THE EXECUTIVE AND ACADEMIC FUNCTIONING OF STIMULANT-MEDICATED AND NON-STIMULANT MEDICATED ADULT COLLEGE STUDENTS WITH ADHD: A NEUROPSYCHOLOGICAL PERSPECTIVE

by

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A Dissertation Presented to the
FACULTY OF THE USC ROSSIER SCHOOL OF EDUCATION
UNIVERSITY OF SOUTHERN CALIFORNIA
In Partial Fulfillment of the
Requirements for the Degree
DOCTOR OF EDUCATION

May 2012

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Acknowledgements

I would like to thank Dr. Pat Tobey, Dr. Stephen Madigan, and Dr. John Monterosso for their guidance during my time at the University of Southern California. To Erik, Eddie, and Sara for being amazing, supportive friends who helped me laugh when I wanted to cry. As for my family, I could not have done it without your love and support….Mom, Dad, Chad, Clint, Grandma Sidney and Grandpa Dorsey, I thank you. Also, to my Aunt Terry and Uncle Tony who have provided me with non-relenting support over the years. Thank you to Grandma Sidney and Mr. Cary Nerelli for teaching me how to write. You have made this process so much easier. Lastly, thank you to my amazing and supporting boyfriend Brian Olson for all the love and compassion a woman could ask for. This dissertation is dedicated to all of you that I love and to every boy, girl, man, and woman coping with ADHD. You are my inspiration and I thank you.

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Abstract

The purpose of this study was to determine whether stimulant-medicated adult college students with ADHD outperformed non-stimulant medicated students on neuropsychological and academic measures. A convenience sample of 33 college students with ADHD volunteered to take two neuropsychological assessments designed to measure executive functioning: The Test of Variables of Attention (T.O.V.A.) and the Kaufman Short Neuropsychological Assessment Procedure (K-SNAP). Participants' academic functioning was assessed by completion of an ADHD Indicator Survey/Questionnaire, requiring disclosure of cumulative Grade Point Average (G.P.A.) Statistical analysis of scores on the measures indicated that there is not a significant difference between the executive and academic functioning of stimulant-medicated and non-stimulant medicated ADHD college students as indicated by T.O.V.A. and K-SNAP scores. There also was not a significant difference between the cumulative G.P.A. of stimulant-medicated and non-stimulant medicated students. However, there was a weak positive relationship between T.O.V.A. scores and self-reported cumulative G.P.A.

Chapter 1: Introduction

Attention-Deficit/Hyperactivity Disorder is a condition that was once thought of as a childhood disorder that eventually is outgrown, but now there is evidence to the contrary. In 2008- 2009, students with ADHD accounted for 18% of the population with disabilities enrolled in postsecondary institutions (NCES, 2011). While learning disabilities (LD) are the most common type of disability reported in postsecondary settings, Attention-Deficit/Hyperactivity Disorder (ADHD) is reported as the second most common (NCES, 2011) and frequently exists in comorbidity with an LD. With post-secondary institutions are reporting high numbers of students possessing this disorder, it is necessary that research which has historically focused on children be further conducted on the adult population. Questions of how ADHD influences the executive and academic functioning of an adult patient remain unanswered, especially when factoring in commonly prescribed treatments such as stimulant medication. ADHD does not appear to be going away or fading with physiological maturity; therefore, additional study is needed in this area.

The Problem

While there is no shortage of empirical research on childhood Attention Deficit Hyperactivity Disorder (ADHD), the same cannot be said for adults with the same condition. Very little research describes the characteristics of ADHD as it persists into adulthood (Doyle, 2006; Ossmann & Mulligan, 2003), resulting in a number of unanswered questions regarding manifestation of the disorder during life's later stages of development. Even greater knowledge gaps exist within the realm of adult ADHD executive and academic functions and the influence stimulant medication used to treat the disorder may have on those functions. The understanding

of certain aspects of ADHD has transferred from child populations to adults; yet, neuropsychological and medication findings are currently unknown. The neuropsychological investigation of adults with this disorder remains in the developing stages (Hervey, Epstein, & Curry, 2004). As a result, additional research in the area of adult ADHD is not only appropriate but necessary.

It is suspected that ADHD has a negative affect on executive functioning, or those cognitive processes thought to guide self-regulated and goal-oriented behaviors such as initiative, motivation, planning, judgment and attention shifting (Gualtieri & Johnson, 2006) In terms of executive function (EF), research indicates that ADHD appears to influence EF, yet which cognitive functions are affected, to what degree they are affected, and what (if any) are the academic implications of these dysfunctions remains a subject of debate. As a result, there is no broad agreement of which neuropsychological deficits are a manifestation of attention deficit hyperactivity disorder (Hervey et al., 2004). Actually, conflicting hypotheses regarding EF dysfunction have resulted from a number of studies yielding inconsistent results on neuropsychological assessments (Barkley, 2007; Boonstra, Oosterlann, Sergeant, Buitelarr, 2005; Sergeant, 2005; Sonuga-Barke, Bitsakou, & Thompson, 2010). Because neuropsychological functioning among adults with ADHD tends to vary with regard to research findings, additional studies in this area are needed.

Though stimulant medications are commonly used to treat both children and adults with ADHD, their efficacy rates among the adult population remain unclear. The response rates adults have to these medications have not been as consist as child and adolescent response rates (Adler & Chua, 2002). A significant lack of controlled studies regarding stimulant medication effects on adults with ADHD currently exists in psychological research (Kinsbourne, De Quiros, &

Tocci Rufo, 2001). Therefore, even less knowledge is available concerning the potential influences of stimulant medication on adult executive functioning. While stimulants appear to positively influence the EF's of adults with ADHD (Barnett, Maruff, Vance, Luk, Costin, Wood, & Pantelis, 2001; Kempton, Vance, Maruff, Luk, Costin, & Pantelis, 1999), the results of other studies failed to provide support for this hypothesis (Biederman, Seidman, Petty, Fried,Doyle, Cohen, Kenealy, & Farone, 2008). The potential influences of stimulant medication on adult ADHD executive functioning requires further research due to inconsistencies among findings and general lack of study.

Purpose

The purpose of this study was to address knowledge gaps in the area of potential stimulant medication influences on the executive and academic functioning of adults with ADHD with a multidisciplinary approach. Using a neuropsychological perspective, this study had implications on educational psychology and higher education considering the measures and population used. Because adult ADHD has not been studied as thoroughly as childhood ADHD, little is known about the executive and academic functions of medicated and medication naïve college students possessing the disorder. This study analyzed the functioning of college students with ADHD using a between groups comparison of stimulant medicated and non-medicated participants.

Research Questions

This study was designed to examine the executive and academic functioning of stimulant medicated and non-medicated adults with ADHD. To guide this research, the following research questions were both proposed and addressed prior to study:

1. Is there a significant difference between stimulant medicated and non-medicated ADHD students' executive functioning as measured by scores on neuropsychological assessments?

    a. It is hypothesized in this study that those taking stimulant medications will out- perform non-medicated controls on tests of executive function.

2. Do ADHD students with higher EF scores have on average a higher grade point average (G.P.A.) than those with lower EF scores?

    b. It is hypothesized that G.P.A. is positively correlated with EF scores.

3. Do stimulant medicated ADHD students have a higher G.P.A. than non-medicated controls?

    c. It is hypothesized that students taking stimulant medication will have higher G.P.A.'s than non-medicated controls.

Guiding Theories for Research

The preceding research questions and hypotheses were based on the following theories: Barkley's (1997) *Inhibitory Hypothesis of ADHD* and the *Dopamine (DA) Hypothesis of ADHD* (Wender, 1971; Levy, 1990). Based on this theory, it was hypothesized that inhibitory deficits will negatively affect EF areas of attention and working memory (WM) as measured by neuropsychological assessment. Because DA is thought to be deficient in the ADHD brain, thus leading to potential executive dysfunction, it was hypothesized that subjects taking stimulant medication will out-perform non-stimulant medicated subjects on academic and EF measures used in the study.

Importance of the Study

The relevance of this study was significant in that a number of individuals in the psychological and educational fields may benefit from its findings. First, knowledge gaps in the area of adult ADHD and the influence of stimulant medications on executive and academic functions were addressed by answering questions posed by the study. This allows college students and psychologists to gain a better understanding of how stimulant medication takers and non-takers function academically and neuropsychologically. As a result, these individuals are more informed regarding stimulant medication as an option for treatment and better able to decide whether these medications are right for them and their patients. College students with ADHD may use this as an additional reference when reflecting upon their own academic and executive function.

In addition, the study was important since it allowed for the collaboration of psychological and educational experts with the goal of achieving additional understanding of ADHD as an adult condition. The cross-disciplinary nature of this study encompassed theories from the fields of education, psychology, and neuroscience and used these theories as a foundation for research hypotheses. However, the ultimate goal was for all interested parties to gain additional insight concerning adult ADHD, its treatment, how those taking (and choosing not to take) medication are functioning from an academic and neuropsychological perspective at the college level.

Definition of Terms

Amygdale: Region of brain that acts as an "alarm" center, relaying signals to the prefrontal cortex regarding potential dangers or punishments.

Attention-Deficit/Hyperactivity Disorder: A disorder typically diagnosed during childhood characterized by inattention, hyperactivity, and impulsivity.

Bottom-Up Cognitive Processes: Constructivist approach to cognitive processing where the individual pieces together information to understand a larger picture (Gaultieri & Johnson, 2006).

Catecholamines: Class of neurotransmitters consisting of norepinephrine, dopamine, and epinephrine (Waxman, 2003).

Caudate Nucleus: Portion of basal ganglia implicated in dopamine synthesis (Waxman, 2003).

Cerebellum: Area of the brain responsible for balance and coordination of movement.

Comorbid: A term used to describe the presence of two or more psychological conditions simultaneously.

Corpus Callosum: Brain structure responsible for linking the left and right hemispheres of the brain.

Crystallized Intelligence: Crystallized intelligence is the ability to use skills, knowledge, and experience (Horn & Cattell, 1966).

Diagnostic and Statistical Manual of Mental Disorders IV and V: Manual used by mental health professionals to diagnose mental and psychological disorders based on a common criteria.

Dopamine: Neurotransmitter influencing motor and cognitive functions which is synthesized in the basal ganglia and suspected to be deficient in the ADHD brain.

Executive Functioning: Executive functions (EF) consist of cognitive processes that facilitate self-regulated, goal-oriented, autonomous behavior.

Fluid Intelligence: The capacity to think logically and solve problems in novel situations, independent of acquired knowledge (Horn & Catell, 1966).

Forethought: Executive function that requires pre-planning and subsequent adjustment of behavior to accommodate for upcoming events.

Frontocerebellar Circuit: Neural circuits linking the frontal lob with the cerebellum.

Frontolimibic Circuit: Neural circuits linking the frontal lobe with the insular lobe.

Frontostriatal Circuit: Neural circuits linking the frontal lobe with the striatum, or deeper structures of the brain.

Functional Magnetic Resonance Imaging (fMRI): A medical imaging procedure used to evaluate the hemodynamic response (blood flow following neuronal firing) in specific regions of the brain when subjects are presented a stimulus.

Genetic Loci: Region of a chromosome where genes suspected to be involved in the manifestation of certain traits or disorders are found.

Half-Life: The amount of time it takes for one half of a medication to be metabolized by the body.

Heterogeneity: A term used to describe the uniqueness of a condition's symptomology as it varies from individual to individual.

Hindsight: The ability to recall prior experiences in various situations to shape and guide behaviors.

Inhibition: The cognitive ability to prevent, stop, or suppress the initiation of a physical response.

Locus Ceruleus: Cells in the brain stem responsible for norepinephrine synthesis.

Neurotransmitters: Chemicals responsible for relaying neural impulses from one neuron to another, such as dopamine and norepinephrine.

Noradrenergic: Neurons activated or stimulated by the release of noradrenaline.

Norepinephrine: Catecholamine responsible for influencing sympathetic cognitive and behavioral responses.

Nucleus Accumbuns: Brain region implicated in reward processing and appraisal.

Occipital Lobe: Region of brain responsible for processing messages from the visual system.

Orbitofrontal Cortex: Region of Prefrontal Cortex involved in decision-making.

Parietal Lobe: Region of the brain containing the somatosensory cortex, which interprets sensations from different areas of the body and contributes to movement of these areas.

Prefrontal Cortex: Area of the brain responsible for regulating executive functions such as planning, prioritizing, sequencing, initiating, and inhibiting actions and behavior (Waxman, 2003).

