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An Electrophysiological Examination of ADHD-Associated Symptoms and Selective Attention in Adults

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AN ELECTROPHYSIOLOGICAL EXAMINATION OF ADHD-ASSOCIATED SYMPTOMS AND SELECTIVE ATTENTION IN ADULTS

by

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ABSTRACT

AN ELECTROPHYSIOLOGICAL EXAMINATION OF ADHD-ASSOCIATED SYMPTOMS AND SELECTIVE ATTENTION IN ADULTS

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A main component of Attention-Deficit/Hyperactivity Disorder (ADHD) is a deficit of inattention. This deficit causes impairment for both children and adults in a variety of settings including school and work. The current study examined auditory selective attention in a community sample of adults. It was the aim of this project to examine possible differences in selective attention for adults with high levels of ADHD-associated symptoms, when compared to adults with low levels of ADHD-associated symptoms, including conditions under which these differences may be an advantage. Specifically, it was expected that adults with high ADHD-associated symptoms would benefit from the high probability condition, whereas they would perform worse in the low probability condition. Results suggested that the high ADHD-associated symptoms group had a slower reaction time overall but, nevertheless, benefited behaviorally from correlated information, as exhibited by an improvement in reaction time for the high probability condition. Electrophysiological differences between the high and low ADHD-associated symptoms groups also emerged such that the high ADHD-associated symptoms group consistently displayed larger N1 amplitudes. Both groups appeared to react differently to distractor tones in the high probability condition although the high ADHD-associated symptoms group was the only group that benefited from this behaviorally.

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CHAPTER I

INTRODUCTION

Attention-Deficit/Hyperactivity Disorder (ADHD) is a disorder commonly first diagnosed in children. ADHD has been shown to affect 3 to 7% of school-aged children with, by definition, at least some symptoms present before the age of 7 years (APA, 2000; Riccio et al., 2004). In addition to the prevalence of ADHD in children, Clarke and colleagues (2008) reported that 2 to 3% of the general population continues to suffer from ADHD into adulthood. Based on *DSM-IV-TR* (APA, 2000) criteria, there are three subtypes of ADHD: Combined Type, Predominantly Inattentive Type, and Predominantly Hyperactive-Impulsive Type. According to Wadsworth and Harper (2007), it appears that symptoms of ADHD may be expressed differently in children and adults. Specifically, they indicated that hyperactivity and impulsivity are not as common in adults, whereas inattentiveness appears to be the most prominent clinical feature in adults suffering from ADHD. In a study by Clarke and colleagues (2008), the authors suggested that adults with ADHD tend to have significantly reduced symptoms of hyperactivity with inattentive components continuing into adulthood. Thus, inattention is of primary concern to many adults with ADHD.

The study of attention, and more specifically selective attention, can be traced back to research by Donald E. Broadbent in the early 1900s. Broadbent, a psychologist known for setting the foundation for studying selective attention is remembered as one of the most influential researchers in the area of selective attention. According to Styles (1997), Broadbent's publications in the area of selective attention were influential in that he developed a theory that the nervous system acts as an information processor and, thus,

set the groundwork for an information-processing approach to psychology. In a study conducted by Broadbent (1952), for example, he was among the first to determine that individuals have the ability to selectively attend to stimuli that possess a common physical feature while inhibiting those stimuli that lack that feature. Broadbent (1952) also found that irrelevant auditory information significantly increases errors. Broadbent believed this occurred because individuals were not successfully discarding or ignoring the irrelevant information. It is agreed upon in the current literature that individuals with ADHD tend to struggle with ignoring irrelevant information relative to the normal population.

According to Pritchard and Neumann (2004), the ability to selectively attend is important for everyday functioning. More specifically, the authors suggest that selective attention is useful in daily functioning due to the overwhelming number of stimuli, both relevant and irrelevant, that are presented within a day. Therefore, the process of selective attention develops, for most individuals, very early in life. Pritchard and Neumann (2004) reported that children as young as five years old are able to inhibit distracting stimuli effectively while successfully selectively attending and performing a Stroop negative priming task. This early development of selective attention, however, may not hold true for individuals with ADHD. As such, a variety of problems and difficulties may emerge.

A deficit in selective attention (i.e., difficulty selectively attend to a target while ignoring a distractor) is particularly found in those individuals with the Predominantly Inattentive Type or Combined Type of ADHD (Jonkman et al., 1997). In addition, Willoughby (2003) reported that, as an individual with ADHD gets older, self-esteem, academic abilities, and social abilities may diminish. In terms of the maladaptive effects

of ADHD, similar to children, adults with ADHD are more likely to achieve academically and occupationally below expectations for their intelligence (Goodman, 2007). According to Barkley, Murphy, and Fischer (2008), the demands of full-time occupations including minimal supervision, training, and skill development are shown to be particularly difficult for adults with ADHD due to their difficulties with self control, organization, deficits in attention, and impulse control. Thus, based on the research base, it is apparent that adult ADHD is an area of concern, and the potential for occupational and academic difficulties are prominent.

Because adults with ADHD are more likely to suffer from deficits of inattention, research in the past may have neglected adult ADHD as a serious disorder. That is, because deficits of inattention are less overt relative to hyperactivity and impulsivity, the symptoms present in adulthood are more likely to go unnoticed. However, even in the absence of overt behavioral problems (i.e., hyperactivity, impulsivity), inattentiveness can be significantly impairing for individuals with ADHD. As found by Biederman, Faraone, and Kiely (1996), adults with ADHD have an increased risk of academic difficulties. More specifically, their research indicated that adults with ADHD had significantly lower vocabulary, block design, digit symbol, arithmetic, and reading scores. Because inattentiveness is associated with ADHD, Thompson and Thompson (2005) suggested that adults with ADHD will more frequently become frustrated at work, lose things easily, underachieve, as well as experience other impairments in occupational functioning (e.g., change jobs frequently).

Thompson and Thompson (2005) also reported that some individuals with ADHD are extremely creative, suggesting that it would be inappropriate to simply conceptualize

individuals with ADHD as being cognitively impaired relative to non-ADHD individuals. That is, rather than describing individuals with ADHD as exhibiting cognitive *deficits*, they perhaps should be described as exhibiting cognitive *differences*. With this in mind, the overall goal of the current study was to consider the notion that symptoms of ADHD may be associated with cognitive differences that, under certain circumstances, may be an advantage rather than a disadvantage. Although there are a variety of symptoms that are present and influencing those with ADHD in their daily functioning, it is of particular interest to look at the deficit of selective attention in adults. As stated previously, selective attention is the ability to ignore distracting or irrelevant information in the environment while responding quickly and accurately to relevant information that is also being provided (Amso & Johnson, 2005; Hooks, Milich, & Lorch, 1994). Thus, the specific goal of the current study was to determine whether a deficit in selective attention may actually be a cognitive difference that can be useful in certain situations. For example, if someone fails to ignore distractors due to a difference in selective attention processing and the distractors are actually correlated with the subsequent target, an enhancement in one's ability to detect the target may occur, thus resulting in a faster reaction time to the target. An additional goal of the current study was to provide physiological evidence of a difference in the way the two groups processed information. Therefore, the current study went beyond examining neurocognitive differences by providing electrophysiological evidence, through the use of an electroencephalogram (EEG), that there are differences between the high and low ADHD-associated symptoms groups in how the brain responds to auditory stimuli.

The idea that a failure to ignore distractors could actually be beneficial in certain situations comes from research showing that humans are naturally drawn to correlated information (Melara & Algom, 2003). This tendency, however, may be diminished for typically functioning individuals under certain conditions. For example, in a selective attention task where a distractor is correlated with a target, a typical individual's natural tendency to be drawn to correlated information may be diminished because of their ability to selectively attend and ignore the distracting stimuli. Conversely, this natural tendency may be augmented in individuals with ADHD in the same type of selective attention task due to their impaired ability to selectively attend. Because of this impairment, those with ADHD should actually attend to the distractors and, thus, benefit from the informative correlation. Therefore, under certain conditions, a deficit in selective attention may actually be useful to the individual. The current study aimed to better understand such conditions by examining the differences in auditory selective attention between adults with both high and low levels of ADHD-associated symptoms, as well as to provide neurophysiological evidence that differences in processing of auditory information exist.

CHAPTER II

REVIEW OF RELATED LITERATURE

Genetic Factors

Consideration of potential risk factors for and predictors of ADHD is an important first step in understanding the underlying basis of ADHD. Given that the focus of the current study was on neurocognitive and electrophysiological functioning as it relates to symptoms of ADHD, it is imperative to consider the genetic predisposition for both subclinical symptoms of ADHD as well as the full onset of the disorder. Indeed, much evidence supports the theory that ADHD has a genetic component (e.g., Biederman et al., 1992; Biederman et al., 1995; Dunn and Kronenberger, 2003). According to the *DSM-IV-TR* (APA, 2000), ADHD is more common among first degree relatives of children with ADHD than in the general population. A study conducted by Biederman and colleagues (1992) suggested that children have an increased risk of developing ADHD if a parent or a sibling also has the disorder. Furthermore, the authors reported a reduction in glucose metabolism in the pre-motor and superior prefrontal cortex (areas that are suggested to be involved in control of attention and motor activity) of parents with children diagnosed with ADHD. This was true even in the cases when the parent was never diagnosed with ADHD. Biederman and colleagues (1995) conducted another study that suggested a genetic predisposition of the development of ADHD, based on results indicating that, among adults diagnosed with ADHD, 84% had at least one child diagnosed and 52% had two or more children diagnosed with the disorder. According to Dunn and Kronenberger (2003), the risk of immediate family members having ADHD ranges from 10 to 35%. This range increases to about 30% between siblings and 57% with parents. The highest

concordance was found in monozygotic (identical) twins, with approximately an 80% risk factor, further underscoring the genetic risk.

Another way of looking at the genetic expression of ADHD is through an evolutionary context. Hartmann (2003) proposed the idea that creativity, impulsiveness, and distractibility are characteristics of ADHD that can be considered highly adaptive and useful. According to Hartmann, the gene responsible for the development of ADHD is known as The Edison Gene, named after Thomas Edison who was thought to meet the criteria for ADHD, but was raised to use his deficits to his advantage. Hartmann proposed that the characteristics of an individual with ADHD were critical in our evolutionary past in terms of survival. In addition to these factors being helpful during hunter-gatherer times, it was proposed that these abilities will be necessary in the future as new challenges emerge within our society. More specifically, Hartmann stated that individuals with the Edison Gene have a genetic predisposition to be superior inventors, entrepreneurs, and explorers.

Shelley-Tremblay and Rosen (1996) also suggested that ADHD has an adaptive genetic trait that has been selected by the environment for survival. According to Shelley-Tremblay and Rosen, and in conjunction with Hartmann (2003), it has been suggested that inattentiveness, impulsiveness, and aggression were useful in the past for hunters. With regard to the deficit in selective attention, it was postulated that a hunter with this trait would constantly observe the environment around him or her and, thus, become more rapid at locating a sudden noise or flash of light indicative of his or her prey. Therefore, hunters would be *more* successful when they do *not* selectively attend to their

environment. Rather, a broader attention span of the entire environment would be deemed more beneficial (Shelley-Tremblay & Rosen, 1996).

Although the hunter theory proposed by Shelley-Tremblay and Rosen (1996) is not an empirical study, research by van Mourik, Oosterlaan, Heslenfeld, Konig, and Sergeant (2007) does offer empirical evidence in support of advantages to having a deficit in selective attention. In their study, the researchers examined the performance of children with and without ADHD on a visual two-choice reaction time task. Twenty-five children diagnosed with ADHD and eighteen control children between the ages of 8 and 12 completed a visual two-choice task that involved an irrelevant sound preceding a visual stimulus. The visual stimulus was a picture of a runner; the runner was either oriented left or right, and the children were asked to indicate which direction the runner was facing via a button press. The children were asked to ignore the irrelevant sounds, which were either a 600 Hz tone or a novel sound. The novel sounds included stimuli such as a dog barking or a bell ringing. Results showed that, when novel sounds preceded the visual stimuli, the ADHD group experienced a significant reduction in errors of omission. Also, in terms of electrophysiological differences, it was found that the ADHD group exhibited a larger P3a in response to both the standard and novel stimuli. Additionally, the ADHD group had a larger late phase P3a, which is only present when the novel stimuli are present. The authors concluded that children with ADHD had more difficulty ignoring the novel stimuli relative to the control group; however, this distracting information appeared to enhance their performance temporarily. Specifically, it was thought that the novel stimuli served to reorient the children with ADHD to the task at hand and to increase their arousal to an optimal level.

Overall, there appears to be a preponderance of evidence in support of a genetic predisposition for the development of ADHD, which is accepted among the majority of researchers in this area of study. In addition, there are evolutionary theories that consider ADHD to be an inherited and potentially useful condition under certain circumstances. As such, it is important to consider the way in which these genetic predispositions and inherited traits would manifest themselves in individuals prone to the development of ADHD symptoms. For this, it is imperative to look at neurological abnormalities associated with symptoms of ADHD.

Neurological Abnormalities

Of the studies mentioned in support of a genetic predisposition of ADHD, many appear to have additional implications for neurological abnormalities in individuals with ADHD. Neurological abnormalities may include differences in brain development or activation. Functional magnetic resonance imaging (fMRI), EEGs, and magnetic resonance imaging (MRI) are all important means for distinguishing potential abnormalities in individuals with ADHD symptoms. Thus, a review of the results from studies using these various data collection techniques is considered.

Casey and colleagues (1997) examined the presence of neurological abnormalities related to ADHD. Specifically, the researchers looked at the relation between the prefrontal cortex and the basal ganglia and deficits in response inhibition shown in participants with a diagnosis of ADHD. With the use of MRI, anatomical images of the frontostriatal area of the brain were examined. The results of three separate response inhibition tasks indicated that participants with ADHD performed significantly worse than that of control participants. Results of the MRI suggested that the right prefrontal

cortex plays an important role in suppressing responses to salient stimuli, whereas the basal ganglia is involved in the execution of these behavioral responses.

More recently, Rubia, Smith, Brammer, and Taylor (2007) investigated the differences in temporo-parietal neural networks in boys with ADHD and a matched control group. The boys completed an oddball task, which is a standard task measuring selective attention, while their brain activation was measured through event-related fMRI. The results indicated that the boys with ADHD showed significantly reduced levels of brain activation when comparing the oddball to the standard. Specific areas where reduced brain activation occurred were in the left and right superior temporal lobes, the basal ganglia, and the posterior cingulate.

Other studies have suggested measurably smaller frontal lobes as a neurological abnormality present in ADHD (Mostofsky, Cooper & Kates, 2002; Shue & Douglas, 1992). Specifically, it was demonstrated that ADHD symptoms are related to a decrease in grey and white matter volume located in the frontal lobe (Mostofsky et al., 2002). MRI studies also suggest that the deficits in ADHD are associated with frontal lobe functioning. Neurological studies have also shown that typically-developing children have slightly larger right frontal lobes than left and that children with ADHD lack this asymmetry; that is, children with ADHD have left and right frontal lobes that are the same size, whereas typically-developing children have a right frontal lobe that is larger than their left (e.g., Hynd, Semrud-Clikeman, Lorys, Novey, & Eliopulos, 1990), underscoring another potential neurological difference associated with ADHD.

Dunn and Kronenberger (2003) noted greater activation of the right frontal cortex in control participants relative to those diagnosed with ADHD in a task involving motor

responses. By use of fMRI, the inferior prefrontal cortex and the caudate nucleus were shown to be specific areas of activation. Casey and colleagues (1997) stated that a deficit in the right prefrontal cortex of children with ADHD may affect their ability to suppress responses to stimuli that are irrelevant in a selective attention task.

As was the interest of the current study, there are various research studies concerning neurological deficits in auditory selective attention with the use of electrophysiological measurement of an event-related potential (ERP) from an EEG. Satterfield, Schell, Nicholas, and Backs (1988) recorded ERPs from 19 electrode sites for six-year-old boys with and without a diagnosis of ADHD. They hypothesized that N2 amplitudes would be abnormally small in children with ADHD. N2 amplitudes are suggested to be related to mismatch negativity (MMN), which is an automatic change-detection response in the brain related to the auditory system. Many researchers have suggested that MMN is elicited by novel events or unattended stimuli (Cheour, Leppanen, & Kraus, 2000; Satterfield et al., 1988). The authors also predicted that the negative difference (Nd) amplitude would be significantly smaller for the boys with ADHD. The Nd amplitude is linked to the processing of the attended stimuli channel. Results indicated that the N2 and Nd amplitudes were both significantly smaller in boys with a diagnosis of ADHD. Implications from the Satterfield and colleagues' study suggest that boys with ADHD have poorer discrimination and poorer processing abilities (i.e., a deficit in selective attention). Lastly, the brain inactivity was directly associated with impairments in the frontal lobe.

In a study conducted by White, Hutchens, and Lubar (2005) adults with and without ADHD were administered the Paced Auditory Serial Addition Test (PASAT),

Wisconsin Card Sorting Test: Computerized Version 3 (WCST), and the Integrated Visual and Auditory Continuous Performance Test (IVA). Throughout testing, brain activity was measured through use of EEGs in order to examine differences in cortical activity during the tasks. Results suggested that adults with ADHD performed at a significantly lower level on tasks involving working memory, processing speed, and sustained attention. Results from the EEG suggested that the presence of an increased theta/beta ratio when administered the IVA was related to poorer attentional abilities. Overall, the results of these studies and others indicate marked physical evidence for neurological abnormalities associated with symptom severity of ADHD. Therefore, it is important to determine the specific neurocognitive and behavioral deficits present as a result of these abnormalities.

Neurocognitive Factors

As reviewed thus far, the literature suggests that there is likely a genetic predisposition associated with the development of ADHD and that neurological abnormalities are present in individuals with ADHD. It is important, therefore, to next consider specific neurocognitive and behavioral deficits associated with ADHD that may result from these genetic and neurological underpinnings. Understanding the neurocognitive correlates of ADHD may be beneficial in advancing the assessment and treatment of ADHD. For example, Thompson and Thompson (2005) suggest that adults with ADHD will commonly suffer from deficits in executive functioning including attention, planning, and inhibition. This information has been used in conjunction with the knowledge of the specific neurological deficits associated with these neurocognitive deficits to establish various treatments using EEGs. Specifically, biofeedback is

suggested as a useful tool to minimize the neurocognitive deficits present in adults. Numerous other researchers suggest that executive functioning, sustained attention, and selective attention are deficits associated with ADHD (e.g., Hooks et al., 1994; Jonkman et al., 1997; Riccio et al., 2004). Sustained attention is one's ability to focus on the task at hand (e.g., reading a book) long enough to complete the task. Selective attention, again, is the ability to ignore distracting stimuli while sustaining attention to the relevant task. Selective attention was the specific focus of the current study because of its theoretical link to evolutionary advantage (Shelley-Tremblay & Rosen, 1996). That is, selective attention appears to be a neurocognitive ability traditionally found to be deficient in individuals with ADHD that best yields itself to test the theory of whether deficits may actually be advantageous under certain circumstances.

Auditory Selective Attention

Because ADHD is a disorder most commonly diagnosed among, and subsequently researched in, children, limited research has been conducted in terms of auditory selective attention in adults. However, research on auditory selective attention that is specific to children may still have implications for adults with ADHD. Satterfield and colleagues (1988) conducted a study of selective attention in six-year-old boys with and without ADHD. The participants (20 in each group) were asked to perform a selective attention task which included both auditory and visual selective attention. The participants were asked to attend to either the auditory or visual stimuli while inhibiting attention to the opposite stimuli. The auditory task used clicks, whereas the visual task used flashes of light. Participants were asked to respond by hitting a button on a hand held control whenever the target stimulus was present. Responses were only considered

correct if they were made before 1.5 seconds after the stimulus was presented. Results suggested that boys with ADHD performed inferior in terms of discrimination and processing of the target stimuli. The researchers considered behavioral data, which indicated that the participants with ADHD had significantly more errors of commission (i.e., responding to a non-target) relative to the control group for the auditory stimuli. The findings by Satterfield and colleagues appear to support the notion that there is a deficit in auditory selective attention for those with ADHD. According to Cheour and colleagues (2000), the deficit in auditory selective attention can be attributed to difficulties of individuals with ADHD to discriminate salient stimuli.

