motivation and arousal. He proposes that children with ADHD have poor inhibitory control because they are unable to modify their emotional reactions to appropriately meet the demands of a task or situation. The inability to properly mediate arousal, emotion, and motivation, to control behavior can be made worse in the presence of an immediately satisfying reward.

Finally, according to Barkley's model, reconstitution is the ability to analyze and synthesize sequences of events or messages into their component parts in order to reconstruct them into new events or messages. Barkley believes that reconstitution is linked to inhibition because a delay in responding is necessary to deconstruct and reconstruct messages to provide meaning. This concept is also highly dependent on the child's ability to hold information in working memory.

Overall, Barkley's theory proposes that the executive system allows the individual to shift control of their own behavior from exogenous environmental demands to endogenous control. His theory of ADHD is based on "top-down" control. In other words, cognitive processes are controlled by thoughts or ideas about the nature of the material being processed rather than stimulus driven processing. The emphasis here is on controlled rather than automatic processing.

Barkley (1997) suggests that ADHD is a developmental disorder of behavioral inhibition that impairs the development of self-regulation and is not chiefly a disorder of attention as its name implies. Barkley defends this position by suggesting that not all forms of self-regulation are fully covert or internalized in the early school-age years and may not be so until early adolescence. Therefore, as we develop, our ability to control behavior shifts from being externally governed to being internally regulated. The behavioral response style of children with ADHD clearly indicates support for Barkley's theory of disruption in the four executive functions that aid inhibition.

The assumptions of Barkley's model are built upon the similar symptoms exhibited by individuals with frontal lobe damages and ADHD (i.e., distractibility, hyperactivity, and impulsivity; Barkley, 1998; Benton, 1991). As it has been noted that individuals with

frontal lobe damage have deficits in their ability to habituate to irrelevant stimuli (Luria, 1973; Fuster, 1997), it is also possible to speculate that the executive deficits noted in ADHD are caused by an impaired ability to habituate to irrelevant stimuli. The role that habituation may play in the cause of ADHD is further supported by the findings that stimulant medications improve functioning of the frontal lobe brain region (see Solanto, 2002).

C. Sergeant's Theory of ADHD

Sergeant and colleagues (1996, 1999, 2000, 2003) have proposed an alternative theory and it is referred to as the cognitive-energetic model of ADHD. The cognitive-energetic model is based on information processing and has been derived from theories proposed by Pribram and McGuiness and Sanders (for a complete review see Sergeant, 1996 and 1999). Information processing models emphasize the cognitive and psychophysiological processes that operate in a stage-like fashion between stimulus input and response output.

In the cognitive-energetic model, the overall efficiency with which an organism processes information is explained in three ordered stages: process, state (energetic), and management. Process factors mediate stimulus input and response output. Process factors are cognitive mechanisms and are described as the following abilities: encoding, search, decision, and motor organization. State factors are considered to be the energy state of the organism and include the following three energetic pools: arousal, activation, and effort. Management factors are control processes which include executive functions, working memory, goal directed behavior, planning, and error-detection.

In contrast to Barkley's hybrid model of ADHD, which proposes the cause of ADHD primarily in terms of top-down inhibitory control deficits, Sergeant and colleagues

attempt to address both the bottom-up and top-down causes of the disorder. To support their theory they argue that because poor inhibitory control is present in a variety of disruptive disorders of childhood and not specific to ADHD it cannot be the primary cause of ADHD. At the same time, even though inhibitory control is not the primary cause of the disorder, it is impaired in individuals diagnosed with ADHD and it can help to differentiate ADHD from other disruptive disorders. Sergeant and colleagues believe that lower level impairments in arousal, activation, and response organization are capable of explaining executive impairments and deficient inhibitory control.

Based on the cognitive-energetic model, the inhibitory deficits commonly found in ADHD can be explained by deficits at the management and energy stages. For example, a deficit in working memory, which has been recognized as an executive function, has been proposed as an alternative explanation to a lack of inhibitory control. According to Sergeant and colleagues (2003), information that is held in working memory represents information that is activated from long-term memory by stimulus-response relationships. Individuals with ADHD have impaired working memory and this affects their ability to monitor errors and make decisions to adjust their performance. Thus, the impairment in working memory inhibits learning over time. They further indicate that the ability to monitor errors is affected by reduced arousal and effort. Additional evidence supporting impaired state regulation in ADHD comes from the findings that individuals with ADHD have delayed responses to time-sensitive tasks and stimuli (Nigg, 2001; 2005).

The state factor dysfunctions proposed in Sergeant and colleague's model could be caused by an impaired ability to habituate to irrelevant environmental stimuli. Habituation is the dampening of arousal and activation (see Stephenson & Siddle, 1983).

If an individual was unable to habituate or had a slowed ability to habituate they would have fluctuating levels of arousal and activation, which would in turn impact attentional focus, as well as learning and memory processes. In the case of ADHD, an impaired ability to habituate would explain over-arousal in the face of novelty and the resultant disruptions in motor preparation. The disruption in motor preparation would be seen as hyperactivity and impulsivity.

D. Halperin and Schulz's Theory of ADHD

More recently, Halperin and Schulz (2006) proposed a developmental theory of ADHD that shifts focus from executive (top-down) function deficits to early noncortical dysfunction as a potential cause of the core pathophysiology of childhood ADHD. The authors argue that evidence from meta-analytic studies indicates that even though executive function deficits are commonly found in children with ADHD, the findings are not robust enough to distinguish ADHD relative to other clinical groups or non-ADHD individuals. Rather, Halperin and Schulz argue that executive function deficits cannot explain ADHD because there is a wide array of "non-executive" cognitive function differences (e.g., motor coordination, perception, visuomotor integration, language, learning and memory) found between individuals diagnosed with ADHD and non-ADHD controls. As individuals diagnosed with ADHD show poorer performance on a wide array of tasks that do not involve executive functions, the symptoms of ADHD may be better accounted for by disturbances to lower-level sensory and motor pathways.

Halperin and Schulz do not propose that executive functions play no role in ADHD. In fact, they are mindful of the reciprocal neural connections between the prefrontal cortex, limbic, and cortical regions and how impairments in these regions or their

connections can be seen to manifest in the behavioral, cognitive, motivational and emotional deficits associated with ADHD. Instead, they argue that the frontal lobe hypothesis of behavioral change after frontal lobe injury cannot be applied to explain the cause of ADHD in children. The frontal lobe hypothesis pertains to injury in the mature adult brain. As the prefrontal cortex in children is not fully developed until the second decade of life, injury to this area will not result in the same pattern of deficits typical of adults with frontal lobe impairments. In fact, research has indicated that young children who receive early injury to the prefrontal cortex rarely develop behavioral and social difficulties, but they can develop behavioral and social deficits in adolescence. In other words, Halperin and Schulz contend that if frontal lobe impairment caused ADHD then we should not see behavioral signs of distractibility, hyperactivity, or impulsivity in young children. Instead, the symptoms of ADHD, especially hyperactivity, diminish as children age.

Halperin and Schulz believe that it is more likely that the later development of the frontal lobes aids in top-down compensatory cognitive and neural self-regulation. Thus, continued maturation of the prefrontal cortex and development of more controlled processes aids in the recovery from ADHD. The development of controlled processes coincides with the diminishing symptoms of the disorder commonly found as children with ADHD mature into adolescents and young adults. The development of controlled processes actually moderates the impact of the more diffuse cognitive deficits found in ADHD populations. In other words, individuals with ADHD would use these effortful processes in situation where most individuals can rely on automatic processes.

Their theory does not imply that the prefrontal cortex is normal in individuals with ADHD as it has developed in a context of impaired lower level systems, just that damage to this area is not primary in ADHD. Instead, the theory posits a number of potential "bottom-up" areas that may contribute to the cause of ADHD. The areas include: the basal ganglia and midbrain dopamine systems, hindbrain noradrenergic mechanisms mediating arousal and activation, and the cerebellum. One of the important features of this theory is its attempt to explain both childhood and adult ADHD.

Disrupted habituation processes also fit well into this model. Early insult to noncortical areas will disrupt the formation and functioning of neural pathways. Impaired neuromodulatory mechanisms will in turn affect regulation and arousal, as well as attention, learning, and memory. In fact, early noncortical insult may predispose children to develop ADHD and thus cause lowered thresholds for activation (i.e., novelty or stress circuits) and this may increase their vulnerability to novel or stressful environmental events.

2. 5 Impact of Current Research Findings on the Present Theories of Attention-Deficit/Hyperactivity Disorder and Future Implications

The current explanatory models of ADHD have attempted to incorporate the research findings to explain the elusive cause of ADHD. Even though a number of models were presented earlier, the present section will focus on models that are most pertinent to the present study. The discussion that follows will attempt to briefly outline how the etiological findings provide support for the current theories and indicate important areas for future research.

Barkley's (1998) Hybrid Model of ADHD, which emphasizes dysfunctional top-down control, is supported by findings of decreased volume (Castellanos, Giedd, Marsh, Hamburger, Vaituzis, Dickstein, Sarfatti, Vauss, Snell, Lange, Kaysen, Krain, Ritchie, Rajapakse, & Rapoport, 1996; Filipek, Semrud-Clikeman, Steingard, Renshaw, Kennedy, & Biederman, 1997; Hesslinger, van Elst, Thiel, Haegele, Hennig, & Ebert, 2002; Mostofsky, Cooper, Kates, Denckla, & Kaufmann, 2002; Pliszka, Lancaster, Liotti, & Semrud-Clikeman, 2006; Seidman, Valera, Makris, Monuteaux, Boriel, Kelkar, Kennedy, Caviness, Bush, Aleardi, & Faraone, 2006) and abnormal activation of the frontal lobe regions of children, adolescents, and adults with ADHD (Vaidya, Austin, Kirkorian, Ridlehuber, Desmond, Glover, & Gabrieli, 1998; Bush, Frazier, Rauch, Seidman, Whalen, Jenike, Rosen, & Biederman, 1999; Durston, 2003; Schulz, Fan, Tang, Newcorn, Buchsbaum, Cheung, & Halperin, 2004).

Further support for this model comes from the numerous neuropsychological studies that have found that both children and adults with ADHD have deficits on measures of executive control compared to non-ADHD controls. These studies have identified impairments in response inhibition, complex problem-solving, and sustained effort on tasks (Barkley, 1997; Denckla, 1993; Seidman, Biederman, Weber, Hatch, & Faraone, 1998; Woods, Lovejoy & Ball, 2002). In addition, Barkley (1998) argues that the failure of numerous studies to find deficits in attentional processes, such as perception, filtering, and selection, further supports executive dysfunctions in ADHD because they have nicely documented impairments in motor control (see van der Meere & Sergeant, 1988a, 1988b).

Barkley's model does explain the hyperactive and impulsive behavior exhibited by children. However, as children with ADHD mature into adults with ADHD, the hyperactive symptoms of the disorder diminish but the inattentive symptoms are still very prominent. This suggests that the inability to inhibit motor behavior is not the primary cause of the disorder. In effect, Barkley theory does not explain the emergence of all of the associated symptoms of the disorder and it does not offer any theoretical explanations for the course and development of ADHD across the lifespan.

Despite the considerable evidence that supports Barkley's model it must be mentioned that there are a number of concerns that further impact the explanatory value of the model. First, the findings from brain imaging studies are inconsistent. There are discrepancies in the brain imaging findings with regard to the location and size of the prefrontal abnormalities and whether they included white matter or gray matter (for a review see Halperin & Schulz, 2006). Second, a number of the studies reported above have each varied in the number of participants included in the investigations (i.e., 57 in Castellanos et al., 1996; 15 in Filipek et al., 1997; 12 in Mostofsky et al., 2002; and 8 in Hesslinger et al., 2002). Third, these same studies have only investigated males, so whether the findings generalize to females who have ADHD is unknown. Fourth, each of the studies report that the participants were diagnosed with "pure" ADHD regardless of whether they were recruited from clinic-based samples or community-based samples. In reality, however, each of the studies may have included individuals with comorbid disorders, such as Conduct Disorder, and Oppositional Defiant Disorder as both disorders occur in high degrees with ADHD (Wender, 1995). Fifth, these studies also differed on the diagnostic measurements used to select participants, for example, some employed

DSM criteria and ICD-9 criteria (Filipek et al., 1997; Hesslinger et al., 2002) and others used DSM criteria and behavioral rating scales (Castellanos, et al., 1996; Mostofsky et al., 2002). Sixth, the brain imaging studies have also found reduced volumes in other brain regions, such as the cerebellum, anterior cingulate and basal ganglia, and Barkley does not address these brain regions and their importance in his model of ADHD. Finally, it must also be pointed out that there are a number of neuropsychological studies that have not found executive dysfunctions in ADHD and there are no executive functions tests that conclusively distinguish ADHD from controls (Halperin & Schulz, 2006).

In contrast, the findings from research studies that have employed psychophysiological measures, such as event-related potentials (ERP's), indicate that individuals with ADHD have atypical sensory and cognitive processes in both visual and auditory modalities (Barry, Johnstone, & Clarke, 2003). Overall, these findings implicate dysfunctional arousal and activation. In addition, it has been noted that individuals with ADHD have impairments in their ability to commit attentional resources; for example, they have preparatory and orienting deficits that partly determine what is selected for processing and what is ignored (Barry et al., 2003). Taken together, these findings suggest a possible diffuse brain stem abnormality and offer support for the theoretical models proposed by Sergeant (1996) and Halperin and Schulz (2006). Both of these theoretical models focus on arousal and activation deficits which affect the ability to receive and respond to information. As the brain stem reticular activating system is the basis for arousal and activation, it seems likely that dysfunctions to this area and areas it has reciprocal connections with (e.g., limbic system, cerebellum, hippocampus, and

prefrontal cortex) are capable of producing the behavioral and cognitive symptoms associated with ADHD. These theoretical models are not limited to explaining only one characteristic of ADHD and are capable of explaining both the behavioral and cognitive symptoms of the disorder.

Future Implications

A recent study conducted by Tamm, Menon and Reiss (2006) may have important implications for theories of ADHD. Tamm and colleagues combined the behavioral techniques typically used in ERP research with functional magnetic resonance imaging (FMRI) to study the allocation and direction of attention to salient stimuli and the ability to inhibit responding to irrelevant stimuli. They compared the behavioral and brain activation data for adolescent males with ADHD and age-matched controls. The data were collected while participants performed a visual oddball task in which they were required to respond by pressing one button to standard stimuli (green circles) and another button to oddball stimuli (green triangles). The behavioral results of the study showed that adolescents with ADHD made more commission errors than controls on the oddball task. The imaging data showed significantly less activation in parietal lobes (including the superior parietal gyrus, the supramarginal and angular gyri and the thalamus) for individuals with ADHD. These findings indicate that adolescents with ADHD demonstrate significant impairment in their ability to allocate, shift, and direct attention to select specific or salient targets. In addition, they found that the ADHD group showed fewer peaks of activation in the frontal cortex and cingulate gyrus. Further, activation of the cingulate gyrus in the ADHD group was more anterior than that observed in the control group. Even though the authors conclude that this area is error-sensitive,

explaining why individuals with ADHD tend to make more commission errors, an alternative explanation is possible.

The cingulate cortex is a part of the brain situated in the medial aspect of the cortex. It is extended from the corpus callosum below to the cingulate sulcus above, at least anteriorly. The cingulate cortex can be divided functionally and anatomically into the four following components: executive (anterior), evaluative (posterior), cognitive (dorsal), and emotional (ventral) (Bush, Luu, & Posner, 2000). The anterior cingulate cortex (ACC) is a brain structure that is located on the medial surface of the frontal lobes (Awh & Gehring, 1999) and may have an important role in executive functions. The ACC has rich interconnections with a number of cortical and subcortical brains areas that include the prefrontal cortex (orbitofrontal and dorsolateral), the parietal cortex as well as the motor systems, limbic regions, basal ganglia and the frontal eye fields (Posner & DiGirolamo, 1998; Awh & Gehring, 1999). FMRI and neurophysiological studies have shown that the ACC is involved in executive processing and higher level cognitively demanding tasks, such as divided attention, conflict resolution, response monitoring and the Stroop task (Awh & Gehring, 1999). These findings suggest that the ACC serves multiple functions.

Research studies conducted on cingulotomy patients offer an alternative explanation to the error-sensitivity hypothesis of the ACC. Cohen, Kaplan, Meadows, and Wilkinson (1994) examined the contribution of the cingulate cortex in the control of the orienting response, arousal and attentional activation by analyzing the habituation and sensitization processes of cingulotomy patients. Their results indicated that bilateral damage to the ACC disrupts habituation to the orienting response. The results also indicated that

cingulotomy patients initially habituate normally but this is followed by a random and variable pattern of resensitization to the stimuli and incomplete habituation. Incomplete habituation is defined as an inappropriate response to stimuli that are no longer novel. Patients with cingulotomies eventually habituate but it takes more trials to reach the complete habituation criteria. This finding suggests that something interfered with the habituation process.