Presynaptic Neuron: Neuron located just before or just prior to the postsynaptic neuron or synapse (Taber's Cyclopedic Medical Dictionary, 2005).

Postsynaptic Neuron: Neuron located after the presynaptic neuron or synapse (Taber's Cyclopedic Medical Dictionary, 2005).

Psychotropic Action: Biochemical mechanisms by which certain medications are thought to exert psychological effects.

Neuropsychology: studies the structure and function of the brain related to specific psychological processes and behaviors.

Set-Shifting: The ability to shift attention from one stimulus or situation to another.

Stimulant Medication: Medications prescribed to treat ADHD by increasing dopamine synthesis in the brain.

Stop Signal Tasks: Tasks assessing motoric inhibition that require the subject to initiate or inhibit a physical response when presented with a specific stimulus.

Temporal Lobe: Area of the brain that interprets messages from the auditory system.

Thalamus: Structure that relays neural impulses traveling up the spinal cord to various brain regions for further processing.

Top-Down Cognitive Processes: Knowledge and expectation guide one's processing of details presented to an individual (Gaultieri & Johnson, 2006).

Ventromedial Prefrontal Cortex: Portion of Prefrontal Cortex also thought to be involved in decision-making processes, possibly those involved in risky decisions and behaviors.

Working Memory: A component of short-term memory thought to play a significant role in processing new or novel information.

Chapter 2: Literature Review

Introduction

The purpose of this review of literature is to highlight some of the major theories, issues, and knowledge gaps associated with adult Attention-deficit/hyperactivity Disorder (ADHD) and its influences on executive and academic functioning in consideration of stimulant medication status. The review begins with an introduction to the topic of ADHD in terms of its definition, prevalence, signs/symptoms, diagnosis, and suspected causes. This background information is followed by a discussion of the neurobiology of the disorder, which highlights neuroanatomical and neurophysiological differences associated with ADHD. The review concludes with diagnosis and treatment protocols. This section is followed by information about executive functions and the theoretical foundations for executive dysfunctions associated with the condition. A discussion of commonly used neuropsychological assessments of executive function follows as does the role these measures play within the ADHD population. A discussion of the action of stimulant medication is then presented with regard to psychotropic action and potential influences on executive function. Lastly, the review concludes with a discussion of ADHD and the potential implications of stimulant medication on the executive and academic functioning of adult students.

Background of Attention-Deficit/Hyperactivity Disorder

The proceeding section consists of a brief background of Attention-Deficit/Hyperactivity Disorder (ADHD). Beginning with a brief history of the disorder, the prevalence of ADHD is discussed, followed by an overview of how the condition is both diagnosed and treated. This information is relevant in order to gain insight as to how the disorder affects the executive

functioning and subsequently, the academic performance of adults with ADHD at the post-secondary level.

Prevalence of the ADHD.

The National Institutes of Mental Health define ADHD as a processing deficit that can result inattention, difficulty controlling behavior, and hyperactivity (NIMH, 2011). For decades, ADHD was considered to be a condition that only affected children but the possibility of the disorder affecting an older population was essentially ignored. Only recently has ADHD been recognized as a valid adult condition (Ossmann & Mulligan, 2003). It was not until the 1970's that the psychology world began to consider the likelihood of ADHD persisting into adulthood (Adler & Chua, 2002). Therefore, research in the area of adult ADHD is relatively new (compared to child and adolescent research) and remains in emerging stages. According to Hervey, Epstein, & Curry (2004), longitudinal studies indicate that ADHD does in fact persist beyond the adolescent years. It is estimated that 30-50% of children with ADHD will continue to meet requirements for the condition as an adult, with persisting problems with inattention, hyperactivity, and impulsivity (Boonstra et al., 2005). Today, an estimated 1-6% of the adult population meets criteria for an ADHD diagnosis (Spencer, Biederman, Wilens, & Faraone, 2002), thus indicating that ADHD is a disorder that is not simply 'outgrown'. Though males tend to be diagnosed more frequently with ADHD as children, the presence of the condition is equally prevalent for the sexes as adults (Weiss & Weiss, 2004).

Diagnostic Procedures for ADHD.

Diagnosing ADHD is complex as there is no simple examination to determine the presence or absence of the disorder. Rather, diagnosis results from consideration and analysis of multiple sources of information documenting ADHD signs and symptoms over the course of an

individual's life. First, an ADHD diagnosis requires that a specialist such as a physician or clinical psychologist examine sources which document the following behaviors: hyperactivity, impulsivity and inattention (Barkley, 2007; Boonstra, Oosterlaan, Sergeant, & Buitlearr, 2005; Faraone, 2000) in home, work, or school environments. However, it is important to note that an individual may not manifest all three behaviors, creating the need for the creation of ADHD sub-types. *The Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV) identifies three sub-classifications of ADHD which include hyperactivity/impulsive inattentive, and combined types (American Psychiatric Association, 1994).

Signs of ADHD are first exhibited prior to the age of 7 (Barkley, 1997; Adler & Chua, 2002). In order to receive an ADHD diagnosis, there are fourteen total symptoms, of which both children and adults must exhibit at least eight to meet criteria for an ADHD diagnosis (Barkley, 2006; DSM-IV; APA, 1994; Faraone, 2000). According to Brown (2002) there are six areas where those with ADHD are thought to be impaired: Activation, focus, memory, effort, action, and emotion. Examples of activation deficits involve the inability to organize tasks, estimate time, and remain organized while difficulties with action are manifested thorough poor self-regulation (Brown, 2002). Brown (2002) also maintains that difficulties focusing, monitoring emotions, challenges with recall from short-term working memory are also classic signs of the disorder. Behavior rating scales such as those created by Brown (2003) are used to evaluate the presence of ADHD characteristics by the adult patient or those who witness his/her behaviors on a daily basis in a variety of settings.

For children, diagnosis has been rather straightforward using the current DSM-IV criteria, yet this has not been the case for adults (Faraone, 2000). ADHD symptoms in adults manifest differently from those of children. Unlike the hyperactive, 'always-in-trouble' child unable to

stay in his/her seat during class, adults with ADHD tend to procrastinate, become irritable after sitting for longer periods of time, violate traffic laws, fail to manage time wisely, or appear to lack motivation to complete tasks (Weiss & Weiss, 2004). In other words, they are not typically hyperactive. Symptoms of the disorder may not be as obvious as they are in children. This makes diagnosis more of a challenge in that adults fail to meet DSM-IV criteria (Barkley, 2006; Faraone, 2000). Researchers have found that hyperactivity and impulsivity components of ADHD improve with age (Hervey, Epstein, & Curry, 2004), resulting in more than 50% of patients no longer meeting the full diagnostic criteria according to the DSM-IV by the time they reach the age of 20 (Spencer et al., 2002). Because of DSM-IV inconsistencies, many specialists consider a personal interview with an adult patient to be the most effective diagnostic tool (Weiss & Weiss, 2004) along with behavior rating scales and the review of personal, academic and medical records when it comes to evaluating adults with ADHD.

Although personal interviews may be integral to diagnosis, one-on-one evaluation is considered in conjunction with a patient's medical and academic history. The consideration of reports from individuals who have observed the patient's behaviors in a variety of contexts and a review of academic records and psychoeducational test results should also be made to rule out other conditions prior to diagnosis (Spencer et al., 2002; Weiss & Weiss, 2004). ADHD is commonly comorbid with other disorders. Many sings and symptoms of ADHD are common to conditions such as Oppositional Defiant Disorder (ODD) or Bipolar Disorder (BPD) (Brown, 2002). It is for this reason that misdiagnosis is frequent. As an example, problems with sustained attention are common to many disorders, not just ADHD (Gualtieri & Johnson, 2006). In fact, it is quite possible that someone with ADHD also has another condition as well. According to Boonstra, Oosterlann, Sergeant, & Buitelarr (2005), 75% of adults with ADHD suffer from

another psychiatric disorder as well. Oppositional Defiant Disorder (ODD) and anxiety or mood disorders frequently affect an individual with ADHD (Whalstedt, Thorell, & Bohlin, 2008; Wilens & Dodson, 2004). According to Brown (2002), as many as 42-61% of those with ADHD have ODD, while 38% battle a comorbid anxiety disorder. In addition, it is estimated that up to 25% of patients with ADHD also suffer from a learning disability (Wilens & Dodson, 2004). This makes the diagnostic process all the more important in that comorbid disorders either need to be ruled out or treated in conjunction with ADHD.

Treatment Protocol.

Once an adult patient has been diagnosed with ADHD, a treatment protocol specifically tailored to each individual must be established. Weiss & Weiss (2004), authors of *A Guide to the Treatment of Adults with ADHD* suggest the following as a guideline for treatment: First, a screening for comorbid disorders must be conducted to determine whether or not another condition is present. This should be followed by the identification of symptoms which impair the individual the most. Ways of treating these symptoms should be identified and may include psychotherapy, medication, environmental changes, or a combination of all three. A review of academic, social, and occupational impairments should then be conducted, followed by a medication trial. In terms of follow-up, frequent, periodic reviews should be conducted to evaluate areas of improved functioning and medication efficacy. Though patients with ADHD tend to exhibit similar behaviors, manifestation of symptoms and response to treatments varies from individual to individual. It is for this reason that proper treatment requires frequent follow-up to determine program effectiveness.

Implications of ADHD

Symptoms of ADHD such as inattention, hyperactivity, and impulsivity cause impairments in occupational, social, and academic functioning (Proctor & Prevatt, 2009), which are most likely due to neuropsychological deficits attributed to the disorder. Though the reasons for poor performance in these areas remains unclear, it is hypothesized that ADHD symptoms hinder coping strategies, lead to poor organization, study skills, and time management (Wilens & Dodson, 2004). These symptoms are thought to stem from cognitive impairments, particularly in the area of executive functioning (EF). Because ADHD has been hypothesized to cause dysfunction in executive functioning (Barkely, 2007; Boonstra et al., 2005; Gaultieri & Johnson, 2006; Wahlstedt, Thorell, & Bohlin, 2008), those with the condition frequently encounter numerous academic and social challenges. These difficulties have led to an extensive amount of EF and academic research on children with ADHD and the influence of stimulant medication on these functions. However, adult literature in these same areas remains minimal at best (Adler & Chua, 2002; Doyle, 2006; Hervey et al., 2004) in spite of its prevalence. At the college level alone, it is estimated that approximately 4-5% of students possess ADHD (Barbaresi, Katusic, & Coligan, 2002; Weyandt, Linterman, & Rice, 1995; Murphy & Barkley, 1996; Barkley, Fischer, & Fletcher, 2006). Due to the frequency of this disorder and its neuropsychological implications in academic contexts, a greater understanding of how ADHD alters adult executive and academic functioning is needed.

The prevalence of ADHD in adults has been both documented and validated (Boonstra et al., 2005; Spencer et al., 2002; Weiss & Weiss, 2004). Thought to result from neuropsychological impairments, which cause executive dysfunction, the symptoms of ADHD can create significant academic challenges for adult students. In addition, neuropsychological

studies of this population are minimal, with even less information available on how stimulant medication may affect these and academic functions (Vitiello, 2001). This study attempts to contribute to knowledge deficits in these areas.

Neurobiology of ADHD

The following section consists of a review of studies which have attempted to establish potential causes of ADHD and risk factors for the disorder. A discussion of anatomical and physiological differences between the ADHD and non-ADHD brain is presented with special attention to how those differences may contribute to executive dysfunction. Key brain regions are discussed, as are neurotransmitters suspected to contribute to ADHD symptomology. Conclusions regarding genetic loci for the disorder are reviewed as they are important for understanding the psychotropic action of stimulant medications and their influences on cognition.

In an attempt to understand the neuropsychological influences of ADHD and pharmacotherapuetic treatments for the disorder, it is important to discuss what researchers hypothesize are both causing the condition as a result of what is occurring in the ADHD brain. This involves an analysis of genetics as well as evidence about how the ADHD brain differs from controls both anatomically and physiologically. In addition to gaining insight regarding the etiology of the disorder, understanding the differences between the ADHD and non-ADHD brain may account for executive and academic dysfunctions associated with the condition.