Jonkman and colleagues (1997) were also interested in investigating auditory selective attention in children with and without ADHD. In their study, a total of 300 auditory stimuli were presented to the participants in either their left or right ear. Each participant was asked to complete two auditory tasks in which a target or distractor channel was determined by which ear (left or right) they were using to attend. The results indicated that participants with ADHD performed worse in terms of errors of omission (i.e., fewer correct responses to target stimuli) and false alarms (i.e., responding to non-target stimuli) relative to the control group. Results from these reviewed studies further help to underscore the notion that there is a deficit in auditory selective attention related to ADHD.

EEGs in Measuring Selective Attention Outside ADHD

Gomes, Barrett, Duff, Barnhardt, and Ritter (2008) indicated that EEGs are useful in obtaining event related potential (ERP) information about both temporal and spatial dynamics of the brain throughout task performance. It has been further suggested by

Luck (2005) that obtaining information with regard to ignored stimuli is particularly difficult for two reasons. The first reason is that it is difficult to assess whether an individual is processing an ignored stimulus without asking them about it, in which case the stimulus is no longer ignored. Secondly, it is difficult to make sense of slower and less accurate responses to ignored stimuli. To aid in solving this problem, Luck suggested that it is beneficial to use EEGs to record brain activity relevant to the ignored stimuli. In a study conducted by Gomes, Duff, Barnhardt, Barrett, and Ritter (2007), it was suggested that the Nd onset and peak latencies occur longer in typical children performing an auditory selective attention task relative to typical adults. In addition, it was found that both children and adults' P3 amplitude was an indicator of attention to targets, but that hits, reaction times, and false alarms improved as a function of age. The authors concluded, through use of ERPs, that auditory selective attention can be explained by improvement in the speed and efficiency of attention allocation as an individual ages. When considering the study by Gomes and colleagues in terms of adults with ADHD, it appears that a developmental lag, which is also supported by Butchner (2002), in the improvement toward this efficiency may account for the deficits in selective attention that are present among individuals with ADHD.

Rao (1998) conducted a study using EEGs to measure neurological functioning during auditory selective attention tasks in adults. Results indicated that a deficit in auditory selective attention in adults is associated with smaller Mismatch Negativity (MMN), which indicates reduced ability to discriminate stimuli. According to Luck (2005), MMN occurs when an individual is exposed to the same stimulus repeatedly with an occasional deviation (e.g., many 600 Hz tones with an occasional 800 Hz tone). The

deviation tends to elicit a negative wave that peaks between 160 and 220 ms. Results found by Rao (1998) indicated that this difficulty with discrimination can be improved through training. It was suggested that auditory selective attention in adults is a plastic ability, meaning that it can be altered and improved through practice (Rao, 1998).

Another interesting study conducted by Sumich and colleagues (2007) examined N100 and P3 amplitudes in siblings discordant for schizophrenia. In their study, an auditory oddball task, which is a common task used to measure selective attention, was administered to adults diagnosed with schizophrenia, their siblings, and a control group. Through use of a mixed-model repeated measures analysis of variance (ANOVA), with diagnosis (i.e. patient, sibling, control) as the between-subjects variable, and task (i.e., go vs. no go), site (i.e., electrodes divided into frontal, central, or parietal scalp locations), and hemisphere (i.e., electrodes divided into either left, right, or midline scalp locations) as within-subjects variables, the authors came to several conclusions on the ERP data collected. It was found that patients diagnosed with schizophrenia had a smaller P3 relative to controls and reduced parietal amplitude relative to siblings, regardless of the task. N100 amplitudes in the frontocentral region were larger for siblings compared to controls. Also, it was found that N100 and P3 amplitudes were positively correlated with anxiety features in schizophrenia. N100 amplitudes were larger across diagnosis for frontal and central scalp locations whereas P3 amplitudes were larger across groups for parietal lobe locations.

Studies have also been conducted on auditory selective attention in children from a typical population. Bartgis, Lilly, and Thomas (2008) conducted a study on differences in auditory selective attention functioning among typically-developing 5, 7, and 9 year

olds. ERPs related to an auditory selective attention task were recorded from frontal, central, and parietal sites. Results suggested that auditory selective attention develops with age. Specifically, 9 year olds showed a greater Nd, processing negativity, to the attend channel relative to the 5 and 7 year olds. In addition, it was found that 7 and 9 year olds showed significantly larger P3 amplitudes during the attend channel relative to the ignore channel.

Overall, it appears that there is a deficit in auditory selective attention that is associated with symptoms of ADHD. The information provided in terms of selective attention and the use of EEGs is beneficial in determining areas of the brain that are specific to selective attention and prone to neurological deficits in those with ADHD. Based on the extant research, it appears that frontal, central and parietal lobe functioning are target areas when researching neurological components of auditory selective attention. In addition, it appears that specific ERP measurements of the P3, N1, N2 amplitudes in addition to the Nd, processing negativity, are key measurements in this area of research. Tannock (1998) suggested that deficits in selective attention in those with ADHD are related to abnormal functioning of the frontal circuit. Frontal lobe deficits have been deemed the cause of some ADHD symptoms by other researchers as well.

For example, research by Shue and Douglas (1992) examined 24 children with and without ADHD on motor control and problem solving abilities, both of which measure deficits in frontal lobe functioning. In addition, tests measuring temporal lobe functioning were administered. Results suggested that deficits in ADHD were specific to frontal lobe functioning and not temporal lobe functioning. Based on the study by Shue and Douglas, as well as evidence from other researchers (e.g., Aman et al., 1998; Barry,

Klinger, Lyman, Bush, & Hawkins, 2001), it appears that ADHD is related to a deficit specific to frontal lobe functioning, but not parietal lobe functioning, which had been associated with ADHD in the past. Both neurological and neurocognitive studies have provided information supporting the expectation that a deficit in auditory selective attention would be present in ADHD because of a deficit in frontal lobe functioning. Because of the minimal research on auditory selective attention in adults, the current study examined frontal lobe functioning on an auditory selective attention task with adults. Furthermore, the current study was the first known study to relate adults' performance on such a task to symptoms of ADHD.

Examining such performance in adults appears relevant given that previous research conducted by Prentkowski (2008) suggests that children (between the ages of 6 and 11) with ADHD are faster at responding correctly to target stimuli in both an auditory and visual selective attention task when compared to a control group of children. This superior performance is not solely explained by impulsive and inaccurate responding. Rather, results indicated that when children with ADHD were successful at choosing the correct response, they continued to perform faster than those without ADHD. Furthermore, when errors of commission were made, children with ADHD did not respond significantly faster than children without ADHD when performing auditory correlated, visual filtering, and visual correlated tasks. Children in the ADHD group were significantly faster relative to children without ADHD on errors of commission only for the auditory filtering tasks. Taken together, these findings suggest that *auditory correlated* information may have *benefited* children with ADHD in that they were able to better inhibit incorrect, impulsive responding (i.e., their reaction time on errors of

commission were congruent with that of the children without ADHD), while simultaneously being able to accurately recognize target sounds and respond at a speed significantly faster than children without ADHD.

The current study built upon the study conducted by Prentkowski (2008) by looking at ADHD symptoms in adults. Studying adult participants, as opposed to children, should improve the percentage of accurate responses to auditory target stimuli. Importantly, in the previous study by Prentkowski (2008), it was evident that the majority of participants did not score at an accuracy level above 50%. Such a low accuracy rate limited the amount of available reaction time data that was usable and may have contributed variability in the data that made it difficult to determine patterns of differences between groups. Furthermore, the low accuracy rate found by Prentkowski (2008) indicates that the task was extremely difficult for children and perhaps some of the participants did not fully comprehend or learn the task. As such, in the current study, frequency of tone presentation was adjusted to allow for easier discrimination among targets. Use of easier discrimination among tones, as well as inclusion of only adult participants, in the current study led to much more accurate responding than in the Prentkowski (2008) study. Finally, the current expands on Prentkowski (2008) by examining both neurocognitive performance and neurological abnormalities through the use of an EEG.

CHAPTER III

CURRENT STUDY

A main goal of the current study was to determine whether a deficit in auditory selective attention could possibly be beneficial for adults with high levels of ADHD-associated symptoms under certain conditions. Evolutionary theories that consider characteristics of ADHD adaptive in certain conditions support this goal (Hartmann, 2003; Shelley-Tremblay & Rosen, 1996). Previous research suggested that individuals with ADHD have considerable difficulty ignoring distracting stimuli, which is thought to be the result of neurocognitive impairments in selective attention. Research in support of this notion is specific to auditory selective attention, suggesting that an individual with ADHD-associated symptoms, particularly inattention, may be especially successful in extracting and using information from distractor stimuli when the distractors are predictive of the target, or task-relevant, stimuli. This, again, is particularly true for auditory selective attention paradigms. According to Melara and Algom (2003), it appears that humans are drawn to correlated information. Thus, the more correlated the information becomes; the more apt the observer is to integrate that information. It is, therefore, thought that this innate tendency may be amplified to an even greater extent in individuals with ADHD due to their overall deficit in the ability to selectively attend. Thus, adults with high levels of ADHD-associated symptoms may be cognitively aware of correlations between information that adults with no or low levels of ADHD symptoms may fail to notice because they are successfully selectively attending.

In addition to these goals, it was also the aim of the current study to determine whether any electrophysiological differences existed between groups when processing

the auditory information presented in both the high and low probability conditions. Differences between the high and low ADHD-associated symptoms groups were expected given the findings of previous research. For example, Satterfield and colleagues (1988) found group differences in electrophysiological data when comparing 6-year-old boys with and without ADHD. More recently, White and colleagues (2005) found differences in EEG data between adults with and without ADHD. These findings, along with information from Luck (2005) suggesting that N1 amplitudes are sensitive to attention, and findings from Bonala, Boutros, and Jansen (2008) suggesting that P3 amplitudes are affected by the order in which target and distractor tones are administered, provide evidence in support of group differences in electrophysiological data for the current study.

Because research suggests that neurocognitive deficits in selective attention of individuals with ADHD are present specifically in auditory selective attention, but may not be as distinct in visual selective attention (Barry et al., 2001; Heaton et al., 2001; Manly et al., 2001), the current study focused solely on auditory selective attention. The auditory selective attention task included the presentation of target stimuli and distractor stimuli, biaurally through headphones. Half of the trials included targets and distractors that were uncorrelated (i.e., low probability task), and the other half of the trials included targets and distractors that were correlated (i.e., high probability task). Currently, there is a paucity of research on auditory selective attention in ADHD among children; moreover, this is the first known study investigating the difference between level of ADHD-associated symptoms with auditory selective attention in adults. Therefore, the current study further contributes to the understanding of this process in adults with varying levels

of ADHD symptoms, including those who have been diagnosed with ADHD. Participants with high and low levels ADHD-associated symptoms were used as opposed to dichotomous groups of ADHD-diagnosed adults and adults not diagnosed with ADHD. This method of grouping was important for a few reasons. The first is that measuring current symptoms of ADHD eliminated the chance that an individual with an ADHD diagnosis who did not currently demonstrate high levels of ADHD (e.g., they were misdiagnosed or were diagnosed as a child and no longer met diagnostic criteria) would be eligible to participate. Likewise, measuring symptoms ensured that an individual, who demonstrated high levels of ADHD according to self-report, was not placed in the control group or eliminated from the study due to a lack of a formal diagnosis. Also, examining a community sample, which did include many participants with a formal diagnosis of ADHD, allows for more general implications. For instance, the results of the current study can be generalized to individuals who struggle with deficits in selective attention, but who do not currently have a diagnosis of ADHD, or who may have subclinical levels of impairment. Therefore, the current study wished to examine the possibility that auditory selective attention processes of adults with high and low levels of ADHD-associated symptoms would be better conceptualized as a difference in processing style, rather than a deficit, given that ADHD symptoms could actually lead to a benefit in performance under certain conditions. It is particularly relevant to examine this question in adults because adults with ADHD are more likely to exhibit inattentive symptoms, including selective attention deficits, relative to hyperactive-impulsive symptoms.

Hypotheses

Based on previous research, it was hypothesized that adults with high levels of ADHD-associated symptoms would experience greater interference (i.e., slower reaction time) relative to the low ADHD-associated symptoms group on the low probability task because they would not be successful at inhibiting true distracting stimuli. Conversely, it was expected that adults with high levels of ADHD-associated symptoms of ADHD would experience greater performance gain (i.e., faster reaction time) relative to the low ADHD-associated symptoms group on the high probability tasks because of their enhanced ability to notice a distractor-target correlation, which would allow them to learn that the non-ignored distractors predict the subsequent targets. Differences associated with levels of ADHD symptoms in response *accuracy* were not expected on any of the tasks presented because it was expected that accuracy would be high for all participants overall.

In addition to the hypotheses based on the neurocognitive performance data, it was expected that ERPs from the EEG would differ between participants with high and low levels of ADHD-associated symptoms. It was hypothesized that a deficit would be found in frontal lobe functioning for individuals performing the auditory selective attention task who report high levels of ADHD-associated symptoms. More specifically, and based on previous research, it was expected that participants with high levels of ADHD symptoms would show greater N1 (given that N1 wave is sensitive to attention) amplitude relative to the low ADHD-associated symptoms group during the presentation of distracting stimuli in both the low and high probability tasks because of the high ADHD-associated symptom groups' difficulty in ignoring distracting stimuli. In the low

probability condition, the distractors are actually distracting and non-informative; therefore, the larger N1 would correspond to worse performance (suggesting that they are not selectively filtering out the distractors). However, in the high probability condition, the distractors are actually informative; therefore, the larger N1 would correspond to better performance (suggesting they are attending to the beneficial distractors). It was also predicted that, within the high ADHD-associated symptoms group only, participants would show greater N1 amplitudes associated with distracting stimuli in the high probability task relative to the size of their N1 amplitudes in the low probability task, corresponding to better performance in the high probability condition. That is, the size of the N1 amplitude was always expected to be larger for the high ADHD-associated symptoms group relative to the low ADHD-associated symptoms group, with the size also increasing in the high probability condition for the high ADHD-associated symptoms group due to their ability to become cognitively aware of the correlated stimuli. No differences between the high and low ADHD-associated symptoms group was expected for N1 amplitudes associated with target stimuli.

In addition, it was expected that participants with high levels of ADHD-associated symptoms would show larger P3 amplitudes for target tones relative to the low ADHD-associated symptoms group (given that the P3 component is elicited in frontal lobe when task-irrelevant stimuli are present along with task-relevant stimuli). Such a finding would reflect the greater ability for individuals with high levels of inattentiveness to pick up correlational information between targets and distractors as a consequence of worse selective attention that leads them to also attend to distractors. Gomes and colleagues (2008) report that the P3 component is sensitive to task difficulty, with the P3 amplitude

being larger for targets that are easier to detect. In addition to this, Bonala and colleagues (2008) reported that P3 amplitudes are affected by the order in which the target and distractor stimuli are presented, such that P3 amplitudes tend to be larger when the target is preceded by a distractor rather than another target. Thus, it was expected that the P3 amplitudes for participants with high ADHD-associated symptoms would be larger for target tones in the high probability tasks relative to the low probability tasks due to the expected use of predictive distractors augmenting their processing of the target stimuli.

In summary, for the *neurocognitive* data, it was hypothesized that (1) a *two-way interaction between group and condition* would occur for the reaction time data such that: (a) the high ADHD-associated symptoms group would be slower in reaction time than the low ADHD-associated symptoms group in the low probability condition; (b) the high ADHD-associated symptoms group would be faster in reaction time than the low ADHD-associated symptoms group in the high probability condition; (c) only the high ADHD-associated symptoms group would benefit (in terms of faster reaction time) from the high probability condition. For the *electrophysiological* data, it was hypothesized that: (2) a *two-way interaction between group and tone* would occur such that the high ADHD-associated symptoms group would demonstrate larger N1 amplitudes, compared to the low ADHD-associated symptoms group, in response to distractors overall; (3) a *three-way interaction between group, condition, and tone* would occur such that only the high ADHD-associated symptoms group would demonstrate larger N1 amplitudes in response to distractors in the high probability condition, relative to the low probability condition; (4) a *three-way interaction between group, site, and tone* would occur such that the high ADHD-associated symptoms group would demonstrate larger N1 amplitudes specifically

within the frontal lobe, compared to the low ADHD-associated symptoms group, in response to distractors overall; (5) a *two-way interaction between group and tone* would occur such that the high ADHD-associated symptoms group would demonstrate larger P3 amplitudes, compared to the low ADHD-associated symptoms group, in response to targets overall; (6) a *three-way interaction between group, condition, and tone* would occur such that only the high ADHD-associated symptoms group would demonstrate larger P3 amplitudes in response to targets in the high probability condition, relative to the low probability condition; (7) a *three-way interaction between group, site, and tone* would occur such that the high ADHD-associated symptoms group would demonstrate larger P3 amplitudes specifically within the frontal lobe, compared to the low ADHD-associated symptoms group, in response to targets overall.

CHAPTER IV

METHOD

Participants

A total of forty participants took part in the current study. Participants ranged from 18 to 59 years of age (M = 24.02, SD = 9.21). A total of 13 participants were male and 27 were female. Participants were recruited for one of two groups: 20 participants were in the high ADHD-associated symptoms group, and 20 participants were in the low ADHD-associated symptoms group. Participants who had been previously diagnosed with ADHD by an independent practitioner (n = 15) and/or exhibited a clinically significant number of symptoms of ADHD (n = 5) on the Barkley's ADHD Checklist were recruited for the high ADHD-associated symptoms group (6 males and 14 females). Participants in the high ADHD-associated symptoms group who were taking medication for their ADHD were asked to refrain from taking the dose of medication prior to their scheduled session. Thirteen participants in this group were taking some form of stimulant medication (e.g., Ritalin, Adderall). Additionally, two participants in this group reported a comorbid diagnosis of a learning disorder, along with one participant who reported having taken special education classes in the past.

Participants with no previous diagnosis of ADHD and scoring below the clinical range on the Barkley's ADHD Checklist were recruited for the low ADHD-associated symptoms group (7 males and 13 females). None of the participants in this group reported a diagnosis of a learning disorder or special education classes. For both groups, none of the participants had a hearing or visual impairment that was not corrected.

Differences between the groups on ADHD symptomatology are discussed in the Results section below.

Participants in the high ADHD-associated symptoms group were 90% Caucasian, 5% African American, and 5% Asian. In the low ADHD-associated symptoms group, participants were 30% Caucasian, 65% African American, and 5% Asian. Family income was coded on a classification system from 0 to 12, with 0 indicating no income and 12 representing an income equal to or higher than $100,000. The average classification for the total sample was between 4 and 5 (M = 4.59, SD = *4.29*), representing an average income of $20,000 to $29,999. See Tables 1 and 2 for these results, as well as comparisons on other demographic variables.

Materials

Participants were asked to complete an auditory selective attention task which was created through STIM Auditory/ Visual Stimulus Presentation and Laboratory Control System (STIM). Within this task, there were three targets and two distractors, each differing in frequency. In addition, ERP data were collected via EEG. The selective attention task and ERP recordings are described in the Procedures section below. Other measures administered include the Barkley's ADHD Checklist (Barkley & Murphy, 1998), the Wechsler Abbreviate Scale of Intelligence (WASI), which is a brief and reliable measure of intelligence (Wechsler, 1999), the Alcohol Use Disorder Identification Test (AUDIT), which is an instrument used to screen for hazardous or harmful levels of drinking (Babor, Higgins-Biddle, Saunders, & Montiero, 2001), and a demographics and background information form. The WASI and AUDIT were administered to obtain a measure of intelligence, which could be a confounding variable

in the neurocognitive data, and a measure of alcohol use, which could be a confounding variable in the ERP data, so that these constructs could be statistically controlled as needed.

Barkley's ADHD Checklist (Barkley & Murphy, 1998)

Barkley's ADHD Checklist is a self-report measure of symptoms of ADHD. The checklist is an 18-item Likert scale, ranging from 0 to 3 (i.e., never or rarely, sometimes, often, and very often), with items assessing attention problems and hyperactivity-impulsivity. Each item directly maps onto the diagnostic criteria for ADHD listed in the *DSM-IV-TR* (APA, 2000). The scale contains nine items assessing inattention and nine items assessing hyperactivity-impulsivity. Consistent with *DSM-IV-TR* (APA, 2000) criteria, endorsement of either six of nine inattention symptoms or six of nine hyperactivity-impulsivity symptoms is considered clinically significant on the Barkley's ADHD Checklist.