The researchers concluded that damage to the cingulate can produce attentional impairments. They further indicate that the cingulate modulates limbic outflow and because of its modulatory influence, a disruption in the ACC impairs the temporal continuity needed to discriminate and coordinate response strength to incoming signals. This finding suggests that the ACC may be the mechanism necessary to override the automatic capture of attention and may serve as a processing point for bottom-up and top-down stimuli.

A number of researchers have also suggested that the ACC is involved in bringing internal and external stimuli together with behavioral goals and plans for action (Botvinick, Nystrom, Fissell, Carter & Cohen, 1999; Corbetta, 1998). The ACC may also be involved in processing competing inputs and inhibiting competing actions (Pardo, Pardo, Janer, & Raichle, 1990) or directing attentional responses when there are multiple or competing inputs and action (Pardo, Fox, & Raichle, 1991). It is clear that the ACC has several roles, but it's most important role may be in optimizing behavioral stability.

As the findings from brain imaging studies have indicated that children, adolescents and adults with ADHD have decreased frontal lobe and ACC volume (see Castellanos et al., 1996; Filipek et al., 1997; Pliszka et al., 2006; Seidman et al., 2006; Tamm et al.,

2006), it is possible to speculate that individuals with ADHD have an impaired ability to habituate to stimuli that are no longer novel. Further, the brain regions that the ACC has interconnections with the (i.e., prefrontal cortex, parietal cortex, motor systems, limbic region, and basal ganglia) have all been implicated in the cause of ADHD.

Summary and Future Implications

The fundamental argument among the present theoretical accounts of ADHD is whether the disorder is fully explained by top-down processes or bottom-up processes. If ADHD was caused strictly by top-down controlled processes, as Barkley suggests, then research evidence should have found specific structural brain damage in the prefrontal cortex. No studies to date have found evidence of gross brain damage in children or adults with ADHD. This suggests that ADHD may be the result of functional impairments. Diffuse brain stem abnormalities in arousal, activation, and control, proposed by Sergeant and colleagues and Halperin and Schulz, are capable of producing both functional and structural impairments in later developing brain regions (i.e., executive functions). At the same time, however, functional impairments in top-down processes are capable of influencing lower level cortical and subcortical processing due to their reciprocal connections. In either case, functional impairments would explain why individuals with ADHD have a variety of symptoms. To date, however, neither a strict view of frontal lobe impairment or brain stem deficiency is able to explain the cause(s) of ADHD. Future research is needed to investigate the basic reciprocal mechanism(s) that link top-down and bottom-up causes of the disorder instead of concentrating on uncovering a single brain region abnormality.

2. 6 Present Study Rationale

A recent study that investigated the allocation of attention to salient stimuli and response inhibition to irrelevant stimuli (Tamm et al., 2006) has hinted that issues with habituation might contribute to both the attentional and behavioral difficulties found in ADHD.

Habituation is an automatic reflexive process that has not received a lot of direct investigation in research on ADHD. Habituation is the loss of orienting response and sensitivity to a stimulus that is repeatedly presented. Habituation is a process that enables us to attend to more salient information in the environment by unconsciously tuning out the familiar sensations that constantly surround us, for example the annoying drip of a faucet as we are trying to sleep, or the feel of wool rubbing against our skin. An individual unable to habituate to familiar stimuli would be constantly experiencing the same sensations over and over as salient and new. It would be literally impossible to complete any one action as all of the stimuli in the environment would remain novel. In essence, the brain would be constantly bombarded by a flood of information and the individual would be unable to properly deploy attention to explore the more salient information in their immediate environment.

Habituation is considered to be the simplest form of non-associative learning (Lieberman, 2000) and is closely related to the concept of the orienting reflex. The orienting reflex was first recognized and reported by Sechenov in the 1850's and was systematically studied by Pavlov. Pavlov originally mentioned what we now call the orienting reflex as an example of a phenomenon that can interfere with a conditioned response. According to Pavlov the orienting response to novelty occurred whenever an organism stopped what it was doing to "turn its sensors to the source of stimulation"

(Pavlov, 1927). Pavlov found that after repeated presentations of the new event it no longer interfered with the conditioned reflex. This decline in amplitude and probability of response as a result of repeated stimulation by the once novel stimulus has generally come to be termed response habituation (Harris, 1943).

Even though for many years habituation has been considered to be strictly a low-level perceptually driven process that involves non-associational learning (Lieberman, 2000) a number of findings suggests that it may be more complicated then previously assumed. First, permanent habituation to a stimulus that is no longer novel would serve no adaptive purpose and it suggests the possibility of an alternate view of habituation that is not strictly non-associative and in this sense incorporates other types of learning (i.e., classical conditioning and instrumental learning). Thus, habituation can additionally be viewed as a process that one learns what to expect in a given context or situation. In this sense it can explain the orientation to novel stimuli in the environment and habituation to previously novel stimuli that occur in the same context or given situation because an expectancy state has been formed (Gray, 1975). This allows for associative learning to occur because an association has been formed between what is expected and what is actually in the environment. Second, this alternative view of habituation suggests that habituation is not strictly a perceptually driven reflexive process and indeed involves some level of inhibitory control as well as both short-term and long-term memory processes and may be impacted by top-down processes.

Findings from neuropsychological studies of patients with bilateral damage to the ACC or frontal lobe damage offer support for this alternative view of habituation. These studies have shown that patients with damage to the ACC (Cohen, Kaplan, Meadows, &

Wilkinson, 1994) or frontal lobe have slowed or incomplete habituation. It has also been well noted that individuals with frontal lobe damage are easily distracted, and so impaired in their ability to regulate behavior that their actions are impulsive and hyperactive (Luria, 1973; Fuster, 1997; Andrewes, 2001). The ACC and frontal lobe regions are intimately connected and also have rich reciprocal relationships with the parietal cortex, limbic regions, basal ganglia, and frontal eye fields (Posner & DiGirolamo, 1998; Awh & Gehring, 1999). The reciprocal interconnection of these different brain areas suggests that habituation may be a key process involved in modulating information between these brain regions.

Individuals with ADHD, like patients with frontal lobe damage, are easily distracted and have an impaired ability to control their behavior. In addition, research findings from brain imaging and volumetric studies of children and adults with ADHD have shown that individuals with ADHD have decreased activation and volume of the ACC (Pliszka, et al, 2006; Seidman et al., 2006; Tamm et al., 2006). Taken together, these findings suggest that the habituation processes of individuals with ADHD should be investigated.

If individuals with ADHD had incomplete or dysfunctional habituation processes this would result in the inefficient inhibition of irrelevant stimuli and it would explain the behavioral hyperactivity and impulsivity as well as the cognitive inattention and working memory deficits associated with the disorder. If individuals with ADHD were unable to inhibit processing of irrelevant stimuli they would be more easily distracted, more prone to impulsive responding and find it extremely challenging to allocate attentional resources. A deficient ability to habituate to events that are no longer relevant to current environmental demands might explain why ADHD appears to be caused by a

combination of widespread brain areas. Finally, a finding of slowed or incomplete habituation in individuals with ADHD may be capable of linking bottom-up (brain stem dysfunction) and top-down (executive dysfunction) causes of the disorder.

2. 6. 1 Theories of Habituation

There are a number of theoretical approaches that have been proposed to account for orientation and habituation. These models range from an examination of simpler reflexes (Sokolov, 1963; Groves & Thompson, 1970) to theoretical accounts of the habituation processes of humans (Wagner 1976, 1978, 1979, 1981; Öhman, 1979). As the present study is not attempting to investigate each theory's explanation of the cause of ADHD, only a brief summary will be provided here. For a more extensive review the reader is referred to Cohen, Sparling-Cohen, and O'Donnell (1993) or Stephenson and Siddle (1983).

The major purpose of the summary is to indicate how important habituation is in cognitive processing and how intimately involved it is in arousal and activation. In fact, habituation is a core automatic mechanism that controls involuntary changes in information processing through decreased levels of alertness and attention. Habituation not only reflects learning per se, but also the efficiency with which the organism can process information at the neural level.

The earliest theoretical model of habituation was proposed by Sokolov (1963). He believed that habituation is dependent on the incongruity of incoming sensory signals compared to existing neural models of the environment stored in memory. If an incoming stimulus matches stored memories then a response to the stimulus is inhibited. If there is a mismatch between the incoming stimulus and the neural model, sensitization

to the stimulus occurs and a response is generated. The neural model is developed and modified by two interacting systems: the comparator system and the amplifying system. The comparator system is believed to be the hippocampus and it determines the degree to which signals match stored memories. The amplifying system is believed to correspond with the reticular activating system. The amplifying system transmits sensory input to the comparator and also prompts the comparator system to modify neural models.

Groves and Thompson (1970) described an alternative neural model to account for habituation of the orienting response. It differs from Sokolov's comparator theory in that it proposes two competing but independent processes for habituation: habituation (inhibitory) and sensitization (excitatory). Sensitization can be thought of as the state of the organism or the organism's readiness to respond. Habituation, on the other hand, is the suppression of a response due to repeated stimulation. Sensitization and habituation in this model can be considered a reflex arc.

Finally, the models proposed by Wagner (1976, 1978, 1979, & 1981) and Öhman (1979) expand on the comparator model to account for the importance of cognitive processes, such as short-term memory and long-term memory. Both models contend that habituation occurs as a result of interactions between short-term and long-term memory. In other words, the orienting response will not be activated if a stimulus is primed (or pre-presented) in a limited capacity short-term store (Cohen et al., 1993; Stephenson & Siddle, 1983).

Summary of Theories of Habituation

The theoretical models of habituation presented above have attempted to explain the physiological mechanisms of habituation in both simple reflexes and humans. These

models suggest that habituation is a mechanism that influences attention and behavioral suppression. Habituation is modulated by arousal and activation and it is highly dependent on intact neural memories (long-term and working memory). These models of habituation have important implications for the cause of ADHD. If individuals with ADHD have deficient habituation mechanisms it would explain the findings of decreased arousal, and activation, as well as explain the inhibitory and working memory deficits.

2. 6. 2 Habituation Measures

A wide variety of measurement techniques have been used to investigate response habituation. Not all of the measures reflect the same underlying habituation processes so it is of the utmost importance to question the measures used and the extent to which they explain the underlying response habituation process. The most commonly used paradigm to study response habituation is termed the repetition-change paradigm. In this paradigm participants are exposed to a number of training trials (habituation trials), either until they no longer respond or until they no longer respond for a fixed number of trials (Siddle, Stephenson, & Spinks, 1983).

The most common measure of response habituation used in research was originally employed by Sokolov (1963). Using this technique, researchers typically measure the number of stimulus presentations that are necessary to reach a preset criterion of habituation. The preset criterion level is usually two or three consecutive presentations in which the elicited responses are smaller than the initial response to the stimulus presentation. In effect, this technique measures an attained state of habituation. This technique is arbitrary as it is defined by the particular sensory system under investigation and the sensitivity of the recording measures, which have generally been electrodermal

and vasomotor responses (Siddle, Stephenson, & Spinks, 1983). Some researchers have extended this methodology by examining the mean response magnitude over a series of habituation trials.

This technique also has a number of problems. First, it examines habituation as a treatment in trials so it provides information about change in response magnitude over time, but it does not provide information on an individual participant's rate of habituation. Second, if there are a sufficient number of trials to reach response habituation then response habituation will be very stable in all groups so the measure of habituation will be relatively insensitive. Some researchers have attempted to correct the sensitivity problem by measuring the response amplitude based on the difference between the first and last trial or between trial blocks. To use this technique a priori justification must be expounded (Siddle, Stephenson, & Spinks, 1983).

All of the above techniques are based on measures of absolute changes. Another technique is to use measures of relative response habituation. Relative measures of response habituation are derived by calculating the ratio of the level of responding after a fixed number of trials to initial response are reached. The relative scores are thus greatly affected by differences in initial responding (Siddle, Stephenson, & Spinks, 1983).

It is important to note a number of criticisms regarding the measurement techniques used to measure response habituation. First, different conclusions will be drawn about habituation depending on whether an absolute or relative measure of response habituation was used. Second, the measurement techniques have been derived without reference to any theory of habituation. As a result the measurement techniques suffer from an over-reliance on statistical procedures without construct validation of measures of response

habituation based on theory and the properties of the central nervous system, which underlie response habituation. Construct validation of response habituation must be based on precise theories of habituation so that choice of measurement and the way it is derived can be guided by sound methodological and statistical consideration (Siddle, Stephenson, & Spinks, 1983).

2. 6. 3 Frontal Lobe Involvement in Habituation: A Troxler Fading Paradigm

The most compelling support of frontal lobe involvement in habituation comes from studies using Troxler Fading paradigms (Mennemeier, Chatterjee, Watson, Wertman, Carter, & Heilman, 1994). The Troxler Fading task examines the rate at which participants habituate to stimuli in their peripheral vision. According to Mennemeier and colleagues, earlier research conducted by Troxler employing the fading task has shown that if an individual fixates on a point in central vision, stationary targets in the periphery will fade from awareness. Further, because the rate of Troxler fading differs from retinal adaptation, it is likely to be cortically rather than retinally based (see Clarke & Belcher, 1962).

Mennemeier and colleagues hypothesized that the parietal cortex was important in sustaining attention and the frontal cortex was important in mediating selective attention through habituation to peripheral stimuli. To test their hypothesis, the researchers compared the performance of patients with lesions to the parietal cortex, the frontal cortex or to both areas with normal controls. Patients with parietal lesions reported accelerated fading (in approximately 5-10 seconds as opposed to healthy participants who reported fading in 17 seconds). In addition, subjects with posterior parietal lesions also reported the fading of moving peripheral stimuli contralateral to their brain lesion. In

contrast, patients with frontal lobe lesions rarely reported Troxler fading. The authors concluded that the parietal cortex may be important in sustaining an image in the peripheral environment especially in response to movement, but the frontal cortex is necessary to habituate attention to objects in the peripheral environment when the objects are redundant and not targeted for action. They further suggest that the frontal lobes may facilitate habituation through two pathways: cortico-thalamic and cortico-cortical. In the first pathway, the lateral geniculate nucleus of the thalamus may mediate habituation through inhibition of the nucleus reticularis. In the latter pathway, the frontal lobes may directly inhibit parietal areas when stimuli are no longer novel or not targeted for action. In either case there are strong connections between the parietal cortex and the frontal lobes and both may have an impact on inhibiting the nucleus reticularis. According to Mennemeier and colleagues, the findings of their study suggest that the frontal cortex facilitates inhibition of the nucleus reticularis and the parietal cortex may reduce inhibition of the nucleus reticularis leading to an inability to sustain attention.

In a more recent investigation, Jansiewicz, Newschaffer, Denckla and Mostofsy (2004) examined whether children diagnosed with ADHD showed an impaired ability to habituate to visual peripheral stimuli using the Troxler Fading paradigm. The researchers found that individuals who have ADHD showed slower fading to peripheral stimuli than did non-ADHD controls. In addition, children diagnosed with ADHD reported fading on a significantly smaller percentage of trials than did control subjects. The authors concluded that the findings of prolonged visual habituation in children who have ADHD may be related to clinical aspects of the syndrome, such as distractibility, impulsivity, and difficulty staying on task. It is important to note that a concern in studying sustained

attention in ADHD is the possibility that participants end a trial early because of task frustration or impulsivity. However, Jansiewicz and colleagues found no evidence to suggest that ADHD participants attempted to rush through the testing conditions because of frustration. This conclusion was inferred from the very slow report of fading by individuals with ADHD.

It is important to note that this finding does not support the conclusion that slowed fading may be related to hyperactive and impulsive responding. If the task was measuring the clinical symptoms of hyperactivity and impulsivity a more variable response pattern should have been reflected. Based on the lack of finding a hyperactive or impulsive pattern of responding an alternative conclusion is possible. It is proposed that slowed habituation reflects the pervasive attentional impairment commonly associated with ADHD.

2. 7 Aims of the Present Study

Although there has been a tremendous amount of research on childhood ADHD, and more recently some on adult ADHD, there are still many unanswered questions. The most important unanswered question is "What causes ADHD?" Barkley (1998) proposes a theory that ADHD is caused by top-down executive control difficulties. Sergeant and colleagues (2003) argue that dysfunctional bottom-up processes (i.e., arousal, activation, and effort) are capable of producing the top-down executive control deficits found in ADHD. Alternatively, Halperin and Schulz (2006) argue that ADHD results from early neural deficits and later developing top-down control may compensate for early impairments. In order to make progress in understanding the cause(s) of ADHD, research must be conducted to assess the specific mechanisms that may be disrupted from

impairments in neural pathways that influence functioning in brain stem mechanisms and executive control.

The review of the literature has indicated quite clearly that ADHD is a neurobiological disorder. Research findings from ERP studies have indicated that individuals with ADHD have global deficits in cortical arousal, and difficulty inhibiting sensory input to irrelevant stimuli, suggesting a brain stem abnormality. In addition, ERP studies have also found that individuals with ADHD have reduced frontal inhibitory control (Barry, Johnstone, & Clarke, 2003). Imaging studies have shown that the frontal lobe volumes of individuals diagnosed with ADHD are smaller than controls. This finding is consistent across studies of children and adults (Castellanos et al., 1996; Filipek et al., 1997; Hesslinger et al., 2002, Mostofsky et al., 2002). These findings taken as a whole suggest that ADHD is caused by a combination of top-down executive dysfunctions and bottom-up diffuse brain stem deficits.