Etiology of ADHD

The exact etiology of ADHD remains a mystery, yet genetics appear to play a role in manifestation of the disorder (Elia & Dovato, 2007; Faraone, 2000). For example, certain individuals are at higher risk for the disorder than others. Research implicates genetics,

pregnancy and/or delivery complications, prenatal factors such as maternal smoking, maternal stress, socioeconomic status, and family dysfunction as potential causes (Elia & Dovato, 2007; Faraone, 2000). In addition to environmental influences, the results of family and twins studies indicate a genetic basis. The results of these studies indicate that 75% of ADHD is attributable to genetic factors, thus indicating that ADHD is a highly heritable disorder (Spencer et al., 2002). Spencer and colleagues suspect that addition to biological factors, manifestation of the disorder is thought to be a combination of genetics *and* the environment (Spencer et al., 2002) In order to determine the extent to which environmental factors influence the manifestation of ADHD, twin studies have been conducted. According to Elia & Dovato (2007) shared genes, not shared environment are suspected to explain ADHD prevalence. If a first-degree relative, such as a mother or father possesses the disorder, there is a 50% chance that the offspring of that individual will also inherit the condition (Adler & Chua, 2002). Additional family studies indicate that there is a 24% prevalence rate for children of father's with the disorder, compared to a 7% prevalence rate for non-ADHD families (Faraone, 2000). In conclusion, causes of ADHD appear to lie within both genetics and the environment.

Neuroanatomical Differences Associated with ADHD.

Geneticists use the equation P = G + E to indicate that phenotype is influenced by a combination of an organism's genotype plus its environment. Phenotypically, the ADHD brain differs in both structure and function from non-ADHD controls. In terms of the ADHD brain, imaging studies using magnetic resonance imaging (MRI) have noted structural differences in overall brain volume within the pre-frontal cortex (PFC), cerebellum, as well as the basal ganglia, all areas required for normal executive function and motor movement (Arnsten, 2006; Barkley, 1997; Bridgett & Walker, 2006). The PFC in particular plays a major role in attention,

working memory, inhibition, and planning in conjunction with all sensory and motor systems (Arnsten, 2006; Fassbender & Schweitzer, 2006). Neuroimaging reveals abnormalities in these areas of the ADHD brain. For example, the ADHD brain is slightly smaller in regions integral to normal executive function. McAlonan and Cheung (2006) found that ADHD subjects possessed abnormalities in terms of function and overall tissue volume within both the PFC and the parietocortical regions, particularly within the right hemisphere.

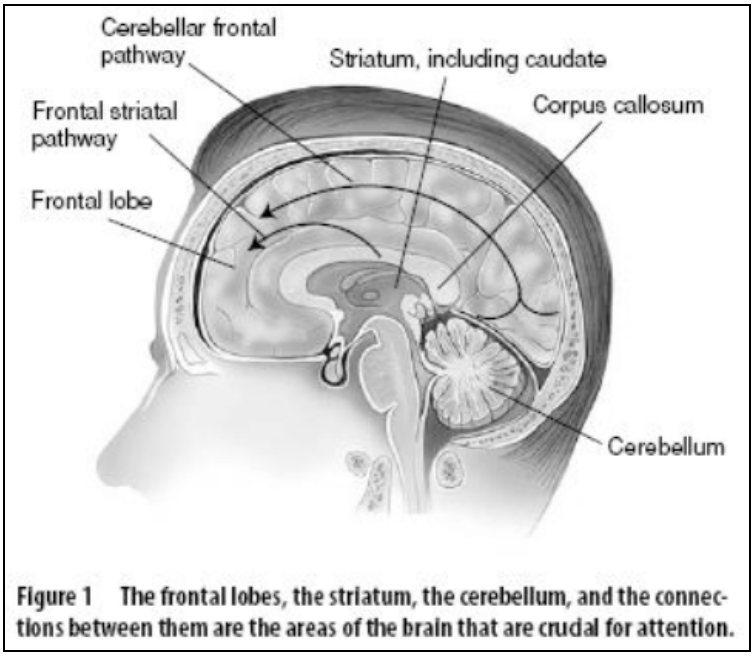

Figure 2.1: Regions of the brain and neural networks involved in executive function. Source: ADHD guides. http://adhd-guides.blogspot.com/2011/04/what-parts-of-brain-are-affected-in.html

In addition to cortical differences, reduced dimensions have been noted in deeper structures of the brain such as the caudate nucleus, corpus callosum, and cerebellar vermis (Tripp & Wickens, 2009). Krain & Castellanos (2006) and Fassbender & Schweitzer (2006) concluded that the overall volume of both grey and white matter is greater in the brains of those without ADHD. This is significant because grey and white matter are integral to functioning of neural circuitry. For the relaying of neural impulses, white matter of the adult cerebral hemisphere

contains myelinated nerve fibers responsible for connecting cortical structures with deeper striatal structures as well as the spinal cord (Waxman, 2003). Gray matter makes up not only the cerebral cortex but the basal ganglia as well. The basal ganglia is a site responsible for influencing motor control and dopamine synthesis (Waxman, 2003) as well, both factors thought to regulate motor movement and influence EF. The PFC and its association areas have neural fibers, which form neural circuits with the basal ganglia, allowing for information processing, planning, and regulation of physical movement (Arnsten, 2006). Normal functioning of the PFC and basal ganglia is fundamental to multiple cognitive functions, especially those associated with executive functioning. As a result, abnormal executive functions associated with ADHD may stem from these neurological differences but the complex nature of the brain is such that the true impact that such differences have on executive function remains in question.

Neurophysiological Differences Associated with ADHD.

Researchers suspect that in addition to anatomical differences, physiological differences within the ADHD brain may also account for executive dysfunctions associated with the disorder. Neural circuits, collectively referred to as frontal-striatal-thalamic-cortical circuitry (FSTC), are suspected to regulate multiple EF's (Roth & Saykin, 2004). The locations of these circuits and anatomical brain regions of interest are found in Figure 2.1. These circuits allow for communication between the PFC, cerebellum, thalamus, and basal ganglia and are suspected to be functioning abnormally in the brains of ADHD patients (Nigg & Casey, 2005; Roth & Saykin, 2004). Analysis of this circuitry using neuroimaging reveals that the ADHD brain appears to function differently than the non-ADHD brain, thus providing possible explanations for abnormal EF.

Each of the frontal-striatal-thalamic-cortical (FSTC) circuits appears to play a specific role in EF. For example, the dorsolateral prefrontal subcircuit has been linked directly with working memory functions (Roth & Saykin, 2004). In terms of regulating emotion and response inhibition, the orbitofrontal subcircuit works with the anterior cingulate gyrus and motor circuits to direct goal-oriented behaviors and physical response (Roth & Saykin, 2004). In addition, frontostriatal, frontolimbic, and frontocerebellar circuits are responsible for providing cognitive representations of *when* and *what* may happen in a given situation, as well as how behaviors should be adjusted in response to what actually *does* occur within a particular context (Nigg & Casey, 2005). According to Nigg & Casey (2005) these loops also are involved in timing, altering an individual to novel stimuli, avoidance conditioning, and reinforcement of learning (Nigg & Casey, 2005). Functional Magnetic Resonance Imaging shows decreased activity in the both the PFC and basal ganglia of the ADHD brain when compared to controls potentially explaining why those with ADHD fail to compute the effects and timing of their actions. This may explain why individuals with ADHD exhibit behaviors impulsively or inappropriately in response to certain stimuli. In conclusion, executive dysfunction potentially arises from differences in neural circuitry activity.

Neurotransmitters Associated with ADHD.

Another important aspect of neuropsychological processes associated with ADHD involves neurotransmitters. Being chemicals responsible for relaying neural impulses from one neuron to another, neurotransmitters dopamine (DA) and to a lesser extent, norepinephrine (NE) appear to play significant roles in ADHD. Animal models of ADHD have shown abnormal dopaminergic activity within the FSTC (Roth & Saykin, 2004), possibly altering the functioning of circuitry required for normal EF. To attest to this, DA and NE are chatecholamines responsible for the

modulation of pre-frontal and striatal functioning (Arnsten, 2006) and are hypothesized to be mediators of reward/punishment reinforcement (Tripp & Wickens, 2009). The PFC requires adequate DA and NE for the PFC to function effectively and efficiently (Arnsten, 2006). DA and NE are also thought to modulate the attention system; (Sergeant et al., 2003; Tripp & Wickens, 2009) however, DA remains the neurotransmitter of greatest interest to researchers in terms of ADHD symptomology and medication development.

With regard to DA, it is suspected that dopaminergic circuits between prefrontal and striate regions function abnormally due to biochemical deficiencies in the ADHD brain, leading to the development of the *Dopamine (DA) model of ADHD* (Wender et al., 1971; Levy, 1991). This hypothesis suggests that proper functioning of FSTC circuits may not be occurring within the ADHD brain due to excessive dopamine reuptake by presynaptic neurons (Swanson & Castellanos, 2002). This DA deficiency leads to abnormal neurological functioning. For example, poor inhibitory control and faulty reward systems are associated with problems within the DA system of neural fibers projecting to the PFC and nucleus accumbuns (Sergeant et al., 2003). In addition, imaging techniques using fMRI and structural MRI scans show smaller, less active neural circuits responsible for regulating attention, working memory, and response inhibition of those with ADHD (Wilens & Dodson, 2004). Compared to controls, Adler & Chua (2002) found low levels of DA in the PFC and overall decreased blood glucose metabolism in the PFC and pre-motor cortex of those with ADHD compared to non-ADHD subjects. These abnormalities are expected to contribute to faulty neural circuitry. Wilens & Dodson (2004) suspect that EF circuits are controlled by the actions of DA and NE release (Wilens & Dodson, 2004). Results such as these are significant because these catecholamines are thought to activate the PFC, stimulate reward processing, influence motor movements, and help humans sustain

attention (Arnsten, 2006; Tripp & Wickens, 2009). In addition to cortical action, DA is suspected to enhance focus, on-task behavior and cognition from the striatum, while NE assists with the filtering of interfering stimuli at the cortical level (Wilens & Dodson, 2004). Therefore, proper synthesis, transport, uptake, and binding of catecholamines in the brain are thought to be integral to successful executive functioning leading to goal-oriented actions. Discrepancies in these processes can offer potential explanations for the cognitive and behavioral characteristics of ADHD.

Executive function is dependent on normal catecholamine activity and appropriate functioning of neural circuitry. Abnormalities in areas of the brain such as the PFC are suspected to contribute to core deficits associated with ADHD. Faulty DA action in the ADHD brain has been supported by PET scans which show DA and noradrenergic pathways (Adler & Chua, 2002). According to Tripp & Wickens (2009) DA cells are supposed to respond to positive reinforcers by creating early cues, which are similar to memories that signal future rewards or punishments when that stimulus is again presented in one's environment. In the ADHD brain, these cues fail to develop early in development and are due to DA transfer deficits (Tripp & Wickens, 2009). As a result, proper behavioral response based on previous experience fails to develop correctly and inappropriate or unsuccessful behaviors and thought processes are maintained. Interestingly, ADHD patients exhibit many of the same characteristics as those with pre-frontal lesions such as poor behavioral inhibition, faulty reward appraisal, and deficits in working memory (WM) (Arnsten, 2006). While non-ADHD individuals are able to adequately assess potential rewards and punishments, it may be a challenge for the ADHD patient to do the same.

Anatomical and physiological differences provide justification for dysfunction of cortical and striatal functioning associated with ADHD. However, it must be noted that information collected using modern imaging techniques has its limitations. ADHD sample sizes using fMRI and PET scans have been rather small, with little research in the area of imaging with simultaneous neuropsychological testing (Roth & Saykin, 2004). Participant medication status and history in these studies was often unknown or not mentioned though such medications alter catecholamine activity. Also like other areas of ADHD research, few studies in this area have used adult participants with ADHD (Roth & Saykin, 2004). In summary, such limitations again highlight the importance of further adult ADHD research, especially in the area of neuropsychological functioning.

Genes Implicated in ADHD.

Because of the role neurotransmitters are thought to play in ADHD, genes regulating catecholamine expression and activity have been targeted as potential 'genetic markers' for the disorder. The problem is that expression of genes in question is not a pre-requisite for manifestation of ADHD traits (Spencer et al., 2002; Swanson et al., 2000) in that not all persons with the disorder possess the genes in question, nor do those with the genetic variant manifest traits of the disorder. Though ADHD is thought to be multifactorial in terms of inheritance (Spencer et al., 2002), the closest scientists have come to determining a genetic locus for the condition has been mapped to chromosome 11 (Spencer et al., 2002; Swanson, et al., 2000). However, genes on other chromosomes have also been hypothesized to contribute to symptoms of the disorder. For example, genes found on chromosomes 5,6,16, and 17 have been areas of ADHD interest, but tend to be genetic regions that overlap with autism and dyslexia (Brown, 2003). Genes such as DAT1, DRD4, SNAP-25, DRD5, 5HTT, HTR1B and DBH have been

genetically mapped to regions of various chromosomes thought to regulate neurotransmitter activity (Elia & Dovato, 2007). Multiple areas of interest on different chromosomes make mapping ADHD to a specific locus incredibly challenging.