After the respondent completed the checklist, the researcher counted the number of inattention symptoms (all odd-numbered items) that had been rated either 2 (often) or 3 (very often). The researcher then counted all of the hyperactivity-impulsivity symptoms (all even-numbered items) that had been rated either 2 or 3. A score of six or more on either the inattention scale or the hyperactivity-impulsivity scale indicated clinical significance. Therefore, those participants with a score of six or higher on the inattentiveness domain, the hyperactivity-impulsivity domain, or both were included in the high ADHD-associated symptoms group, whereas those participants with less than six symptoms on both domains were included in the low ADHD-associated symptoms group. These group assignment decisions were based on *DSM-IV-TR* criteria (APA, 2000),

which indicate that an individual must have at least six of the nine symptoms of inattentiveness to be diagnosed with ADHD, Predominantly Inattentive Type, six of nine symptoms of hyperactivity-impulsivity to be diagnosed with ADHD, Predominantly Hyperactive-Impulsive Type, or six of the nine symptoms in both domains to be diagnosed with ADHD, Combined Type. Barkley's ADHD Checklist was used as an indicator of level of ADHD symptoms for each group. These reports were used to divide the participants into two groups (i.e., high and low ADHD-associated symptoms).

Interobserver agreements on the Barkley's ADHD Checklist have ranged from .77 to .85. In addition, the interrater reliability phi coefficient has ranged from .52 to .95 (Pelham, Fabiano, & Massetti, 2005). In the current sample, internal consistency for both domains was excellent, with a Cronbach's alpha coefficient of .94 for inattention and .91 for hyperactivity-impulsivity.

Wechsler Abbreviated Scale of Intelligence (WASI; Wechsler,1999)

The WASI is an individually administered IQ test that has been normed for use with individuals ranging in age from 6 to 89 years. The WASI is a quick way to obtain an estimate of an individual's IQ, with a total administration duration of approximately 30 minutes, and one of its stated purposes is for use in research. The two-subtest version (Vocabulary and Matrix Reasoning) of the WASI was used, yielding an estimate of Full Scale IQ (FSIQ). This measure was included in the current study to determine differences between groups in FSIQ, as this may be a confounding variable in the interpretation of neurocognitive data.

The reliability of the WASI obtained from an adult sample is consistent with that of the WAIS-III. At the subtest level, the reliability is .90 to .98 for Vocabulary and .88 to

.96 for Matrix Reasoning. Reliability for the FSIQ is .96 to .98. Correlations between the WASI and WAIS-III are .88 for Vocabulary, .66 for Matrix Reasoning, and .92 for the FSIQ, showing adequate validity (Wechsler, 1999).

Alcohol Use Disorders Identification Test (AUDIT; Babor et al., 2001)

This screening tool is sensitive to the early detection of high risk drinking. It was used in the current study to determine differences between groups in alcohol use, as this may be a confounding variable in the interpretation of the ERP recordings. Specifically, research suggests that those individuals considered to be alcoholics and those with at-risk levels of alcoholism tend to have a smaller P3 amplitude when measured in ERP studies (Porjesz et al., 2005). Therefore, without controlling for level of alcohol consumption if group differences were to be found in consumption, interpretation of the difference between groups on P3 amplitude would not be meaningful. Internal consistency reliability on the AUDIT was found to be .76 for college students in a study conducted by Conley and O'Hare (2006). In the current sample, internal consistency for the AUDIT was good, with a Cronbach's alpha coefficient of .83.

Demographics and Background Information Form

A demographic form created for the current study included the participants' age, gender, race, and socioeconomic status (SES). Along with this demographic information, questions regarding general cognitive and academic ability, learning disabilities, visual and hearing impairments, illicit drug use, use of nicotine, and details about ADHD and other behavioral disorder diagnoses (if applicable) were included (see Appendix A).

Procedure

Recruitment

College students attending The University of Southern Mississippi were recruited through Experimetrix, a web-based computer system that is available to undergraduates. Time, location, and a brief description of what was expected of participants were listed in Experimetrix, and students were given the option to sign up to participate. Once participation was complete, research credit was assigned through Experimetrix by the experimenter, which translated to either research requirement points or extra credit points in their psychology courses, as specified by the individual instructor. Participants were given one Experimetrix credit for every thirty minutes of participation. Participants were also recruited to participate through Experimetrix by first completing a mass screener, which included the Barkley ADHD Checklist. The experimenter contacted potential participants via email, if they met criteria on the screener, to participate in the study. In addition, other adult participants were recruited through advertisements through the university and greater community. Participants recruited through means other than Experimetrix were entered into a drawing to win a $25 gift card to a local store. The total duration of this experiment lasted approximately 90 to 120 minutes per participant.

Screening

Participants with a current diagnosis of ADHD were asked what type, if any, of ADHD medication they were currently taking. Any participant taking a psychotropic medication other than a stimulant medication to treat ADHD was not able to participate. This exclusion criterion also applied to those individuals taking non-stimulant medication, such as Strattera (atomoxitine), for their treatment of ADHD. Non-stimulant

medications are known to take weeks to reach their full therapeutic effect. Thus, asking the participant to refrain from taking their non-stimulant medication may be detrimental to their functioning (Waxmonsky, 2005). On the other hand, participants currently taking stimulant medications [e.g., Ritalin (methylphenidate), Adderall (amphetamine)] for the treatment of ADHD were eligible to participate. In addition, participants were asked to refrain from alcohol consumption at least 24 hours prior to participating in the study. None of the participants reported a hearing impairment that was not corrected, history of seizures, or a traumatic brain injury.

In a study conducted by Knott and colleagues (2006) on the neural effects of nicotine during an auditory selective attention task, it was reported that, in general, nicotine does not have a significant effect on behavioral task performance of EEG measures specific to selective attention. In their study, participants, all of whom were smokers, were asked to refrain from smoking for a duration of eight to eleven hours prior to their scheduled experiment. Upon arrival, half of the participants were randomly given either a piece of nicotine gum or a placebo and were asked to complete an auditory selective attention task. Specifically, results showed that N1 amplitudes and P3 amplitudes were not affected by the use of nicotine during a selective attention task. It was discovered, however, that early Nd latencies were affected by the use of nicotine. Late Nd latencies, on the other hand, were not affected. Specific implications of the effect of the early Nd latencies indicate that nicotine is potentially enhancing one's ability to focus on the target in addition to enhancing ability to filter out distracting stimuli. Based on these findings, the use of nicotine was monitored through the demographics form, but nicotine use was not an exclusion criteria.

Prior to testing, participants recruited through the mass screener were contacted via email or telephone to determine whether they had a current diagnosis of ADHD and whether they were currently taking medication to help with their ADHD. These individuals were also asked whether they were taking any other medication to treat a mental health disorder other than ADHD, as individuals who responded yes to this were not eligible to participate (n = 3). This screening helped to determine if participants were eligible to participate given their current ADHD medication. Participants not taking any medication for the treatment of ADHD were eligible to participate. Participants taking a psychotropic medication, including non-stimulant medication (e.g., Strattera) for the treatment of ADHD, were not eligible to participate. However, individuals taking stimulant medications, such as Ritalin (methylphenidate), to treat ADHD were eligible. Each participant was asked to refrain from taking their afternoon does of their stimulant medication if scheduled in the late afternoon or early evening, or the morning does if scheduled early in the day or on a weekend. The effects of stimulant medications will dissipate after a duration of time ranging from 3 to 7 hours; for sustained release medication the range is from 5 to 8 hours (Bezchlibnyk-Butler & Jefferies, 1997). Thus, in order to maximize differences between the high ADHD-associated and low ADHD-associated symptoms groups, participants were scheduled when it was clear that their medication should not be yielding a therapeutic benefit.

Paper-and-pencil measures, IQ testing, and participant prepping for EEG

Participants meeting the initial screening requirements were asked to participate in the study. The date and time of the participation was confirmed. Appointments were scheduled throughout the day, however, the number of morning, afternoon, and early

evening appointments were matched between the high ADHD-associated and low ADHD-associated symptoms groups. This experiment was conducted in the Clinical Studies Lab at The University of Southern Mississippi (USM). Upon arrival to the lab, participants were asked to complete an informed consent. After obtaining informed consent, the experiment began. First, the participant was asked to complete the paper-and-pencil measures (i.e., ADHD checklist, AUDIT, demographics form). All of the participants obtained a score of less than 8 on the audit indicating a low risk of alcohol related problems. As mentioned previously, the Barkley ADHD Checklist is part of a screener used for other studies at USM and, therefore, was already available for some participants recruited through Experimetrix. In these cases, it was not re-administered. Likewise, the checklist was scored immediately to obtain the level of ADHD-associated symptoms. Participants were only asked to continue in the study if their scores were classified into a group (high or low ADHD-associated symptoms) for which data were still needed. One participant was excluded at this point because he had a diagnosis of ADHD, but did not meet the study criteria on the Barkley ADHD Checklist of current symptoms.

After meeting all criteria to continue in the study, the participants were administered the WASI. After completion of the WASI, the participant was fitted with an electrocap in which recordings were obtained from the frontal (F3, F4, Fz, F5, F6, F7, F8), central (C3, Cz, C4), parietal (P3, P4), and occipital (O1, O2) regions. After the size of the electrode cap was determined, each electrode site listed above was filled with electrode gel and the skin beneath the electrode was lightly brushed with a blunt needle in order to improve impedance at each site. According to lab standards, each electrode site

read at an impedance of less than 5k Ω. Additionally, the left mastoid electrode site was used as a reference site and read at less than 5k Ω on the impedance meter. The right mastoid electrode site was also prepped with an impedance reading of less than 5k Ωs and was used in conjunction with the right mastoid to create an average of both mastoids used as the final reference. According to Luck (2005), using the average of both mastoids as a reference aids in avoiding the left or right hemisphere bias often found when using just one reference site (i.e., using only the right mastoid will lead to an imbalance between active electrodes because that site is in the right hemisphere). Therefore, in the current study, the left and right mastoid sites were averaged together by using the formula a' = a- (r/2). In this equation, a' is the average of both sites, a is the original waveform for any site that might be referenced (e.g., Cz) with a reference to the right mastoid, and r is the original waveform for the left mastoid with the right mastoid as the reference. What this formula explains in regard to the active electrode sites is that an ERP waveform is never an electrical property of that site alone; rather, it is the difference between the active site and the averaged reference sites (Luck, 2005). Lastly, both vertical and horizontal electro-ocular grams (EOG) were measured (with the vertical electrodes being placed just above and below the left eye and the horizontal being placed on the outer canthi of the left and the right eye) in order to detect the occurrence of noise due to eye blinks. Impedance on the EOG sites was also determined and met the lab standards of less than 10kΩ. Participants were given opportunities to rest and move their eyes between trials and were asked to refrain from moving their eyes during testing to ensure that contamination of the data due to eye movement was minimized. Through the experimental trials, a fixation point was displayed on the screen, and the participant was

asked to focus on that point and refrain from eye movement other than regular blinking. Electrodes placed vertically and horizontally for the measurement of EGOs were placed on the face, with the examiner first abrading the skin, adding get to the electrode, placing the electrode in the appropriate spot on the face, and adding an adhesive collar around the electrode to hold it in place. After the experiment was completed, the researcher manually scored and eliminated artifacts that were presented through the EOG channels. Once all impedances were at appropriate levels, a 10 second calibration was performed prior to the administration of the experiment. The calibration before or immediately after each participant was important in that it eliminated differences in the gain settings for all of the electrode channels. The gains for this experiment were set at 2000; however, gains change slowly over time and may not be the same for each channel.

Auditory Selective Attention Task

After the participant was prepped and all necessary EEG equipment was connected, the researcher started the computer program presenting the auditory selective attention task and provided verbal instructions along with written instructions displayed on the computer monitor on how to complete the task. The participant was provided an opportunity to listen to the three target tones three times each in order to prepare for the task. Once the participant was ready to begin, he or she began the practice trial. The practice trial included 6 of each target tone and 5 high distractor tones. The practice trial included 1.5 second delays between the presentations of each tone. Throughout the practice trial, feedback on the participant's accuracy was displayed on the screen. The feedback included information such as "Correct" or "Sorry, that was the medium tone." Next, the participant was instructed to press the start button on the response box to begin

the task. The researcher monitored the testing room for the duration of the experiment through a video camera.

The experimental task included a total of six blocks of trials. Of these six blocks of trials, three blocks of trials were low probability and three were high probability. Again, there was a small likelihood that the target and distractor tones were correlated in the low probability condition, whereas there was a large likelihood that the target and distractor tones were correlated in the high probability condition. The blocks of trials alternated between low and high probability. In addition to these six blocks of trials, a practice trial was completed prior to beginning the experimental trials. Within in each block of trials, the participants were presented with three target (i.e., high = 800 Hz, medium = 500 Hz, low = 200 Hz) and two distractor (i.e., high = 600 Hz, low = 400 Hz) tones that differed in frequency. In addition to the tones differing in frequency, the target and distractor tones differed in timbre, or sound quality. Specifically, the target tones were sine waves and the distractor tones were square waves. Each tone had a 10 ms rise and 10 ms decay time. These times are necessary because, without the rise and decay, a noticeable click sound would be presented through the headphones. The maximum sound pressure quality through the experiment was 95 dB. This is considered the lowest position on the headphones that encompasses all the dBs and maximizes sound quality.

The low probability trials included two distractor tones and three target tones presented pseudo-randomly in that there were no more than two of the same distractor tones presented consecutively. The high probability trials included two distractor tones and three targets presented in a way such that some distractors were randomly assigned to precede, and thus predict, a specific target. That is, in each high probability condition a

different distractor tone was paired with a target tone and that distractor preceded the target every time the distractor was presented. For example, in one high probability condition, the high distractor tone (600 Hz) was correlated with the medium target tone (500 Hz); therefore, every time the 600 Hz tone was presented (which would be 15 times total for one condition), it was followed by the 500 Hz tone as would be the case for the 400 Hz distractor tone and the target with which it was paired.

Each tone was presented for 500 milliseconds (ms) with a 1500 ms delay between tones. The participants were allotted 1000 ms to respond to the tone before a response was considered incorrect (i.e., no response recorded). Therefore, from the onset of the tone, the participant had one second to respond, followed by a one second delay where no response could be recorded before the next tone was presented.

Within each block of trials, each distractor tone was presented 15 times and each target tone was presented 30 times per trial. Prior to beginning the first experimental trial, each participant had the opportunity to listen to the target stimuli. In the high probability condition, the two distractors were paired with two of the three targets. From trial to trial, the specific target that each distractor predicted varied. Because three targets were used, a distractor-target correlated pairing occurred less frequently than if there were only two targets. The use of a third target helped to ensure that the correlated trials were not too explicit. For instance, in a situation in which there are only two targets, it may be fairly easy for all participants to notice a correlation between the tones, therefore making it difficult to determine whether there is a relationship between level of ADHD symptoms and ability to pick up on correlated stimuli. On the other hand, with three targets, the correlation may not be as easily noticed if a participant is selectively attending. Also, in

the high probability condition, a distractor was always followed by a target; however, a target may have occurred without a preceding distractor.

Participants were instructed to ignore the distractor tones while responding as quickly and accurately as possible to the target tones. Participants were asked to press the button associated with each of the three target tones using the index, middle, and ring finger of their dominant hand. Feedback was provided only during the practice trial. Specifically, the computer provided a written indicator on the computer screen for correct responses (correctly responding to a target), misses (not responding to a target), correct rejection (not responding to a distractor), and false alarms (responding to a distractor or an incorrect target). After the practice trial, it appeared that each participant understood the task; thus, no additional training was provided. After the practice trial (i.e., once the experimental trials began), feedback on accuracy of responses was not provided. Performance on these tasks was measured using reaction time (measured to the millisecond) and accuracy, including number of correct responses, misses, correct rejections, and false alarms.

EEG Data

In conjunction with the neurocognitive performance data, neurological data was also obtained through the use of an EEG. As mentioned previously, the N1 amplitude was measured to determine whether there was a difference between participants with high and low levels of ADHD-associated symptoms. According to Knott and colleagues (2006), the N1 amplitude can be measured through the Cz channel of an electrode cap located in the central area of the brain. Also being measured electrophysiologically was the P3 amplitude.

CHAPTER V

RESULTS

Differences between Groups on Behavioral Data

Independent samples *t*-tests were conducted to compare the high ADHD-associated symptoms group and the low ADHD-associated symptoms group on the Barkley's ADHD Checklist to ensure that the groups differed on ADHD symptoms (i.e., as a criterion check). As expected based on how the groups were formed, the high ADHD-associated symptoms group reported more hyperactivity (M = 4.58, SD = 2.477) than the participants in the low ADHD-associated symptoms group (M = .50, SD = .89), $t(38) = -7.39$, $p < .001$. Likewise, participants in the high ADHD-associated symptoms group displayed more inattention (M = 6.60, SD = 1.43) than participants in the low ADHD-associated symptoms group (M = .50, SD = .95), $t(38) = -15.91$, $p < .001$. It is important to note that the means represent the average number of symptoms endorsed that were scored as a 2 or 3 on the Likert scale ranging from 0 to 3 and are not an indicator of average symptom severity.

Difference between Groups on Demographic and Background Variables

Independent samples *t*-tests were conducted to compare the high ADHD and low ADHD groups on relevant demographic and background variables. There were no significant differences between groups when comparing age, family income, or total score on the AUDIT. However, the groups did differ significantly on FSIQ, with the high ADHD-associated symptoms group displaying a higher FSIQ (M = 108.35, SD = 10.90) relative to those in the low ADHD-associated symptoms group (M = 95.80, SD = 10.43), $t(38) = -5.72$, $p < .01$ (see Table 1).

Table 1

Differences Between High ADHD-associated and Low ADHD-associated Symptoms Groups on Demographics and Background Variables

	Total Sample (N = 40)		High ADHD (n = 20)		Low ADHD (n = 20)		
	Mean	SD	Mean	SD	Mean	SD	*t*-value
Age	24.03	9.21	23.15	8.21	24.90	10.26	.60
Income	4.44	4.31	4.59	4.29	4.32	4.44	-.19
Nicotine use	.28	.45	.25	.44	.30	.47	.35
AUDIT Total	3.88	5.20	3.60	3.84	4.15	6.38	.33
WASI FSIQ	102.08	12.30	108.35	10.90	95.80	10.43	-3.72***

Note. AUDIT = Alcohol Use Disorders Identification Test; WASI FSIQ = Wechsler's Abbreviated Scale of Intelligence Full Scale IQ.

*** $p < .001$.

Additionally, a Pearson Chi-Square analysis (Table 2) was conducted to determine if there were any differences between groups on relevant categorical variables. Race was dichotomized into a Caucasian and Non-Caucasian group due to the extremely low number (n = 2) of Asian participants. These results revealed that the high and low ADHD-associated symptoms groups significantly differed on race. That is, the high ADHD-associated symptoms group had significantly more Caucasians, whereas the low ADHD-associated symptoms group had significantly more Non-Caucasians, χ^2 (1, N = 40) = 15.00, $p < .01$. A Chi-Square analysis indicated that there was not a significant difference between groups on gender, as both groups had more female participants relative to male, χ^2 (1, N = 40) = .11, $p = .74$. Thus, based on group differences between

FSIQ and race, both of these variables were used as controls in all subsequent analyses using group as an independent variable.

Table 2

Demographic Characteristics of the High ADHD-associated and Low ADHD-associated Symptoms Groups

Characteristic	Total Sample ($N = 40$) *n* (%)	High ADHD ($n = 20$) *n* (%)	Low ADHD ($n = 20$) *n* (%)	Chi Square (χ^2)
Race				15.00*
Caucasian	24 (60%)	18 (90%)	6 (30%)	
Non-Caucasian	16 (40%)	2 (10%)	14 (70%)	
Gender				.11
Male	13 (32.5%)	6 (30%)	7 (35%)	
Female	27 (67.5%)	14 (70%)	13 (65%)	

* $p < .05$

Relations of Neurocognitive Data with Demographic and Background Variables

Correlation analyses were conducted between the neurocognitive data and demographic/background variables to determine if any additional controls needed to be included when analyzing the neurocognitive data. Specifically, age (continuous), race (dichotomous), gender (dichotomous), family income (continuous), FSIQ (continuous), and AUDIT total (continuous) were correlated with the four neurocognitive outcome variables that were to be included in the mixed model analyses of variance (ANOVAs; i.e., high probability target reaction time, low probability target reaction time, high probability target accuracy, and low probability target accuracy).