It is possible that none of the theoretical models to date are able to explain all the symptoms of ADHD because they attribute cause to specific brain areas rather than considering processes that may simultaneous activate both bottom-up and top-down brain areas. A key problem in ADHD research is the inconsistent conceptualization of inhibitory processes in the literature (Nigg, 2001). Some researchers investigate controlled effortful processes (i.e., deliberate suppression to achieve an internally guided goal), while others investigate automatic processes (i.e., uncertainty in the presence of a novel stimulus). Although these processes may be partially distinct, they rely on the same underlying neural systems (i.e., basal ganglia, prefrontal cortex, and associated

cortical and subcortical structures). As such, in any real world behavior, the relationship between these systems is reciprocal (Nigg, 2005).

Habituation is considered to be a low-level automatic process, but its potential to explain attentional impairments has been overlooked. The ability to habituate to irrelevant stimuli in the environment is a necessary determinant of selective attention (Cohen et al., 1993). Even though habituation is an unconscious process it involves learned associations and automatic skill generation that only become efficient through the formation of short and long-term memory traces. In other words, habituation is a learning process that involves associating past experiences and expectations with current situations.

Habituation is not only a gating mechanism that is extremely critical in attentional processes but it also dampens arousal (e.g., reticular activating system) and motor behavior (Sokolov, 1963; Grove & Thompson, 1970; Stephenson & Siddle, 1983). Thus habituation is a process that also involves inhibitory control. Stated differently, the same mechanisms of habituation would be recruited to sustain willed attention to an activity (e.g., habituating to environmental distractors in order to read a book) and to automatically capture attention if the demands of the environment change (e.g., sound of a fire alarm that pulls our attention from reading).

As previous research findings have also indicated that habituation involves the frontal lobes (i.e., executive control) (Luria, 1973; Fuster, 1997; Mennemeier et al, 1994; Jansiewicz et al., 2004) and the ACC (Cohen et al., 1994); it may be capable of explaining both the bottom-up (i.e., arousal, activation) and top-down (i.e., behavioral disinhibition, poor working memory) deficits attributed to ADHD. The inability to

habituate to or filter irrelevant signals can disrupt both bottom-up automatic and top-down control processes and could cause both the cognitive inattention and behavioral hyperactivity-impulsivity commonly seen in the disorder.

The present study investigates habituation processes in adults with ADHD. Adults were chosen because their executive control processes are more fully developed compared to children with ADHD. The structure and function of the prefrontal cortex changes significantly across the lifespan; by age 10 the ability to inhibit attention to irrelevant stimuli is nearly complete with full mastery by age 12 (Passler, Isaac, & Hynd, 1985). In addition to possibly controlling for developmental factors, adults with ADHD may be a better population to study automatic selective attention deficits as research suggests inattention is the most prominent symptom of adult ADHD as opposed to the excess motor hyperactivity and impulsivity more commonly found in ADHD children (Barkley, 1998; Wender, 2000).

To this end, the first aim of this dissertation is to replicate the findings of slower visual habituation in children diagnosed with ADHD in a sample of adults with ADHD using the Troxler Fading Paradigm. Further, if prolonged visual fading found in childhood ADHD suggests a generalized impairment in habituation processes, then more evidence is needed to provide insight into the habituation processes of other modalities. The second aim of this dissertation is to extend the examination of habituation processes into the auditory modality using a modified Troxler Fading Paradigm created for this project. It is hypothesized that adults who are diagnosed with ADHD will show slower habituation to visual and auditory signals than adult controls.

If a generalized impairment in habituation processes is found for adults with ADHD it will have a significant impact on the potential cause of ADHD and important implications for theories of ADHD. First, it would suggest that impaired habituation may be the most clinically significant impairment in ADHD across the lifespan. Second, it would suggest that at least some ADHD may be caused by widespread brain involvement that combines both brain stem and executive control deficits. On the other hand, if adults do not exhibit the same prolonged habituation found in children it will suggest that ADHD symptoms may be alleviated by mature executive functions that compensate for lower level brain stem deficits (Halperin & Schulz, 2006).

The third aim of this dissertation is to assess both self-report and empirical measures of habituation. As sensory gating is believed to be an index of habituation (Waters, McDonald, and Koresko, 1977) the Sensory Gating Inventory (SGI; Hetrick and Smith (unpublished manuscript) will be administered to determine if adults with ADHD report higher occurrences of sensory gating disturbances. It is hypothesized that adults with ADHD will report greater impairment on the overall SGI and all four subscales.

Chapter III

Method

Participants: Forty-one participants between the ages of 18-40 met selection criteria for the study (30 females and 11 males). Demographic characteristics of the ADHD and control group appear in Table 2. Of the 41 participants, 21 were diagnosed ADHD (12 females and 9 males) and 20 were controls (18 females and 2 males). Independent t-tests revealed no significant difference between the two groups in age ($t(40) = 1.32$, $p = .19$) or Full Scale IQ ($t(40) = .863$, $p = .86$). The reported ethnicity of the participants is as follows: 31 Caucasian, 5 Hispanic, 4 African American, and 1 Asian. Although not reported on the history questionnaire, participants in both groups met criteria for a number of comorbid disorders on the Adult Semi-Structured Interview. The comorbid disorders are also listed in Table 2.

Table 2

Demographic characteristics of the sample

		ADHD* (N=21)	Control (N=20)	Combined (N=41)
Gender		12 Females 9 Males	18 Females 2 Males	30 Females 11 Males
Ethnicity				
Caucasian		15	16	31
Hispanic		5	0	5
African American		1	3	4
Asian		0	1	1
Ages in Years				
	Mean	23.95	27.55	25.71
	SD	6.10	7.45	6.96
	Range	18-39	18-40	18-40
WASI FSIQ				
	Mean	107.42	102.10	104.83
	SD	13.81	14.37	14.16
	Range	89-128	81-129	81-129
Comorbid Diagnosis**				
Oppositional Defiant		10	0	10
Conduct Disorder		2	0	2
Antisocial Personality Disorder		1	0	1
Major Depressive Disorder		5	1	6
Generalized Anxiety Disorder		3	4	7
Social Anxiety		2	2	4
Substance Abuse		2	0	2

* Gender broken down by ADHD subtype showed 8 female Inattentive type; 1 male Inattentive type; 4 Female Combined type; 8 male Combined type; and no Hyperactive/Impulsive Type. ** It is important to note that none of the participants reported being diagnosed with any of these conditions on the History Questionnaire or during the Adult Semi-Structured Interview.

A total of 57 individuals were recruited for the project. Inclusionary criteria were as follows: (a) a Full Scale IQ score of 80 or above on the Wechsler Abbreviated Scale of Intelligence (WASI; The Psychological Corporation, 1999), (b) normal or corrected to normal vision, and (c) normal hearing. Exclusionary criteria included the following: psychosis, neurological disease, chronic physical illness, taking prescription systemic medications (including psychoactive medications), and diagnosis of schizophrenia. Recruits were also excluded if they could not be calibrated on the eye tracking device and if they made errors of habituation or anticipation on the auditory method-of-limits task. Adults taking prescription medication for ADHD were excluded from the study if they took medication on the day of testing. If the ADHD participants were currently taking prescription medication to relieve their symptoms they were asked not to take medication on the day of testing. Adults in the ADHD group met research criteria for ADHD if they were previously diagnosed by a psychologist/physician with ADHD. In addition, the presence of ADHD symptoms was verified from a retrospective self-report measure and a current functioning self-report measure. Sixteen of the 57 recruits were excluded from the study for failing to meet research criteria. The reader is referred to Table 3 for a list of these criteria. The exclusionary criteria for specific procedures (eye tracking and method-of-limits) will be discussed in more detail in the materials and procedures sections.

Table 3

List of the exclusionary criteria and frequency of failing for the 16 participants not included in the sample

Exclusionary Criteria	Frequency of Failing
Medical Conditions	5
Epileptic	1*
ADHD with comorbid Bipolar Disorder	3*
ADHD with comorbid Major Depression	1*
Taking Prescription Systemic Medication	5
ADHD	5*
Controls	0
Eye tracking Calibration Errors	5
ADHD	1*
ADHD with Bipolar	1*~
Controls	3^
Auditory method of limits	4
ADHD	1*
ADHD with Bipolar Disorder	2*
Control	1
ADHD Rating Scales	3
Control participants with elevated scores on the WURS and/or CAARS	3
Scheduling Difficulties	3
ADHD	2
Controls	1

Note: * indicates that participants were excluded on multiple criteria
^It must be noted that controls excluded for eye tracking calibration errors wore colored contacts lens.
~indicates errors of anticipation on the auditory method of limits task in addition to errors of habituation.

Participants were recruited through newspaper announcements in the New York–New Jersey Metropolitan area, community referrals, bulletin board announcements on Kean University campus, and students enrolled in Introductory Psychology courses at Kean University (copies of recruitment announcements are included in Appendix A). All participants in the study received $20 remuneration to partially compensate them for their time. All of the ethical guidelines of the APA were strictly adhered to in conducting this research study.

Materials/Measures:

Demographic and Developmental History Questionnaire: Participants were asked to complete an in-depth history questionnaire to gather demographic and developmental information (see copy included in Appendix B). The demographic section asked questions regarding ethnicity, family composition, and socioeconomic status. The developmental history section asked questions to capture the participant's behavioral style during his/her childhood years. In addition, the developmental history section asked questions regarding chronic medical conditions, substance use, and behavioral or learning problems that are often comorbid with ADHD or misdiagnosed as ADHD. The questionnaire also included questions regarding current functioning (i.e., in school, on the job, and social relationships). The History Questionnaire was used to validate diagnosis, make inclusionary decisions (normal or corrected to normal vision, and normal hearing), and exclusionary decisions (psychosis, neurological disease, chronic physical illness, taking prescription systemic medications, taking psychoactive medications, and diagnosis of schizophrenia).

Adult Semi-Structured Interview: A short semi-structured interview was administered to each participant using a modified version of Barkley and Murphy's Adult Interview (Barkley, R. A. & Murphy, K. R., 1998). The semi-structured interview asked questions to further establish degree of ADHD symptoms and to probe for behavioral, conduct, anxiety and mood disorders that might better address the attentional difficulties. As ADHD is often joined with a wide range of comorbid disorders, it is necessary to assess whether the concentration difficulties, hyperactivity, and impulsivity are primary symptoms of ADHD or are secondary symptoms of another medical or psychiatric condition. In adulthood, ADHD is often comorbid with substance abuse, Antisocial Personality Disorder, anxiety and mood disorders, or may be related to medical conditions, such as diabetes, high blood pressure, or hypo- or hyperthyroidism. The interview was used to further validate degree of ADHD symptoms and aid in making exclusionary decisions. Participants' answers to the interview questions were written by the test administrator on the interview form, and participants were informed that they did not have to answer any questions that made them feel uncomfortable.

ADHD Screening Measures: The Wender Utah Rating Scale (WURS) was developed by Ward, Wender, and Reimherr (1993) and is useful as a retrospective assessment of an individual's childhood self-report of ADHD symptoms. In order to make a diagnosis of adult ADHD the symptoms of ADHD must present in childhood. The WURS short version was used for the present study. The WURS short version consists of 25 behavioral descriptions of attentional deficit symptoms, such as "Anxious, worrying", "Concentration problems, easily distracted" and the individuals are asked to respond to the statements by indicating to what degree these behavioral symptoms affected them as

children. The following five choices are possible: Not at All, Mildly, Moderately, Quite a Bit, and Very Much (see Appendix C for a copy of the WURS). Participants were asked to fill out a WURS self-report assessment and to have a parent or someone who knew them well (i.e., sibling or significant other) complete a WURS Observer questionnaire before their scheduled appointment to further aid diagnosis.

The Conner's Adult ADHD Rating Scale – Long Version (CAARS-LF; Conners, Erhardt, & Sparrow, 1999) is a self-report measure that assesses current ADHD symptomatology across the following important clinical domains: home, work, school, and interpersonal relations. The CAARS-LF self report (CAARS-S:L) and observer (CAAR-O:L) versions were collected for each participant. There are three DSM-IV ADHD symptom measures on the CAARS that assess ADHD symptoms according to the criteria listed in the DSM-IV (APA, 1994) as follows: a 9-item Inattentive Symptom subscale, a 9-item Hyperactivity-Impulsivity Symptom subscale, and an 18-item Total ADHD symptoms subscale (combination of the 9-item Inattentive Symptom and Hyperactivity/Impulsivity subscales). In addition, the CAARS-LF contains a 12-item ADHD Index. The ADHD Index contains the best set of items for distinguishing ADHD adults from nonclinical adults. The long forms also contain an Inconsistency Index, which is useful in determining random or careless responding (for a more detailed review of the CAARS-LF the reader is referred to Conners, Erhardt, & Sparrow, 1999).

In order to be included in the study adults diagnosed with ADHD had to meet one of the following criteria assessed from self-report or observer report: (a) a score of 36 or higher on the WURS (b) a T-score of 61 or higher on the CAARS total DSM-IV ADHD Symptom subscale or (c) a T-score of 61 or higher on the CAARS ADHD Index. Adults

were included in the control group if they met all of the following criteria assessed from self-report and observer report: (a) a score of 35 or fewer on the WURS, and (b) a T-score of 55 or below on the CAARS DSM-IV ADHD Symptom subscale, or a T-score of 55 or below on the CAARS ADHD Index. The WURS cutoff score for inclusion in the ADHD or control group was based on the findings of Ward, Wender and Reimherr (1993). The researchers indicated that when the cutoff score was set at 36 or higher they were able to correctly identify 96% of normal subjects, and 96% of the ADHD subjects.

Intelligence Quotient: The Wechsler Abbreviated Scale of Intelligence (WASI, The Psychological Corporation, 1999) was administered to establish IQ. The WASI can be used with a broad age range (6-89). The WASI consists of the following four subtests: Vocabulary, Similarities, Block Design, and Matrix Reasoning. The WASI is nationally standardized and yields the three traditional Verbal, Performance, and Full Scale IQ scores.

Sensory Gating Inventory: The Sensory Gating Inventory (SGI: Hetrick & Smith, unpublished manuscript) is a self-report questionnaire consisting of 36 questions that measure sensory inundation, the ability to filter stimuli and inhibit processing of irrelevant stimuli. The following four factors are measured: Perceptual Modulation, Distractibility, Overinclusion, and Fatigue/Stress Vulnerability (A copy of the SGI can be found in Appendix D).

Experimental Measures:

Visual Fading Task (Troxler Fading): The computer program for the visual task was created using Presentation software developed by Neurobehavioral Systems. In the visual task, participants were instructed to maintain focus on a central fixation point (a 9

mm yellow crosshair) on a medium gray background screen throughout the experiment. A black dot (a 2 dimensional circle 5mm in size) appeared in one of four positions in their peripheral vision (45, 135, 225, and 315, degrees); participants were told not to shift their attention to the black dot in their peripheral vision. The participants were asked to report when the black dot in their peripheral vision faded from awareness by pressing a button (left click) on a computer mouse held in their dominant hand. The circle was presented in a stationary, moving or validity condition. In the stationary condition, the black dot appeared near the edge of the visual field and did not move. The stationary black dot stayed on the screen for 30 seconds or until the subject reported fading by pressing the mouse button. Participants were expected to report visual habituation or fading in response to the stationary black dots. In the moving condition, the black dot appeared near the edge of the visual field and moved in an orbit at a rate of 3.16 cycles per second for 30 seconds or until the participants reported fading by pressing the mouse button. In the moving condition participants were not expected to report visual habituation or fading; if they did report fading it was expected to be slower than fading of the stationary dots. In the validity condition, the black dot appeared at the edge of the visual field and was blended into the gray background color within 5 seconds after the start of the trial. In the validity condition participants were expected to report visual habituation or fading in response to the black dots only after the 5-second mark.

The visual task consisted of all possible combinations of the 12 conditions of the visual experiment (stationary–four peripheral locations; moving-four peripheral locations; validity-four peripheral locations). The stimuli were presented in each of the four possible quadrants one at a time and there were 1500 milliseconds between stimulus

presentations. The visual task was administered in a completely randomized and counterbalanced order across participants. The visual task consisted of 2 blocks of 24 stimuli with a 5-minute break between block presentations. All participants performed a short visual practice trial which consisted of three moving, three stationary and three validity stimuli.

Visual stimuli were presented on a 19 inch flat panel LCD monitor measuring with stand 17.8 x 13.1 x 7.9 inches. The screen resolution was 1280 x 800 32 bit. The monitor was level with tilt set to be perpendicular with the floor. The monitor was placed on a table and the distance from the top of the monitor to the floor was 48 inches. Participants were seated in a stationary chair (without wheels) at a distance of 24 inches from the monitor to assure that the visual angle was the same across participants (1° of visual angle from seated position to 9mm center crosshair and 16º of visual angle from seated position with eyes fixated on crosshair to 5mm dot in peripheral vision). Room and computer monitor luminance was standard across all participants. The visual task was controlled by the experimenter from a laptop connected to the monitor.