In light of possible differences in terms of catecholamine expression, genetics studies have tended to focus on genes regulating the action of these biochemicals. Genes of greatest interest are those thought to be involved in the regulation of DA levels in the brain (Brown, 2003; Elia & Dovato, 2007; Spencer et al., 2002). It is hypothesized that a faulty dopamine (DA) receptor gene contributes to ADHD, but presence of this allele is not necessary for the condition as it is only present in about 50% of reported ADHD population (Spencer et al., 2002; Swanson et al., 2000). It is suspected that abnormal expression of the dopamine receptor gene (DRD4) and dopamine transporter gene (DAT or DAT1) decrease the amount of DA available between neurons, or synapses (Brown, 2003; Elia & Dovato, 2007). DATs are responsible for terminating DA signals between cells, (Tripp & Wickens, 2009) causing inhibitory action in neural tissue. Specifically, DAT1 is implicated in the reuptake of DA and over-active transporters may increase that action, making less DA available to fuel cognitive processes at cortical and striatal levels (Elia & Dovato, 2007; Tripp & Wickens, 2009). The presence of DAT has a dramatic effect on DA action, indicating that altered transporter expression would change the strength and time-course of this chemical's action on post-synaptic neurons (Tripp & Wickens, 2009).

Other attempts to locate ADHD genes have been conducted using animal models of the disorder. Animal models involve the use of mice, rodents, or monkeys, which are genetically engineered to display behaviors typical of a specific disorder. Animal studies have led scientists to focus on the SNAP-25 gene, a gene suspected to regulate neurotransmitter release (Elia & Dovato, 2007). According to Elia & Dovato (2007) the SNAP-25 gene has not been confirmed

in humans but could alter DA expression or reuptake in rodents, causing less biologically available DA to support neurological processes. Though a viable hypothesis, human studies have failed to confirm similar SNAP-25 action in humans with ADHD.

Recent attention has been shed on the LPHN3 gene located on chromosome 4 as a another potential genetic marker for ADHD (Arcos-Burgos, Jain, Acosta, Shilvely, Stanescu, Wallis, Domene, Velez, Karkera, Balog, Berg, Kieta, Gahl, Roessier, Long, Lie, Pineda, Londono, Palacio, Arbelaez, Lopera, Elia, Hakonarson, Johansson, Knappskig, Haavik, Ribases, Cormand, Baynes, Casas, Ramos-Quiroga, Hervas, Maher, Faraone, Seitz, Freitg, Palmason, Meyer, Romanos, Walitz, Hemminger, Warnke, Romanos, Renner, Jacob, Lesch, Swanson, Vortmeyer, Bailey-Wilson, Castellanos, & Muenke, 2010). Using an international, multigenerational sample of over 6000 participants (approximately half with ADHD and half without), Arcos-Burgos et al. (2010) found LPHN3 gene variants in key regions implicated in neural circuitry modulating attention, activity, and metabolism of the ADHD brain. Portions of the brain that are involved in both emotion and motor control such as the amygdale, cerebellum, and caudate nucleus expressed the genetic variant (Arcos-Burgos et al., 2010). However, it was unclear whether the gene in question was mapped to regions implicated in EF (Arcos-Burgos et al., 2010) such as the PFC. The failure to confirm expression of the gene in cortical regions of the brain makes it difficult to account for executive dysfunction that is typical of those with ADHD.

The human genome is incredibly complex, causing explicit identification of ADHD genes to be presently non-existent. While genes responsible for catecholamine activity are investigated most often, these genes are located on different chromosomes in various regions. In addition, the actions of these genes are still being investigated. What complicates matters further is not all ADHD patients possess target genes, nor do all those possessing the gene exhibit signs of

ADHD. In spite of large-scale studies, geneticists cannot pinpoint specific ADHD genes common to all ADHD patients (Elia & Dovato, 2007; Spencer et al., 2002; Swanson et al., 2000). Because genetic markers common to all ADHD patients have yet to be found, the genetic basis for this condition remains a mystery.

ADHD and Executive Functioning

This section discusses the executive functioning deficits associated with the condition of ADHD. The section begins with a definition of executive functions and is followed by popular models of executive functioning associated with ADHD. Emphasis is placed on the EF constructs of attention, inhibition, and working memory as they relate to the condition of adult ADHD. The strengths and weaknesses of these models is also discussed to provide support for future EF and adult ADHD study.

Neuroscientists hypothesize that the primary job of the PFC is to regulate a significant number of executive functions (Barkley, 2007). Executive functions (EF) consist of neurological processes, which facilitate self-regulated, autonomous behavior such as initiative, motivation, planning, judgment, goal directed behavior, flexibility, and self-monitoring, all of which are cognitive constructs important for academic success (Gaultieri & Johnson, 2006). EF's are required not for simplistic, automated tasks but for complex, novel tasks which are cognitively demanding (Henry & Bettany, 2010). Pennington & Ozonoff (1996) categorized executive function as consisting of the following processes: planning, working memory/updating, problem solving, self-monitoring, mental flexibility, fluency, and inhibition. Efficient and effective executive functioning leads to the manifestation of goal-oriented and self-directed behaviors, which happen to be problem areas for those with ADHD. Because challenges with cognitive and motor processes associated with self-regulation are often symptoms of ADHD, it is likely that

executive dysfunction lies at the core of the disorder. This dysfunction may stem from poor inhibition, problems with sustained attention, and limited working memory (Barkley, 1997; Holmes, Gathercole, Place, Alloway, Elliot, & Hilton, 2010).

Inhibition is regarded as an executive function drawing much attention from ADHD researchers. It is hypothesized that all executive functions require inhibition, a cognitive construct to resist distraction and sustain attention (Barkley, 1997). Because ADHD patients struggle with both attention and inhibitory deficits, executive dysfunction is likely. Deficits in EF are thought to lie at core of ADHD (Holmes et al., 2010); however which EF's are predominantly affected as a result of the disorder is unknown. Though Nigg (2005) and Rhodes, Coghill, & Matthews (2005) found that deficits consistently occur in attention, working memory, planning, strategy formation and motivation among the ADHD population, literature supporting the hypothesis that ADHD symptoms arise from EF deficits is not well established (Tripp & Wickens, 2009). Inconsistencies in neuropsychological research warrant further study of adult ADHD to better understand the relationship between executive dysfunction and the disorder.

Executive dysfunction models associated with ADHD are rooted in the suspected neurobiology of the disorder. For example, symptoms of the disorder that have been implicated in executive dysfunction may be due to faulty integration of attentional systems with higher order cognitive functions (Gaultieri & Johnson, 2006). Hypotheses related to executive dysfunctions associated with ADHD are based on theories of 'top-down' and 'bottom-up' cognitive processes (Gaultieri & Johnson, 2006). Cognitive Processes considered to be 'top down' are those thinking patterns guided by larger ideas, beliefs, or expectations about the environment. However, 'bottom-up' processes involve the piecing together of information to construct larger mental representations (Gaultieri & Johnson, 2006). For example, four ADHD

models have been proposed in an attempt to account for EF discrepancies associated with ADHD. These include the inhibition (or frontostriatal model), cognitive energetic, delay aversion, and the behavioral inhibition/activation models (Sergeant et al., 2003). Though empirical support exists for such hypotheses in children, studies generalizing findings to adult populations are both lacking and inconsistent in their findings.

Inhibititory Model of Attention.

One of the most commonly referenced hypotheses of ADHD is the inhibition or frontostriatal model of attention proposed by Barkley (1997). As the name of the disorder implies, inattention and hyperactivity are key symptoms of the disorder but researchers now claim that poor inhibition, not inattention is the primary symptom of ADHD (Barkley, 1997; Boonstra et al., 2005). The frontostriatal hypothesis of ADHD attempts to explain why those with the condition exhibit impulsivity and poor inhibitory skills. Frontostriatal control systems regulate mechanisms of arousal and attention in conjunction with sensory information needed for the integration of goal- orientation behavior (Gaultieri & Johnson, 2006). Barkley (2001) hypothesizes that these areas of the brain function abnormally in ADHD patients, creating inhibitory deficits that lead to inattention and further executive dysfunction. Measures of inhibitory processing indicate that those with ADHD show deficits in areas of inhibitory processing when compared to controls (Ossman & Mulligan, 2003). Holmes et al. (2010) found that children with ADHD performed poorly compared to normal developing children on measures of cognitive inhibition, motor inhibition, set shifting, planning, card sorting, and working memory. It is important to note that participants in the majority of these studies were children with ADHD, not adults.

With regard to the inhibitory hypothesis among adult populations, the few studies that have been conducted indicate that those with the disorder also have impaired attentional function compared to controls when called upon to sustain attention on continuous performance tests (Hervey, Epstein, & Curry, 2004). Rapport et al. (2001) concluded that adults with ADHD were unable to inhibit inappropriate responses and demonstrated greater difficulty when faced with EF tasks of attention. Barkley (1997) hypothesizes that poor attention results from goal-setting impairments stemming from inhibition difficulties. When EF's function abnormally, self-regulation suffers (Barkley, 2001). However, when inhibitory mechanisms are functioning normally, irrelevant stimuli are ignored, while the PFC plays out possible physical, social, and emotional outcomes for various decisions prior to taking action, (Barkley, 1997) thus leading to better self-regulation. Because inhibition appears impaired in ADHD patients, the cognitive suppression of irrelevant information is not occurring (Ossmann & Mulligan, 2003), resulting in actions or decisions being made pre-maturely. This results in incorrect or inappropriate exhibition of behavior. In clinical settings adults with ADHD had difficulty with tests of motoric inhibition (Hervey et al., 2004), a sign of impulsive behavior. Pennington & Ozonoff (1996) also found that several deficits in EF are associated w/ ADHD related to inhibition and planning. Based on poor reaction times using twenty stop signal tasks, deficits in inhibition were found to be a core EF dysfunction in ADHD (Pennington & Ozonoff, 1996). Those with the hyperactive sub-types of Attention Deficit are especially prone to impaired inhibition, and often exhibit behavioral impairments in the areas of self-control of actions and verbalizations (Gathercole & Alloway, 2006).

The effort to control impulses also appears to slow reaction times of ADHD patients. For example, Gualtieri & Johnson (2006) also found that ADHD patients were slower in tests of

psychomotor speed than controls, and made fewer correct responses on attention shifting and continuous performance tests. This suggests that more concentrated effort was needed by ADHD patients to suppress impulses. Taking its toll on an individual's self-regulation, lack of inhibition allows external and internal stimuli to interfere with other key executive functions (Barkley, 1997). Based on Barkely's (1997) hypothesis, it would appear that better inhibitory skills could improve executive function and subsequently, self-regulation.

The inhibitory hypothesis of ADHD is not without limitations. In the area of adult ADHD research, sample composition and selection becomes a concern in Ossman & Mulligan's (2003) study in that participants were college students receiving psychology credit in exchange for participation. Therefore, it is possible that participants were intelligent individuals motivated to participate in the research in order to receive a grade. Research conducted by Barkley (1997) as well as Gathercole & Alloway (2006) specifically focus on children with ADHD, rather than adults. From a developmental perspective, executive functioning may change with age, making it challenging to apply all aspects of the inhibitory model to adults possessing the same disorder. Additional studies must be conducted to test this hypothesis among adults with ADHD.

In addition to inhibitory deficits, Barkley (1997) goes further to suggest that four other executive functions result from cognitive inhibition: working memory, internalization of speech, and self-regulation of affect and motivation. With regard to working memory (WM) two types of processes can be distinguished, maintenance and manipulation of information (Sergeant et al., 2003). Maintenance involves simple cognitive recall like active selection, comparison, and evaluation of stimuli held in short and long-term memory (Sergeant et al., 2003). Manipulation is the monitoring of higher-level executive processes (Sergeant et al., (2003). For example, recalling when a final research paper is due for a course would involve maintenance, while

manipulation would be required to formulate a plan for completing the paper in well-done, yet timely manner while blocking out distractions and irrelevant stimuli. Working memory (WM) and short-term memory systems store and manipulate visuo-spatial information and verbal information needed for the learning of science/mathematical and literacy skills (Gathercole & Alloway, 2006).