Results are presented in Table 3. Race (already a control variable due the group differences on race) was significantly correlated with accuracy in the high probability condition (i.e., Caucasians were more accurate). In addition, age was significantly correlated with accuracy in both conditions (i.e., younger participants were more accurate), and gender was significantly correlated with reaction time in the high probability condition (i.e., females were faster). Based on these findings, it was determined that the analyses of the neurocognitive data should control for age and gender, in addition to race and FSIQ.

Table 3

Correlations Between Demographic/Background Variables and Neurocognitive Data

	Gender	Race	Age	Income	Nicotine	AUDIT	FSIQ
Reaction Time							
High Prob Target	.33*	.15	.26	.08	.03	-.20	-.10
Low Prob Target	.24	.19	.28	.90	.02	-.24	-.12
Accuracy							
High Prob Target	-.15	-.33*	-.47**	-.21	.00	.26	.08
Low Prob Target	-.11	-.31	-.52**	-.21	-.03	.29	.07

Note. Gender was coded 0 = male and 1 = female; race was coded 0 = Caucasian and 1 = Non-Caucasian; AUDIT = Alcohol Use Disorders Identification Test; WASI FSIQ = Wechsler's Abbreviated Scale of Intelligence Full Scale IQ.

* $p < .05$, ** $p < .01$.

Analyses of Neurocognitive Data

The neurocognitive data were analyzed using repeated measures analyses of covariance (ANCOVAs). Specifically, a 2 x 2 repeated measures ANCOVA was used with group as a between-subjects factor with two levels (high ADHD-associated symptoms and low ADHD-associated symptoms). Condition (high probability or low probability) was the within-subjects factor. Covariates included gender, race, age, and FSIQ. The dependent variables included the mean reaction time for responding to high probability targets and low probability targets. Importantly, for the reaction time outcomes, only reaction times for *accurate* responses were used. The two-way interaction (group x condition) on the reaction time outcome was of particular interest to test the main hypothesis of the current study regarding the neurocognitive data. Other significant interactions with covariates are reported but were not explored through post-hoc probing, given that they were not a focus of the study. Finally, it should be noted that an alpha level of .05 was considered statistically significant for these analyses. However, results at an alpha of .10 are noted and discussed, given the small sample size and subsequent low power for the statistical tests.

Reaction Time when Responding to Targets

Results of the repeated measures ANCOVAs indicated that there was no main effect for condition (high probability and low probability). A main effect approaching significance was found, however, for group, $F(1, 34) = 3.12$, $p < .10$, partial $\eta^2 = .11$, with the low ADHD-associated symptoms group reacting faster ($M = .706$) than the high ADHD-associated symptoms group ($M = .774$). A main effect approaching significance was also found for gender, $F(1, 34) = 4.03$, $p < .10$, partial $\eta^2 = .11$, such that

male participants responded faster (M = .698) than female participants (M = .760) across both high and low probability conditions. Results also indicated a main effect approaching significance for age, $F(1, 34) = 3.89$, $p < .10$, partial $\eta^2 = .10$, indicating that younger participants were faster at responding relative to older participants.

As predicted, a condition x group interaction approaching significance was found, $F(1, 34) = 4.13$, $p = .05$, partial $\eta^2 = .08$ (see Figure 1). Post-hoc repeated measures ANCOVAs did not yield significant slopes for either group. This lack of significance may be caused by the decrease in degrees of freedom that is caused by including covariates in the analysis. Therefore, ANOVAs (without covariates) were conducted to test for simple effects. These analyses yielded a main effect approaching significance, $t(19) = -1.74$, $p < .10$, for the low ADHD-associated symptoms group and a significant main effect, $t(19) = -3.50$, $p < .05$, for the high ADHD-associated symptoms group. These results suggest that the low ADHD-associated symptoms group's reaction time was faster overall and was slightly faster in the high probability condition relative to the low probability condition. For the high ADHD-associated symptoms group, notable improvement marked by faster reaction time when responding accurately to targets was found in the high probability condition relative to the low probability condition.

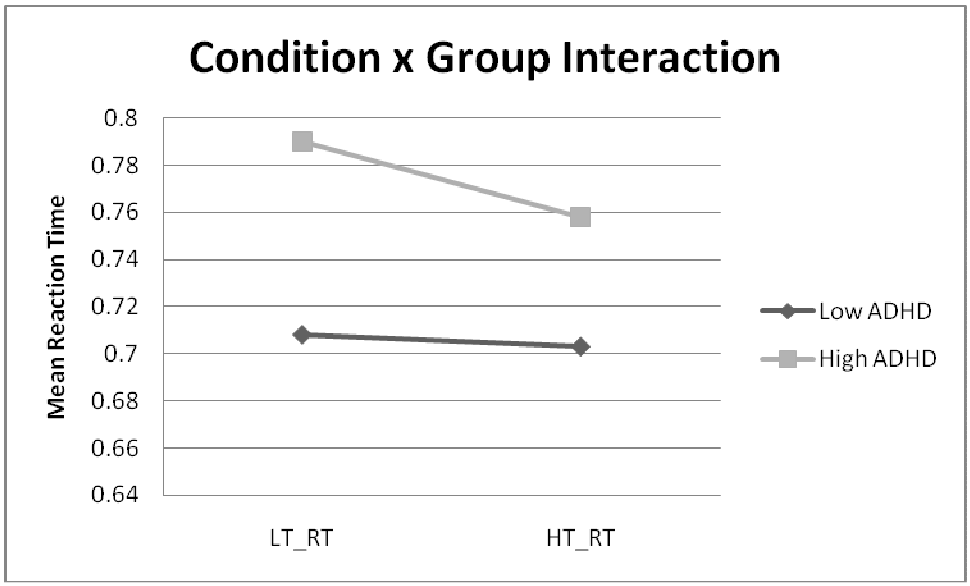

Figure 1 . Condition x Group Interaction

Note. Post-hoc analyses revealed a significant main effect for the high ADHD-associated group, t(19) = -3.50, p < .05, and a main effect approaching significance for the low ADHD-associated symptoms group, t(19) = -1.74, p < .10; LT_RT = Mean reaction time for target tones in the Low Probability Condition; HT_RT = Mean reaction time for target tones in the High Probability Condition.

A condition x gender interaction approaching significance was also found, $F(1, 34) = 3.01$, $p < .10$, partial $\eta^2 = .08$, again with male participants responding faster than female participants. No other significant main effects or interactions were found (see Table 4).

Accuracy.

Next an examination of participants' accuracy to target tones was conducted. Results of the repeated measures ANCOVAs also indicated that there was no main effect for condition. There was, however, a significant main effect for race, $F(1, 34) = 5.68$, $p < .05$, partial $\eta^2 = .14$, such that Caucasian participants were significantly more accurate (*M*

= .890) than Non-Caucasian participants (M = .792). A significant main effect for age was also found, $F(1, 34) = 12.47$, $p = .001$, partial $\eta^2 = .27$, indicating that accuracy level decreased as the participants' age increased. No other significant main effects or interactions were found (see Table 4).

Participants' accuracy level for distractor tones was also analyzed through repeated measures ANCOVA. Results indicated that a main effect approaching significance for race was found, $F(1, 34) = 2.98$, $p < .10$, partial $\eta^2 = .08$. This indicates that Caucasian participants were more accurate (M = .935) at ignoring distractor tones relative to Non-Caucasian participants (M = .766). An additional main effect approaching significance was found for age, $F(1, 34) = 3.06$, $p < .10$, partial $\eta^2 = .08$, indicating that younger participants more accurately ignored distractor stimuli than older participants . A condition x age interaction approaching significance was also found, $F(1, 34) = 3.67. <$ .10, partial $\eta^2 = .10$. No other significant main effects or interactions were found (see Table 4).

Table 4

Repeated Measures ANCOVAs Examining Differences between High ADHD-associated and Low ADHD-associated Symptoms Groups on Neurocognitive Variables [a]

	Reaction Time (Targets)			Accuracy (Targets)		Accuracy (Distractors)			
Source	Sum of Squares		Mean Square		*F*(1, 34)	Sum of Squares		Mean Square	*F*(1, 34)
	Sum of Squares		Mean Square		*F*(1, 34)				
Covariate									
Gender	.069	.069	4.03†	.067	.067	2.09	.176	.176	2.34
Race	.047	.047	2.73	.182	.182	5.68*	.225	.225	2.98†
Age	.067	.067	3.89†	.400	.400	12.47**	.231	.231	3.06†
WASI FSIQ	.004	.004	.26	.002	.002	.08	.026	.026	.34
Main Effects									
Group	.054	.054	3.12†	.041	.041	1.27	.001	.001	.01
Condition	.001	.001	1.03	.001	.001	.37	.000	.000	.06
Interactions									
Condition x Gender	.002	.002	3.01†	.001	.001	1.03	.000	.000	.06
Condition x Race	.001	.001	2.07	.002	.002	1.67	.003	.003	1.62

Table 4 (continued).

	Sum of Squares		Mean Square		$F(1, 34)$	Sum of Squares		Mean Square	$F(1, 34)$
	Sum of Squares		Mean Square		$F(1, 34)$				
Condition x Age	8.42	8.42	.17	.000	.000	.13	.007	.007	3.67†
Condition x WASI FSIQ	.000	.000	.57	8.69	8.69	.06	.001	.001	.68
Condition x Group	.002	.002	4.13††	.001	.001	.55	.004	.004	2.23

Note. WASI FSIQ = Wechsler's Abbreviated Scale of Intelligence Full Scale IQ.

[a] Controlling for race, gender, age, and FSIQ.

† $p < .10$, †† $p = .05$, *$p < .05$, ** $p < .01$.

Analysis of Electrophysiological Data

Prior to analyzing the N1 and P3 amplitudes, a grand mean waveform for each electrode site was created. Based on visual inspection of the grand mean waveform, along with findings from previous research, appropriate latency time intervals were determined (Johnstone, Barry, Markovska, Dimoska, & Clarke, 2009; Luck, 2005). Specifically, N1 amplitudes were measured by computing the average amplitude between a latency of 90 and 170 ms. Moreover, P3 amplitudes were measured by computing the average amplitude between a latency of 390 and 400 ms. According to Luck (2005), computing the mean amplitude within a predetermined latency is superior to measuring peak amplitude. Therefore, in the current study, all ERP analyses were conducted with mean amplitudes for each electrode site.

The mean amplitudes were computed for each electrode site by use of the STIM analysis program ERPSCORE. The mean amplitudes were then entered into SPSS and were organized by site (F3, F4, Fz, P3, P4, C3, C4, or Cz), tone (target or distractor), condition (high probability or low probability), and amplitude (N1 or P3). After creating these new variables, descriptive statistics were computed on all eight sites for each tone, condition, and amplitude to determine which scalp locations would be included in subsequent analyses. Luck (2005) suggests that researchers refrain from including electrode sites in their analyses when there is no amplitude present because including these sites increases error in the analyses. Therefore, determining which scalp regions to include in the analyses was the next step in preparing the data. N1 amplitudes for all eight scalp locations (F3, F4, Fz, C3, C4,Cz, P3, P4) were included in subsequent analyses due to the presence of negative mean amplitudes in all three areas, for all tones and

conditions. N1 amplitudes are negative going evoked potentials and are thought to be elicited by unpredictable stimuli. Descriptive statistics for the P3 amplitudes, however, suggested that this amplitude was only present in the parietal lobe sites (P3, P4), given that these scalp locations were the only ones of the eight that elicited a positive going evoked potential across tones and conditions. This finding is consistent with previous research in which the P3 amplitude was most prominent at the parietal sites (Polich & Criado, 2006; Sumich et al., 2008). Therefore, in subsequent analyses, P3 amplitudes were only examined in the parietal lobe. Lastly, as recommended by Luck (2005), the Greenhouse-Geisser epsilon adjustment was used to address issues of artificially low *p*-values for all within-subjects effects. Results using the Greenhouse-Geisser adjustment followed the same pattern as the non-corrected results, therefore the non-corrected results are presented. It was reported by Luck (2005) that the use of this adjustment is imperative when there are more than two levels of a factor in an ANOVA, particularly when one of the factors is the electrode site.

Finally, race and FSIQ (the only demographic/background variables shown to differ between the high ADHD-associated and low ADHD-associated symptoms groups) were included as covariates in all ANCOVAs examining the electrophysiological data. As with the neurocognitive data, post-hoc probes of significant interactions between the variables of interest in the study were conducted, whereas significant interactions with covariates are reported but were not explored through post-hoc probing. That is, any significant interactions with the primary between-subjects and within-subjects variables (group, condition, tone, and site) were further examined, even if they were not predicted *a priori*. As before, an alpha level of .05 was considered statistically significant, but results at an

alpha of .10 are also noted and discussed (i.e., considered to approach significance, which may be meaningful in light of the low power for the statistical tests). To test the hypotheses involving the electrophysiological data, two-way interactions (group x tone) and three-way interactions (group x condition x tone; group x site x tone) were of primary interest. In the first of these analyses, specific electrode sites were examined, No predictions were made about main effects for or interactions with specific site of the electrodes; however, site was examined as a within-subjects factor in order to determine if variation in electrophysiological functioning in response to the stimuli occurred by electrode site.

Analysis of N1 Amplitudes by Site

Data from each N1 electrode site was analyzed in a 2 x 8 x 2 x 2 repeated measures ANCOVA in attempt to draw conclusions about more specific scalp distribution of the N1 amplitude. Group was the between-subjects factor with two levels (high ADHD-associated symptoms and low ADHD-associated symptoms). Site (with eight levels of location), condition (high probability or low probability), and tone (target or distractor) were the within-subjects factors. The dependent variable was mean N1 amplitude.

Results (presented in Table 5) indicated a main effect for site approaching significance, $F(7, 27) = 2.36$, $p = .05$, partial $\eta^2 = .38$, suggesting that the size of the N1 amplitude decreased as the electrodes were closer to the parietal lobe. In other words, the N1 amplitude was found to be largest in the frontal lobe, slightly smaller in the central lobe, and smallest in the parietal lobe. A main effect approaching significance for group was also found, $F(1, 33) = 3.73$, $p < .10$, partial $\eta^2 = .10$, such that the high ADHD-

associated symptoms group had a larger N1 amplitude (M = -4.39) across electrode sites relative to the low ADHD-associated symptoms group (M = -2.91). A main effect approaching significance for FSIQ was also found, $F(1, 33) = 3.70$, $p < .10$, partial $\eta^2 = .10$. Specifically, the size of the N1 amplitude appears to be larger for those participants with a higher FSIQ. A site x FSIQ interaction approaching significance was found, $F(7, 27) = 2.11$ $p < .10$, partial $\eta^2 = .35$. In addition, a site x tone x FSIQ interaction approaching significance was found, $F(7, 27) = 1.99$ $p < .10$, partial $\eta^2 = .34$.

An interaction approaching significance for tone x condition was also found, $F(1, 33) = 4.00$, $p = .05$, partial $\eta^2 = .11$(see Figure 2). Post-hoc repeated measures ANCOVAs revealed a main effect approaching significance for distractor tones, $F(1, 34) = 2.90$, $p < .10$, partial $\eta^2 = .08$. The main effect for target tones was not significant, $F(1, 34) = .71$, p = ns, partial $\eta^2 = .02$. These results suggest that the difference between the size of the N1 amplitude for target tones in the high (M = -3.87) and low (M = -3.74) probability conditions was not significant; however, the N1 amplitude for high probability distractors was significantly larger (M = -3.80) than that of low probability distractors (M = -3.43).

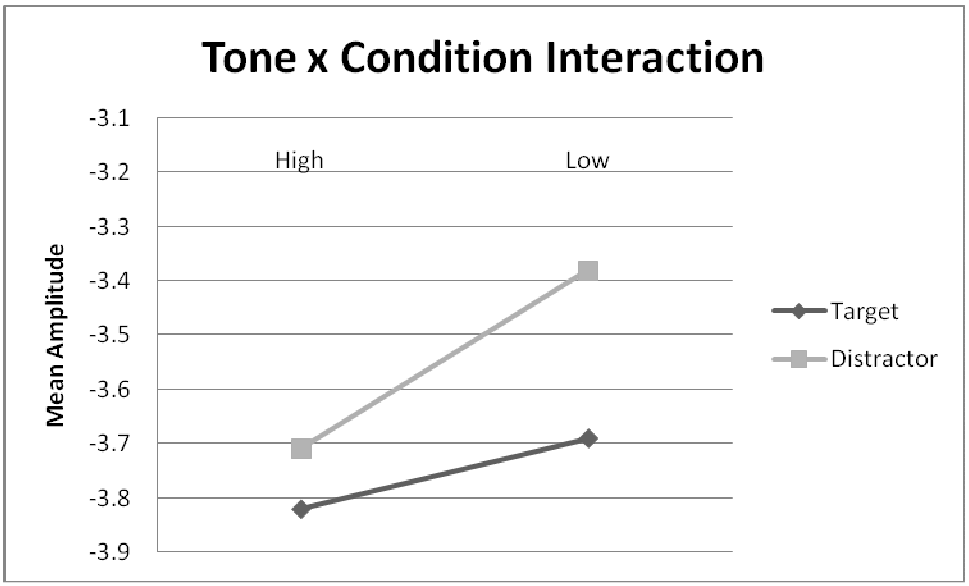

Figure 2. Tone x Condition Interaction

Note. Post-hoc analyses revealed a main effect approaching significance for distractor tones, $F(1, 34) = 2.90$, $p < .10$, partial $\eta^2 = .08$. The main effect for target tones was not significant $F(1, 34) = .71$, p = ns, partial $\eta^2 = .02$.

Lastly, a tone x condition x FSIQ interaction approaching significance was found, $F(1, 33) = 3.78$, $p < .10$, partial $\eta^2 = .35$. No other main effects or interactions, including the predicted two-way and three-way interactions, were found to be significant (see Table 5).

Analysis of P3 Amplitudes by Site

A 2 x 2 x 2 x 2 repeated measures ANCOVA was conducted to examine scalp distribution of the P3 amplitude within the parietal region of the brain (see Table 5). Group was the between-subjects factor with two levels (high ADHD-associated symptoms and low ADHD-associated symptoms). Site (P3 or P4), condition (high

probability or low probability), and tone (target or distractor) were the within-subjects factors. The dependent variable was mean P3 amplitude.

Results of this analysis revealed two interactions approaching significance but no significant main effects. First, a site x tone interaction approaching significance was found, $F(1, 36) = 2.97$, $p < .10$, partial $\eta^2 = .08$ (see Figure 3). Post-hoc repeated measures ANCOVAs were first conducted, but revealed non-significant results for the two slopes. Therefore, post-hoc repeated measures ANOVAs were conducted to examine simple effects. A significant main effect for tone was found for the P3 amplitude at the P4 scalp location, $F(1, 39) = 30.47$, $p < .001$, partial $\eta^2 = .49$. The main effect for the P3 amplitude for the P3 scalp location was also significant, $F(1, 39) = 37.03$, $p < .001$, partial $\eta^2 = .44$. Both lines sloped in the same direction, indicating that the distractor tones for both the P3 ($M = 3.54$) and P4 ($M = 1.90$) site locations yielded a larger P3 amplitude relative to the target tones for both the P3 ($M = 1.90$) and P4 ($M = 2.41$) site locations. Thus, the pattern was more suggestive of an effect for tone than an interaction between site and tone. This pattern, combined with the fact the interaction was only approaching significance, indicates that the interaction is not interpretable.