A Quick Glance II-SH Eye Tracker by Eye Tech Digital Systems was used to monitor participant's eye movements during the visual task. Quick Glance II-SH is a high resolution video camera with strobe, and a dark pupil eye tracker that uses infrared illumination to track eye movements. The video camera was mounted on the bottom center panel of the monitor and the infrared illuminators were attached on the left and right side of the monitor 5cm from the top of the monitor. The 25mm standard lens was replaced with a 16mm lens to compensate for the participants' seating distance from the monitor. In addition, the 16 mm lens allowed capture of binocular eye movements. The

Quick Glance II-SH Eye Tracker has excellent lighting and motion tolerance which allows for head movement up to 10 x 10 cm. In addition, the Quick Glance II-SH Eye Tracker captures up to 30 samples per second, and has a gaze point accuracy of within ±.5 degrees of actual gaze point.

Even though the eye tracker allows for greater head movement, participants were asked to place their chin on a chin rest to minimize head movement. The chin rest was made expressly for the purpose of the experiment and was based on a design created by Matt Peterson. (http://vision.nyu.edu/Tips/ChinRests.html). The design is created by attaching three pieces of wood to form a U-shape and secured to a table with an L-bracket and a C-clamp. A belt is attached to the inside of the U-shape by hook and eyes. The belt can be adjusted for head size, comfort, and maintenance of eye gaze at level with the center of the computer monitor to assure consistent visual angle across all participants. The last is accomplished by creating several additional notches in the belt. In the present experiment the belt was further cushioned by covering the area the participants would be asked to rest their chin on with a layer of soft foam and a fabric sleeve. The eye tracking device was interfaced with Presentation software using Eye Science by Eye Tech Digital systems. The Eye Science control panel controls the following features: adjustment of eye capture to a maximum of 30 samples per second; starting and stopping eye gaze capture; playing back eye gaze data in real-time to illustrate the user's gaze path; saving eye gaze data to a text-file; and reading of eye gaze data from a text file. The researcher was able to monitor a participant's eye movements in real-time on the laptop connected to the monitor while the participants were completing the visual task. If the participants broke visual fixation from the center crosshair at any time during the stationary condition

trials, those trials were deleted and replaced by other trials of the same type later in the testing session. The range of rejection for determining a broken fixation was set at eye movements of more than 2º of visual angle in any of the four peripheral quadrants of the display where the black dots appeared (for more information on determining eye fixation the reader is referred to Duchowski, 2003). If participants moved their gaze slightly on any trial when the dot first appeared and immediately reoriented their gaze to the central crosshair the trial was not deleted. Blinking was also permitted. A record of the number of replaced trials was kept for each participant. Overall, participants in the ADHD group broke fixation from the center crosshair on 25% of the stationary trials compared to 11% for controls.

Each participant's eye gaze was calibrated using an Eye Science calibration screen. The Eye Science software displays 16 disk shaped targets on the screen. The targets are displayed one at a time in succession across the entire screen display. Participants were asked to look directly at the center of each target when it appeared. The Eye Science window provides a visualization of the calibration mapping to tell quickly and easily if there is a good calibration. If the calibration points are not good for any specific points on the screen they can easily be repeated to improve calibration. Each participant's calibration setting was saved to be used in subsequent trials.

In addition to the Eye Science calibration and before beginning the visual experiment the researcher calibrated the visual task to determine the extent of each participant's visual fields in all four quadrant locations. During calibration of the visual task a black dot appeared in the center of the monitor screen over the crosshair and it was moved to the edge of the screen in each of the four locations the dots would be appearing in during

the visual trials one at a time. Participants were asked to report if they could see the dot using their "side vision", while the researcher made sure they were maintaining fixation on the center crosshair. If the participants could see the dot the calibration was accepted and a visual test trial was conducted. If the participants reported that they could not see the dot it was gradually moved closer to the crosshair and assessed again until the participants were able to see the dot clearly in all four locations. Once the participants reported seeing the dots in peripheral vision, the dots were once again moved to the location they would appear in during the experimental trials. If the participants still reported that they did not see the dots their field of vision was modified to an acceptable calibration and they were given the experimental trials. Any participant whose visual field was modified from the standard stated above (i.e., 1° of visual angle from seated position to 9mm center crosshair and 16º of visual angle from seated position with eyes fixated on crosshair to 5mm dot in peripheral vision) was excluded from data analysis.

Auditory Fading Task: The auditory task was comparable in design to the visual task and was also created using Presentation Software by Neurobehavioral Systems. The participants were asked to maintain focus on a central fixation sound that was presented continuously through a speaker positioned at a central location in front of where the participants were seated throughout the experiment. The central fixation sound consisted of 70 db high frequency white noise that was filtered to remove the low frequencies using a high pass bandwidth. A simple tone (30 db 1000 Hz sine tone either 8 or 30 seconds in duration) was presented through speakers situated at four different locations (front, back, left, or right of seated position). Both the white noise and simple tones had a 50 millisecond rise and fall time. Participants were asked to report when the simple tones

faded from their awareness by pressing a button on a computer mouse held in their dominant hand. The stimuli were presented in a stationary, moving, or validity condition. In the stationary condition, sounds of constant intensity and frequency were presented in one of the four speaker locations and remained in that location for 30-seconds. Participants were expected to report auditory habituation or fading in response to the stationary simple tones. In the moving condition, tones of constant intensity and modulated frequency were presented for 30 seconds from one of the four speaker locations. In this condition the 1000 Hz sine tone frequency was modulated at a 60% depth and a 5 second phase to simulate the 5mm black dot moving in an orbit at 3.16 cycles. Participants were not expected to report auditory habituation or fading in response to the moving simple tones. In the validity condition, simple tones were presented from one of the four speaker locations and were blended into the background sound within 8 seconds after the start of the trial. The validity tones in the auditory condition were of a longer duration than in the visual condition as research indicates that it takes longer for auditory stimuli to habituate (Thompson, 1993). All participants were expected to report auditory habituation or fading in response to the simple tones after the 8-second mark.

The auditory test was administered using the same procedures as the visual test. The auditory task consisted of all possible combinations of the 12 conditions of the auditory experiment (stationary–four speaker locations; moving-four speaker locations; validity-four speaker locations), randomized and counterbalanced across participants. The stimuli were presented in each of the four possible speaker locations one at a time with 1500 milliseconds between stimulus presentations. The auditory task consisted of 2 blocks of

24 stimuli with a short 5-minute break between block presentations. All participants performed a short auditory practice trial which consisted of three moving, three stationary and three validity stimuli.

The sine-wave stimuli for the stationary, moving, and validity condition and the white noise fixation sound were generated using LabVIEW Full Development System by National Instruments. The auditory task was delivered using a Sony notebook computer with a Creative Labs Audigy 2ZS PCMCIA sound card for notebooks. Audigy 2ZS maintains a high signal-to-noise ratio exceeding 104db using high linearity, low distortion 24-bit converters with resolution up to 192 kHz. The digital audio converter filters at a 44.1 kHz sampling rate. The sound card was attached to Creative Labs Inspire P7800 7.1 surround sound speakers. The main speaker that played the fixation sound was placed in a centrally located position in front of the participants. The four speakers that were used to present the tones were positioned in front, back, left and right of the participants seated position. To maintain consistency with the visual condition the four speakers were placed on wall shelves that were 48 inches from the floor.

Each participant's absolute threshold (i.e., the intensity that they could just barely detect sound) was measured for the auditory stimuli used in the study both before and after the experiment. Descending and ascending method-of-limits procedures were used to test threshold judgments to the stationary and moving stimuli in quiet and in noise (i.e., the presence of the constant white noise fixation sound). Threshold judgments were not measured for the validity stimuli as they were identical to the stationary stimuli except for presentation time. In the quiet condition, the 1000 Hz stationary and moving tones were played consecutively one at a time from 70 to 0 decibel (descending order) and from 0 to

70 decibels (ascending order) in 5 decibel increments through Sennheiser noise cancellation headphones connected to the headphone output on the Audigy soundcard. Participants were asked to raise their hand to indicate "Yes" if they heard a tone and the researcher marked the participant's responses on a graph. In the noise condition, participant's threshold judgments were examined using the same procedures with the inclusion of the white noise constant fixation sound both before and after the experimental auditory task. The white noise fixation sound and the stationary and moving stimuli were mixed together using the Audacity editing program. The stationary and moving stimuli were mixed with the white noise and were presented at random intervals (i.e., every 30 to 90 seconds) to prevent the participant's expectation of tone presentation.

The method-of-limits procedure was employed for various reasons. First, measuring participant's absolute threshold judgments to the same stimuli used in the auditory experiment was analogous to the calibration procedure used to track participant's eye movements in the visual portion of the study. In addition, the method-of-limits procedure is particularly sensitive to two biasing factors. The first biasing factor is called the error of habituation. If an error of habituation is present the participant may fall into the "habit" of continuing to respond "Yes" they can hear the stimuli in an ascending series and "No" in a descending series. This type of responding would lead to a shift in the individual's threshold judgment (i.e., higher threshold in the "Yes" condition and a lower threshold in the "No" condition). Participants were excluded from the study if they made habituation errors on the method-of-limits test. The second type of biasing error is called the error of anticipation. If this type of error is present, the participant would tend to

respond prematurely simply because they expect a change to occur. (It is important to note that one of the excluded ADHD/Bipolar disorder participants made anticipation errors. In addition, during the actual auditory experiment she refused to remain seated and walked around the room trying to determine which speaker the sound would be coming from next. She stated that she knew she should remain seated but it was more interesting to see if she could figure out where the sound would come next.) Finally, using the threshold procedure before and after allowed the investigator to examine if there was a shift in the participant's threshold judgments caused by exposure to the auditory stimuli. If individuals with ADHD shifted threshold it would have suggested a lower level perceptual processing deviance. Neither control nor ADHD participants shifted threshold judgment after exposure to the auditory task.

The reader is referred to Appendix E for a review of the pilot studies for both the visual and auditory Troxler Fading Task.

Procedures: Upon scheduling an appointment all participants were asked to have a parent or someone who knew them well (i.e., sibling or significant other) complete a copy of the WURS and CAARS before their scheduled appointment. If the participant was not able to have a parent or someone who knew them well complete the forms they were told that they would not be able to participate in the study. A letter indicating that the individual was participating in a research project was mailed to each participant along with an observer copy of both the WURS and CAARS. The letter explained that completion of the forms was required for participation in the study and that the individual would not be allowed to participate if the forms were not completed. The letter also indicated that it would take a relatively small time commitment (approximately 10-15

minutes) to complete both of the forms. Participants were asked to give the letter and forms to a parent or someone who knew them well to complete and to return the forms on the day of their appointment.

On the day of their appointment, after participants completed a consent form (see Appendix F for a copy of the consent form), they were asked to complete the following forms: CAARS-S: L, WURS and Developmental History Questionnaire. After completing these forms the Adult Semi-Structured Interview was administered to each participant.

Upon completion of the Adult Interview, the battery of tests (WASI, Visual and Auditory Experimental measures) was administered to each participant in a small, separate testing room. The test battery was administered in the following order: WASI Vocabulary and Block Design, Visual or Auditory Experimental Test (depending on counterbalanced order), WASI Similarities and Matrix Reasoning, and Visual or Auditory Experimental Test (depending on counterbalanced order). After the test battery was completed participants were asked to complete the SGI.

Data Analysis: Means and standard deviations were calculated for all continuous variables. First, a 2 (Modality) x 2 (Group) repeated measures ANOVA was calculated using mean report of fading time to validity stimuli to confirm that the participants were engaging in the task as expected. A 2 (Modality) x 2 (Stimulus Type) x 2 (Group) repeated measures ANOVA was then calculated to examine mean report of fading to stationary and moving stimuli. A total of seven participants did not respond to all stationary and moving stimuli presented in the visual or auditory modality. Data were missing as follows: 2 visual stationary trials, 12 visual moving trials, and 3 auditory

stationary trials. Group means for the appropriate stimuli replaced missing data for the omnibus statistical tests. To assess whether there were differences in performance variability between the ADHD and control groups a 2 (Modality) x 2 (Stimulus Type) x 2 (Group) repeated measures ANOVA of the mean standard deviations across trials was calculated. The data from the visual stationary fading task were analyzed to investigate whether there were fading differences between the groups based on visual hemifield. As stimuli were presented in two locations on the right side of the screen and two locations on the left side of the screen a 2 (Right or Left Dot Locations) x 2 (Group) repeated measures ANOVA was calculated. The data from the auditory stationary fading task were also analyzed to investigate whether there were fading differences based on speaker location. As four speaker locations were used in the auditory task a 4 (Right, Left, Front, and Back Location of Sound) x 2 (Group) repeated measures ANOVA was calculated. An ANOVA was performed to examine whether the overall SGI score differed by group. In addition, multivariate tests were calculated for each of the four subscales of the SGI to further investigate group differences. Finally, the overall SGI and four subscale scores were correlated with mean fading time on the stationary and moving stimuli in both modalities across groups to examine if there were relationships between sensory gating reports, and fading time.

Chapter IV

Results

Preliminary Analysis:

ADHD and Control Group Comparisons on the ADHD Rating Scales

Group means and standard deviations for self- and observer reports of attentional and behavioral disturbances are presented in Tables 4 and 5 respectively. As expected, both self- and observer reports of attentional and behavioral symptoms were significantly greater for the ADHD sample. For the ADHD group, the mean scores on all of the CAARS subscales were at the 97th percentile relative to general population norms, indicating that they were on the high end of experiencing attentional and behavioral difficulties. Not only were the means at the high end but the range of their scores was from above average to very elevated. The ADHD group's mean score on the WURS self-reports was well above the diagnostic cut-off of 36, and the group mean was similar to the sample mean from the field study (62.2 ± 14.6; see Ward et al., 1993). The group mean on the WURS observer reports was also above the 36 point cut-off score; however, as the original study did not collect data on the observer version of the WURS no direct comparison can be made. In comparison to the sample mean for self-reports; the group mean on the observer reports was slightly lower. In fact, observer's ratings for 4 of the 21 participants diagnosed with ADHD did not reach the cut-off score of 36 needed for diagnosis on the WURS. The fact that some parental observer ratings were low may explain why eight of the individuals in the present study were not identified with ADHD in childhood. For the control group, the mean scores fell within normal limits across all the ADHD rating scales, ranging from much below average to average.

Table 4

ADHD and Control Group Comparisons for Self-reported ADHD Symptoms

	ADHD (n=21)		Controls (n=20)		t (39)
Variable	M	Range	M	Range	
WURS	48.47 (11.73)	24-69	17.35 (10.69)	2-35	8.87*
CAARS-S:L:					
DSM-IV ADHD Symptom Subscale	71.38 (8.45)	60-88	45.15 (7.65)	31-55	10.39*
ADHD Total Index	66.14 (7.10)	50-85	45.50 (7.63)	33-55	8.97*
DSM-IV Inattentive Subscale	71.29 (9.64)	56-89	44.60(7.50)	29-55	9.85*
DSM-IV Hyperactive Subscale	64.90 (7.89)	54-79	45.95 (7.35)	31-61	7.94*

Note: WURS is the Wender Utah Rating Scale.
CAARS-S:L is the Conner's Adult Rating Scale Self-Report Long Form Version.
*p = .001 (2-tailed).

Table 5

ADHD and Control Group Comparisons for Observer Report of ADHD Symptoms

	ADHD (n=21)		Controls (n=20)		t (39)
Variable	M	Range	M	Range	
WURS	42.71 (12.85)	9-62	15.15 (11.83)	2-34	7.14*
CAARS-O:L					
DSM-IV ADHD Symptom Subscale	65.38 (6.15)	53-75	45.00 (6.79)	36-55	10.08*
ADHD Index	62.66 (6.11)	49-73	46.05 (6.50)	36-55	8.44*
DSM-IV Inattentive Subscale	62.09 (7.14)	50-72	45.03 (6.18)	35-55	8.03*
DSM-IV Hyperactive Subscale	63.76 (5.99)	52-75	45.35 (5.88)	36-55	9.92*

Note: WURS is the Wender Utah Rating Scale
CAARS-O:L is the Conner's Adult Rating Scale Observer Report Long Form Version.
*p = .001 (2-tailed).

Primary Hypothesis Testing

Group Comparisons on the Visual and Auditory Troxler Tasks

It was predicted that adults with ADHD would be slower to report fading to visual and auditory stationary stimuli than controls. Before testing these hypotheses, a 2 (Modality) x 2 (Group) repeated measures ANOVA for mean report of fading time to validity stimuli was calculated to examine if participants were attentive to the task. Group means and standard deviation are presented in Table 6. No significant group differences were found for report of fading to validity stimuli. This finding suggests that the task was engaging participants' attention.

A 2 (Modality) x 2 (Stimulus Type) x 2 (Group) repeated measures ANOVA was applied to analyze mean report of fading time to stationary and moving stimuli. Group means and standard deviations are also presented in Table 6. A significant Modality x Stimulus Type x Group interaction was found ($F(2, 39) = 17.28$, $p = .01$). As expected, post hoc independent t-tests revealed that the ADHD group was slower overall to report fading to visual stationary stimuli ($t(39) = 1.93$, $p = .03$). This is consistent with past findings of slower report of fading to visual stationary stimuli in children. In addition, adults with ADHD were slower than controls to report fading to auditory stationary stimuli ($t(39) = 2.36$, $p = .02$). The findings confirmed our expectation that ADHD adults would be slower to report fading to visual and auditory stimuli, and extended past report by indicating that slowed habituation processes are not specific to the visual modality, but may be indicative of more generalized habituation impairments in ADHD.