While some research indicates that those with ADHD suffer from WM impairments, other studies failed to observe a significant difference in WM function between those with and without ADHD. For example, Biederman et al. (2008), Ossman & Mulligan (2003), and Barnett, Maruff, Vance, Luk, Costin, Wood, & Pantelis (2001) found that adults and children with the disorder demonstrate significant WM deficits on neuropsychological assessments; however, other researchers claim that WM deficits are insignificant between these groups (Gathercole & Alloway, 2006; Hervey et al., 2004). Those asserting that WM deficits result from ADHD claim that limited WM is linked with attention, thus restricting the amount of information to be processed at one time (Barkley, 1997; Gathercole & Alloway, 2006). Limited WM stores could also explain why many with ADHD have difficulty managing their time, adhering to schedules, and making deadlines. Additionally, WM is also thought to influence an individual's hindsight, forethought, sense of time, and goal-directed behaviors (Barkley, 1997). From this, it can be inferred that WM processes contribute to individual self-regulation. If WM deficits are in fact associated with ADHD, the poor self-regulation often seen within this population may stem from executive dysfunction in this area.

To account for inconsistent conclusions regarding WM research, additional neuropsychological studies among the adult ADHD population are needed to provide support for WM claims. In addition to research with older ADHD subjects being limited, previously

discussed WM studies failed to account for consistency among crystallized and fluid participant intelligence among homogenous samples (Sergeant, 2003). For example, Biederman's (2008) research results were drawn from a ten-year follow-up study of predominantly Caucasian children ages 6-18 years old. Between the initial time of testing and follow-up, participants may have suffered head trauma, abused substances, or developed compensations strategies that affect EF. With regard to Barnett et al. (2001) children who participated in the study were very young (< 12 years of age), had varying levels of cognitive ability and possessed comorbid disorders such as oppositional defiant and conduct disorder. Such limitations call for additional research among ADHD adults with similar levels of intelligence, fewer comorbid disorders that can alter WM assessment results, and a short follow-up time for drawing conclusions.

In addition to WM deficits hypothesized to stem from poor inhibition is the internalization of speech. Internalization of speech is part of what Barkley (1997) calls *reconstitution*. Reconstitution is an EF involving the understanding of verbal messages being sent and received prior to initiating a behavior (Barkley, 1997). Such processes allow the individual to process and comprehend what is being said to them in both conversation and instruction. Working in conjunction with the internalization of speech is the separation of affect. This involves the processing of emotions in ways, which allow the individual to exhibit socially appropriate behaviors that may in fact be contradictory to how one is feeling (Barkley, 1997). Preventing us from acting out of emotional impulse, separation of affect allows humans to exhibit actions, which are not direct reflections of negative or aggressive emotional states. Reconstitution and the separation of affect allow for the processing, interpretation, and guidance of appropriate action based on the environmental stimuli being presented. It can be inferred that potentially faulty processes in these areas can lead to context-specific inappropriate behavior.

ADHD patients often respond to immediate contexts and consequences prior to using EF's like hindsight and forethought, making deficits in the separation of affect and internalization of speech a possible explanation for inappropriate behaviors (Barkley, 1997).

While it appears that inhibitory deficits contribute to additional EF problems, a number of questions remain in this area. First, the amount of executive dysfunction that stems from poor inhibition is unclear. If in fact poor inhibition leads to poor attention and working memory, then the results should be clear and consistent across various studies. However, this is not so. There are also developmental issues that must be considered when examining adults with ADHD in comparison to children. As an individual matures both physiologically and psychologically, it becomes possible that EF's too may change. Therefore, the changes that take place over the course of maturation must be accounted for with additional study of older participants. Also, the overall strength of the relationship between ADHD and EF also remains unknown, especially among adult populations. The only way to answer such questions is through studies involving neuropsychological assessment of adults with ADHD.

Cognitive Energetic Model of Attention.

Other hypotheses regarding how ADHD is implicated in executive dysfunction have been proposed recently. As an alternative to the inhibition hypothesis proposed by Barkley (1997), the *cognitive energetic model* (CEM) proposes that deficient cognitive energy is the potential cause of executive dysfunction in the ADHD brain (Sergeant, 2005). According to Sergeant (2005) ADHD causes deficits in behavioral response output and energetic mechanisms that are considered to be activation and effort control systems of EF (Sergeant et al., 2003). Rather than resulting from impaired cognition as suggested by the front-striatal model, it is suggested that tasks involving attention require activation, energy, and arousal in order to fuel cognitive

resources (Doyle, 2006; Sergeant, 2005). Sergeant (2005) proposes that those with ADHD have abnormal executive functioning because energetic pools fueling arousal, effort, and activation are deficient. Arousal involves signal detection and activation constitutes a reward response (Nigg, 2005). Arousal and activation involves noradrenergic neurons ascending from the locus coeruleus to the cortex; alerting persons to relevant stimuli in order to elicit a response output (Nigg, 2005). Nigg (2005) found that children with ADHD show slower reaction times during neuropyschogical assessment. This indicates that slower reaction times may be due to insufficient cognitive arousal and activation. Such deficits cause alterations in the perception of what constitutes a reward and what constitutes a punishment. Linked to effort pools, the effects of rewards and punishment are considered to be a 'bottom up system' which sends feedback to orbital frontal cortex to determine whether a certain stimulus-response events is satisfying or not (Sergeant et al., 2003). ADHD patients tend to respond for an immediate reward rather than wait a longer period for a greater reward, thus indicating that rewards become low in reinforcing power (Nigg, 2005).

While this hypothesis attempts to explain some of the heterogeneity surrounding ADHD, additional research is needed to support CEM assertions. Data needed to support aspects of this model are currently limited and require further study (Doyle, 2006). Because CEM is based on neural circuitry involving reward and punishment, developmental and cognitive considerations made and accounted for. Because a significant amount of CEM studies have involved children only, further research must be conducted using adult participants to confirm that adolescent findings either parallel or differ from those involving older individuals.

The Delay Aversion Model of Attention.

Along the lines of CEM, and directly related to cognitive systems associated with reward, punishment, and inhibition, is the *delay aversion model of attention*. Sonuga-Barke et al. (2010) proposed an addition to a dual pathway model involving EF and delay aversion to involve a third neural pathway. Originally, it was suggested that dysregulation of fronto-striatal circuitry is altered by two faulty neural circuits, resulting in delayed rewards and poor delay aversion (Sonuga-Barke et al., 2010). However, authors now suggest that a third pathway is functioning abnormally in temporal regions, causing additional processing deficits (Sonuga-Barke et al., 2010). In support of this hypothesis, Sonuga-Barke et al. (2010) found that ADHD children were unwilling to delay their need for gratification and choose small, immediate rewards over larger, delayed rewards. Researchers stipulate that these processes should not be seen as overarching characteristics of the disorder but as one of multiple possible symptoms (Songua-Barke et al., 2010). ADHD should be seen as a heterogeneous disorder where EF deficits vary from person to person, particularly in connection with inhibition and delay aversion (Sonuga-Barke et al., 2010). According to CEM, faulty neural circuitry may explain why those with ADHD have difficulty delaying behavioral responses to receive a specific reward and switching attention from one stimulus to another.

A number of limitations regarding *Delay Aversion* are a concern. First, delay aversion research has been heavily grounded in animal models (Doyle, 2006). While there are similarities, animal brains differ from that of humans, especially those of a higher-order primate. Also, most environmental factors can be controlled for during experimentation with animals, while few of those factors can regulated among human subjects. Therefore, applicability to humans may be different. In addition, studies that have been conducted using human subjects

have yet to involve adults with ADHD (Boonstra et al., 2005). Possessing many aspects of behaviorist learning theory, delay aversion's emphasis on reward, punishment, and reinforcers makes sense, yet requires further testing using human subjects.

Behavioral Inhibition/Activation Model of Attention.

The Behavioral Inhibition/Activation model proposed by Gray (1972, 1981) suggests that two neurological systems are responsible for an organism's reaction to various environmental stimuli. Thought to be regulated by norepinephrine (NE), the behavioral inhibition system (BIS) signals whether a particular stimulus will bring about pleasure or punishment, while the behavior activation system (BAS) signals the organism's emotional state (Sergeant et al., 2003). Once the BIS determines the novelty and/or potential reward of an environmental signal, it inhibits behaviors that would lead to potential undesirable outcomes (Gray, 1972, 1981). Such outcomes are often social in nature, causing the individual to get into trouble, offend someone, or forget a deadline. This system guides human behaviors toward goal-attainment through the avoidance of inappropriate response by working in conjunction with the BAS. Influenced by dopaminergic action, the BAS is thought to be responsible for the experience of positive emotions associated with behaviors which bring about reward and pleasure (Carver & White, 1994; Gray, 1972, 1981). Together, these systems allow an individual to extinguish behaviors that prevent goal achievement and initiate behaviors that perpetuate accomplishment of objectives.

As the model suggests, dysfunction of the BAS and BIS systems would cause faulty regulation of behavior. This is precisely the concern in the study of ADHD. Behavioral inhibition/activation accounts for ADHD in that the BIS is hypothesized to be underactive, while the behavioral activation system (BAS) is overactive (Sergeant et al., 2003). An over-active BAS may cue hypersensitivity to rewards as the under-active BIS fails to activate inhibitory

mechanisms, thus resulting in the manifestation of ADHD behaviors (Carver & White, 1994). Examples of these behaviors include the need for instant gratification, challenges waiting for far-off rewards, and other impulsive actions. If these systems are impaired in the ADHD brain, those with the condition would be less successful in determining which stimuli bring about reward or punishment. These deficits potentially result in the failure to inhibit well-learned, yet inappropriate patterns of behavior and replace them with new behaviors when presented with specific social cues (Henry & Betteny, 2010). As a result, appropriate adjustment of behavior fails to occur in response to specific environmental cues.

The BAS and BIS models account for some of the major behavioral symptoms of ADHD, but fall short of explaining all of the condition's symptomology. For example, BAS and BIS does not account for certain aspects of poor self-regulation such as challenges with time management, being unorganized, and the inability to sustain attention in various contexts that are associated with ADHD. From the perspective of behavior, BAS and BIS accounts for impulsivity and poor inhibition but these are not the only symptoms of ADHD. BAS and BIS fails to explain why specific self-regulated behaviors necessary for academic success are a challenge for those with ADHD to exhibit.

In summary, multiple models of ADHD attempt to explain executive dysfunctions that are linked with the disorder. While some have more evidence in support than others, it remains clear that additional research using heterogeneous samples is needed to support or refute these claims. Again, many of these hypotheses have been formulated using younger populations, animal models, and small, homogenous samples. As a result, many questions about the nature of the disorder with regard to executive and academic dysfunction remain. The neuropsychological

testing of adult ADHD patients may answer questions about executive dysfunction associated with the disorder.

Neuropsychological Measures of Executive Function and ADHD

This section begins reinforcing the need for EF assessment among adults with ADHD in addition to an overview of neuropsychological assessment purposes. This is followed by a synopsis of EF theory and the neuropsychological assessments designed to capture and measure various elements of these theories. In addition, this section describes how these measures are used to assess the EF of those with ADHD. Special attention is paid to assessments created for the purpose of assessing attention, inhibition, and working memory as those constructs will be assessed in this study.

There has been very little research in the area of EF assessment among adults with ADHD. According to Rapport, VanVoorhies, Tzelepis, & Friedman (2001), very few studies have directly assessed the executive functions of ADHD adults. This is partially due to the fact that such measures were not originally designed for ADHD assessment. Originally, neuropsychological assessment developed out of the need to measure executive functions of individuals suffering from neurological impairments (Sturm, 2007), most likely stemming from some sort of trauma. Today, such examinations are designed to assess a variety of cognitive processes, including those of executive function of those with a variety of disorders. Because ADHD is suspected to cause executive dysfunction, many of these assessments can also be used with adult populations. These tests measure basic and higher order sensory functions, intelligence, memory, attention, sensorimotor functions, and executive functions (Sturm, 2007). Measures were developed to assess EF's associated with emotion, such as those cognitive processes activated in response to reward, punishment, and regulation of behavior as well as

those considered to be non-emotional, logical thought processes (Chan, Shurn, Toulopoulou, & Chen, 2007). Sturm (2007) asserts that neuropsychological examination must include an intellectual profile, attention assessment, and memory functions measures in conjunction with EF assessment and emotional affectivity. In the case of this research, the purpose of these measures is to evaluate the executive functioning of adults both medicated and non-medicated for ADHD.