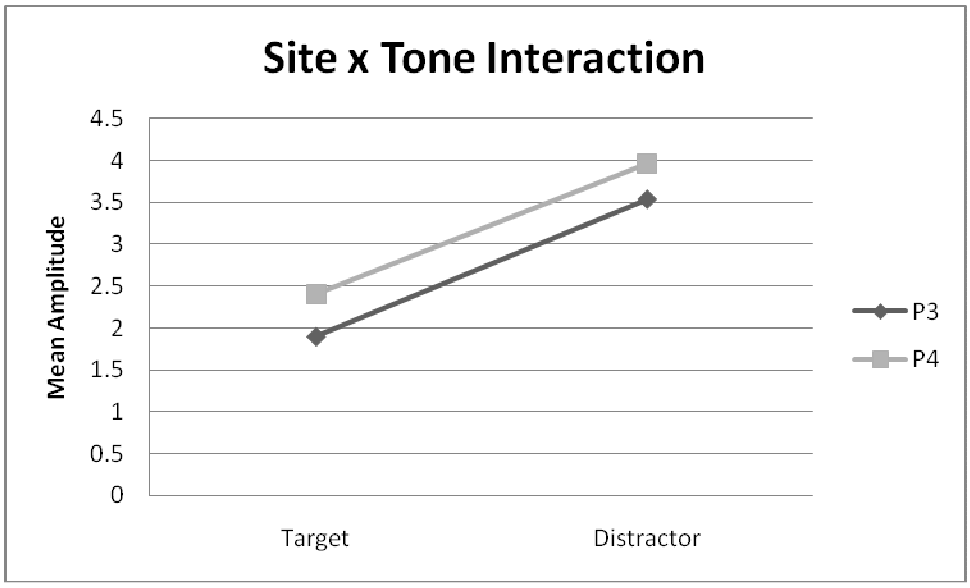

*Figure 3***.** Site x Tone Interaction

Note. Post-hoc analyses revealed that the P3 amplitude for the P3 electrode site was significantly larger for distractor tones relative to target tones, $F(1, 39) = 37.03$, $p < .001$, partial $\eta^2 = .44$. Simple effects also revealed that P3 amplitude for the P4 electrode site was significantly larger for distractor tones relative to target tones $F(1, 39) = 30.47$, $p < .001$, partial $\eta^2 = .49$.

Second, a site x condition x group interaction approaching significance was found, $F(1, 36) = 3.35$, $p < .10$, partial $\eta^2 = .09$ (see Figure 4). Post-hoc analyses for group x condition were conducted. These analyses revealed that the slope for both the high ADHD-associated symptoms group, $F(1, 19) = 4.02$, $p < .05$, partial $\eta^2 = .17$, and the low ADHD-associated symptoms group, $F(1, 19) = 15.72$, $p < .001$, partial $\eta^2 = .45$, were significant for the P3 electrode site. Likewise, the slope for both the high ADHD-associated symptoms group, $F(1, 19) = 11.01$, $p < .05$, partial $\eta^2 = .37$, and the low

ADHD-associated symptoms group, $F(1, 19) = 20.72$, $p < .001$, partial η^2=.52, were significant for the P4 electrode site. These results revealed that the mean P3 amplitude increased significantly for both electrode sites and both groups in the low probability condition. The patterns of these findings were more suggestive of an effect for condition rather than an interaction between group, site, and condition. This pattern, combined with the fact this three-way interaction was only approaching significance, indicates that the interaction is not interpretable. No other significant main effects or interactions (including the predicted interactions) were found (see Table 5).

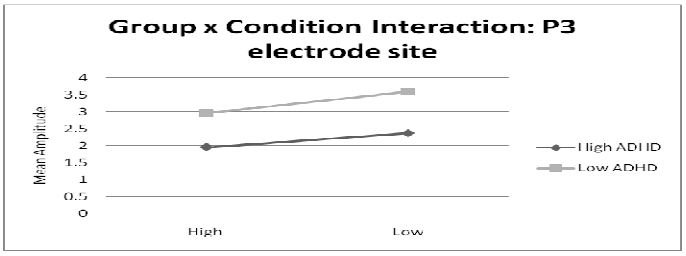

*Figure 4***.** Group x Condition Interaction: P3 electrode site

Note. Post-hoc analyses revealed that the slope for both the high ADHD-associated symptoms group, F(1, 19) = 4.02, $p < .05$, partial $\eta^2 = .17$, and the low ADHD-associated symptoms group, F(1, 19) = 15.72, $p < .001$, partial $\eta^2 = .45$, were significant.

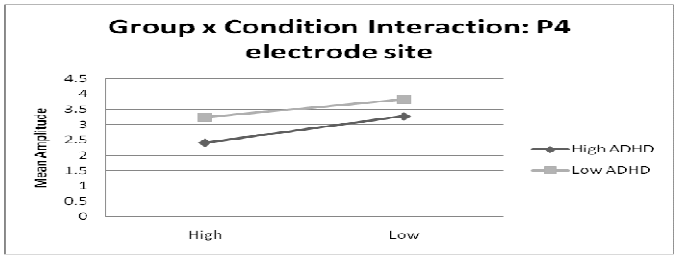

Figure 4. Group x Condition Interaction: P4 electrode site

Note. Post-hoc analyses revealed that the slope for both the high ADHD-associated symptoms group, F(1, 19) = 11.01, $p < .05$, partial $\eta^2 = .37$, and the low ADHD-associated symptoms group, F(1, 19) = 20.72, $p < .001$, partial $\eta^2 = .52$, were significant.

Table 5

Repeat*ed Measures ANCOVAs Examining High ADHD and Low ADHD-associated Symptoms on N1 and P3 Amplitude by Site* [a]

	N1 Mean Amplitude (all sites)			P3 Mean amplitude (P3 and P4 sites)		
Source	Sum of Squares	Mean Square	*F*	Sum of Squares	Mean Square	*F*
Covariate			*F*(1, 33)			*F*(1, 36)
Race	84.04	84.04	.80	9.03	9.03	.19
WASI FSIQ	387.44	387.44	3.70†	65.87	65.87	1.41
Main Effects			*F*(7, 27)			*F*(1, 36)
Group	390.29	390.29	3.73†	36.87	36.87	.79
Site	60.51	8.64	2.36††	2.71	2.71	.61
Tone	8.28	8.28	.18	159.34	4.43	.92
Condition	12.67	12.67	2.30	.01	.01	.01
Interactions			*F*(7, 27)			*F*(1, 36)
Site x Race	5.33	.76	.66	.48	.48	.11
Site x FSIQ	20.49	2.93	2.11†	1.51	1.51	.34

Table 5 (continued).

	Sum of Squares	Mean Square	*F*	Sum of Squares	Mean Square	*F*
Site x Condition	1.25	.18	.54	.54	.54	2.45
Site x Tone x Race	1.10	.16	.60	.03	.03	.09
Site x Tone x FSIQ	11.44	1.63	1.99†	.97	.97	3.10
Site x Tone x Group	1.04	.145	.56	.05	.05	.18
Site x Condition x Race	.36	.05	.34	.03	.03	.14
Site x Condition x FSIQ	.92	.13	.53	.49	.49	2.21
Site x Condition x Group	1.68	.24	.62	.74	.74	3.35†
Tone x Race	4.04	4.04	.18	.48	.48	.46
Tone x FSIQ	5.94	5.94	.26	.91	.91	.15
Tone x Group	5.05	5.05	.22	.00	.00	.00
Tone x Condition	25.84	25.84	4.00††	3.09	3.09	1.43
Tone x Condition x Race	2.35	2.35	.36	.05	.05	.02
Tone x Condition x FSIQ	24.42	24.42	3.80†	2.61	2.61	1.21

Table 5 (continued).

	Sum of Squares	Mean Square	*F*	Sum of Squares	Mean Square	*F*
Condition x FSIQ	10.86	10.86	1.97	.17	.17	.12
Condition x Group	7.29	7.29	1.32	.01	.01	.01

Note. WASI FSIQ = Wechsler's Abbreviated Scale of Intelligence Full Scale IQ.

[a] Controlling for race and FSIQ, † $p < .10$, †† $p = .05$, * $p < .05$

Analysis of Electrophysiological Data Grouped by Lobe

Next, in order to reduce the number of factors entered into the ANCOVA, thus decreasing the chance of an experimentwise Type 1 error, the electrode sites were grouped according to scalp location (i.e., frontal, central, and parietal lobes). This organization also allowed testing of the hypothesis predicting a group x site x tone interaction, in which it was expected that N1 amplitudes in the frontal lobe would be larger for distractor tones for the high ADHD-associated symptoms group. The hypothesis in which it was expected that P3 amplitudes in the frontal lobe would be larger for target tones, however, was not able to be tested due to the lack of P3 amplitudes in the frontal and central lobes. This grouping resulted in three groups of averaged amplitudes for N1, separating the groups by tone and condition. The frontal lobe included F3, F4, and Fz, the central lobe included C3, C4, and Cz, and the parietal lobe included P3 and P4 electrode sites. In addition, this grouping resulted in one group of averaged amplitudes in the parietal lobe for P3, separating the groups by tone and condition. As before, race and FSIQ were entered as covariates for each of these ANCOVAs because these variables differed between the groups.

Analyses of N1 Amplitudes

N1 amplitudes were analyzed with a 2 x 3 x 2 x 2 repeated measures ANCOVA, with group as a between-subjects factor with two levels (high ADHD-associated symptoms and low ADHD-associated symptoms). Site location (frontal, central, or parietal), condition (high probability or low probability), and tone (target or distractor) were within-subjects factors. The dependent variable for this analysis was mean N1 amplitude.

Results of the analysis (see Table 6) indicated that there was a main effect approaching significance for group, $F(1, 33) = 3.53$, $p < .10$, partial $\eta^2 = .10$ (see Figure 5). Specifically, this main effect suggests that the high ADHD-associated symptoms group had larger N1 amplitudes overall ($M = -4.13$) relative to the low ADHD-associated symptoms group ($M = -2.75$). For descriptive purposes, images of the grand mean ERPs are presented (Figures 6 through 11) to compare group differences for the N1 amplitude for each lobe and for both distractors and targets independently.

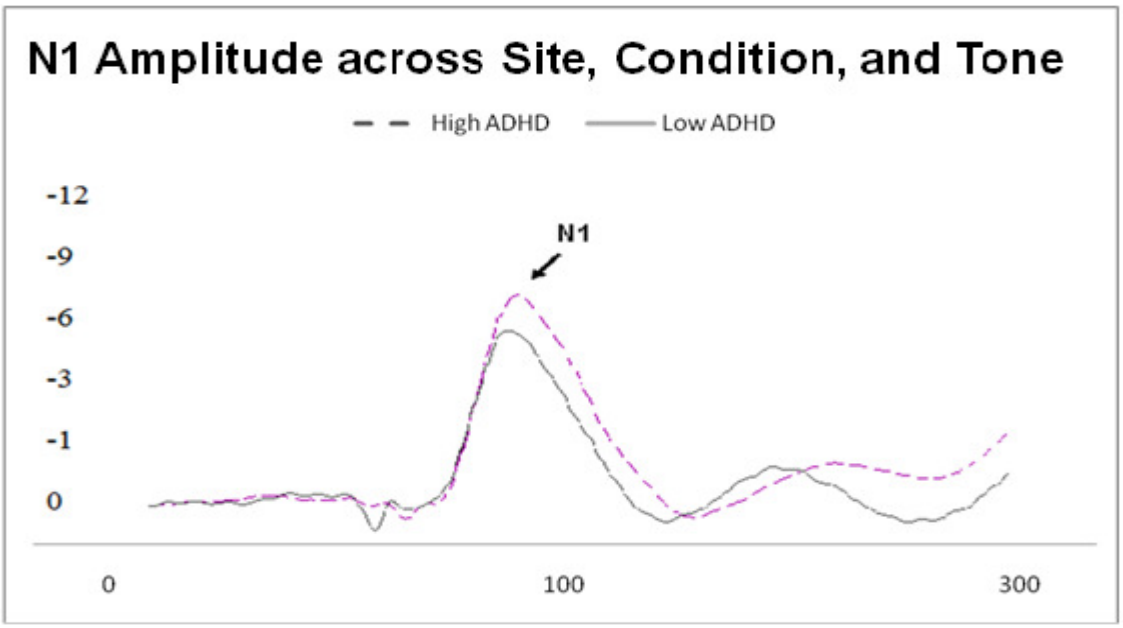

Figure 5. N1 Amplitude across Site, Condition, and Tone

Note. The X-axis represents latency in milliseconds and the Y-axis represents the mean amplitude in µVs; N1 refers to the N1 negative amplitude, which displays in a positive direction.

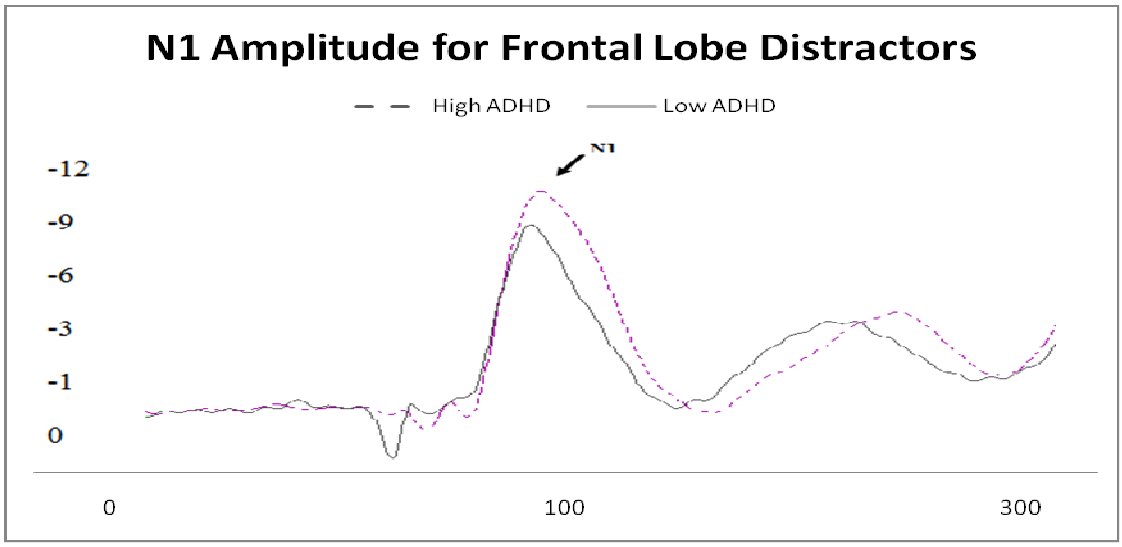

Figure 6. N1 Amplitude for Frontal Lobe Distractors

Note. The X-axis represents latency in milliseconds and the Y-axis represents the mean amplitude in µVs; N1 refers to the N1 negative amplitude, which displays in a positive direction.

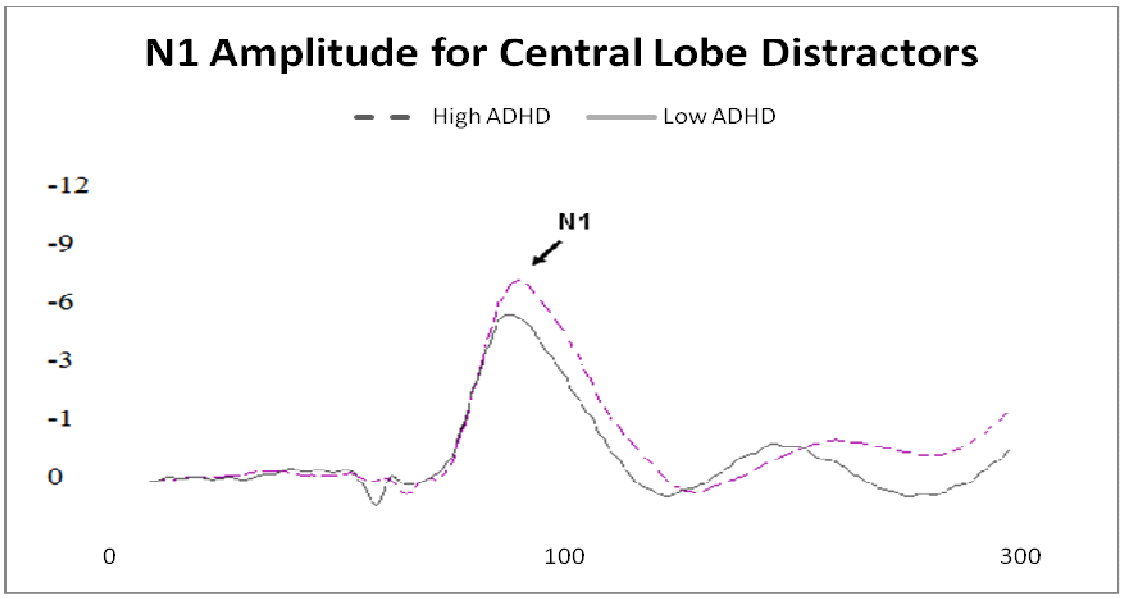

Figure 7. N1 Amplitude for Central Lobe Distractors

Note. The X-axis represents latency in milliseconds and the Y-axis represents the mean amplitude in µVs; N1 refers to the N1 negative amplitude, which displays in a positive direction.

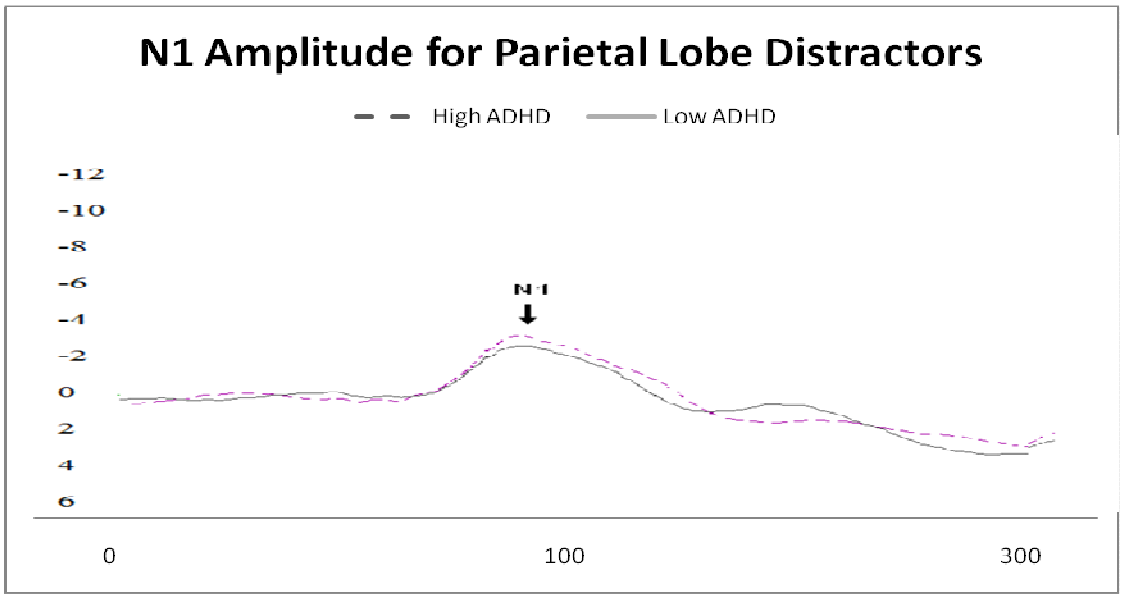

Figure 8. N1 Amplitude for Parietal Lobe Distractors

Note. The X-axis represents latency in milliseconds and the Y-axis represents the mean amplitude in µVs; N1 refers to the N1 negative amplitude, which displays in a positive direction.