Unexpectedly, post hoc tests revealed that the ADHD group was faster to report fading to auditory moving stimuli than was the control group ($t(39) = -6.37$, $p = .001$). It

was expected that adults in both groups would have the same performance on moving and validity stimuli in both modalities. As can be seen from Table 6 and as anticipated, the ADHD and control groups did have similar performance on the visual moving, visual validity, and auditory validity stimuli. These results indicate that the visual and auditory Troxler fading tasks were valid measures. The finding of faster report of fading to auditory moving stimuli suggests that participants in the ADHD group were not as distracted by these signals as were the controls. Thus, the ADHD group may have had more difficulty discriminating the moving signals from the background noise.

Qualitatively, it must also be noted that the ADHD group did report that the auditory stationary stimuli were more annoying than the auditory moving stimuli and the control group reported the reverse (i.e., auditory moving more annoying than auditory stationary stimuli).

Performance Variability

A 2 (Modality) x 2 (Stimulus Type) x 2 (Group) repeated measures ANOVA using the standard deviations of fading report across trials was calculated to examine if there were any meaningful performance differences between the groups. The average standard deviations are also listed in Table 6. A significant Group x Modality interaction was found ($F(2, 39) = 6.61$, $p = .01$). Post hoc tests revealed that ADHD participants had greater response variability on auditory stimuli ($t(39) = -2.08$, $p = .04$) than controls.

Table 6

Means, Standard Deviations, and Average Standard Deviations for Visual and Auditory Stimuli by Group

		Group			
		ADHD (n=21)		Controls(n=20)	
Modality					
	Type	M	MSD	M	MSD
Visual					
	Stationary	17.34 (5.32)*	4.89	15.52 (3.18)	4.44
	Moving	24.41 (5.39)	4.02	24.54 (4.32)	3.61
	Validity	5.86 (.32)	.49	5.63 (.31)	.48
Auditory					
	Stationary	20.11 (5.09)*	6.09	17.01 (3.03)	4.78
	Moving	15.45 (4.41)*	4.38	23.65 (3.79)	3.32
	Validity	6.21 (1.17)	.96	6.13 (1.11)	.72

Note: MSD is the average standard deviation.
*significant effect $p < .05$

Troxler Fading: A Measure of Habituation or Dishabituation?

A 2 (Modality) x 2 (Block) x 8 (Time) x 2 (Group) repeated measures ANOVA of fading report to stationary stimuli was calculated to examine whether the findings of slowed habituation to stationary stimuli in participants with ADHD were confounded by periods of dishabituation. Group means and standard deviations for each stimulus presentation by trial block are presented in Table 7. A significant Time x Group interaction was found ($F(1, 34) = 7.02$, $p = .01$). The findings indicated that control participants' fading reports to stationary stimuli steadily declined over time. This finding suggests that Troxler fading is a measure of habituation. In comparison, the ADHD group's fading reports were much more variable from trial-to-trial. This suggests that individuals with ADHD cannot sustain habituation.

Table 7

Means and Standard Deviations for Visual and Auditory Stationary Stimuli by Modality, Block, Time, and Group

		Group	
Block 1		ADHD (n=21)	Controls(n=20)
Modality			
	Time	M	M
Visual	1	15.27 (4.85)	18.31 (6.83)
	2	17.34 (4.76)	17.32 (4.76)
	3	16.21 (7.40)	16.90 (5.79)
	4	15.98 (5.98)	16.62 (5.84)
	5	16.51 (6.40)	16.00 (5.27)
	6	17.16 (5.91)	15.92 (5.38)
	7	16.84 (5.48)	15.72 (4.77)
	8	17.34 (5.27)	13.96 (3.65)
Auditory	1	20.61 (7.79)	21.26 (6.09)
	2	24.07 (7.07)	19.28 (5.43)
	3	25.30 (7.20)	18.99 (3.27)
	4	25.83 (6.95)	18.06 (5.38)
	5	20.96 (9.24)	16.74 (2.97)
	6	22.70 (9.09)	16.46 (4.81)
	7	22.93 (7.93)	15.01 (4.36)
	8	15.18 (4.33)	14.58 (5.12)

		Group	
Block 2		ADHD (n=21)	Controls(n=20)
Modality			
	Time	M	M
Visual	1	17.35 (6.38)	17.62 (6.72)
	2	17.65 (6.68)	16.50 (5.37)
	3	17.82 (6.48)	15.59 (6.12)
	4	20.11 (6.60)	14.27 (4.77)
	5	17.93 (5.59)	13.96 (5.90)
	6	18.81 (4.53)	13.62 (3.73)
	7	17.49 (5.71)	13.56 (5.96)
	8	17.36 (5.30)	12.43 (3.73)
Auditory	1	17.74 (5.45)	18.23 (5.73)
	2	19.89 (4.79)	17.10 (3.78)
	3	19.09 (5.69)	16.47 (5.57)
	4	18.84 (5.85)	16.36 (5.63)
	5	16.73 (5.75)	16.20 (4.31)
	6	15.35 (5.55)	16.11 (4.51)
	7	17.52 (6.20)	15.76 (5.13)
	8	19.05 (6.19)	15.54 (3.92)

Group Comparison of Fading to Stationary Stimuli in Right versus Left Hemifields on the Visual Troxler Task

As a past report found significant group differences for report of fading to stationary stimuli in right versus left visual hemifields, a 2 (Location of dot: right vs. left) x 2 (Group) repeated measures ANOVA was calculated for the visual stationary data to examine if there were fading differences between groups based on dot screen location. The means and standard deviations are presented in Table 8. No significant main or interaction effects were found for Location.

Table 8

Means and Standard Deviations for Visual Stationary Stimuli by Right and Left Screen Location and Group

	Group	
	ADHD (n=21)	Controls (n=20)
Location		
	M	M
Right	17.44 (5.35)	15.51 (3.09)
Left	17.24 (5.65)	15.54 (3.50)

Group Comparison of Fading to Stationary Stimuli by Speaker Location on the Auditory Troxler Tasks

As the auditory stimuli were presented in four different speaker locations (front, back, right, and left) a 4 (Location) x 2 (Group) repeated measures ANOVA was calculated to further explore any potential location differences between the groups. The means and standard deviations are presented in Table 9. A significant Location x Group interaction was found ($F(3, 37) = 6.86$, $p = .01$). The ADHD group was slower than controls to report fading to auditory stationary stimuli presented from the left ($t(39) = 3.19$, $p=.01$ and right ($t(39) = 3.34$, $p= .01$) speakers. No significant differences were observed for the front or back speaker locations.

Table 9

Means and Standard Deviations for Auditory Stationary Stimuli by Speaker Location and Group

	Group	
	ADHD (n=21)	Controls (n=20)
Location	M	M
Left	21.64 (5.82)*	16.92 (3.28)
Right	21.63 (5.20)*	16.90 (3.68)
Front	18.46 (4.66)	17.32 (4.29)
Back	18.70 (7.57)	16.89 (3.73)

Note: *significant effect $p<.05$

Secondary Hypothesis Testing:

Group Comparison on the Sensory Gating Inventory

The means and standard deviations for the overall SGI score and each subscale by group appear in Table 10. It was predicted that individuals with ADHD would report more sensory gating disturbances overall and on all four subscales (Perceptual Modulation Index, Distractibility Index, Overinclusion Index, and Factor-Stress Vulnerability) of the SGI than controls. To test this hypothesis, a univariate ANOVA was calculated using the overall score of the SGI as the dependent variable and group as the independent variable. A significant effect of group was found ($F(1, 39) = 51.40$, $p = .01$). As outlined in Table 10 and supporting the prediction, the ADHD group reported significantly more sensory gating disturbances than controls.

A MANOVA was then calculated using the four subscales of the SGI (Perceptual Modulation Index, Distractibility Index, Overinclusion Index, and Factor-Stress Vulnerability) as dependent variables and group as the independent variable. A significant multivariate effect of group was found (Wilks' $\Lambda = .33$; $F(4, 36) = 18.70$, $p = .01$). Post hoc univariate main effects were found for all four subscales of the SGI as follows: Perceptual Modulation Index ($F(1, 39) = 46.32$, $p=.01$); Distractibility Index ($F(1, 39) = 74.14$, $p = .01$); Overinclusion Index ($F(1, 39) = 32.10$, $p = .01$); and Factor-Stress Vulnerability Index ($F(1, 39) = 26.96$, $p = .01$). Again in support of the prediction, the ADHD group reported significantly more sensory gating disturbances on all four subscales compared to controls (see Table 10).

Table 10

Means and Standard Deviations for the Overall SGI and Four Subscales by Group

	ADHD (n=21)	Controls (n=20
Category	M	M
Perceptual Modulation Index	2.20 (.87)*	.57 (.63)
Distractibility Index	3.32 (.65)*	1.16 (.93)
Overinclusion Index	2.70 (.96)*	1.08 (.86)
Factor-Stress Vulnerability Index	2.64 (1.23)*	.99 (.74)
Overall SGI	2.62 (.76)*	.90 (.77)

Note: *significant effect $p<.01$

Correlations Between Reports of Sensory Gating Disturbances and Performance on Visual and Auditory Troxler Fading Tasks

Although the primary interest of this study was to examine report of slowed habituation as measured by the visual and auditory Troxler fading tasks, it is also important to examine whether the subjective experience of sensory gating difficulties is correlated with the experimental habituation measures, as past research has indicated that the ability to gate sensory information is also an index of habituation. The present study examined the relationship between the SGI (overall score and four subscales) and mean report of fading on the stationary and moving Troxler fading tasks in both modalities using Pearson Product Moment correlations (see Tables 11 and 12). The correlations for mean report of fading to visual stimuli are presented in Table 11. As can be seen from this table, no significant correlations were found between fading reports to visual stationary or moving stimuli and the overall SGI score or the four subscales.

The correlations for mean report of fading to auditory stimuli are displayed in Table 12. As can be seen from this table, significant negative correlations were found between fading time to auditory moving stimuli and the overall SGI score, and the four subscales of the SGI. No other significant correlations were found.

Table 11

Pairwise Correlations Between Mean Fading Time on Visual Stimuli and Report of Sensory Gating Disturbances

	Visual Stimuli	
Variable	Stationary	Moving
SGI		
PMI	.09	.04
DI	.01	.03
OI	-.02	-.12
FVI	.11	.05
Overall Score	.07	.01

Note: SGI is the Sensory Gating Inventory. The subscale abbreviations are as follows: PMI is the Perceptual Modulation Index; DI is the Distractibility Index, OI is the Overinclusion Index; and FVI is the Factor-Stress Vulnerability Index.

Table 12

Pairwise Correlations Between Mean Fading Time on Auditory Stimuli and Report of Sensory Gating Disturbances

	Auditory Stimuli	
Variable	Stationary	Moving
SGI		
PMI	.08	-.59**
DI	.26	-.65**
OI	.14	-.45**
FVI	.15	-.37*
Overall Score	.13	-.59**

Note: SGI is the Sensory Gating Inventory. The subscale abbreviations are as follows: PMI is the Perceptual Modulation Index; DI is the Distractibility Index, OI is the Overinclusion Index; and FVI is the Factor-Stress Vulnerability Index.
** p = .01
* p = .05

Summary of the Findings

The present study extended past findings of slower visual habituation to stationary stimuli in ADHD children to an adult ADHD population. In addition, the present study extended the past report by finding slower habituation to auditory stationary stimuli in adults with ADHD. Interestingly and unexpectedly, the present findings indicated that adults with ADHD were faster to report fading to auditory moving stimuli than were controls. The ADHD group, however, also showed greater performance variability on auditory stationary stimuli than did controls. The ADHD group was also slower than controls to report fading to auditory signals in the right and left speaker locations.

The secondary analysis showed that individuals with ADHD reported more sensory gating disturbances than controls, again as might be expected. Significant negative correlations were found between mean report of fading time on auditory moving stimuli and the overall SGI and four subscale scores.

Chapter V

Discussion

The primary goal of this dissertation was to examine the visual and auditory habituation processes of adults with ADHD. It was proposed that impaired habituation might explain some of the cognitive and behavioral symptoms displayed by individuals with ADHD. It was predicted that adults with ADHD would be slower to report habituation to visual and auditory stationary stimuli than controls. A secondary goal of the present study was to examine sensory gating deficits in an adult ADHD sample. As the ability to gate sensory information is an important index of habituation (Waters et al., 1977), it was predicted that adults with ADHD would report more sensory gating disturbances than controls.

Consistent with these predictions, we found that adults with ADHD were slower than controls to report fading to visual and auditory stationary stimuli. This finding supports and extends previous reports of slower habituation to visual stimuli in children diagnosed with ADHD (Jansiewicz et al., 2004). The results also supported the second prediction: ADHD participants reported greater occurrence of sensory gating disturbances than did controls. These findings have important implications for the etiology of ADHD and will be discussed in detail below.

Visual and Auditory Troxler Fading Tasks

Visual Task

The replication of slowed habituation in an adult ADHD population is an important finding of this dissertation. First, the finding is consistent with the notion that ADHD is a

life long disorder. Second, and perhaps more important, the finding may help to shed light on the neuroanatomical basis of ADHD.

A number of theories have been proposed to explain the cause of ADHD. These theories range from dysfunction in lower level brain regions (Sergeant et al, 1996, 2003; Halperin & Schulz, 2006) to dysfunctional top-down executive control (Barkley, 1998). Previous research using the Troxler Fading paradigm to investigate habituation processes in frontal lobe patients and children with ADHD has been interpreted to support dysfunctional executive control (see Mennemeier et al., 1994; Jansiewicz et al., 2004). This finding logically follows the neuroimaging evidence of reduced frontal lobe volume and activation in children and adults with ADHD (Castellanos et al., 1996; Filipek et al., 1997; Mostofsky et al., 2002; Seidman et al., 2006) and the similar hyperactive and impulsive behaviors displayed by frontal lobe patients and individuals with ADHD.

It has been proposed that visual Troxler fading is mediated by the lateral geniculate nuclei (LGN) of the thalamus (Clarke & Belcher, 1962), and the frontal cortex receives projections from the thalamus through interconnected cortical-thalamic pathways. Watson, Valenstein, & Heilman (1981) have proposed that connections between the frontal lobe and the nucleus reticularis (NR) of the thalamus might play a role in the habituation process. The NR is involved in arousal and activation, and is closely linked to the motor system (e.g., basal ganglia, and frontal lobes). Mennemeier and colleagues (1994) further proposed that the NR may facilitate habituation when stimuli are unchanging by inhibiting thalamic transmission to the cerebral cortex. On the other hand, the frontal cortex sends excitatory signals to the NR and may facilitate habituation of selected stimuli but, if stimuli are novel or biologically adaptive, the NR will be inhibited

by the thalamus. This proposal suggests that damage to the frontal cortex may facilitate the inhibition of the NR causing a disruption in habituation. It must be noted, however, that these conclusions may not fully explain mechanisms of habituation and as these conclusions were based on examination of patients with frontal lobe lesions they may not generalize to ADHD. Alternatively, it is possible that impairments in lower level brain areas, (i.e., reticular activating system, thalamus) may be capable of having a direct impact on the later developing frontal lobe regions and executive control (see Sergeant, 2003; Halperin & Schulz, 2006). These early brain impairments could adversely affect mechanisms of habituation.

Even though neuroimaging evidence has indicated that individuals with ADHD have reduced volume and activation in the frontal lobe region, the neuroimaging findings of additional brain region abnormalities in ADHD cannot be ignored. Brain imaging studies of children and adults with ADHD have also found abnormalities in the ACC, basal ganglia, and cerebellum (Castellanos et al., 1996; Filipek et al., 1997; Vaidya et al., 1998; Durston, 2003; Pliszka, et al., 2006; Seidman et al., 2006).

Previous research has shown that the ACC is involved in a number of cognitive processes (e.g., attention, salience, interference, and response competition) (see Bush, Luu, & Posner, 2000; Casey, Thomas, Welsh, Badgaiyan, Eccard, Jennings, & Crone, 2000; Downar, Crawley, Mikulis, & Davis, 2002)). Studies that have investigated cingulotomy patients have found that despite having intact processing in a number of cognitive domains (e.g., language, visual perceptual skills, motor, and memory); these patients have a variable and incomplete pattern of habituation (Cohen et al., 1994). Additional research on cingulotomy patients has also found impairments in selective and

sustained attention as well as executive control deficits in response generation, intention, and persistence (Cohen, Kaplan, Zuffante, Moser, Jenkins, Salloway, & Wilkinson, 1999).