Theories of Executive Function and Measures

The assessment of cognitive construct such as executive functioning can be challenging using one measure alone. As a result, multiple assessments are commonly used to capture one's EF ability. Attempts to measure EF involve tasks requiring attention, inhibition, and working memory, among other cognitive processes. There are a significant number of neuropsychological measures available, but some are used more often than others with the ADHD population. A gold standard in terms of ADHD neuropsychological measures does not exist; however, assessments have been developed based on five theories of executive function in order to capture various aspects of the EF construct (Chan et al., 2007). These theories provide a foundation for assessments designed to measure cognitive tasks associated with EF.

The first theory of executive function is based on what is known about neuroanatomy. According to Luria (1966, 1973) the brain has three basic functional units that are linked. The first unit consists of the brain stem and arouses the cortex, while the second is responsible for processing information and storage. This unit is composed of the temporal, parietal, and occipital lobes. Making up the third unit are anterior structures of the brain (the PFC) that work to regulate mental programming, activities and behaviors (Luria, 1966, 1973). Taking these neurological regions into consideration, assessment was designed to test the functioning of these brain structures. Measures based on this theory attempt to evaluate attention and inhibition

skills. These include assessments of inhibition such as the Wisconsin Card Sort Test (WCST), which is a measure of mental sequencing and motor control (Chan et al., 2007). Such activities can be challenging for those with ADHD, potentially signifying EF deficits. It is for this reason that assessments like the WCST are frequently used with ADHD patients. However, because other regions of the brain are activated in response to this task, WCST's cannot be used as a purely "frontal" or EF assessment (Chan et al., 2007) and should be used in conjunction with other measures designed to assess executive dysfunction.

The ability to maintain focus and switch tasks mentally is a vital aspect of normal EF. Some assessments of attention and mental multitasking are based on Norman and Shallice's (1986) *Supervisory Attention Model* (SAM). According to this theory, programming and regulation of human behavior requires both scheduling and supervisory attention (Norman & Shallice, 1986). The process of scheduling involves cognitive functions required to plan and perform routine behaviors while supervisory attention is responsible for processes needed to perform new, non-routine tasks (Norman & Shallice, 1986). Assessments such as the Continuous Performance Test (CPT) and digit span tests when used among the ADHD population were designed to assess sustained attention and attention switching (Boonstra et al., 2005). Because attention deficits and task switching in various contexts are a concern with ADHD, such tests provide valuable information about the skills of an individual in these EF areas.

In addition to assessing inhibition and attention, neuropsychological tests can also contain a self-regulation component, which is accounted for by the Triparitite model. In Stuss and Benson's (1986) Tripartite model, three systems of the brain interact to monitor attention and EF. They include the anterior reticular activating system (ARAS), diffuse thalamic projection

system, and the fronto-thalamic gating system. Composed of complex neural circuits, the first two systems help maintain alertness, while the fronto-thalamic gating system is involved in executive control (Stuss & Benson, 1986). This region also regulates higher level processing like planning, stimulus response selection, and monitoring of one's daily progress (Stuss & Benson, 1986). Based on this theory, assessments were created that involve tasks of mental conflict such as the Stroop Test, mental switching (WCST), Trail Making Test, and verbal fluency (Chen et al., 2007; Stuss & Benson 1986). These examinations require self-regulation to sustain and monitor attention over a long period of time (Chen et al., 2007; Stuss & Benson, 1986). Because problems with sustained attention, inhibition, and self-regulation are typical of those with ADHD, assessments of such abilities are often conducted.

The fourth theory of EF is also based on self-regulation but contain extra emphasis on working memory (WM) function. Duncan's (1986) Goal Neglect Theory posits that human behavior is goal-oriented and is regulated by a list of mental objectives. Once these goals are formulated, they are cognitively stored to guide future behavioral response in WM (Duncan, 1986). Much of this process is thought to involve WM. Based predominantly on animal studies; Goldman-Rakic (1992) developed a WM model, which has been used as a foundation for WM assessments. It was suggested that the PFC is responsible for WM but is divided into sub-regions where useful information is neurologically excited by catecholamines such as DA, while useless information is inhibited (Goldman-Rakic, 1992). Because self-regulation and WM are thought to be impaired as a result of ADHD, numerous neuropsychological assessments administered to ADHD patients contain working memory components. Though neuropsycholgical assessments have been designed to measure self-regulation, many aspects of poor self-regulation may be difficult to capture in the case of ADHD. Therefore, such

assessments should most likely accompany behavior-rating scales that assess behaviors at school, work, and home to successfully evaluate one's self-regulation as it impacts daily life.

To account for self-regulation of emotion, Damasio's *Somatic Marker Hypothesis* (1995) makes up the sixth theory of executive function. According to this theory, the role of the ventromedial prefrontal cortex is to regulate emotion, social behaviors, and decision-making through links with the subcortical structures. Damasio (1995) goes further to suggest that damage to this area results in the inability to use emotion-related somatic markers to guide behaviors. Poor regulation of behaviors related to emotion is very common among those with ADHD. Measures assessing such functions and behaviors may involve rating scales such as those created by Brown (1996, 2001). Rating scales are similar to questionnaires and can be completed by the patient, teachers, family, or parents (Brown, 1996, 2001) to gain an idea of how an individual regulates his/her emotions and behaviors at home, work, or school. The challenge; however, with behavior rating scales among adults with ADHD is that it may be difficult to receive adequate evaluation from those witnessing the behaviors. As an example, someone with ADHD may be reluctant to have their behavior rated by a boss, coworkers, roommates, or professors where there is a feeling of competition or fear of being stigmatized. Therefore, adult assessment should encompass a battery of measures to adequately assess EF.

Commonly Used Neuropsychological Assessments

Neuropsychological tests of attention disorders are based on the four components of attention: *Sensory selective attention, response selection and control, attentional capacity and focus, and sustained attention* (Cohen, Malloy, Jenkins, & Paul, 2006; Snyder, Nussbaum, & Robbins, 2006). Each aspect of attention differs slightly in terms of function and purpose.

Neuopsychological assessments of attention are designed to capture and assess each component of this construct to identify areas of EF strength and need for each ADHD patient.

The first component of attention, *sensory selective attention* is the process by which specific environmental stimuli are chosen and selected for further cognitive processing. This encompasses the filtering of other distracters, the enhancement or additional focus on the relevant stimuli, and ultimately, the disengagement of attention once a more relevant stimulus is presented. Those with ADHD are often administered the Continuous Performance Test (CPT) to assess this aspect of attention. One type of CPT, the Connors' Continuous Performance Test (2nd ed.; CCPT-II, 2000) is an exam of attention, vigilance and inhibition. This test requires participants to push a computer key immediately after a certain stimulus is presented (Homack & Riccio, 2006). Individual scores are calculated based on error and response rates. Close attention to detail and adequate inhibition is required to attain higher scores on the exam.

Response selection and control allows for an individual to allocate resources required for attention needed to regulate behavioral responses. This allows for the appropriate physical response to various situations after situational analysis. Key cognitive processes include inhibition, persistence, and the initiation of specific behaviors. The Go/No-Go assessment, sorting tasks such as the WCST, as well as the CPT are measures used to assess this feature of attention. In the Go/No-Go task, patients with ADHD are presented visually with items, which either belong in a certain category or fail to belong in that category (Nosek & Banaji, 2001). If the item presented on the computer screen belongs in a given category the 'go' or space bar is pressed; however, those that do not belong are a 'no-go' and no key is pressed (Nosek & Binaji, 2001). This measure calls for attention and inhibition to make controlled yet timely responses. Another examination of response selection and control is the WCST. The WCST measures the

problem-solving and flexibility skills of ADHD participants as they are asked to sort cards in a particular fashion (Rapport et al., 2001). Also known as set-shifting, examinees are required to match cards based on shape, color, or numerosity to four given base cards (Henry & Bettany, 2010). As matching demands change throughout the exam, participants must successfully switch from one matching dimension to another (Henry & Bettany, 2010). Like the Go/No-Go exam, attention, inhibition, and cognitive flexibility are needed to successfully implement a response at the correct time without forsaking accuracy.

Once a stimulus has been selected for additional cognitive processing *attentional capacity and focus* mechanisms are activated, as one working memory resource. Depending on the demands of the task, different levels of attention and focus will be required. Higher levels of focus usually result in better task performance. Measures of such focus and attention include digit span tests, Stroop color tests as well as reaction time assessments. According to Rapport et al (2001), digit span tests are examinations of working memory. After reading a set of numbers or letters, the examinee must say or place them in a given order. Stroop tests require that participants read a series of color words printed in black and then identify the colors of a series of squares. After, the participant must read the color ink in which color words are printed. The words do not always match the colors, requiring selective attention and response inhibition to accurately name the appropriate word.

The last component of attention is *sustained attention.* This involves extended focus and attention to a certain task for a lengthy period of time. Optimum performance is dependent on sustained attention and the ability to maintain focus on a task depending on its complexity and subsequent reinforcement. The most commonly used sustained attention measure is the CPT. Specifically the CPT can measure vigilance, which is a special aspect of sustained attention,

consisting of the ability to remain focused for extended periods of time. This is a challenge for those with ADHD, especially when presented with uninteresting and challenging stimuli.

In addition to using such assessments, ADHD symptom checklists are also used as indicators of EF dysfunction among adults. Symptom checklists are self-report questionnaires where patients rate the intensity of their symptoms. For example, Brown's ADHD checklist (1996, 2001) assesses symptomology using six different clusters: *Activation, Focus, Effort, Emotion, Memory, and Action.* These clusters assess individual organization, focus, management of emotions, memory, and self-regulation through the use of rating scales (Brown, 1996, 2001).

Because one assessment fails to capture all aspects of ADHD, multiple measures should be used when conducting neuropsychological assessments. These measures should also be conducted in conjunction with a behavior-rating scales, case history, and individual interviews to adequately evaluate the impact of ADHD on one's executive and academic functioning. It is for this reason that this study will use a battery of neuropsychological assessments in conjunction with an academic self-report measure.

Limitations of Neuropsychological Testing with the Adult ADHD Population

Neuropsychological assessment of adult ADHD patients is not without limitations. Much variability in the executive functioning of the ADHD population exists based on results of various measures (Doyle, 2006). Though neuropsychological assessment offers insight regarding EF, these measures cannot be used solely as a diagnostic tool. To demonstrate this, Holdnack et al. (2005), Rapport et al. (2001), and Weyandt, Rice, Linterman, Mitzlaff, & Elmert (1998) report that the Stroop, digit span tests and WCSTs failed to reliably identify individuals with ADHD based on their scores alone. However, reliable classification results were found by Holmes, Gathercole, Place, Alloway, Elliot, & Hilton (2010) where EF measures accurately

predicted group identification for 90% of participants with and without ADHD in children. With regard to older ADHD populations, studies involving college students indicated that those with ADHD performed just as well on neuropsychological assessments as those without the disorder, (DuPaul, Weyandt, O'Dell, & Varegjao, 2009) a result which differs substantially from those of Holmes' et al. (2010) study where differences between ADHD and controls could be observed. Adult ADHD executive functioning assessment results are very inconsistent. It is for these reasons that further research is needed to adequately evaluate executive and academic functioning of those with ADHD using valid and reliable EF assessments.

Variable results and conclusions from the neuropsychological assessment of ADHD populations are suspected to be due to a number of factors. First, there are methodology problems. For example, methodological concerns have centered on smaller samples sizes consisting of highly intelligent participants (Rapport et al., 2001) who may have developed compensatory mechanisms by adapting academic strategies that work for them. Also, it can be a challenge to assess EF constructs not easily measured in a lab or clinical setting (Brown, 2002) such as those displayed in occupational, academic, or social settings. This may explain why abnormal EF deficits are only found within a sub-set of adults affected with ADHD (Doyle, 2006). Neuropsychological tests are clinical exams given in a lab setting. Therefore, neuropsychological testing does not measure one's study habits, driving ability, or performance at work. These are all activities where executive dysfunction may be manifested in adults with the disorder, but assessment is virtually non-existent in those environments. Clinicians also have a difficult time assessing EF areas associated with self-regulation needed for academics such as discipline, establishment and maintenance of routines, and organizational strategy (Wolf & Wasserstein, 2001). In addition, adult EF has not been studied as much as youths with ADHD

(Doyle, 2006; Rapport et al., 2001), so it is difficult to generalize findings from studies from younger ADHD populations to their older counterparts and identify which EF measures' results accurately distinguish between those with ADHD and controls. To clarify questions involving EF deficits among adults with ADHD, additional research should be conducted (Rapport et al., 2001). Therefore, studies involving the neuropsychological assessment of adults with ADHD are both justified and appropriate.