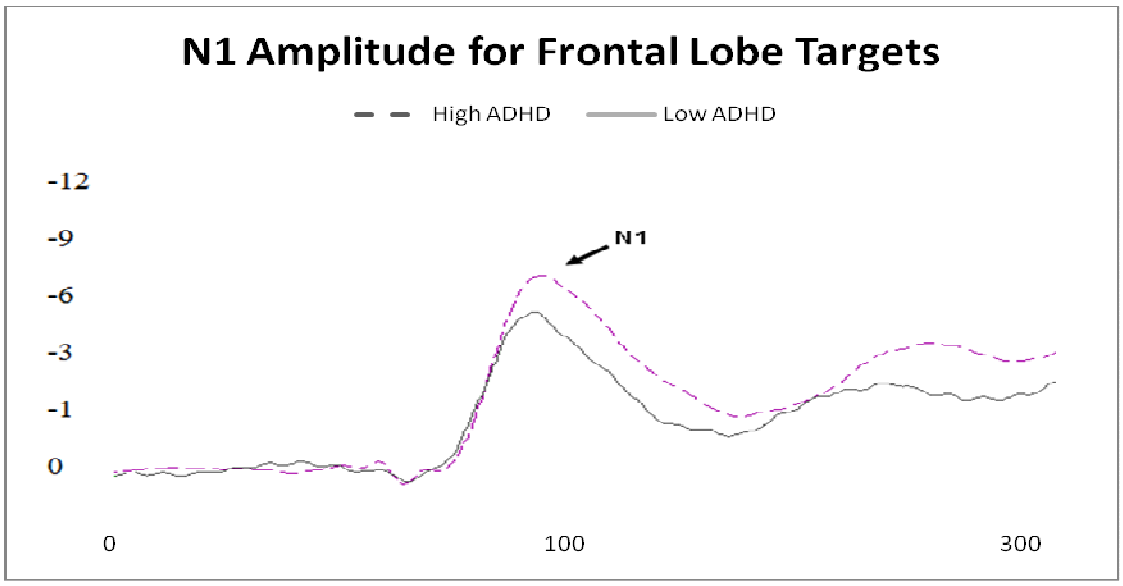

Figure 9. N1 Amplitude for Frontal Lobe Targets

Note. The X-axis represents latency in milliseconds and the Y-axis represents the mean amplitude in µVs; N1 refers to the N1 negative amplitude, which displays in a positive direction.

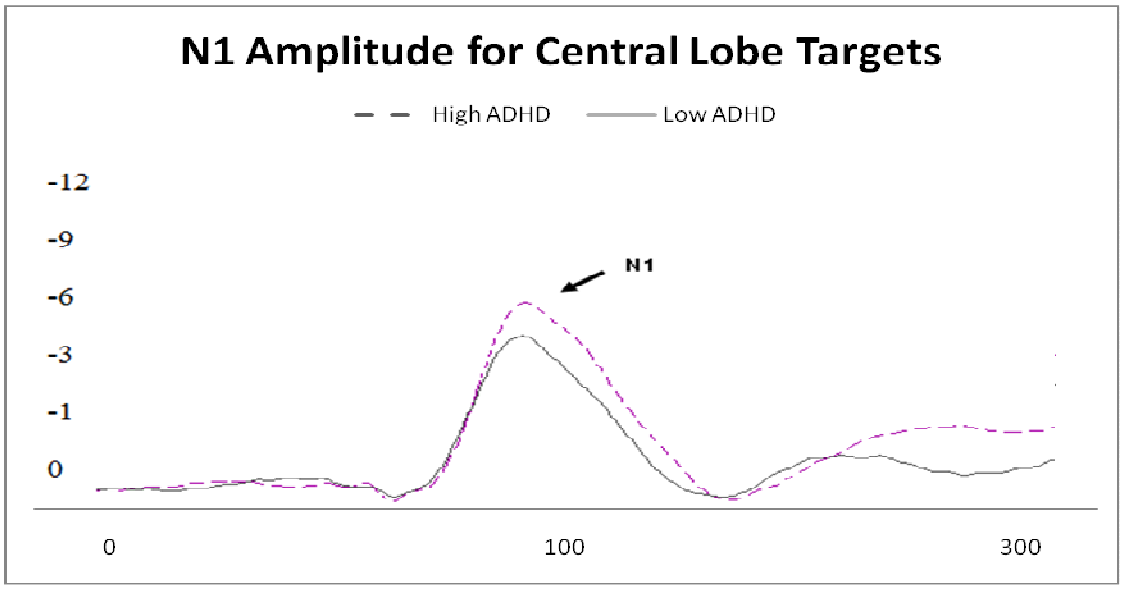

Figure 10. N1 Amplitude for Central Lobe Targets

Note. The X-axis represents latency in milliseconds and the Y-axis represents the mean amplitude in µVs; N1 refers to the N1 negative amplitude, which displays in a positive direc

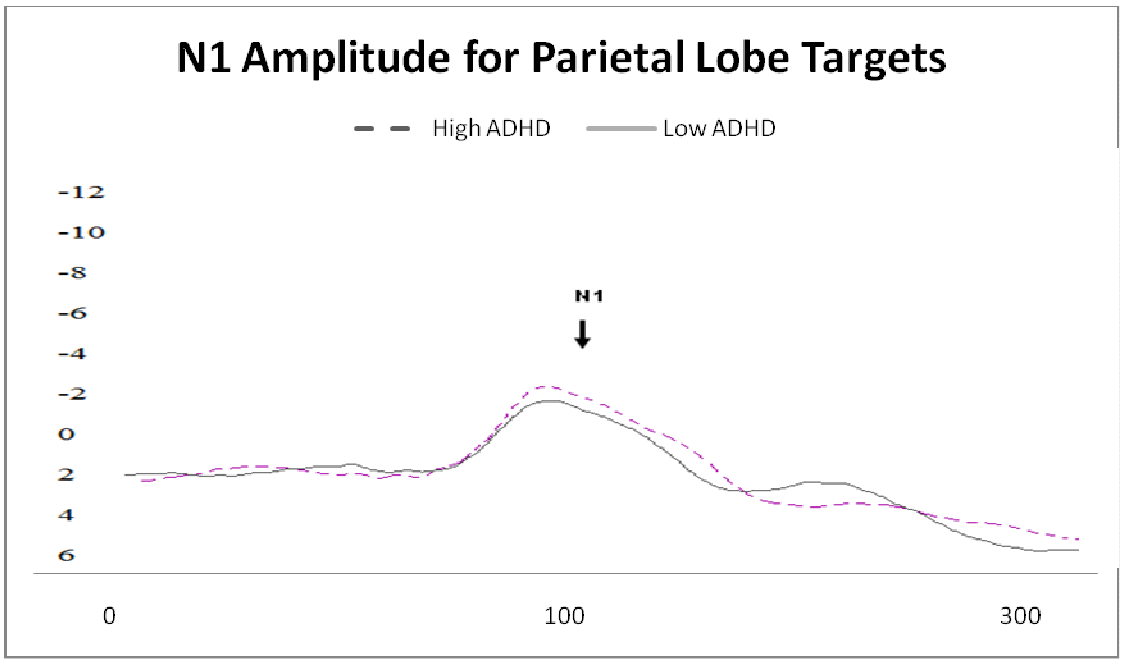

Figure 11. N1 Amplitude for Frontal Lobe Targets

Note. The X-axis represents latency in milliseconds and the Y-axis represents the mean amplitude in μVs; N1 refers to the N1 negative amplitude, which displays in a positive direction.

A main effect approaching significance was also found for the control variable FSIQ, $F(1, 33) = 3.77$, $p < .10$, partial $\eta^2 = .11$, such that participants with higher FSIQs had larger N1 amplitudes. Results indicated a tone x condition interaction approaching significance, $F(2, 32) = 3.90$, $p < .10$, partial $\eta^2 = .11$. The post-hoc plot of the interaction suggests that the N1 amplitudes for the target tones were larger overall, but the amplitude for the distractor tones increased more in the high probability condition (see Figure 12). However, results of the post-hoc ANCOVAs for both target, $F(1, 36) = 1.36$, p = ns, partial $\eta^2 = .04$, and distractor, $F(1, 36) = 1.94$, p = ns, partial $\eta^2 = .05$, tones were not significant.

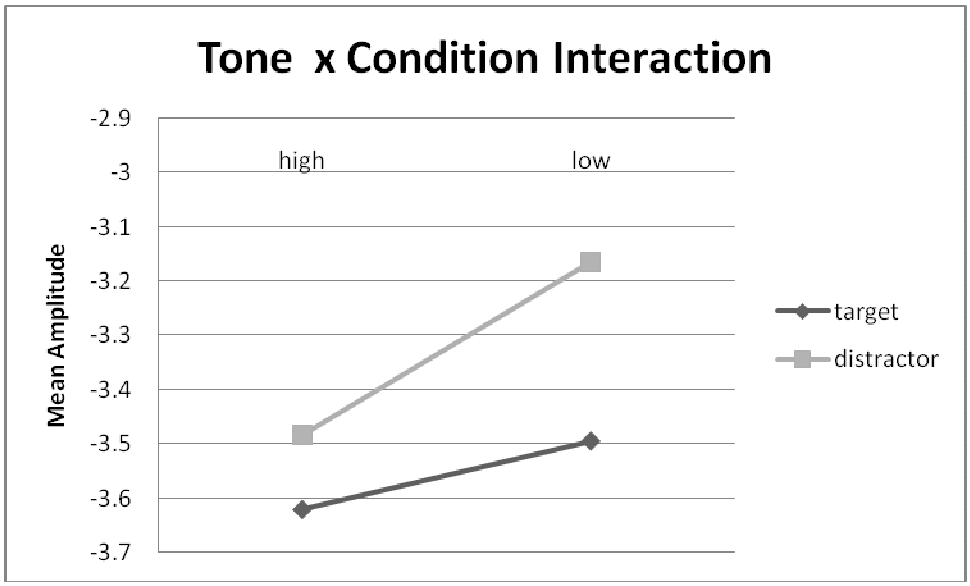

Figure12. N1 Amplitude for Frontal Lobe Targets

Note. Post-hoc analyses yielded non-significant slopes for both target, $F(1, 36) = 1.36$, p = ns, partial η^2= .04, and distractor, $F(1, 36) = 1.94$, p = ns, partial η^2=.05, tones.

A tone x condition x FSIQ interaction approaching significance was also found, $F(1, 33) = 3.72$, $p < .10$, partial $\eta^2 = .10$ (see Table 6). No other main effects or interactions (including the predicted interactions) were found.

Table 6

Repeated Measures ANCOVAs Examining Differences between High ADHD and Low ADHD-associated Symptoms Groups on N1 Amplitudes for Frontal, Central, and Parietal Lobes [a]

	N1 Mean Amplitude		
Source	Sum of Squares	Mean Square	*F*
Covariate			*F*(1, 33)
Race	32.85	32.85	.91
WASI FSIQ	136.14	136.14	3.77[†]
Main Effects			*F*(2, 32)
Group	127.31	127.31	3.53[†]
Site	22.13	11.07	2.42
Tone	2.25	2.25	.28
Condition	4.34	4.34	2.31
Interactions			*F*(2, 32)
Site x Race	1.08	.54	.15
Site x FSIQ	5.25	2.63	.61
Site x Group	26.66	13.33	2.16
Site x Tone	2.37	1.18	1.32
Site x Condition	.38	.19	.43
Site x Tone x Race	.23	.11	.48
Site x Tone x FSIQ	3.15	1.57	1.42
Site x Tone x Group	.25	.12	.13
Site x Condition x Race	.27	.27	.07
Site x Condition x FSIQ	.27	.14	.34
Site x Condition x Group	.11	.06	.88

Table 6 (continued).

	Sum of Squares	Mean Square	*F*
Tone x Race	1.33	1.33	.17
Tone x FSIQ	1.44	1.44	.18
Tone x Group	2.13	2.13	.26
Tone x Condition	8.83	8.83	3.90†
Tone x Condition x Race	.59	.59	.26
Tone x Condition x FSIQ	8.42	8.42	3.72†
Tone x Condition x Group	2.19	2.19	.97
Condition x Race	.02	.02	.01
Condition x FSIQ	3.73	3.73	.01
Condition x Group	2.82	2.82	1.50

Note. WASI FSIQ = Wechsler's Abbreviated Scale of Intelligence Full Scale IQ.

[a] Controlling for race and FSIQ.

† $p < .10$

Analyses of P3 Amplitudes

P3 amplitudes were analyzed for the parietal lobe with a 2 x 2 x 2 repeated measures ANCOVA, with group as a between-subjects factor with two levels (high ADHD-associated symptoms and low ADHD-associated symptoms). Condition (high probability or low probability) and tone (target or distractor) were within-subjects factors. The dependent variable for this analysis was mean P3 amplitude. Results indicated that there were no main effects or interactions for the P3 amplitudes (see Table 7).

Table 7

Repeated Measures ANCOVAs Examining Differences between High ADHD and Low ADHD-associated Symptoms Groups on P3 Amplitude for the Parietal Lobe [a]

	P3 Mean amplitude		
Source	Sum of Squares	Mean Square	*F*
Covariate			*F*(1, 36)
Race	4.52	4.52	4.52
WASI FSIQ	32.94	32.94	1.41
Main Effects			*F*(1, 36)
Group	18.44	18.44	.79
Tone	2.86	2.86	.92
Condition	.01	.01	.01
Interactions			*F*(1, 36)
Tone x Race	.81	.81	.26
Tone x FSIQ	.45	.45	.15
Tone x Group	.00	.00	.00
Tone x Condition	1.55	1.55	1.43
Tone x Condition x Race	.02	.02	.02
Tone x Condition x FSIQ	1.31	1.31	1.21
Tone x Condition x Group	.63	.63	.59
Condition x Race	.03	.03	.05
Condition x FSIQ	.08	.08	.20
Condition x Group	.00	.00	.01

Note. WASI FSIQ = Wechsler's Abbreviated Scale of Intelligence Full Scale IQ.

[a] Controlling for race and FSIQ.

Follow-up Analyses of Electrophysiological Data Grouped by Region

Finally, follow-up analyses were conducted to examine any differences between groups that may have been present in the left, right, or midline regions of the brain. These analyses were exploratory in nature, and no *a priori* hypotheses were made.

Analysis of N1 Amplitudes

N1 amplitudes were grouped by region of the brain (left, right, or midline) and entered into a 2 x 3 x 2 x 2 repeated measures ANCOVA with group (high ADHD-associated symptoms or low ADHD-associated symptoms) as the between-subjects factor and region (left, right, or midline), tone (target or distractor), and condition (high probability or low probability) as the within-subjects factors. The dependent variable was mean N1 amplitude. Results of this analysis (see Table 8) revealed a main effect approaching significance for group $F(1, 33) = 3.73$, $p < .10$, partial $\eta^2 = .10$, indicating that the high ADHD-associated symptoms group had a significantly larger N1 amplitude ($M = -4.47$) across all regions relative to the low ADHD-associated symptoms group ($M = -2.97$). A main effect approaching significance for FSIQ was also found, $F(1, 33) = 3.58$, $p < .10$, partial $\eta^2 = .10$.

In addition, several significant interactions were found. First, a significant site x tone interaction was found, $F(2, 32) = 4.37$, $p < .05$, partial $\eta^2 = .22$ (see Figure 13). Post-hoc repeated measures ANCOVAs yielded non-significant results. Therefore, post-hoc repeated measures ANOVAs (without covariates) were conducted to test for simple effects. The results indicated that there was a main effect approaching significance for tone in the right hemisphere of the brain, $F(1, 39) = 3.81$, $p < .10$, partial $\eta^2 = .09$. The main effects for tone in the left hemisphere, $F(1, 39) = 1.16$, $p < .10$, partial $\eta^2 = .03$, and

midline, $F(1, 36) = 2.66$, p = ns, partial $\eta^2 = .07$, locations were not significant. The post-hoc plot indicated that the N1amplitudes increased for targets relative to distractors, particularly in the right hemisphere of the brain when compared to other regions. A significant site x tone x FSIQ was also found, $F(2, 32) = 5.02$, $p < .05$, partial $\eta^2 = .24$.

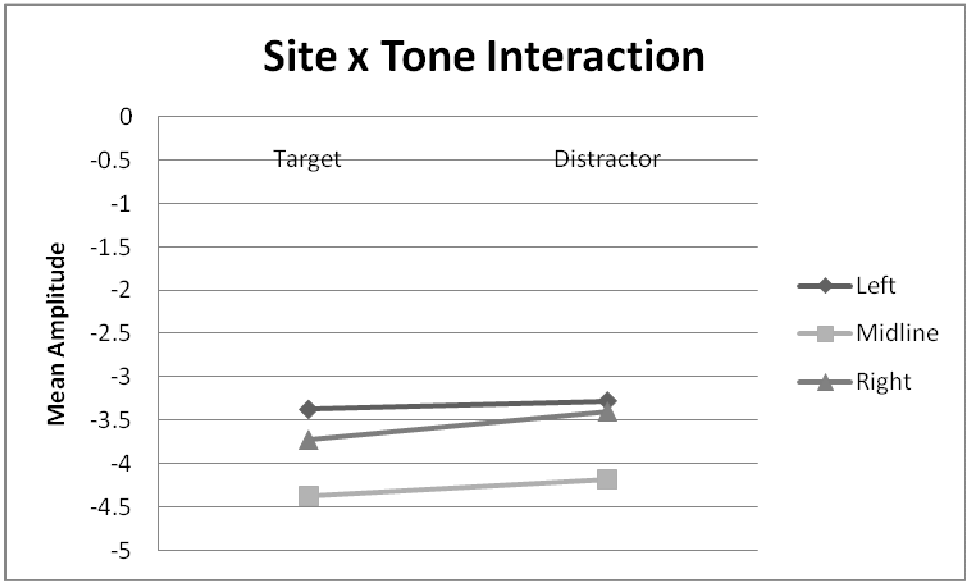

Figure 9. Site x Tone Interaction

Note. Post-hoc tests of simple effects found an effect approaching significance for tone in the right hemisphere of the brain, $F(1, 39) = 3.81$, $p < .10$, partial $\eta^2 = .09$. The simple effects for tone in the left hemisphere, $F(1, 39) = 1.16$, $p < .10$, partial $\eta^2 = .03$, and midline, $F(1, 36) = 2.66$, p = ns, partial $\eta^2 = .07$, locations were not significant.

Furthermore, a tone x condition interaction approaching significance was found, $F(1, 33) = 3.84$, $p < .10$, partial $\eta^2 = .10$ (see Figure 14). Results of post-hoc repeated measures ANCOVAs revealed a main effect approaching significance for tone in the low probability condition, $F(2, 33) = 3.08$, $p < .10$, partial $\eta^2 = .08$, whereas the main effect

for tone in the high probability condition was not significant, $F(2, 33) = .02$, p = ns, partial $\eta^2 = .00$. Specifically, although the mean N1 amplitude for target tones was larger than the amplitude for distractor tones in both conditions, the difference between targets and distractors was most marked in the low probability condition.

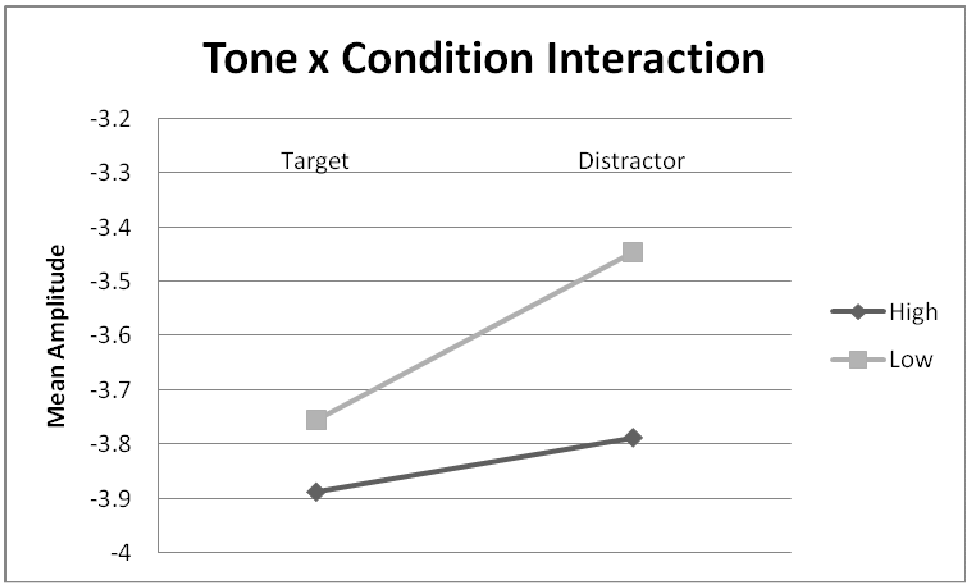

Figure 14. Tone x Condition Interaction

Figure 14. Post-hoc ANCOVAs revealed a main effect approaching significance for tone in the low probability condition, F (2, 33) = 3.08, $p < .10$, partial $\eta^2 = .08$. The main effect for tone in the high probability condition was not significant, F (2, 33) = .02, p = ns, partial $\eta^2 = .00$.