As the anterior cingulate has rich interconnections with the frontal cortex (orbitofrontal and dorsolateral), the parietal cortex, the motor systems, limbic region and basal ganglia, it was proposed that damage to this area in cingulotomy patients indicates that the ACC influences both attention and executive control by modulating the salience of external signals so that attention can be directed to the most important environmental events (Cohen et al., 1999). Overall, this suggests that damage to the ACC may disrupt the temporal continuity needed to discriminate and coordinate response strength to incoming signals (Cohen et al., 1994).

The finding of decreased volume and abnormal activation of the ACC and slowed habituation in children and adults with ADHD suggests that deficits in ACC modulation may be able to explain both the cognitive (i.e., inattention, working memory) and behavioral (i.e., hyperactivity, impulsivity) symptoms of ADHD. In fact, it has been reported that damage to the ACC mimics the behavioral symptoms commonly displayed by individuals with frontal lobe damage (Cohen et al., 1999). Further, the inability to habituate to or filter irrelevant signals can cause disruption in both bottom-up (arousal and activation) and top-down (executive control) processing.

Although the present study cannot offer any direct evidence supporting the underlying neural correlates of habituation, it does indicate that further investigation of visual habituation in ADHD may reveal important insights into both the neuroanatomical basis of ADHD and the brain mechanisms involved in habituation.

Visual Task Lateralization

Prior research using Troxler fading showed that ADHD children were slower to habituate to stimuli in the right visual field (Jansiewicz, et al., 2004) and suggested greater involvement of the left hemisphere in habituation. The present study failed to replicate the finding of prolonged Troxler fading in the right visual field. In fact, no fading differences were found between the right and left fields. The lack of finding a difference in fading to right and left visual stimuli may be attributed to the mature frontal lobe functioning of adults with ADHD relative to children with ADHD. This is supported by the neuroimaging evidence of greater white matter volume in adults with ADHD compared to children with ADHD. The increased white matter volume may reflect developmental changes as white matter continues to grow until the late 20's (Seidman et al., 2006). The findings of the present study along with the neuroimaging differences between children and adults with ADHD suggest that adults with ADHD may use controlled processes to compensate for disruptions in more automatic processes (see Halperin & Schulz, 2006).

Auditory Task

The findings of the present study showed that adults with ADHD were also slower to report fading to auditory stationary stimuli. This finding is an important contribution of the dissertation as it provides evidence for impaired habituation in other modalities, and suggests a general impairment of habituation processes in ADHD.

The finding that individuals with ADHD are slower to report habituation to auditory signals compared to controls is capable of explaining why they are more easily distracted by less relevant sounds in their environment as well as their hyper-responsiveness to

distracting stimuli. For example, we note the difficulty individuals with ADHD have focusing on their teacher's voice when their attention is alerted by the sound of a lawn mower outside the classroom window that they cannot ignore.

Overall, we are much more alerted by auditory stimuli because we are not able to shut off our auditory senses to unwanted inputs the same way we can shut off our visual senses by closing our eyes. In addition, we cannot move our ears in the same manner we can move our eyes to scan the environment. The alerting effect of auditory stimuli may be a remnant of our evolutionary history as auditory signals warn us of life threatening events (e.g., oncoming predator, approaching automobile); however, in comparison to individuals with intact habituation processes, individuals with ADHD may not be as effectively able to modulate irrelevant alerting signals.

As a large majority of early auditory processing occurs subcortically, slower habituation to auditory signals in adults with ADHD offers support for theories that propose impairments in lower level brain systems. This, however, does not rule out the involvement of the ACC in auditory habituation. Recent investigation has shown increased activation of the ACC and auditory sensory cortex during processing of irrelevant auditory stimuli (Crottaz-Herbette & Menon, 2006). Overall, this finding suggests that the ACC may also modulate auditory habituation; ACC impairments in ADHD may lead to slower habituation of auditory signals.

Auditory Task Lateralization

The findings of the present study show that individuals with ADHD were slower than controls to report fading to stimuli in right and left speaker locations. As the ADHD participants' fading reports to stimuli in the right and left speaker locations were almost

identical, the findings do not suggest underlying hemisphere lateralization for habituation to tones. This is not surprising given that previous research has indicated that habituation to speech stimuli is lateralized in the left hemisphere but is not lateralized for tones (Teisman, Soros, Manemann, Ross, Pantev, & Knecht, 2004).

The finding of slower habituation by the ADHD group suggests that they were more alerted by the right or left signals because they "popped-out" against the continuous background noise. The continuous background noise was centrally located and reached both ears simultaneously but the tones presented through the right or left speakers only directly reached the respective ear. This may have caused these tones to be more alerting and attentionally demanding for the ADHD participants, thus more difficult for them to suppress.

Validity of Troxler Fading Tasks

Compared with the research protocols used by Mennemeier and colleagues (1994) and Jansiewicz and colleagues (2004), the protocol used in the present study allowed for more reliable methods and greater control of eye movements. First, computerized display of stimuli replaced the examiner-controlled method (e.g., manual manipulation of stationary, validity and moving peripheral stimuli) used by Mennemeier and colleagues (1994). Second, in comparison to the video camcorder methods used by Jansiewicz and colleagues (2004) to control eye movement, the use of a state of the art eye tracking device in the present study allowed for more accurate calibration of the task for each participant and also reduced human error in monitoring eye movements on a television screen. These improved methods helped to resolve concerns that difficulties with visual fixation may have led to slower fading time for individuals with ADHD compared to

controls reported in both previous studies. Overall, the finding that participants in both groups were fairly consistent to report fading to visual moving, visual validity, and auditory validity stimuli lends weight to the notion that the Troxler fading tasks are assessing visual and auditory habituation.

It was anticipated that both the ADHD and control group would not report fading to the auditory moving stimuli because of the novelty of the tones. Even though the control group reported fading slightly faster to the auditory moving stimuli than visual moving stimuli, the difference was not significant. Unexpectedly, however, the ADHD group reported faster fading to auditory moving stimuli than visual moving stimuli and they were significantly faster to report fading compared to controls. It is possible that individuals with ADHD were not able to attend to the auditory moving stimuli because they had more difficulty discriminating the auditory moving tone stream from the continuous background noise stream, suggesting a potential deficit in figure-ground analysis.

Difficulties with figure-ground analysis may suggest that the ADHD participants had auditory processing deficits. This is not surprising given the high comorbidity of ADHD and central auditory processing disorder (CAPD). Even though the exact co-occurrence of ADHD and CAPD is unknown, it is suspected to be 41% for individuals who have been diagnosed with ADHD and 43% for individuals suspected of having ADHD (Geffner, 2006).

Alternatively, it is also possible that the auditory moving tones were not as alerting to the ADHD group because the tones were modulated. The rise and ebb of the modulated frequency may have made it easier for them to habituate to the moving tones or may

reflect impulsive responding. Qualitatively the ADHD group did report that the auditory stationary signals were far more annoying than the auditory moving tones. Interestingly, the control group reported the opposite effect. Finally, it is also possible that the findings indicate that individuals with ADHD have auditory short-term memory deficits and may thus explain why individuals with ADHD have a higher incidence of language-learning disorders (for a review see Barkley, 1998).

Summary and Conclusion of Visual and Auditory Troxler Fading

Overall, the results of the study indicate that the automatic process of habituation is impaired in ADHD for both visual and auditory stimuli. Together with the findings from the literature, this suggests that the impairment is life long. Further, the inability to automatically habituate to stimuli in the periphery facilitates the explanation of the characteristic symptoms of inattention, distractibility, hyperactivity, and impulsivity commonly displayed by individuals with ADHD. The attentional and behavioral symptoms could be a consequence of a system that does not provide the automatic inhibition of irrelevant internal or external signals to provide greater focus on relevant stimuli.

At the present time however, the underlying neural correlates of Troxler fading are not known. Additional research is needed to provide insight into the neural mechanisms of impaired habituation in both the visual and auditory modalities using ERP and FMRI technology. Future research should also explore evidence for impaired habituation in somatosensory and multimodal processing.

Sensory Gating Disturbances and ADHD

The finding that adults with ADHD reported greater sensory gating disturbances on the overall SGI and all four subscales indicates that individuals with ADHD are aware that they have an impaired ability to filter external and internal sensations. This is an important finding for several reasons. First, no known study to date has investigated self-reporting of sensory gating disturbances in an ADHD population. Second, as adults with ADHD are not always good at reporting their own symptoms and it is often difficulty to obtain parental corroboration (see Barkley, 1998), an index of sensory gating impairments may be helpful in distinguishing adults with ADHD.

The additional finding of significant correlations between the SGI and fading to auditory moving stimuli offers empirical support for disrupted habituation processes in ADHD because sensory gating is an index of habituation. This is an important contribution of the dissertation because no known study to date has found a correlation between habituation and self-reported sensory gating impairments. Although correlations were found for the SGI and Troxler fading, no correlations were found for stationary stimuli as anticipated. Additional research is needed to explore the correlation between Troxler fading and subjective reporting of sensory gating difficulties.

Limitations of the Study

Performance Variability

A concern in studying deficits in ADHD, and a potential limitation in the present study, is the use of tasks that may confound motor performance with attention and the difficulties of operationalizing attentional constructs (Barkley, 1998). In the present task, like many in the research literature, participants were required to perform a motor response in order to report a change in a subjective attentional task (i.e., report fading by

pressing a button). It is unclear from the results of previous studies, and the present study, whether true attentional processes or motor responses are being investigated.

This has led many researchers to argue that comparing the mean reaction time performance of ADHD participants with controls does not adequately assess meaningful differences in performance because mean reaction time scores are often positively skewed (Leth-Steenson, Elbaz, & Douglas, 2000). A number of studies have indicated that it is the variability in reaction time between ADHD and control participants which indicates attentional inconsistency (Douglas, 2005; Castellanos & Tannock, 2002). Further, it is often the lack of stability in the reaction times of ADHD participants over the course of trials that indicates a reduction in the control of action (Zimmermann & Fimm, 2002). Given the possible confound between report of fading and motor performance, the present study examined the variability in performance between ADHD participants and controls.

The present study did not find any performance variability differences between the groups on the visual fading tasks. As only one other study has measured the visual habituation processes of individuals with ADHD, and the researchers did not measure performance variability, no direct comparisons can be made. It is possible to argue that the lack of performance variability differences across groups on the visual tasks suggests that prolonged fading to stationary stimuli by the ADHD group is a measure of the slower engagement of their motor processes rather than attentional impairments. However, this is unlikely given the comparable reaction times between the groups on the visual validity trials. On the other hand, it is equally plausible to argue that the eye tracking procedures used in the present experiment may have contributed to the response consistency across

the groups. The results from post hoc analysis did show that individuals with ADHD had significantly more visual stationary trials deleted and replaced because they moved their eye gaze to peripheral stimuli ($t(1,39) = 4.63$, $p = .001$) compared to controls. As individuals with ADHD were more easily attracted to the stimuli in peripheral vision, it is likely that they would also have had more response variability without the use of the eye tracking rejection procedures. As fading times on these rejection trials were not retained, no analysis could be conducted, so it is currently unknown if this may have impacted the results of the present study.

The results of this study did indicate that ADHD participants demonstrated more performance variability on auditory stationary stimuli than controls. Again, it is possible that prolonged fading by the ADHD group is indicating the slower engagement of their motor than attentional processes, but this is unlikely given the comparable fading times between the tested groups on the auditory validity trials.

Overall, the finding of slower habituation to both visual and auditory stationary stimuli by the ADHD group suggests that Troxler fading is measuring inattention. The findings, however, do not rule out potential motor confounds. In fact, it may not be possible to separate motor control from inattention using behavioral tasks, like the Troxler Fading paradigm, that require participants to make an overt motor response to a subjective measure, such as habituation. However, it is argued that the act of habituating, even though it is relatively automatic, involves the dampening of the motor system. As such, it is possible to speculate that an impaired ability to habituate not only leads to inattention but also causes related behavioral and motor difficulties. Stated differently, an impaired ability to habituate involves both motor and attentional processes. Thus,

Troxler fading may be measuring both the attentional and motor impairments commonly displayed by individuals with ADHD.

Dishabituation

Another concern in both the present study and prior reports is the possibility that mean Troxler fading scores may be confounded with measures of dishabituation. Dishabituation is defined as an increase in responsiveness to previously habituated stimuli following the presentation of another stimulus (Thompson & Spencer, 1966). In both the present study and past reports, stimulus type (stationary, moving, and validity) and stimulus location were randomly counterbalanced across trials and trial blocks. Dishabituation is a concern because it can arise from changes in stimulus type, location, and temporal occurrence from trial-to-trial or between trial blocks (Siddle, Stephenson, & Spinks, 1983).

An analysis of fading reports to stationary stimuli over trials indicated that control participants' fading reports declined steadily over time. Evidence of decreasing responsiveness to the same stimuli over trials reflects both habituation and elementary learning.

The findings indicated that the ADHD groups fading reports were much more variable from trial-to-trial suggesting a complex pattern of habituation and dishabituation. Rather than suggesting a dishabituation confound, it is much more likely that this response variability would be expected if habituation processes were impaired. In order for habituation to occur, the organism must recognize that the repeated presentation of the same stimulus is no longer novel. If habituation was impaired, an organism would not be able to consistently filter out no longer novel stimuli resulting in responding to some but

not all presentations of the same stimuli. In other words, a very variable pattern of responding would result.

Previous research on cingulotomy patients has indicated that they also showed a variable pattern of habituation from trial to trial compared to the steady decline in responding for controls. The variable responding by cingulotomy patients was interpreted to suggest incomplete habituation caused an impaired ability to discriminate signal importance (Cohen et al., 1994). In contrast to the present study, these researchers used a more traditional repetition-change paradigm in which a preset criterion of habituation was reached before a dishabituation trial was intentionally presented. By using this methodology the researchers were able to obtain separate measures of habituation and dishabituation. Interestingly, they did not find any group differences for dishabituation.

As the neuroimaging findings indicate reduced volume and activation of the ACC in individuals with ADHD (Castellanos et al., 1996; Filipek et al., 1997; Vaidya et al., 1998; Durston, 2003; Pliszka, et al., 2006; Seidman et al., 2006), the findings of variable habituation in the present study may suggest the involvement of the ACC in the habituation process.

Additional studies are needed to further examine the potential confound of dishabituation in Troxler fading. Future studies using Troxler fading to investigate habituation may wish to counterbalance the start of the trials to prevent order effects rather than completely counterbalancing the presentation of all trials by modality, type and location. This may prevent a potential dishabituation confound because participants will be asked to report fading to all trials of a stimulus type, location, and modality before

a new stimulus is presented. It is also suggested that in between trials of different stimuli types the researcher may wish to present a series of previously habituated stimuli to measure dishabituation. It may also be effective to reduce the alerting nature of the stimuli themselves by including a warning cue (e.g., a directional arrow in the center of the screen on the visual task or a distinct tone from the specific speaker location on the auditory task) to alert the participants that a stimulus will be presented and where it will be appearing.

Group Characteristics and ADHD Rating Scales

The results of the study should also be considered in light of the following issues regarding group characteristics. First, in contrast to the literature, the present study found no significant IQ differences between the groups. The similarity in IQ scores across the groups is not surprising given that college students were recruited for the present study. A higher IQ may allow individuals with ADHD to overcome the attentional obstacles they face in completing necessary course requirements to successfully obtain their bachelors degree.

Second, even though the participants in the ADHD group did not report previous diagnosis of any psychiatric disorders which commonly coexist with ADHD, they did meet criteria for a number of these disorders on the semi-structured interview (e.g., Oppositional Defiant Disorder, and Conduct Disorder in childhood; Major Depressive Disorder, and Anxiety Disorder in adulthood). In addition, a number of the control participants also met criteria for anxiety and mood disorders. The presence of these disorders may impact the interpretation of the findings, as the study was thus not limited

to a comparison of "pure" ADHD and normal controls as had been intended. However, it may be impossible to find a sample of "pure" ADHD (Wender, 1998).

Third, the results of this study may not generalize to both genders as the present study included more females in both groups and far more females than males in the ADHD group than expected. At the same time, however, it should be noted that the finding of slower habituation in childhood ADHD was based on a much larger male than female population (see Jansiewicz et al., 2004). Taken together the results of both studies suggest that habituation processes are impaired in ADHD for both genders.

More interesting is the fact that the present study supports the contention that females are less likely to be diagnosed with ADHD in childhood because they do not exhibit the behavioral signs of the disorder (Nadeau and Quinn, 2002; Adelizzi, 1998). In fact, more than 50% of the females in the ADHD group were not diagnosed with ADHD until entering college and experiencing academic difficulties. Females may go undiagnosed until they realize that they are unable to effectively cope with the demands of school or work. In addition, the larger sample of females may explain why participants in the present study met criteria for undiagnosed anxiety and mood disorders. Prior research findings have indicated that females with ADHD are much more likely to have higher incidences of anxiety and depression compared to males (Rucklidge & Tannock, 2001). The findings of the present study highlight the need to further explore the emergence and course of ADHD in females to better explain why their symptoms are not recognized in childhood and are more likely to be present with anxiety and mood disorders.

Fourth, although the present study recruited participants who had been diagnosed with ADHD by a professional (medical or psychological), it was decided that they also had to

meet criteria on two popular ADHD rating scales to be included in the project. The additional criteria were included because the diagnosis of ADHD is very subjective and thus not reliable.