Efficacy and Action of Stimulant Medication

Stimulant medications are frequently prescribed to treat adult ADHD (Heal, Cheetham, & Smith, 2009) and are suspected to alter both academic and executive functions (Meaux, Hester, Smith, & Shoptaw, 2006; Roth & Saykin, 2004). The following begins with an introduction to commonly prescribed stimulants and their efficacy. The psychotropic action of these medications is reviewed to provide connections to the *dopamine hypothesis of ADHD*. The section concludes with a discussion of how stimulants may effect the executive functioning of those with ADHD.

Effects of Psychostimulants.

To treat ADHD, stimulants such as Methylphenidate (Mph) and amphetamines are among the most commonly prescribed drugs for both children and adults (Heal et al., 2009). Stimulant medication prescribed for the treatment of ADHD is considered to be one of the safest and effective treatments for children with the disorder. However, little research in the way of controlled studies has been conducted among adults taking the same medications (Adler & Chua; Boonstra et al. 2005; Kinsbourne, De Quiros, & Rufo, 2001). Overall, there are very few long-term ADHD medication studies targeting older ADHD populations (Santosh & Taylor, 2000). Shorter-term studies indicate that 50% of adults respond favorably to stimulant medication,

compared to 70% of children, despite some minor side effects (Faraone, 2000; Santosh & Taylor, 2000). Adult-centered studied tend to concentrate on behavioral improvements as a result of the medication rather than on cognitive changes (Kinsbourne et al., 2001). Though stimulants appear to be effective ADHD treatments, it is unclear whether they affect EF (Biederman et al., 2008). As a result, additional studies focusing on the potential influence of medication on the executive functioning of adults with ADHD are needed.

Psychotropic Action of Stimulant Medications.

Standard stimulants are fast-acting, with a relatively short half-life (Santosh & Taylor, 2000). Once they enter the bloodstream, stimulant medications alter the levels of neurotransmitters within the brain. Many stimulant medications are formulated in an attempt to remedy DA deficits in the brain by boosting norepinephrine NE and DA simultaneously. The most efficacious drugs are those that enhance transmission of DA and NE from synaptic vesicles of catecholanergic neurons (Wilens & Dodson, 2004). Taken orally, these medications cross the blood/brain barrier as cleaved d-amphetamines to exert effects on DA and NE transporters (McBurnett & Weiss, 2011) via mesocortical and frontostriatal pathways (Wilens & Dodson, 2004). Once this occurs, presynaptic terminals of catecholinergic neurons displace DA and NE from storage vesicles, allowing them to be released into synapses to stimulate neural activity (Heal et al., 2009). Pyschostimulants such as amphetamines also prevent the reuptake of catecholamines to extend the life of actions exerted by these neurotransmitters (McBurnett & Weiss, 2011; Vaida, Austin, Kirkorian, Ridlehuber, Desmond, Glover, & Gabrieli, 1998; Wilens & Dodson, 2004). This allows for DA and NE to remain active for longer periods of time in key EF areas of the brain.

The most important regulator of DA is the dopamine transporter (DAT), which is located in the plasma membrane of neurons responsible for DA synthesis (Santosh & Taylor, 2000). DA concentration is regulated when DAT's remove the chemical from extracellular space by relocating them to the cytoplasm (Santosh & Taylor, 2000). This process decreases overall levels of DA. Stimulant medications block the action of DAT in the ADHD brain, thus increasing the amount of DA available in key cognitive areas of the brain (Elia & Dovato; Santosh & Taylor, 2000). In humans, the stimulant medication methylphenidate can block 60% or more of DAT to open DA receptors and increase levels of extracellular DA (Tripp & Wickens, 2009). By making more DA and NE biochemically available for cognitive processes stimulants may have a positive effect on executive dysfunction implicated in ADHD (Coghill & Markovitch, 2004).

Effects of Stimulants on Executive Function

Because ADHD patients possess certain EF deficits, the potential impact stimulant medications have on these functions is highly important. Overall, improvements in EF are thought to be one positive effect of stimulant medication, but the extent and specificity of these effects remains in question. There is evidence to suggest that EF's can improve as the result of psychostimulant medication because of its effects on FSTC circuitry (Roth & Saykin, 2004). Double-blind medication trials suggested that the neuropsychological functioning of those with ADHD is higher than non-medication takers (Wilson, Cox, Merkel, Moore, & Coghill, 2006). The problem area appears to lie in the relationship between stimulants and executive function. For example, which EF's are positively affected by stimulant medication and which are not is a source of scholarly debate. In the ADHD adult, the EF areas to be examined include inhibition, attention, and working memory.

To measure the potential effects of stimulant medication on executive functioning, studies have compared ADHD patient performance on neuropsychological measures to non-medicated ADHD participants and non-ADHD controls. It is suspected that atypical frontal-striatal function contributes to symptoms of ADHD, including problems with inhibition (Vaidya, Austin, Kirkorian, Ridlehuber, Desmond, Glover, & Gabrieli, 1998). Researchers have found that commonly prescribed stimulants such as methylphenidate (Mph) and extended release amphetamines (such as Adderall®) improve response inhibition in children on stimulus-controlled tasks such as the Go/No-Go and Delayed-Matching-to-Sample assessments (Vaidya et al., 1998; Wilson, et al., 2006). Similarly, Bedard, Ickowicz, Logan, Hogg-Johnson, Schachar, & Tannock (2003) concluded that ADHD children taking stimulant medication outperformed controls on measures of inhibition such as the stop-signal task. In contrast to these findings, results of other studies involving ADHD boys suggested that Mph failed to demonstrate a significant effect on sensitive tests of inhibition (Rhoades, Coghill, & Matthews, 2006). Therefore, it cannot be claimed that stimulant medication improves the inhibitory skills of all ADHD patients, especially adult patients.

In addition to improving response inhibition, stimulant medication had positive effects on attention according to Stop Signal Tests or Change Tests results when compared to non-medicated controls (Boonstra et al., 2005; Vaida et al., 1998). Teenage ADHD subjects taking medication also scored higher on neuropsycholgical measures of attention and verbal memory than non-medicated peers (Biederman et al., 2008). Interestingly, Rhodes et al. (2006) concluded that Mph *did not* affect attention-shifting skills of ADHD participants though sustained attention effects were not mentioned. Overall, it appears that stimulant medication helps many with ADHD sustain attention for longer periods of time, and shift attention more

efficiently. Unfortunately, overall findings are somewhat mixed and the majority of studies have focused on younger patients, rather than older populations. EF assessments of medicated adults with attention deficits could yield different findings than children taking similar medications.

Though a number of studies suggest that stimulant medication has a positive influence on attention and inhibition, others have yielded inconsistent findings in the area of WM. Mehta, Owen, Sahakian, Mavaddat, Pickard, & Robbins (2000) found that stimulants exerted cerebral blood flow changes to key areas of the PFC, suggesting that improvement of participants' WM processes was the result of medication. This resulted in better WM storage and information retrieval as well as increased active manipulation of cognitive information (Mehta et al., 2000). These findings were similar to those of Mehta, Goodyear, & Sahakian (2004) where ADHD children placed on Mph improved their performance on tasks of WM as well as attentional set-shifting following a 16 hour medication withdrawal period. In spite of these results, not all researchers have been able to conclude that stimulants improve WM processes (Rhodes et al., 2006). For example, Biederman et al. (2008) found that stimulants have limited effects on executive functioning deficits, including WM function. However, because some researchers have found no significant discrepancies in WM among the ADHD population, (Gathercole & Alloway, 2006) it is difficult to assess the overall impact of stimulant medication on this construct. As a result, further EF assessments with WM components are needed in adult ADHD medication studies.

Adult EF has not been studied as much as it has in children; (Doyle, 2006) potentially explaining why executive dysfunction has not been specifically proposed for adults with ADHD, though it is expected that such deficits would apply (Boonstra et al., 2005). Another issue is that abnormal EF scores are usually predictive of ADHD but there are those with ADHD who do well

on EF measures (Doyle, 2006). When stimulant medication is considered, even more questions arise as to how EF's are altered. Neuropsychological difficulties in ADHD may not be confined to executive functioning alone (Boonstra et al., 2005) suggesting that additional assessments are needed using older, stimulant medicated and non-medicated participants as subjects to make further conclusions.

Academic Implications of Self-Regulation and Stimulant Medication

Academic success requires additional cognitive resources that allow an individual to focus, study, remain organized, and elicit goal-oriented behaviors. Though someone with ADHD may have an average to above average I.Q., symptoms of the disorder can impede academic achievement. The following section discusses the role of self-regulation as it relates to executive function, ADHD, stimulant medication, and success in school.

The Importance of Self-Regulation on Academic Functioning.

The relationship between self-regulation, academic functioning, and ADHD is not entirely understood; however, self-regulation (a component of EF) appears to be integral to academic success. Because self-regulated behaviors are often a challenge for those with ADHD, negative impacts on academics are possible. For example, allocating time to study, remaining organized when faced with multiple coursework demands, establishing and maintaining routines without parental guidance, and staying disciplined in spite of temptation are all components of self-regulation needed for adult student success (Wolf & Wasserstein, 2001). Research suggests that self-regulation is closely related to EF. According to Barkley (2001) EF's consist of general classes of self-directed actions that humans use in self-regulation. Self-regulation has been correlated with EF ability leading to assertions that cognitive effort required for self-regulation is the same as that needed for executive function (Kaplan & Bergman, 2010). Kaplan & Bergman

(2010) hypothesize that self- regulation and EF may depend on common cognitive resources such as frontal and parietal control mechanisms. Therefore, self-regulation, EF, and academic functioning appear to be viewed as elements that are constantly interacting with one another. EF information provides a sense of time, behavior towards future goals, and away from immediate rewards (Barkley, 1997). Within the academic realm, student success requires individual focus on distant goals with long-term rather than immediate benefits. Good students realize that rewards for academic commitment may not to come to fruition for months or years to come. Continued commitment and progress toward goals over the course of time are examples of self-regulative behaviors (Koo & Fishbach, 2008). The EF aspect of response inhibition sustains goal-oriented behaviors, which requires self-disciplined, guided actions toward future actions (Barkley, 1997). Self-directed processes and goal-directed behaviors are preserved through resisting or ignoring distractions. Therefore, response inhibition is an important aspect of both EF and academic success and life in general.

Academic Implications of ADHD

The true extent that EF has on academic performance remains unclear. However, those with ADHD do appear to struggle to a greater degree in academics than non-ADHD peers. Those with the disorder are at greater risk of academic failure, more likely to drop out of college prior to completing a degree program, and face tremendous difficulty at higher education level (DuPaul, Weyandt, O'Dell, & Varejao, 2009). It appears that those with ADHD potentially struggle in higher education settings due to EF deficits. Lower grades in college may be the result of impulsivity interfering with self-regulation to complete academic activities (Norwalk, Norvilitis, & MacLean, 2009). Once the ADHD teenager leaves the structure of both the school and the home for flexible college life, academic challenges tend to mount. Young adults with

ADHD report making poor decisions that lead to academic failure (Green & Broussard, 2009). Faced with the need to get organized (Wilens & Dodson, 2004), college students with the disorder have difficulties with time management, organization, and higher-order thinking. Such challenges are potentially due to EF deficits in self-regulation and goal orientation (Proctor & Prevatt, 2009; Wilens & Dodson, 2004). Meaux et al. (2006) found that when ADHD participants entered college, increased autonomy and academic demands lead to procrastination and poor sustained attention. Due dates for assignments were missed, absences were frequent, and the motivation to study was found to be rather low. Results from studies conducted among younger participants with the disorder are consistent with these findings. Children and young adults with the disorder have lower grades, more academic struggles in general, and are more likely to be on academic probation than non-ADHD youths (Biederman et al., 2004; DuPaul et al., 2009; Proctor & Prevatt, 2009). Norwalk, Norvilitis, & MacLean (2008) also found a negative correlation between ADHD symptoms, study skills, and overall adjustment to academics. These may explain why ADHD students also report difficulty taking tests, dealing with time constraints for tests and assignments, and believe they must work harder than non-ADHD peers to do well in school (Du Paul et al., 2009).