Lastly, a tone x condition x FSIQ interaction approaching significance was found, $F(1, 33) = 3.62$ $p < .10$, partial $\eta^2 = .10$. No other main effects or interactions (including the predicted interactions) were found (see Table 8).

Table 8

Repeated Measures ANCOVAs Examining Differences between High ADHD and Low ADHD-associated Symptoms Groups on N1 amplitudes for Left, Right, and Midline Regions [a]

	N1 Mean Amplitude		
Source	Sum of Squares	Mean Square	*F*
Covariate			*F*(1, 33)
Race	30.69	30.69	.76
WASI FSIQ	144.98	144.98	3.58†
Main Effects			*F*(2, 32)
Group	150.89	150.89	3.73†
Site	1.11	.56	.43
Tone	3.77	3.77	.42
Condition	4.76	4.76	2.18
Interactions			*F*(2, 32)
Site x Race	.21	.10	.11
Site x FSIQ	.38	.19	.15
Site x Group	1.43	.71	.63
Site x Tone	1.91	.96	4.37*
Site x Condition	.05	.03	.20
Site x Tone x Race	.20	.20	.42
Site x Tone x FSIQ	2.13	1.07	5.02*

Table 8 (continued).

	Sum of Squares	Mean Square	*F*
Site x Tone x Group	.20	.10	.41
Site x Condition x Race	.05	.03	.22
Site x Condition x FSIQ	.03	.01	.12
Site x Condition x Group	.44	.22	1.75
Tone x Race	1.67	1.67	.19
Tone x FSIQ	2.80	2.80	.31
Tone x Group	1.72	1.72	.19
Tone x Condition	9.68	9.68	3.84*
Tone x Condition x Race	.93	.93	.37
Tone x Condition x FSIQ	9.12	9.12	3.62†
Tone x Condition x Group	1.86	1.86	.75
Condition x Race	.01	.01	.00
Condition x FSIQ	4.07	4.07	1.86
Condition x Group	2.70	2.70	1.23

Note. WASI FSIQ = Wechsler's Abbreviated Scale of Intelligence Full Scale IQ.

[a] Controlling for race and FSIQ.

† $p < .10$, * $p < .05$.

Analysis of P3 Amplitudes

P3 amplitudes were not analyzed in the follow-up analyses due to a lack of P3 amplitude at many of the electrode sites. P3 amplitudes were only found at the P3 and P4 electrode site locations. Therefore, grouping the P3 amplitude by left, right, and midline hemispheres would result in meaningless findings.

CHAPTER VI

DISCUSSION

The present study examined the neurocognitive and electrophysiological functioning of adults with high and low levels of ADHD-associated symptoms. The current study tested the theory that adults with high levels of ADHD-associated symptoms may exhibit superior performance on a selective attention task in which distracting stimuli are predictive of subsequent target stimuli. In terms of accuracy of responding, it was expected that both groups would perform well (certainly above a 50% accuracy and level) and, thus, no significant differences between groups were expected on accuracy. Indeed, both groups, on average, responded accurately (above 80%), and no significant differences between groups on accuracy emerged. This shows that the task difficulty level was appropriate and that a high number of trials yielded usable data for the important reaction time analysis. It should be noted that the majority of the findings discussed below are not statistically significant. Rather, the majority of the findings reported only approached significance, showing a trend in the expected direction. These findings were reported due to small effect sizes that the current study may not have been able to detect due to low power. Therefore, although these findings are trending in the expected direction, conclusions or implications made on these findings should be interpreted with caution.

Support for Hypotheses Regarding Neurocognitive Data

First, it was hypothesized that those participants with high levels of ADHD-associated symptoms would have slower reaction times relative to the low ADHD-symptoms group in the low probability condition, with their reaction times becoming

faster than the low ADHD-symptoms group in the high probability condition. Specifically, it was expected that only high ADHD-associated symptoms group would benefit from the high probability condition, as demonstrated by a faster mean reaction time for that group compared to the group's mean reaction time in the low probability condition. Results lend some support to this hypothesis such that a group x condition interaction approaching significance was found. As expected, the high ADHD-associated symptoms group was slower than the low ADHD-associated symptoms group in the low probability condition. However, contrary to prediction, the high ADHD-associated symptoms group was not faster than the low ADHD-associated symptoms group in the high probability condition. That is, the high ADHD-associated symptoms group tended to have slower reaction times overall relative to the low ADHD-associated symptoms group (i.e., there was a main effect for group that approached significance). Nevertheless, as predicted, the condition x group interaction indicated that the high ADHD-associated symptoms group's reaction time tended to improve to a greater extent, relative to the low ADHD-associated symptoms group, when the probability of a distractor tone predicting a target tone was high. As mentioned previously, reaction times were only computed for trials with correct responses, and there was no group difference on accuracy. Therefore, these faster reaction times do not appear to be associated with impulsive, inaccurate responding.

Although these results suggest that the high ADHD-associated symptoms group did not perform at a superior level relative to the low ADHD-associated symptoms group in the high probability condition, the high ADHD-associated symptoms group did tend to benefit from high probability trials over and above that of the low ADHD-associated

symptoms group. Therefore, these results (although only approaching significance) may be interpreted such that the high ADHD-associated symptoms group was less successful at fast responding, relative to the low ADHD-associated symptoms group, due to a deficit in selective attention that makes filtering out distracting stimuli and responding quickly to target stimuli more difficult. At the same time, *because* of a deficit in selective attention, the high ADHD-associated symptoms group was able to improve their reaction time in the high probability condition. Therefore, the presence of correlated information was found to be beneficial only for those individual with high levels of ADHD-associated symptoms, thus suggesting that the impairment associated with a deficit in selective attention may reduce in conditions where distracting stimuli are no longer distracting but, rather, are informative. These findings fit well with the research conducted by van Mourik and colleagues (2007), in that both studies have implications that non-ignored but useful information can enhance an individual's performance if the individual has difficulty selectively attending.

Support for Hypotheses Regarding Electrophysiological Data

To test the hypotheses regarding the electrophysiological data, ERPs for N1 and P3 amplitudes were analyzed in three separate ways: (1) by site; (2) by lobe; and (3) by region. Again, two-way interactions between group and tone, as well as three-way interactions between group, condition, and tone, were of interest for both the N1 and P3 amplitude data. In addition, an interaction between group, site, and tone for N1 data analyzed by lobe was also of interest. An interaction between group, site, and tone for P3 data was also of interest, but again, because P3 amplitudes were only present in the parietal lobe, these analyses were not able to be conducted. Although none of the two-

way and three-way interactions that were predicted *a priori* were significant, the pattern of findings for the electrophysiological data does shed light on the nature of individuals' neurological functioning associated with the auditory selective attention task, as well as some differences between individuals with high and low levels of ADHD. Thus, the significant findings are discussed.

Findings by Site

First, N1 and P3 ERPs were analyzed through repeated measures ANCOVA, with site, condition, and tone as within subject's factors and group as a between subject factor. Analyses examining the N1 amplitude revealed a main effect approaching significance for electrode site. In particular, the size of the N1 amplitude tended to be largest for the frontal lobe electrodes (F3, F4, and Fz), slightly smaller for the central lobe electrode (C3, C4, and Cz), and smallest for the parietal lobe electrode (P3 and P4). What this finding suggests is that the N1 amplitude is most prominent in the frontal lobe. As mentioned by Luck (2005), the N1 amplitude is sensitive to attention. Therefore, these findings suggest that the function of attention, particularly auditory selective attention, is found primarily in the frontal region of the brain. This finding is consistent with previous research, which suggests that the measurement of attention and deficits in selective attention can be found in the frontal lobe (Barry et al, 2001; Hynd et al., 1991; Shue & Douglas, 1992). Likewise, Posner and Rothbart (2000) suggest that attentional processes are likely mediated by frontal lobe development.

A main effect approaching significance was also found for group, such that the high ADHD-associated symptoms group had larger N1 amplitudes across all electrode sites, regardless of whether the tone was a target or a distractor, relative to the low ADHD-

associated symptoms group. This finding lends some partial support to the hypothesis that the high ADHD-associated symptoms group would display larger N1 amplitudes for distractor tones in both conditions, relative to the low ADHD-associated symptoms group. However, a group x tone interaction (rather than a main effect for group) had been predicted, and the larger N1 amplitudes for the high ADHD-associated symptoms group for target tones were not expected. That is, no differences were predicted between groups on the N1 amplitude of target tones. Therefore, what these findings suggest is that the high ADHD-associated symptoms group is paying more attention to both target and distractor tones, when compared to the low ADHD-associated symptoms group. One important implication of these findings is that the high ADHD-associated symptoms group is exhibiting electrophysiological evidence that they are attending more to the distracting stimuli. Therefore, these results provide electrophysiological evidence (in addition to evidence provided from the behavioral data) that the processing of the distractor tones was different between groups. Such evidence suggests that increased attention to the distractor stimuli, as indicated by larger N1 amplitudes, is likely the mechanism behind the high ADHD-associated symptoms groups' improvement in reaction time in the high probability condition. As for the larger N1 amplitude for the high ADHD-associated symptoms group for target tones, it appears that this group has a heightened arousal to the tones overall. An alternative reason for the differences in N1 amplitudes between groups may be a failure to habituate for the high ADHD-associated symptoms group.

An interaction approaching significance for tone x condition also was found for the N1 amplitude. This finding suggests that the N1 amplitude for high probability distractors

was significantly larger than that of low probability distractors, whereas the difference between N1 amplitude for target tones was not significant across conditions. Therefore, it appears that although the high ADHD-associated symptoms group exhibited larger N1 amplitudes overall, both groups reacted differently to the correlated distractor tones. Although it appears that the high ADHD-associated symptoms group was the only group to benefit behaviorally from the correlated distracting stimuli, these results may be interpreted cautiously as a ceiling effect may be present for the low ADHD-associated symptoms group, leaving improvement in reaction time difficult to accomplish.

For the P3 amplitude, a site x tone interaction approaching significance was found. However, the plot of the interaction indicated that the P3 amplitude for both scalp locations (P3 and P4) was larger for distractor tones relative to target tones. According to Escera, Alho, Winkler, and Naatanen (1998), distractor stimuli elicit a P3 component in adults. Therefore, it appears that distractor tones elicited larger P3 amplitudes for both groups. A site x condition x group interaction approaching significance was also found. When probed in post-hoc analyses, however, this three-way interaction revealed that the mean P3 amplitude increased for both P3 and P4 electrode sites and both groups in the low probability condition, but not the high probability condition. So, the interaction appears non-interpretable and the findings are suggestive of an effect for condition. As mentioned previously, P3 amplitudes are thought to be elicited by distractor tones, which suggest that distractor tones in the current study were more distracting in the low probability condition, thus resulting in larger P3 amplitudes, relative to the high probability condition where the distractors are more helpful and are not truly distracting. In summary, marginally significant findings along with a pattern in the post-hoc analyses

more consistent with effects for tone (in the two-way interaction) and condition (in the three-way interaction) deemed these two interactions not interpretable. This lack of significant interactions, however, is not surprising as differences between parietal lobe site locations on condition and group does not make sense theoretically. Likewise, differences between site and tone do not make sense theoretically.

The hypotheses that larger P3 amplitudes would be present for the high ADHD-associated symptoms group in the high probability condition relative to the low probability condition and that the high ADHD-associated symptoms group would have larger P3 amplitudes across conditions relative to the low ADHD-associated symptoms group were not supported. However, because in preliminary analyses the P3 amplitude was only present in the parietal lobe electrode site, this hypothesis was no longer expected as it is suggested in previous research that attentional deficits are most prominent in the frontal lobe (Posner & Rothbart, 2000). Therefore, lack of meaningful group differences further support theories that deficits in selective attention are primarily a result of frontal lobe dysfunction and that parietal lobe function appears to be intact in individuals with symptoms of ADHD.

Findings by Lobe

The electrode sites were next grouped together by lobe (frontal, central, and parietal) and N1 and P3 amplitudes were analyzed. Grouping together the electrodes was effective in decreasing the chance of a Type 1 error because the number of factors entered into the ANCOVA was reduced. These analyses not only allowed a replication of the tests for the two-way and three-way interactions for the N1and P3 amplitudes which had been tested at the electrode site level, but also involved a direct test of the hypothesis

predicting a significant interaction between group, tone, and site for the N1 amplitude. Therefore, these results are considered the most meaningful for the current study. Again, none of the *a priori* hypotheses were directly supported; however, some meaningful results did emerge.

Specifically, results of the N1 analyses revealed a main effect approaching significance for group, with the high ADHD-associated symptoms group having a larger N1 amplitude overall relative to the low ADHD-associated symptoms group. As with the analyses conducted on the eight separate electrode sites, this finding lends some partial support for the hypotheses presented in the current study. Specifically, this main effect suggests that the high ADHD-associated symptoms group yielded larger N1 amplitudes for distractor tones in both conditions. On the other hand, group differences on the N1 amplitude for target tones were found, which was not predicted. Additional results suggest a tone x condition interaction approaching significance. Post-hoc analyses, however, resulted in non-significant findings.

Results of the P3 amplitudes analyzed by lobe yielded non-significant findings. Because only the parietal lobe sites were found to have P3 amplitudes in preliminary analyses, only the parietal lobe data were analyzed. As mentioned previously, research on attention and selective attention report that deficits are found mostly in the frontal lobe (Barry et al., 2001, Hynd et al., 1990; Shue & Douglas, 1992). Thus, when considering the hypotheses *a posteriori*, no differences between groups would be expected when examining the parietal lobe. As such, no significant main effects or interactions were found for P3 amplitudes in the parietal lobe.

Findings by Region

Lastly, follow-up analyses were conducted to examine any differences between groups that may have been present in the left, right, or midline regions of the brain. As these analyses were more exploratory in nature, no *a priori* hypotheses were made for these data. Because preliminary analyses indicated that only P3 and P4 electrode sites presented P3 amplitudes, this amplitude could not be analyzed by the left, right, and midline hemisphere grouping. Therefore, N1 amplitudes were the only amplitudes analyzed in the follow-up analyses. A main effect approaching significance for group was found. This result suggested, as expected from previous findings, that the high ADHD-associated symptoms group had larger N1 amplitudes overall relative to the low ADHD-associated symptoms group. A significant site x tone interaction was found such that the N1 amplitudes in the right hemisphere of the brain were larger for target tones relative to distractor tones. The amplitudes for the left and midline hemispheres did not change significantly across tones. These results are supported by previous research suggesting that auditory selective attention tasks elicit greater right hemisphere activity relative to left hemispheric activity (Petit et al., 2007). Finally, a tone x condition interaction approaching significance was found. Results suggested that the mean N1 amplitude for target tones was larger than the amplitude for distractor tones in the low probability condition. The results for the high probability condition were not significant. As stated by Luck (2005), N1 amplitudes are often elicited by attending to something. Therefore, what these results suggest is that, in the low probability condition, all of the participants were attending more to the target tones. However, in the high probability condition, the difference between the target and distractor tones was not significant. Therefore, it

appears that both groups were attending more to the distractor tones in the high probability condition.

Overall Summary of Findings

The main findings of the current study were that the high ADHD-associated symptoms group had a slower reaction time, but benefited behaviorally from correlated information, as exhibited by the group's improvement in reaction time for the high probability condition. In terms of the electrophysiological findings, there appear to be differences between the high and low ADHD-associated symptoms groups such that the high ADHD-associated symptoms group consistently displayed larger N1 amplitudes relative to the low ADHD-associated symptoms group, suggesting greater distractibility from the distractor tones as well as greater arousal from the target tones. Both groups appeared to react differently to distractor tones in the high probability condition, although the high ADHD-associated symptoms group was the only group benefiting from this behaviorally. These results partially support an evolutionary hypothesis that there may have been some advantage to having symptoms of ADHD in the past.

Other relevant findings suggest that the N1 amplitude, which is associated with attention, is largest in the frontal lobe. The P3 amplitude was larger for distractor tones. However, in the high probability condition, the P3 amplitudes were not as large, suggesting that the distractor tones were less distracting when the probability of a correlation was high. Lastly, the right hemisphere appeared to have the largest N1 amplitude across groups.

Implications

Results from the neurocognitive task suggest that correlated distractors are beneficial for adults who have difficulty selectively attending. Hartmann (2003) describes individuals who are easily distracted as "easily attracted to new stimuli" (p. 4). Therefore, in the current study, it was shown that adults with high levels of symptoms of ADHD (including attentional deficits) are easily attracted to distractor stimuli, which under some circumstances can be helpful.

Therefore, it may be beneficial for adults who have difficulty sustaining and selectively attending to have auditory signals related to the task they wish to complete. For example, a college student may have a specific alarm tone that goes off throughout the night when they are working on a term paper. The alarm, which may be distracting in some situations, would serve as a reminder to the student to get back on task and continue working on the assignment. This is similar to the findings by van Mourik and colleagues (2007) in which they suggest that distracting stimuli can stimulate a child with ADHD enough to get them back on task.

The findings of the current study also have implications as far as careers in which individuals with ADHD may wish to pursue. For instance, an individual with ADHD may perform better in a career where they have a variety of duties and are not required to focus on one project for long durations. Having a deficit in selective attention may also be useful in a job where flexibility and changing strategies at a moment's notice are required. As shown in the current study, individuals with high levels of ADHD-associated symptoms are stimulated more by target and distractor stimuli (i.e., larger N1

amplitude across tones and conditions). Therefore, the ability to attend to a lot of information at once may be useful in some careers.

Although the current study focused on ADHD symptoms in adults, there are some implications that can be generalized to children with ADHD-associated symptoms. For example, teachers and educators may wish to engage these children in classroom activities that involve correlated information. A potential classroom activity might involve the children raising their hand throughout the class period to answer a question; the first child to raise his or her hand gets to answer the question. However, the teacher could set it up so that the children are only to raise their hand following the presence of some cue that is typically considered distracting and filtered out (e.g., when they hear a door slam shut in the hallway). It is likely that the children in the classroom who have difficulty selectively attending will be among the first to respond to a door slamming shut. Therefore, by increased opportunities to respond, these children will begin to feel more successful in the classroom. If a teacher chose to implement an activity that would highlight strengths of the student with ADHD, it may make learning more enjoyable and encourage the student by creating a situation in which they experience more success and less failure.

Limitations and Directions for Future Research

The current study has several notable limitations that can be addressed in future research. A relatively low sample size in the current study may be a limitation in that there was low power for detecting weak effects. For instance, with a larger sample, the data may have exhibited a P3 amplitude in the frontal and central lobe as well as the parietal lobe. Likewise, it may have been difficult to detect two-way and three-way

interactions (the main hypotheses of the study) with the current sample size. The use of a larger sample size in the future would increase power, thus making it more likely to detect true differences and interaction effects. In addition, future studies may wish to include only those individuals who have a current diagnosis of ADHD in the high ADHD-associated symptoms group rather than relying on a self-report measure of symptomatology. Specifically, rather than just rely on either a reported diagnosis or a self-report of symptoms (the latter of which was used in the current study), it may be most beneficial for future researchers to diagnose the participants with ADHD (i.e., establishing that a criterion number of symptoms are present in at least two settings; determining severity of impairment; confirming an onset of symptoms prior to the age of 7 years; APA, 2000) before allowing them to participate. The use of the Barkley ADHD checklist alone as a screener is limiting in that the symptoms endorsed may also be indicative of anxiety and depression; therefore, more specific screening of ADHD symptoms should be used in the future.

Although it was mentioned that P3 amplitudes are the most prominent in the parietal lobe (Polich & Criado, 2006; Sumich et al., 2008), it was expected that P3 amplitudes would differ between groups in the frontal lobe. A limitation to the current study that may have contributed to the lack of P3 amplitudes across electrode sites is that there were not enough tones presented. P3 amplitudes are difficult to detect if the number of trials is limited (J. M. Long, personal communication, October 10, 2008). Therefore, future research that wishes to examine P3 amplitudes in the frontal lobe should increase the number of tones presented in each condition to maximize the chance of this occurring. An alternative theory as to the lack of P3 in the frontal lobe may be due to the

participants orienting to the distractor stimuli in such a way that the distractor tones actually became a proxy for the target (i.e., because they typically signaled the onset of the target), thus causing an earlier onset of the P3 amplitude that was not detected in the analyses.