Originally, to be included in the ADHD group, the participants were expected to meet diagnostic criteria on both the self- and observer reports of the WURS and the CAARS (either the DSM-IV ADHD Symptom Subscale or Total ADHD Index of the CAARS). The WURS was used to measure retrospective childhood symptoms of the disorder as diagnostic protocol indicates that in order to be diagnosed with adult ADHD the symptoms must be present in childhood. The CAARS was used to measure current function of the individual presenting with ADHD symptomatology.

As both rating scales measure attentional and behavioral disturbances, it was assumed that adults with ADHD would meet criteria on both measures. It was quickly noted, however, that it was not possible to meet this protocol. It was discovered that the observers' ratings were much higher on the WURS than on the CAARS. The reverse was noted for the self-report scales (lower on WURS than CAARS). Although unexpected based on the diagnostic criteria and the similarity of the questions on the two rating scales, the fact that there were discrepancies on the self- and observer reports should not have been surprising based on the instructions to complete each of the rating scales.

On the WURS, the instructions indicate that the person reporting the symptoms should base their observations on childhood patterns. The observers (participant's parents) were better able to report these symptoms than the ADHD participant because they were basing their reports on observable characteristics of their child. Even though it could be argued that both observer and self-ratings were distorted by retroactive recall bias, a

better explanation may be that children are not able to accurately judge or verbally encode for storage their own attentional and behavioral difficulties and this impacts their later recall. On the other hand, the ADHD participants would be better able to describe their present functioning on the CAARS than an observer because adults with ADHD are better able to hide their deficits and exhibit far less outward (hyperactive/impulsive) signs of the disorder.

To accommodate for the disparate ratings, the present study protocol was modified to include participants in the ADHD group if they scored within the elevated range on either self- or observer reports on the WURS or the CAARS (see chapter 3 for specific details). In addition, diagnosis of ADHD was further verified through the administration of a semi-structured interview. The original inclusionary criteria did not need to be modified for the control participants.

Finally, it must also be mentioned that the ADHD participants in the present study may not be representative of a clinic-based sample for a number of reasons. First, a large majority of the sample (more than 50%) were not diagnosed with ADHD until entering college or due to unsatisfactory academic progress. Second, none of the ADHD participants was currently being treated with prescription medication to relieve their attentional deficits. Even though the overall results from group comparisons of both the self- and observer rating scales did show that the ADHD group met diagnostic criteria and the control group fell within normal limits, caution is warranted in interpreting the present findings as there are academic benefits (e.g., more time to take tests, tailored tests, and longer time to complete assignments) associated with documented learning disorders.

Summary and Concluding Remarks

In conclusion, the finding of slower visual and auditory habituation along with the finding of higher subjective report of sensory gating disturbances for adults with ADHD has important implications for current theoretical models of the etiology of ADHD.

First, the findings suggest that ADHD may be caused by the involvement of widespread brain regions. Adults with ADHD, who have more fully developed frontal brain regions, have the same impaired habituation processes as children with ADHD. This suggests that fully developed frontal functioning in adults does not ameliorate habituation deficits in ADHD. The similar pattern of responding by children and adults with ADHD suggests that a disruption in automatic processes may be able to explain both the bottom-up (arousal and activation) and top-down (self-control) deficits associated with ADHD.

Second, as hyperactivity and impulsivity are not prominent symptoms of adult ADHD it is possible to speculate that impaired habituation produces an "approach" behavioral style in childhood ADHD. This behavioral style would cause children with ADHD to approach and respond to all attractive stimuli in their environment. The mature frontal control processes of adults with ADHD would explain why they do not exhibit the same behavioral "approach" style as ADHD children. In other words, adults with ADHD use top-down control processes to compensate for impaired automatic processes. Mature executive functions would explain the decreased behavioral disturbances found in ADHD adults, but not the unremitting distractibility, suggesting that inattention is the most prominent symptom of ADHD across the lifespan.

Given the education level, and gender differences in the present study, caution must be used in generalizing the findings. At the same time the investigation of the emergence of ADHD in a female population is extremely important. In addition, the ADHD sample size was too small to investigate habituation deficits across subtypes of ADHD. It is possible that habituation deficits may be specific to inattentive or combined type ADHD and not hyperactive-impulsive type. A larger sample of adult ADHD participants with sufficient distribution across subtypes should be examined to further explore impaired habituation. However, it must be mentioned that it may not be possible to find an adult ADHD sample of hyperactive-impulsive type, as the research literature indicates that adults with ADHD predominantly fall into inattentive or combined subtypes (Barkley, 1998).

Finally, to date, this is the only known report of visual Troxler fading in an adult ADHD population. Further, it is the only known study investigating auditory Troxler fading and reporting of sensory gating disturbances in an adult ADHD population. Future research is needed to provide greater understanding of the impact of habituation on the development and course of ADHD over the lifespan.

Appendix A

Recruitment Announcements

See attached copies

RESEARCH PARTICIPANTS WANTED

Have you been diagnosed with Adult Attention-Deficit/Hyperactivity Disorder or do you have attentional difficulties?

A research study is being conducted to examine visual and auditory attention in adults who have Attention-Deficit/Hyperactivity Disorder. Qualified participants will be paid $20 for the 60-minute study.

Participants must be:

- Age 18 - 40
- English Speaker
- Diagnosed with Adult ADHD or experience attentional difficulties
- Have Normal or corrected to normal vision
- Have Normal hearing

If you are interested in volunteering for or learning more about this study, Please contact Jacqueline Massa at 201-618-3509 or send email inquires to jmassa@kean.edu

RESEARCH PARTICIPANTS WANTED

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Appendix B

History Questionnaire

See attached copy.

Visual and Auditory Habituation Processes in Adults

HISTORY QUESTIONNAIRE

The following questionnaire is designed to provide us with some basic information about your development and current everyday functioning. Some of the questions may not be relevant to you but maybe relevant to others in the study, so please fill out the questions on the form as completely as possible. We will go over it with you in order to clarify your responses.

Please be assured that all information contained in this form will be kept confidential.

ID#_____

Date_____

ID#_____

BACKGROUND INFORMATION

1. What is your date of birth? _____/_____/_____

2. What is your gender? Male Female

Please answer both Ethnicity and Race Questions

3. Ethnically, I consider myself to be:

_____Hispanic or Latino American

A person of Cuban, Mexican, Puerto Rican, South or Central American or other Spanish culture regardless of race.

_____Not Hispanic or Latino

4. Racially, I consider myself to be:

_____American Indian or Alaska Native:

A person having origins in any of the original peoples of North, South, or Central America and maintain tribal affiliation or community.

_____Asian:

A person having origins in any of the original peoples of the Far East, Southern Asia, or the Indian subcontinent including, for example Cambodia, China, India, Japan, Korea, Malaysia, Pakistan, the Philippine Islands, Thailand, and Vietnam.

_____Black or African American:

A person having origins in any of the black racial groups of Africa.

_____Native Hawaiian or other Pacific Islander:

A person having origins in any of the original peoples of Hawaii, Guam, Samoa, or other Pacific Islands.

_____White:

A person having origins in any of the original peoples of Europe, North Africa, or the Middle East.

5. a. What is your current marital status? (Circle the one that best describes you)

Single Never Married
Married
Separated
Divorced
Widowed

b. If married, what is the length of your current marriage?
_____

6. Do you currently live with anyone?
Yes No

If you answered yes to the above question, please list the names, ages and relationship of the individuals that live in your household below.

First Name Age Relationship

7. What is the first language that you spoke?

Asian Dialect English Spanish Other _____

8. Do you speak any other languages? Yes No

a. If you answered ""YES"" to question 8 what other languages do you speak?"

Asian Dialect English Spanish Other_____

Medical History
The following questions are related to your childhood and present medical history

9. Have you ever had any to the following conditions?

Please circle all that apply.

Allergies	Coma	Injury to the head
Anemia	Convulsions or fits	Lead poisoning
Asthma	Drug/poison injections	Sickle Cell
Brain Infections	Fever over 104	Staring Spells
Heart Problems	Epilepsy/Seizure	High Blood Pressure
Broken Bones	Migraine/Headaches	Loss of Consciousness
Thyroid Problems	Diabetes	

If yes to any, please describe and indicate whether the conditions occurred in childhood, or adulthood and are currently causing you distress: _____

_____

_____

_____

_____

_____

10. Did you ever have ear infections or fluid in your ears?
Yes No
If yes, how often?
_____

11. Were you ever hospitalized and/or had an operation?
Yes No

If yes, please list below:

a. Hospitalization(s):
Date: _____
_____
_____

b. Operation(s):
Date: _____
_____
_____

12. Did you ever have any of the following tests or special exams?

a. CT Scan or MRI Yes No
b. EEG (brain waves) Yes No
c. X-rays (not including those done by a dentist) Yes No
d. Other Yes No
e. Chromosome Tests Yes No

"If yes to any, why was the test done?
_____

13. Did you have any other chronic medical conditions that have not been mentioned?

Yes No

"If yes, please describe: _____

_____

14. Are you currently taking any prescription medications?
Yes No

If yes, please list the medications: _____

_____

15. Do you currently take any vitamin supplements?
Yes No

If yes, please list:
_____

16. When was your last eye exam?
_____

Was your vision normal? Yes No

17. When was your last hearing test?
_____

Was your hearing normal Yes No

18. Do you have any other concerns about your health now or in the past that have not been mentioned? Yes No

If yes, please describe:
_____

19. Have you ever sought psychiatric treatment?
Yes No

If yes, for what purpose? _____

20. Have you had or do you currently have any of the following difficulties?

1. Alcohol abuse Yes No
2. Arrests (Multiple) Yes No
3. Drug Abuse Yes No

a. If yes to any of the above, please describe: _____
_____

b. If yes to Drug or Alcohol abuse have you ever sought treatment? _____

21. Has any doctor or other professional ever told you that you have any of the following?

a. Depression or Mania	Yes	No
b. Generalized or Social Anxiety	Yes	No
c. Attention-Deficit/Hyperactivity Disorder	Yes	No
d. Schizophrenia	Yes	No
e. Obsessive Compulsive Disorder	Yes	No
f. Antisocial Personality Disorder	Yes	No
g. Learning Disability or Dyslexia	Yes	No
h. Speech problems	Yes	No
I. Sleep Disorders	Yes	No

22. Have you had or currently have any of the following sleep disturbances?

a. Nightmares	Yes	No
b. Sleepwalking	Yes	No
c. Recurrent dreams	Yes	No

If yes to any of the above, please describe: _____
_____

23. Do you have trouble sleeping at night?

Yes No

If yes, please describe: _____

_____

24. Do you experience any difficulty with the following?

a. Daytime sleepiness	Yes	No
b. Difficulty waking in the morning	Yes	No
c. Difficulty staying awake during the day	Yes	No
d. Difficulty staying awake in the evening	Yes	No

If yes to any question, please describe: _____
_____

FAMILY HISTORY

25. Mothers Medical History

a. What is your mother's age? _____

b. What is your mother's occupation? _____

c. What is the highest school grade your mother completed?
_____

d. Did your mother have any learning difficulties in school?

Yes No

If yes, please indicate what type of problems she had_____

e. When she was a child did your mother have behavioral problems?
Yes No

If yes, please indicate what type of problems she had_____

f. Does your mother currently have any chronic medical conditions?
Yes No

If yes, please describe: _____

g. Did your mother have any chronic medical conditions when she was a child? Yes No

If yes, please describe:_____

h. Has mother ever sought psychiatric treatment?
Yes No

If yes, for what purpose?_____

i. Has your mother ever experienced any difficulties with any of the following problems?

1. Alcohol abuse	Yes	No
2. Arrests (Multiple)	Yes	No
3. Severe Depression	Yes	No
4. Severe Anxiety	Yes	No
5. Drug Abuse	Yes	No
6. Mania	Yes	No
7. Schizophrenia	Yes	No
8. Excessive Fears or Phobia	Yes	No
9. Panic Attacks	Yes	No
10. Obsessions/Preoccupations	Yes	No
11. Delusions	Yes	No
12. Hallucinations	Yes	No

j. Have any of your mother's blood relatives ever had any learning problems or psychiatric problems including such things as alcohol/drug abuse, depression, anxiety, or psychiatric hospitalizations?

Yes No

If yes, please describe: _____

26. Fathers Medical History

a. What is your father's age? _____

b. What is your father's occupation?
_____

c. What is the highest school grade your father completed?
_____

d. Did your father have any learning difficulties in school?
Yes No

If yes, please indicate what type of problems he had _____
_____

e. When he was a child did your father have behavioral problems?
Yes No

If yes, please indicate what type of problems he had _____
_____

f. Does your father currently have any chronic medical conditions?
Yes No

If yes, please describe: _____

g. Did your father have any chronic medical conditions when he was a child? Yes No

If yes, please describe:_____

h. Has your father ever sought psychiatric treatment?
Yes No
If yes, for what purpose? _____

i. Has your father ever experienced any difficulties with any of the following problems?

1.	Alcohol abuse	Yes No
2.	Arrests (Multiple)	Yes No
3.	Severe Depression	Yes No
4.	Severe Anxiety	Yes No
5.	Drug Abuse	Yes No
6.	Mania	Yes No
7.	Schizophrenia	Yes No
8.	Excessive Fears or Phobia	Yes No
9.	Panic Attacks	Yes No
10.	Obsessions/Preoccupations	Yes No
11.	Delusions	Yes No
12.	Hallucinations	Yes No

j. Have any of your father's blood relatives ever had any learning problems or psychiatric problems including such things as alcohol/drug abuse, depression, anxiety, or psychiatric hospitalizations?

Yes No

If yes, please describe _____

DEVELOPMENTAL HISTORY

27.If you are able, please describe your parent's attitudes toward their pregnancy with you _____
_____

28. Are you aware of any complications your mother may have had during her pregnancy with you? (For example, extreme morning sickness, bleeding, infection, x-rays, etc.)

Yes No

If yes, please describe:_____
_____

29. Are you aware of any problems your mother may have had during your birth? (For example, forceps delivery, trauma, excess bleeding etc.)

Yes No

If yes, please describe:_____
_____

30. Did your mother use alcohol or other drugs during her pregnancy with you?

Yes No

If yes, please give details: _____
_____

31. Did your mother smoke cigarettes during her pregnancy with you?

Yes No

If yes, please give details: _____

32. Did you have any significant delays in your development? (For example, walking, talking, or sitting up)

Yes No

If yes, please provide details:_____
_____

33. Please circle any of the following that you believe you had significant difficulties with as a child:

Defiant	Aggression	Eating
Hyperactive	Impulsive	Stubborn
Shy	Withdrawn	Inattention
Fearful	Lying	Depression
Learning	Language	Stealing
Fighting	Motor Skills	Memory
Anxious	Distractibility	Destructive

34. In a short statement, please describe yourself as you think others see you. _____
_____

35. Is there anything that we have not asked or discussed that you feel would be of help to us in Better understanding you?
_____
_____

School History

36. What is the highest level of education that you have completed?

Less than High school
High school Diploma
Technical School
2 year of college/Associates Degree
4 years of College/BA or BS degree
Masters Degree (MA)
Doctoral Degree (Ph.D.)
Professional Degree (MD, JD)

37. If you have attended college, have you ever dropped out or stopped taking courses?
Yes No

If yes, please list the number of times you have started college course and failed to complete them
_____

_____

38. Would you say your grades in grade school were: (Circle the one the best describes you)

a. A's and B's
b. B's and C's
c. C's and D's
d. D's and F's
e. Widely varied

39. Did you ever skip a grade in school?
Yes No

If yes, please specify which grade and why: _____
_____

40. Did you ever repeat a grade?
Yes No

If yes, please specify which grade and why: _____
_____

41. What subjects in school would you consider you were strong in?
_____

42. What subjects in school would you consider you were weak in?

_____

43. Did you ever have any trouble with reading?
Yes No

If yes, please describe the problem: _____

44. Did you ever have any trouble with math?
Yes No

If yes, please describe the problem: _____

45. Did you have trouble organizing and completing homework assignments? Yes No

If yes, please describe the problem_____

46. Were you ever in a class for children with special needs?
Yes No

If yes, what kind of program was it? _____

47. Did you ever receive any of the following services in school?

a. Counseling (help with behavior) Yes No
b. Help with mathematics Yes No
c. Help with reading Yes No
d. Occupational Therapy (help with movement) Yes No
e. Social Skills(help with getting along with others) Yes No
f. Speech Therapy Yes No
g. Other Yes No

If “other”, please describe: _____

48. Did you ever receive any of the following services outside of school?

a. Counseling (help with behavior) Yes No
b. Help with mathematics Yes No
c. Help with reading Yes No
d. Occupational Therapy (help with movement) Yes No
e. Social Skills (help with getting along with others) Yes No
f. Speech Therapy Yes No
g. Other Yes No

Please describe: _____

49. Were you considered a discipline or behavior problem in school (For example, class clown or mischief maker)?
Yes No

If yes, please describe _____

_____

50. Did your teacher(s) ever say you were capable of doing much better than you did?

Yes No

51. Were you ever truant from school?

Yes No

52. Were you ever expelled or suspended from school?

Yes No

If yes, please describe_____

53. Did you ever get into any physical fights in school?

Yes No

If yes, please give details: _____

54. Did you ever have any trouble getting along with your peers in school?

Yes No

If yes, please explain_____

55. In general, what do you think your teachers would say about your performance in school? _____
_____

Employment History

56. What is your current employment status?

a. Full-time
b. Part-time
c. Unemployed
d. Student
e. Homemaker
f. Disabled

57. What is your current occupation?_____

58. How long have you worked in your present position? (Please list in months and years) _____

59. What is your longest period of employment at one place? (Please list in months and years)_____

60. If you are currently employed what is your overall satisfaction with you current job?

High Moderate Low

a. If your overall satisfaction is low, please describe: _____

_____

61. Summarize the jobs you have had, list from most favorite to least favorite._____
_____
_____

62. Have you ever had any work related problems?

Yes No

If yes, please list the types of problems you have experiences at your workplace _____
_____

63. Have you ever been fired from a job?

Yes No

If yes, please describe the reason _____
_____

64. Have you ever quit a job without giving a formal resignation? (For example, went to lunch and decided not to return)

Yes No

If yes, please describe the reason _____
_____

65. What would your employers or supervisors say about you?
_____
_____

SOCIAL HISTORY

66. Do you have trouble making friends? Yes No

67. Do you have trouble keeping friends? Yes No

68. Do you have trouble with your intimate relationships?
Yes No

69. If you answered yes to questions 66, 67, or 68, please describe your difficulties _____

_____
_____

70. Do your moods change very frequently?
Yes No

If yes, please provide details _____
_____
_____

71. Do you have problems with your temper?
Yes No

If yes, please describe _____
_____
_____

Thank you for completing the History Questionnaire.

Appendix C

Wender-Utah Rating Scales: Self-report and Observer report

See attached scales.

Wender Utah Rating Scale (WURS)
Self-Report Version

Date

	Not at all or very slightly	**Mildly**	**Moderately**	**Quite a bite**	**Very much**
As a child I was (or had):					
1. Concentration problems, easily distracted					
2. Nervous, fidgety					
3. Inattentive, daydreaming					
4. Hot- or short- tempered, low boiling point					
5. Shy, sensitive					
6. Temper, outbursts, tantrums					
7. Trouble with stick-to-it-tiveness, not following through, failing to finish things started					
8. Stubborn, strong-willed					
9. Sad, blue, depressed, unhappy					
10. Disobedient with parents, rebellious, sassy					
11. Low opinion of myself					
12. Irritable					
13. Moody, ups and downs					
14. Angry					
15. Acting without thinking, impulsive					
16. Tendency to be immature					
17. Guilty, feeling regretful					
18. Losing control of myself					
19. Tendency to be or act irrational					
20. Unpopular with other children, didn't keep friends for long, didn't get along with other children					
21. Trouble seeing things from someone else's point of view					
22. Trouble with authorities, trouble with school, visits to principal's office					
23. Overall, a poor student, slow learner					
24. Trouble with math or numbers					
25. Not achieving up to potential					

Wender Utah Rating Scale (WURS)
Observer Report Version

Date

As a child the person being described was (or had):

	Not at all or very slightly	Mildly	Moderately	Quite a bite	Very much
1. Concentration problems, easily distracted					
2. Nervous, fidgety					
3. Inattentive, daydreaming					
4. Hot- or short- tempered, low boiling point					
5. Shy, sensitive					
6. Temper, outbursts, tantrums					
7. Trouble with stick-to-it-tiveness, not following through, failing to finish things started					
8. Stubborn, strong-willed					
9. Sad, blue, depressed, unhappy					
10. Disobedient with parents, rebellious, sassy					
11. Low opinion of myself					
12. Irritable					
13. Moody, ups and downs					
14. Angry					
15. Acting without thinking, impulsive					
16. Tendency to be immature					
17. Guilty, feeling regretful					
18. Losing control of myself					
19. Tendency to be or act irrational					
20. Unpopular with other children, didn't keep friends for long, didn't get along with other children					
21. Trouble seeing things from someone else's point of view					
22. Trouble with authorities, trouble with school, visits to principal's office					
23. Overall, a poor student, slow learner					
24. Trouble with math or numbers					
25. Not achieving up to potential					

Appendix D

Sensory Gating Inventory

See attached inventory

Sensory Gating Inventory

W. P. Hetrick

Participant:_____ Gender: _____

Date: _____ Age: _____

Never True	Almost Never	Sometimes True	Almost Always	Always True
0	1	2 3	4	5

Please circle your response

1. Every now and the colors seem more vivid to me than usual. 0 1 2 3 4 5
2. Sometimes I find it difficult to focus on one visual sight to the exclusion of others. 0 1 2 3 4 5
3. I find it hard to concentrate on just one thing. 0 1 2 3 4 5
4. The silliest things that are going on interest me. 0 1 2 3 4 5
5. At times I have feelings of being flooded by sounds. 0 1 2 3 4 5
6. There are times when I can't concentrate with even the slightest sounds going on. 0 1 2 3 4 5
7. Sometimes it seems like someone has turned the volume up – things seem really loud. 0 1 2 3 4 5
8. There are days when indoor lights seem so bright that they bother my eyes. 0 1 2 3 4 5
9. I notice background noises more than other people. 0 1 2 3 4 5

Never True	Almost Never	Sometimes True	Almost Always	Always True
0	1	2 3	4	5

10. I Hear sounds but I can't make sense of them all because it's like. 0 1 2 3 4 5

11. For several days at a time I have such heightened awareness of sights and sounds that I cannot shut them out. 0 1 2 3 4 5

12. It seems like I hear everything at once. 0 1 2 3 4 5

13. I am easily distracted. 0 1 2 3 4 5

14. It seems like I take in too much. 0 1 2 3 4 5

15. When I am driving at night, I am bothered by the bright lights of oncoming traffic. 0 1 2 3 4 5

16. It is hard to keep my mind on one thing when there's so much else going on . 0 1 2 3 4 5

17. When I am in a group of people I have trouble listening to one person. 0 1 2 3 4 5

18. My hearing is so sensitive that ordinary sounds become uncomfortable. 0 1 2 3 4 5

19. It's not bad when just one person is speaking but if others join in, then I can't pick it up at all. I just can't get into tune with that. 0 1 2 3 4 5

20. Sometimes I notice background sounds more than usual. 0 1 2 3 4 5

21. Not only the color of things fascinates me but all sorts of little things, like markings in the surface, attract my attention, too. 0 1 2 3 4 5

22. I find it difficult to shut out background noise and that makes it difficult for me to concentrate. 0 1 2 3 4 5

23. I seem to always notice when automatic appliances turn on and off (like the refrigerator or the heating & cooling systems). 0 1 2 3 4 5

Never True	Almost Never	Sometimes True	Almost Always	Always True
0	1	2 3	4	5

24.	I have feelings of being flooded by visual experiences, sights, or colors.	0 1 2 3 4 5
25.	When I am tired, the brightness of lights bothers me.	0 1 2 3 4 5
26.	There have been times when it seems that sounds and sights are coming in too fast.	0 1 2 3 4 5
27.	I can't focus on one sound or voice to the exclusion of others.	0 1 2 3 4 5
28.	At times I have trouble focusing because I am easily distracted.	0 1 2 3 4 5
29.	Background noises are just as loud or louder than the main noises.	0 1 2 3 4 5
30.	I cannot focus on visual images when I am tired or stressed.	0 1 2 3 4 5
31.	I have more trouble concentrating than others seem to have.	0 1 2 3 4 5
32.	Maybe it's because I notice so much more about things that I find myself looking at them for a longer time.	0 1 2 3 4 5
33.	Everything grips my attention even though I am not particularly interested in any of it.	0 1 2 3 4 5
34.	I seem to hear the smallest details of sound.	0 1 2 3 4 5
35.	When I'm tired sounds seem amplified.	0 1 2 3 4 5
36.	It seems that sounds are more intense when I'm stressed.	0 1 2 3 4 5

Appendix E

Visual and Auditory Troxler Fading Task Pilot Studies

See attached copy

Pilot Studies

Thirty students from an Introductory Psychology course at Kean University piloted the visual and auditory Troxler fading tasks. Students received course credit for their participation. The visual task was piloted as described in the above method section. The 10 participants reported fading within 12.78 (4.25) seconds to the stationary stimuli, 27.5 (1.72) seconds to the moving stimuli and 5.67 (.77) seconds to the validity stimuli. Participants reported no difficulties with the visual task. There were also no difficulties with calibration of the peripheral dot or calibration of the eye tracker.

Many different combinations were implemented to construct an auditory analog of the visual task. Initially, a carnival atmosphere sound was used as the continuous background sound, but as will become evident the sound was masking the tones. The continuous noise was originally presented from all four speaker locations and remained constant. A number of sine tones ranging for 200, 400, 800, 1000 Hz and 20, 25, and 30db each were played through one speaker at a time (front, back, left or right). The same 10 participants who performed the visual task reported that they were unable to discriminate the sounds from the background noise on the visual task. When the data was examined they appeared to be randomly pressing the response button.

Tones were then tested separately (without the continuous background noise) and the same 10 participants reported that the 1000 Hz 30db tones were the most pleasant. The 1000 Hz 30 db tones were then played again along with the continuous noise and participant's reported fading to the stationary tones within 15.45 (4.75) seconds and the validity tones within 7.87 (2.45) seconds However, participants reported fading to the moving tones within 4 seconds. Initially, the moving tone was created by modulating the

amplitude (60% depth) to be consistent with the visual movement. However, participants reported that they had pressed the response button when they could no longer hear the tone. On many occasions after they pressed the response button they remarked that they thought they made a mistake because they started to hear the tone again. Many participants also reported that the moving tone was piercing in sound. At this point a new moving tone was created by modulating the frequency instead of the amplitude. The same 10 participants were asked to retest the stimuli at a later date. The participants still reported fading quicker than expected to the moving stimuli (m = 7.6 seconds, sd = 4.67). They also still reported difficulty distinguishing the moving tone from the continuous background sound. The conclusion that was drawn from their data and reports was that the continuous background sound, which consisted of multiple frequencies and amplitudes, was masking the moving tone.

In order to prevent masking of the tones a new continuous background noise and presentation design was implemented. High frequency continuous white noise was chosen so that the lower frequency sine tones would not be masked. In addition, an adjustment was made to the computer program to allow the researcher to play the continuous noise alone from a separate speaker placed at a central location. This adjustment would also help prevent masking of the sine tones. Ten new participants were asked to pilot both the visual and auditory task. The participants mean report of fading on the visual task was as follows: stationary = 13.53 (sd = 4.51), moving = 28.23 (sd = 2.34); and validity = 5.89 (sd = 1.76). The report of fading to all visual stimuli was similar to the first pilot group. Again, none of the participants reported any difficulties seeing the dot in peripheral vision and no eye tracking difficulties were noted. The mean

report of fading time in seconds for the auditory task was as follows: stationary stimuli = 14.68 (sd = 4.02); moving stimuli = 28.11 (3.34); and validity stimuli within 6.54 (sd = 1.49).

Appendix F

Consent Form

See attached copy

Consent Form to Participate in Research

Kean University Psychology Department

Project: Visual and Auditory Habituation Processes of Adults: A Comparison of ADHD, Elevated ADHD Scores, and Controls

Principle Investigator: Jacqueline Massa

Background Information:

We are asking you to take part in a research study on attention. The study has been designed to help us to better understand auditory and visual attention process differences. Three groups of adults will be enrolled in the study: Adults diagnosed with attention-deficit/hyperactivity disorder, adults who evidence elevated scores on attention-deficit/hyperactivity disorder scales, and adults who evidence normal range scores on attention-deficit/hyperactivity disorder scales. This research is being done under the supervision of Dr. Suzanne Bousquet from Kean University and will be conducted using the office facilities of the Psychology Department (J-330).

Procedures:

If you agree to participate in the study you will be asked to fill out a History Questionnaire, two adult attention difficulties scales (Wender-Utah Rating Scale, and Conner's Adult ADHD Rating Scale), and a Sensory Gating Inventory, which is an adult sensory modulation scale. You will also be asked to take part in a semi-structured interview. The History Questionnaire will ask you to write down very sensitive information pertaining to your own and/or your family member's medical and psychiatric history. In addition, you will be asked to write down information pertaining to your employment, social, and school history. During the semi-structured interview the test administrator will ask you questions pertaining to very sensitive information regarding your past and present psychiatric history, such as "have you in the past or do you presently suffer from excessive anxiety" or "have you in the past or do you presently have prolonged periods of sadness". In addition, the test administrator may ask you questions regarding past and present criminal behavior ("were you ever physically cruel to people or animals"), sexual abuse ("have you ever forced someone into sexual activity") and substance abuse (have you ever had problems with alcohol/drug abuse). The administrator will write your answers to the interview questions on the interview form; no other recording devices will be used. You do not have to answer any questions on any of the questionnaires or during the interview that make you feel uncomfortable.

You will also be given a standardized test that assesses verbal and performance abilities. In addition, you will be asked to view some basic shapes and listen to some tones through speakers and to push a button when the shape or tone fades from awareness. During this task your eye movements will be tracked using Eye Tech Digital Systems Quick Glance 2 eye tracking device. The eye tracking device consists of a video camera that is mounted on a stand and placed on the computer/laptop keyboard. An infrared light source which is attached to the back of the computer/laptop will record your

eye movements. No device is attached directly to you. If you take medication for attention-deficit/hyperactivity disorder, you will be asked not to take the medication on the day of testing and provide a letter from your physician stating that it is both safe and acceptable to discontinue medication on the day of testing. It is important that you know that refraining from taking your medication may affect your academic performance on the day of testing or your competency in any other activity that requires you to take your medication. Finally, we ask that you return the completed parent or significant other attentional difficulties forms that were mailed to you before your appointment. The attentional difficulty forms should take approximately 10-15 minutes for your parent or significant other to complete. You will not be able to participate in the study if you do not have or cannot have the attentional difficulties forms completed by a parent or significant other. Your appointment is expected to last about 90 minutes.

Risks and Benefits of being in the Study:

There are more than minimal risks (i.e. than those risks encountered in everyday life) involved in this study. Specifically, you are being asked to provide sensitive and private information about your personal and your family members' medical and psychiatric history, as well as your past or present criminal behavior, drug abuse, or sexual conduct. In addition, you may become somewhat bored with the repetitiveness of the auditory and visual tasks. There are no direct benefits to you for participating in this research study. There are a number of indirect benefits to you for participating in the research study. First, you will be provided with information on attention-deficit/hyperactivity disorder and if you meet ADHD criteria. Second, you will also be provided with any information about your results and the research findings if you choose to. Finally, you will have the satisfaction of helping improve the understanding of attentional processes in the auditory and visual modalities. The benefit to the scientific community is the better understanding of the deficits underlying attentional processing.

Compensation:

You will be given $20.00 for your participation in the study to reimburse you for your time. You will receive payment immediately upon completion of testing. If you decide to withdraw from the study prior to completing testing you will still receive full payment.

Confidentiality:

Participant's names will not be recorded on any of the test material. The records of this study, including all test scores, history questionnaires and interview answers, master list of names, addresses and codes, will be kept entirely private and confidential to the extent permitted by law. In any sort of report that might be published, no information will be included that will make it possible to identify a participant. Research records will be stored securely in a locked cabinet in a locked office located in the Psychology Department (room J-330) at Kean University, and only researchers involved in the study will have access to the records. When the study is completed all forms and information related to the project will be destroyed.

Voluntary Nature of the Study:

Participation in this study is entirely voluntary. Your decision whether or not to participate will not affect your current or future relations with Kean University. If you decide to participate, you are free to not answer any question or to withdraw your participation at any time without penalty.

Contacts and Questions:

If any questions arise related to this study, I can call Jacqueline Massa at 201-618-3509 or 908-737-4000 or Dr. Suzanne Bousquet at 908-737-4000. I have also been told that I may contact, Toufic Hakim, Kean University Institutional Review Board Administrator, at 908-737-3364, if any problems or questions about my rights as a subject in the research study arise.

Consent Statement:

By signing this form I have agreed to participate as a subject in a research study entitled Visual and Auditory Habituation Processes of Adults: A Comparison of ADHD, Elevated ADHD scores and Controls to be carried out by Jacqueline Massa under the supervision of Dr. Suzanne Bousquet. Dr. Bousquet's office is located in Building J, Room 330, Kean University.

I have read and understood the above information. I have asked questions and have satisfactorily received answers. I have been told that my participation in this study is entirely voluntary and that I may withdraw from the study at any time without penalty. I consent to participate in the study. A copy of this consent form will be given to me for my records.

_____	_____	_____
Signature of Participant	Printed name	Date
_____	_____	_____
Signature of Investigator	Printed name	Date

Important Study Note:

The tests and forms you are being asked to complete are being used for research purposes only. If over the course of the study, and in the event that we find something of clinical significance, you would like to be informed please indicate by checking the appropriate line below. As our findings will not be diagnostic we will also be happy to provide you with a referral.

_____ Yes, I would like to be informed and provided with a referral.

_____ No, I do not wish to be informed and provided with a referral.

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