Another limitation to the current study is that the experiment was not conducted in a sound-proof room. The experimenter made efforts to provide a quiet testing environment; however, noise outside of the lab (e.g., people talking, doors slamming) may have created noise in the ERP recordings. This noise may have also provided unwanted distracting stimuli to the participants. Future research in this area should strive to provide the quietest experimental environment possible, which may include use of a sound-proof room or limiting the running of the experiment to times of the day when hallway traffic volume is low (i.e., early morning, weekends, and evenings).

An additional manipulation, such as including motivational factors (including immediate and delayed reinforcement), may provide new and interesting findings in terms of the high ADHD-associated symptoms group's performance when motivation to perform well is added to the experiment. Future researchers may also wish to conduct a similar experiment on children with ADHD. Moreover, comparing different subtypes of ADHD may lead to interesting findings. For example, results may be different for individuals with ADHD, Predominantly Hyperactive/Impulsive Type, and more similar for those who are in the Predominantly Inattentive or Combined Types because individuals without inattention deficits may not exhibit deficits in auditory selective attention. Also, it may be beneficial to collect data in the future with adults from the community rather than the majority of participants being students enrolled in a four-year

university (as was the case with the current study). University students may not be the best representation of the overall population, in which case, the generalization of these findings may be limited. If future researchers conducted a similar study with participants diagnosed with ADHD, it would be interesting to examine the differences in performance between groups of ADHD-diagnosed individuals on and off stimulant medication relative to a control group. Examination of the effects of other drugs such as caffeine, alcohol, and nicotine in this selective attention paradigm may also be interesting for future research. Potential benefits due to a deficit in selective attention may also be present in other mental health disorders (e.g., depression and anxiety), and future research may wish to examine these disorders in a similar study to determine whether there are any evolutionary advantages to having these seemingly impairing disorders. Future research should aim to match the groups by race and gender as opposed to holding them constant in the analyses.

Lastly, in terms of the analyses of electrophysiological data, the current study examined the difference between N1 and P3 amplitudes. Future studies may also wish to analyze Nd (difference negativity) between target and distractor tones. In order to do this, the methodology would have to be modified such that the same tones are used as both target and distractor tones across blocks of trials. For example, in order to examine the size of the Nd between targets and distractors, the same tone (e.g., 400 Hz) would be a target tone in one block of trials and a distractor tone in another block of trials. Therefore, in future research it may be hypothesized that the Nd would be smaller for the high ADHD-associated symptoms group due to their inability to selectively attend.

Conclusions

The present study expands on the current literature concerning the relation of neurocognitive, electrophysiological functioning, and ADHD-associated symptoms. To this date, the current study appears to be the first study to examine potential neurocognitive benefits of having symptoms of ADHD in an adult population. There is, however, one published study that examined potential neurocognitive benefits of having symptoms of ADHD in children (van Mourik et al., 2007). The current study yielded a similar pattern of findings as the research conducted by van Mourik and colleagues (2007) and provides important implications as to how a deficit in selective attention can be used in such a way that individuals with ADHD may significantly improve in their ability to selectively attend. That is, if distracting information is actually informative, a person with a deficit in selective attention may be able to perform at a level comparable to that of a person without this deficit. In addition, the current study provides evidence that the neurological processing of auditory information is different between individuals with high and low levels of ADHD-associated symptoms, thus adding further support to the literature that ADHD is a neurological disorder. This area of research is relevant in that children and adults have severe difficulties and impairments on a daily basis with regard to social, academic, and work functioning. Any information that may benefit or aid these individuals in using their abilities and/or deficits to their advantage is considered useful. Continued research should be done in this area to determine whether other deficits associated with ADHD may be beneficial under certain circumstances.

APPENDIX A

INSTITUTIONAL REVIEW BOARD APPROVAL

APPENDIX B

DEMOGRAPHICS AND BACKGROUND INFORMATION FORM

General Information:

Age: _____

Sex: Male Female

Race: Caucasian African American Asian

Hispanic Other

Date of Birth: _____

Current Occupation: _____

Behavioral History

Have you ever been diagnosed with ADHD (or ADD)? YES NO

If yes, at what age were you **diagnosed** with ADHD? _____

If yes, where or by whom were you diagnosed with ADHD?

_____

_____

Have you ever been on medication to treat attention problems or hyperactivity? YES NO

If yes, are you currently on medication to treat ADHD symptoms?

If yes, type: _____ Daily Dosage: _____/mgs ____/times per day

Is the medication sustained-release? YES NO

When was the last dosage given? _____
(day/time)

Have you ever been diagnosed with another mental health disorder (e.g., depression, anxiety)?

YES NO

If yes, are you currently taking medication to treat a mental health disorder?
YES NO

If yes, what type of medication are you currently taking? -
_____

Academic History

Have you ever been diagnosed with a learning disorder (LD)? YES NO

If yes, at what age were diagnosed with LD? _____

If yes, where or by whom was he/she diagnosed with LD?
_____
_____
_____

Have you **ever** been in any special classes at school? YES NO

Have you ever repeated a grade? YES NO

If yes, what grade(s) were you retained? _____

Highest grade completed in school:
_____

[If attended college, please enter 12+ 1 for each year attended. For example, if 2 years of college, enter 14. Bachelor's degree, enter 16; Master's degree, enter 18, Ph.D. or higher degree, enter 20.]

Medical History:

Do you require a hearing aid? YES NO

If yes, are you wearing your hearing aid today? YES NO

Do you have a hearing problem that is not corrected by a hearing aid? YES NO

If yes, please describe: _____

Do you have a history of seizures? YES NO

Do you have a history of a traumatic brain injury (TBI)? YES NO

Family Information:

Current Marital Status:

___ Never married
___ Currently married
___ Currently living together
___ Separated
___ Divorced
___ Widowed

Taking into account all sources of income (wages, interest, government assistance, child support, etc.), please estimate the total family income on a yearly basis BEFORE taxes. *(This is for research purposes **ONLY**. No identifying information will be paired with these data*)

(**Enter corresponding Number from column at right)**

_____

0= Earns no income/dependent on welfare
1=Earns less than $10,000
2= $10,000- $14,999
3= $15,000- $ 19,999
4= $20,000- $ 24,999
5= $25,000- $29,999
6=$30,000- $ 34,999
7= $35,000- $39,999
8= $40,000- 49,999
9= $50,000- $59,999
10= $60,000- $ 74,999
11= $ 75,000- $99,999
12= Earns $100,000 or more

Are you receiving any form of government assistance (e.g. AFCD, SSI)? YES NO

*(This is for research purposes **ONLY**. No identifying information will be paired with these data*)

<u>Drugs and Alcohol:</u>

Do you drink caffeinated beverages? YES NO

If yes, what is the <u>average number</u> of caffeinated beverages that you consume in a day?

_____

If yes, when was the <u>last time</u> that you consumed a caffeinated beverage?

_____

(day/time)

If yes, what was the amount of your last consumption of caffeine ?

_____

For example: 1 cup of coffee.

Do you smoke or use other nicotine products? YES NO

If yes, what is the average amount of nicotine that you use in a day?

_____

If yes, when was the last time that you used nicotine?

_____

(day/time)

If yes, what was the amount of your last consumption of nicotine?

_____

Do you drink alcoholic beverages? YES NO

If yes, what is the average number of alcoholic beverages that you consume in a day?

_____

If yes, when was the last time that you consumed an alcoholic beverage?

_____

(day/time)

If yes, what was the amount of your last consumption of alcohol?

_____

For example: 1 beer.

Do you use any illicit drugs? YES NO

If yes, what is the average amount of illicit drugs that you use in a day?

_____

If yes, when was the last time that you used illicit drugs?

_____

(day/time)

If yes, what was the amount of your last consumption of illicit drugs?

_____

REFERENCES

Aman, C. J., Roberts, R. J., & Pennington, B. F. (1998). A neuropsychological examination of the underlying deficit in attention deficit hyperactivity disorder: Frontal lobe versus right parietal lobe theories. *Developmental Psychology, 34*, 956-969.

American Psychiatric Association (2000). *Diagnostic and statistical manual of mental disorders.* (4th ed., rev.). Washington, DC: Author.

Amso, D., & Johnson, S. P. (2005). Selection and inhibition in infancy: Evidence from the spatial negative priming paradigm. *Cognition, 95*, B27-B36.

Babor, T. F., Higgins-Biddle, J. C., Saunders, J. G., & Montiero, M. G. (2001). *AUDIT: The alcohol use disorders identification test; guidelines for use in primary care* (2nd ed.). Geneva, Switzerland: World Health Organization Department of Mental Health and Substance Dependence.

Barkley, R. A., Murphy, K. R., & Fischer, M. (2008). ADHD in adults: what science says. New York: Guilford Press.

Barkley, R. A,. & Murphy, K. R. (1998). *Attention-deficit hyperactivity disorder*: *A clinical workbook* (2nd ed.). New York: Guilford Press.

Barry, T. D., Klinger, L. G., Lyman, R. D., Bush, D., & Hawkins, L. (2001). Visual selective attention versus sustained attention in boys with attention deficit/hyperactivity disorder. *Journal of Attention Disorders, 4*, 193-201.

Bartgis, J., Lilly, A. R., & Thomas, D.G. (2008). Event-related potentials and behavioral measures of attention in 5-, 7-, and 9-year-olds. *The Journal of General Psychology, 130*, 311-335.

Bezchlibnyk-Butler, K. Z., & Jefferies, J. J. (1997). *Clinical handbook of psychotropic drugs* (7th ed.). Ashland, OH: Hogrefe & Huber Publishers.

Biederman, J., Faraone, S. V., Keenan, K., Benjamin, J., Krifcher, B., Moore, C., et al. (1992). Further evidence for family-genetic risk factors in attention deficit hyperactivity disorder: Patterns of comorbidity in probands and relatives in psychiatrically and pediatrically referred samples. *Archives of General Psychiatry*, *49*, 728-738.

Biederman, J., Faraone, S. V., & Kiely, K. (1996). Comorbidity in outcome of attention-deficit/hyperactivity disorder. In Hechtman, L. T. (Ed). *Do they grow out of it? Long term outcomes of childhood disorders* (pp. 39-75). Washington, DC: American Psychiatric Association.

Biederman, J., Faraone, S. V., Mick, E., Spencer, T., Wilens, T., Kiely, K., et al. (1995). High risk for attention deficit hyperactivity disorder among children of parents with childhood onset of the disorder: A pilot study. *American Journal of Psychiatry, 152,* 431-435.

Bonala, B., Boutros, N. N., & Jansen, B. H. (2008). Target probability affects the likelihood that a P3 will be generated in response to a target stimulus, but not its amplitude. *Psychophyisiology, 45*, 93-99.

Broadbent, D. E. (1952). Listening to one of two synchronous messages. *Journal of Experimental Psychology, 44,* 51-55.

Butchner, A. T. (2002). Symptom dimensions and neurocognitive functioning in adult ADHD. *Dissertation Abstracts International: Section B: The Sciences and Engineering, 62,* 3373.

Casey, B., Castellanos, F., Giedd, J., Marsh, W., Hamburger, S., & Marsh, W. L. (1997). Implication of right frontostriatal circuitry in response inhibition and Attention-Deficit/Hyperactivity Disorder. *Journal of the American Academy of Child and Adolescent Psychiatry, 36*, 374-383.

Cheour, M., Leppanen, P. H. T., & Kraus, N. (2000). Mismatch negativity (MMN) as a tool for investigating auditory discrimination and sensory memory in infants and children. *Clinical Neurophysiology, 11,* 4-16.

Clarke, A. R., Barry, R. J., Heaven, P. C. L., McCarthy, R., Selikowitz, M., & Byrne, M. K. (2008). EEG coherence in adults with attention-deficit/hyperactivity disorder. *International Journal of Psychophysiology, 67*, 35-40.

Conley, T. B. & O'Hare, T. (2006). Factoral validity of the alcohol use disorders identification test: Drunk drivers versus college students. *Journal of Social Work Practice in the Addictions, 6*, 25-36.

Dunn, W. D., & Kronenberger, W. G. (2003). Attention-deficit/hyperactivity disorder in children and adolescents. *Neurologic Clinics*, *21*, 933-940.

Escera, C., Alho, K., Winkler, I., Naatanen, R. (1998). Neural mechanisms of involuntary attention to acoustic novelty change. *Journal of Cognitive Neuroscience, 10*, 590-604.

Gomes, H., Barrett, S., Duff, M., Barnhardt, J., & Ritter, W. (2008). The effects of interstimulus interval on event-related indices of attention: an auditory selective attention test of perceptual load theory. *Clinical Neurophysiology, 119*, 542-555.

Gomes, H., Duff, M., Barnhardt, J., Barrett, S., & Ritter, W. (2007). Development of auditory selective attention: event-related potential measures of channel selection and target detection. *Psychophyisiology, 44*, 711-727.

Goodman, D.W. (2007). The consequences of attention-deficit/hyperactivity disorder in adults. *Journal of Psychiatric Practice, 13*, 318.

Hartmann, T. (2003). *The Edison gene: ADHD and the gift of the hunter child.* Rochester, VT: Park Street Press.

Heaton, S. C., Reader, S. K., Preston, A. S., Fennell, E. B., Puyana, O. E., Gill, N., et al. (2001). The test of everyday attention for children (TEA-CH) patterns in performance in children with ADHD and controls. *Child Neuropsychology, 7,* 251-264.

Hooks, K., Milich, R., & Lorch, E. P. (1994). Sustained and selective attention in boys with attention deficit hyperactivity disorder. *Journal of Clinical Child Psychology, 23*, 69-77.

Hynd, G. W., Semrud-Clikeman, M., Lorys, A. R., Novey, E. S., & Eliopulos, D. (1990). Brain morphology in development dyslexia and attention deficit disorder/hyperactivity. *Archives of Neurology, 47,* 919-926.

Johnstone, S. J., Barry, R. J., Markovska, V., Dimoska, A., & Clarke, A. R. (2009). Response inhibition and interference control in children with AD/HD: a visual ERP investigation. *International Journal of Psychophyisiology, 72,* 145-153.

Jonkman, L. M., Kemner, C., Verbaten, M. N., Koelega, H. S., Camfferman, G., v.d.-Gaag, R. J., et al. (1997). Event-related potential and performance of attention-

deficit hyperactivity disorder: Children and normal controls in auditory and visual selective attention tasks. *Biological Psychiatry, 41*, 595-611.

Knott, V., Blais, C., Scherling, C., Camarda, J., Millar, A., Fisher, D., et al. (2006). Neural effects of nicotine during auditory selective attention in smokers: an event-related potential study. *Neuropsychobiology, 53*, 115-126.

Luck, S. J. (2005). *An introduction to the event-related potential technique.* Cambridge, MA: MIT Press.

Manly, T., Anderson, V., Nimmo-Smith, I., Turner, A., Watson, P., & Robertson, I. H. (2001). The differential assessment of children's attention: The test of everyday attention for children (TEA-CH), normative sample and ADHD performance. *Journal of Child Psychology, 42,* 1065-1081.

Melara, R. D., & Algom, D. (2003). Driven by information: A tectonic theory of Stroop effects. *Psychological Review, 110*, 422-471.

Mostofsky, S. H., Cooper, K. L., & Kates, W. R. (2002). Smaller prefrontal and premotor volumes in boys with attention-deficit/hyperactivity disorder. *Biological Psychiatry, 52*, 785-794.

Pelham, W. E., Fabiano, G. A., & Massetti, G. M. (2005). Evidence-based assessment of attention deficit hyperactivity disorder in children and adolescents. *Journal of Clinical Child and Adolescent Psychology, 34*, 449-476.

Petit, L., Simon, G., Joliot, M., Andersson, F., Bertin, T., Zago, et al. (2007). Right hemisphere dominance for auditory attention and its modulation by eye position: An event related fMRI study. *Restorative Neurology and Neuroscience, 25,* 211-225.

Polich, J., & Criado, J. R. (2006). Neuropsychology and neuropharmacology of P3a and P3. *International Journal of Psychophysiology, 60*, 172-185.

Porjesz, B., Rangaswamy, M., Kamarajan, C., Jones, K. A., Padmanabhapillai, A., & Begleiter, H. (2005). The utility of neurophysiological markers in the study of alcoholism. *Clinical Neurophyisiology, 116,* 993-1018.

Posner, M. I., & Rothbart, M. K. (2000). Developing mechanisms of self regulation. *Development and Psychopathology, 12*, 427-441.

Prentkowski, E. (2008). *Attention-Deficit/Hyperactivity Disorder: Potential advantages in auditory selective attention*. Unpublished masters thesis, The University of Southern Mississippi, Hattiesburg.

Pritchard, V. E., & Neumann, E. (2004). Negative priming effects in children engaged in nonspatial tasks: Evidence for early development of an intact inhibitory mechanism. *Developmental Psychology*, *40,* 191-203.

Rao, A. (1998). Plasticity of auditory selective attention in adults: A behavioral and physiological analysis. *Dissertation Abstracts International: Section B: The Sciences and Engineering, 58,* 4701.

Riccio, C. A., Wolfe, M., Davis, B., Romine, C., George, C., & Lee, D. (2004). Attention deficit hyperactivity disorder: manifested in adulthood. *Archives of Clinical Neuropsychology*, *20*, 249-269.

Rubia, K., Smith, A. B., Brammer, M. J., & Taylor, E. (2007). Temporal lobe dysfunction in medication-naïve boys with attention-deficit/hyperactivity disorder during attention allocation and its relation to response variability. *Biological Psychiatry, 62*, 999-1006.

Satterfield, J. H., Schell, A. M., Nicholas, T., & Backs, R. W. (1988). Topographic study of auditory event-related potentials in normal boys and boys with attention deficit disorder with hyperactivity. *Psychophysiology, 25*, 591-606.

Shelley-Tremblay, J. F., & Rosen, L. A. (1996). Attention deficit hyperactivity disorder: an evolutionary perspective. *The Journal of Genetic Psychology, 157*, 443-453

Shue, K. L., & Douglas, V. I. (1992). Attention deficit hyperactivity disorder and the frontal lobe syndrome. *Brain and Cognition, 20,* 104-124.

Sumich, A., Kumari, V., Dodd, P., Ettinger, U., Hughes, C., Zachariah, E., & et al. (2008). N100 and P3 amplitude to go and no-go variants of the auditory oddball in siblings discordant for schizophrenia. *Schizophrenia Research, 98*, 265-277.

Styles, E. A. (1997). *The psychology of attention.* New York: Psychology Press.

Tannock, R. A. (1998). Attention deficit hyperactivity disorder: Advances in cognitive, neurobiological, and genetic research. *Journal of Child Psychology an Psychiatry, 39,* 65-99.

Thompson, L., & Thompson, M. (2005). Neurofeedback intervention for adults with ADHD. *Journal of Adult Development, 12,* 123-130.

van Mourik, R., Oosterlaan, J., Heslenfeld, D. J., Konig, C. E., & Sergeant, J. A. (2007). When distraction is not distracting: a behavioral and ERP study on distraction in ADHD. *Clinical Neurophysiology, 118,* 1855-1865.

Wadsworth, J. S., & Harper, D.C. (2007). Adults with attention-deficit/hyperactivity disorder: assessment and treatment strategies. *Journal of Counseling and Development, 85*, 101-108.

Waxmonsky, J. G. (2005). Nonstimulant therapies for attention-deficit hyperactivity disorder (ADHD) in children and adults *Essential Psychopharmacology, 6*, 262-276.

Wechsler, D. (1999). *Wechsler Abbreviated Scale of Intelligence-manual.* San Antonio, TX: Pearson.

White, J. N., Hutchens, T. A., & Lubar, J. F. (2005). Quantitative EEG assessment during neuropsychological task performance in adults with attention deficit hyperactivity disorder. *Journal of Adult Development, 12*, 113-121.

Willoughby, M. T. (2003). Developmental course of ADHD symptomatology during the transition from childhood to adolescence: a review with recommendations. *Journal of Child Psychology and Psychiatry, 44,* 88-106.