Because EF is suspected to be such an important part of self-regulation, Petersen, Lavelle, & Guarino (2006) propose that results from EF measures may be used to understand college learning in terms of planning, strategy use, and self-regulation. Researchers found that self-regulation strategies such as time management and concentration were related to EF performance as was test-taking skills and study habits (Lavelle & Guarino, 2006). Metacognitive judgments regarding material complexity and perceived time available were found to influence one's study time allocation (Son & Metcalfe, 2000). Therefore, it would

make sense that poor self-regulation would lead to less time allotted to academic study. ADHD symptomology associated with executive dysfunction may impair self-regulatory abilities needed for academic achievement. The failure to manage one's actions can have detrimental effects on multiple academic outcomes. Therefore, it is important that researchers acquire a greater understanding of the relationship between ADHD, and the EF construct.

Academic Implications of Executive Dysfunction

When the ADHD individual reaches college, many symptoms of the disorder continue to persist. 3-4% of college students report having symptoms of ADHD (Proctor & Prevatt, 2009). Proctor & Prevatt (2009) assert that ADHD often leads to academic, social, and occupational dysfunction of adults. Because ADHD is thought to impair a variety of executive functions, questions arise regarding the implications of executive dysfunction on ADHD students' academic functioning. However, the extent to which these functions affect academic performance is debated within the research. Some researchers have suggested that normal EF is necessary to achieve in English, math and science (St. Claire, Thompson, & Gathercole, 2006). Biederman, Monuteaux, Doyle, Seidman, Wilens, Ferrero, Morgan, & Faraone (2004) reported that ADHD children with and without EFD's performed worse on all academic outcomes assessed compared to controls. Even controls with EFD's did not perform significantly worse than those with ADHD on academic measures (Biederman et al., 2004). Latzman, Elkovitch, Young, & Clark (2009) and Proctor & Prevatt (2009) found a significant correlation between EF composites and academic achievement. Using multiple regression, the results on EF measures of inhibition predicted academic scores in science and math of adolescent males with ADHD, while WM results were correlated with reading comprehension ability (Latzman et al., 2009) However, DuPaul et al. (2009) claims that research on college student executive functions indicates that

there is no statistical difference between ADHD and non-ADHD controls on certain EF measures. Unfortunately, not enough research regarding adult ADHD and executive dysfunction exists to make a sound conclusion either way. As a result, efforts are needed to investigate executive function deficits (EFD's) in adult samples with ADHD to provide a better understanding of how such processes impact academics (Biederman et al., 2004). Therefore, the extent to which EF's affect overall indicators of academic success or failure remains in question.

The studies to date are valuable in that there is an attempt to evaluate the connection between EF and academic functioning. However, with regard to the adult ADHD population such findings may not be applicable. As an example, Latzman et al. (2010) evaluated only male subjects whose mean age was 13.64 years old, which is far from the age of most college students. Also, since none of the participants reported having ADHD, the connection between EF and academic functioning may be different due to the effects of the disorder. Research has also indicated that over 70% of subjects were raised by Caucasian college-educated parents (Latman et al., 2010). This limits the applicability of results to students whose parents or guardians fail to have a college degree and/or were raised in a lower SES environment. According to the authors, the cross-sectional and correlational methods of the experiment do not allow for causal conclusions to be made (Latzman et al., 2010). Though Petersen et al. (2006) attempted to assess more a heterogeneous, college-aged population, the EF and academic measures were self-report only and therefore raise questions of validity. Self-report items rely solely on participant honesty for evaluation of complex psychological and cognitive constructs. The relationship between EF and academic functioning of ADHD adults at the collegiate level requires a heterogeneous sample with more objective measures of evaluation.

Stimulant Effects on Academic Performance

Because of the pressure associated with success in college, many ADHD student turn to stimulant medication for help. Stimulant medication is thought to have a positive influence on the academic performance of those with ADHD. Improvements in daily class performance have been linked to improvements in critical thinking skills and concentration based on self-report measures and interviews with young adults (Meaux et al., 2009). Better problem-solving skills associated with stimulant medication use have positive effects on academic achievement most likely because ADHD symptoms such as hyperactivity, inattention, and impulsivity are reportedly reduced (Meaux et al., 2006). Stimulants such as Mph for example, appear to exert effects by improving one's regulatory ability (Sinead et al., 2006). Also, there appears to be a greater likelihood of student engagement in the learning process when these symptoms are controlled for through medication. In case studies, adults with ADHD report improved academic success as a result of taking stimulant medication (Meaux et al., 2006). This is especially the case if medication trials began in childhood. For example, Powers, Marks, Miller, Newcorn, & Halperin (2008) found that consistently medicated ADHD adolescents performed better than their non-medicated ADHD counterparts but failed to reach non-ADHD achievement levels. Consistent with these findings, Barbaresi, Katusic, Colligan, Weaver, and Jacobsen (2007) claim that those with ADHD with a history of taking stimulant medication had higher levels of achievement in reading, lower school absenteeism, and less grade retention compared to non-medicated adolescents with ADHD. Stimulant-medicated subjects also fared better on additional measures of academic achievement such as subtests of the WIAT-II and high school G.P.A. (Powers et al., 2008). These results indicate that though limited, academic performance gains for ADHD patients can be attributed in part to stimulant medication use (Powers et al., 2008).

Additional research has suggested that adults ADHD taking stimulant medication on a regular basis demonstrated significant improvements in organization skills (Roth & Saykin, 2004). College students with the disorder stated that ADHD medication helped them study, maintain attention in courses that were longer in duration, and complete their course work (Meaux et al., 2009). Therefore, while many stimulant-medication takers report improved grades and study skills, additional research is needed to support such findings among adult ADHD subjects.

Though research suggests that ADHD adults struggle with academics at the college level, it is difficult to analyze just how many of these struggles are due to study habits, medication status, and other personal or environmental factors (Du Paul et al., 2009). Covariates such as gender also need to be taken into consideration as they too may influence academic success. When Biederman et al. (2004) conducted a 4-year follow-up on academic outcomes of ADHD on adult students, only males were included in the analysis, in spite of having 140 female participants in his original study. Gender differences in academic outcomes may be evident and must be accounted for. Also, some of the participants were not given all assessment measures due to time restraints, lack of desire to participate, or scheduling conflicts (Biederman et al., 2004), thus leaving only motivated participants who were readily available. Also, it is important to note that although academic assessments were used in the study as an indicator of achievement, G.P.A., and scores on academic assessments such as the *Stanford Achievement Test* (SAT) were not considered.

Most would agree that at the college level, the main indicator of academic success is one's grade point average, not whether the student had an extra support class in his/her schedule or had repeated a course, as the investigators had indicated. Therefore, assessing the academic functioning of medicated and non-medicated ADHD college students must involve data

collection of subject G.P.A. In addition to grades, analysis of academic data should also include interviews and self-report questionnaires to examine the true effect of the disorder and stimulant medication on academic functioning (Du Paul et al., 2009). However, even studies that have included self-report and interviews in this area are somewhat flawed methodologically. Though Meaux et al. (2009) conducted a qualitative study of adults with ADHD and academic functioning complete with such measures, only 18 volunteers participated and demographic information such as ethnicity and gender were not included. This limits the generalizability of study conclusions to any subset of the ADHD population. Once again, G.P.A. data were not collected though the study focused on ADHD academic functioning at the college level.

Summary

ADHD in adults has not received the attention from researchers that adolescent ADHD has garnered. The ADHD brain appears to differ from that of controls in structure and function, potentially resulting in executive dysfunctions such as inhibitory, attention, and working memory deficits. Such deficits are thought to contribute to difficulty with self-regulation, a cognitive construct thought to be a component of executive function and academic success. Neuropsychological measures attempt to measure EF deficits among adults with ADHD, but possess limitations and have yielded inconsistent findings. Because stimulant medication is often used to treat ADHD, the role these medications play in adult executive and academic function is important, yet remains unknown. In summary, very little is known about adult ADHD. There is even less of an understanding of this population's neuropsychological and academic functioning under the influence of stimulant medication. As a result, additional research is required to understand how stimulants affect the executive and academic functioning of adults possessing ADHD at the college level.

Chapter 3: Methodology

The primary purpose of this study was to analyze a potential relationship between stimulant medication and executive and academic functioning of adults with ADHD. The relationship between medication status and executive and academic functioning was analyzed using quantitative research methods, which include the following: A Goodness-of-fit test (Kolmogorov-Smirnov test) for normality, two-sample t tests, tests for equal and unequal variances, and Fisher's Exact Tests were also conducted. In order to analyze relationships between executive and academic functioning in addition to medication status, least-squares regression was used.

Design Summary

This study is designed to follow Cresswell's (2009) *experimental method plan* for quantitative research and experimentation. This chapter consists of the following information: A description of participants, materials, measures, and experimental procedures to be used, as well as potential threats to internal and external validity. From a methodological perspective, this study has been designed to answer the following research questions using the subsequent hypotheses:

1. Is there a significant difference between the executive functioning of stimulant-medicated ADHD college students compared to non-medicated ADHD college students as measured by neuropsychological assessments?
    a. It was hypothesized that stimulant-medicated participants would outperform non-medicated subjects on tests of executive function.
2. Do students with higher scores on assessments of executive function have on average a higher Grade Point Average than students with lower scores?

a. It was hypothesized that G.P.A. and executive functioning scores were positively correlated.

3. Do stimulant-medicated participants have a higher Grade Point Average than non-stimulant medicated participants?

    a. It was hypothesized that stimulant-medicated participants would have a higher Grade Point Average than non-stimulant medicated participants.

Participants and Setting

Using convenience sampling at one large, urban, private university in southern California and one smaller rural university in central California, a call for participants requested adult volunteers with ADHD to take part in this correlational study. To achieve an adequate sample size, this study consisted of 33 participants 18 years of age or older. In order to take part, each individual must have an ADHD diagnosis based on DSM-IV criteria by a mental health professional such as a medical doctor or clinical psychologist. Due to the fact that medication status was not be altered by the experimenter, participants were matched to a group based on their current medication status. The goal was to achieve and maintain a 50-50 split between participants who take stimulant medication on a regular basis (regular basis is considered as medication taken daily) and those who do not take stimulant medication regularly if at all. Non-medication takers consisted of the control group, while stimulant medicated participants made up the experimental group in this study.

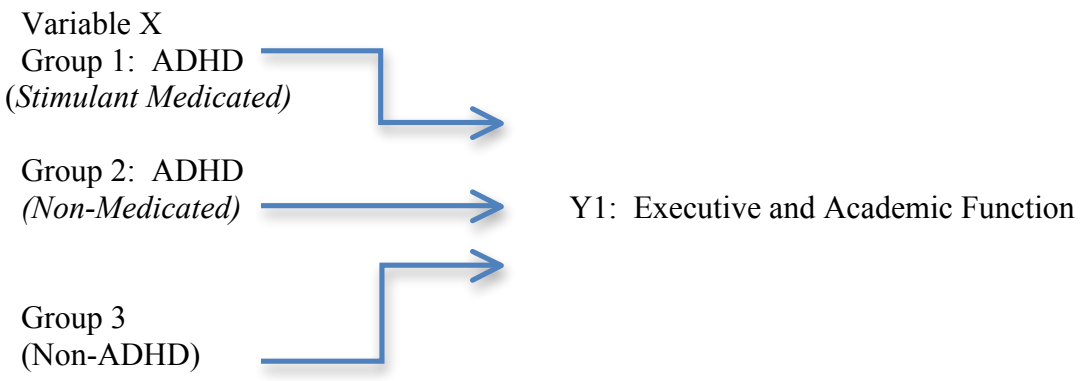

Figure 3.1. Independent and Dependent Variables used for ADHD Between Groups Comparisons adapted from Cresswell (2009) p. 55. Copyright 2009 by Sage Publications.

Variables

This study consisted of one independent variable (IV) and two sets of dependent variables (DV). The IV was stimulant medication status of participants. Therefore, two groups will be formed based on self-disclosed stimulant medication status. Stimulant medicated subjects were considered members of Group 1. The second group (Group 2) individuals were medication-naïve ADHD college students at the time of testing. Group 3 consisted of non-ADHD college students. The first dependent variable (DVa) was all three groups' performance on tests of executive function as measured by the *Kaufman Short Neuropsychological Assessment Procedure* (K-SNAP) and *Test of Variables of Attention* (T.O.V.A). Each groups' performance was denoted as (DVa1, 2, or 3). The second dependent variable (DVb) was the mean of all three groups' cumulative grade point average. This served as the primary measure of academic function for the study.

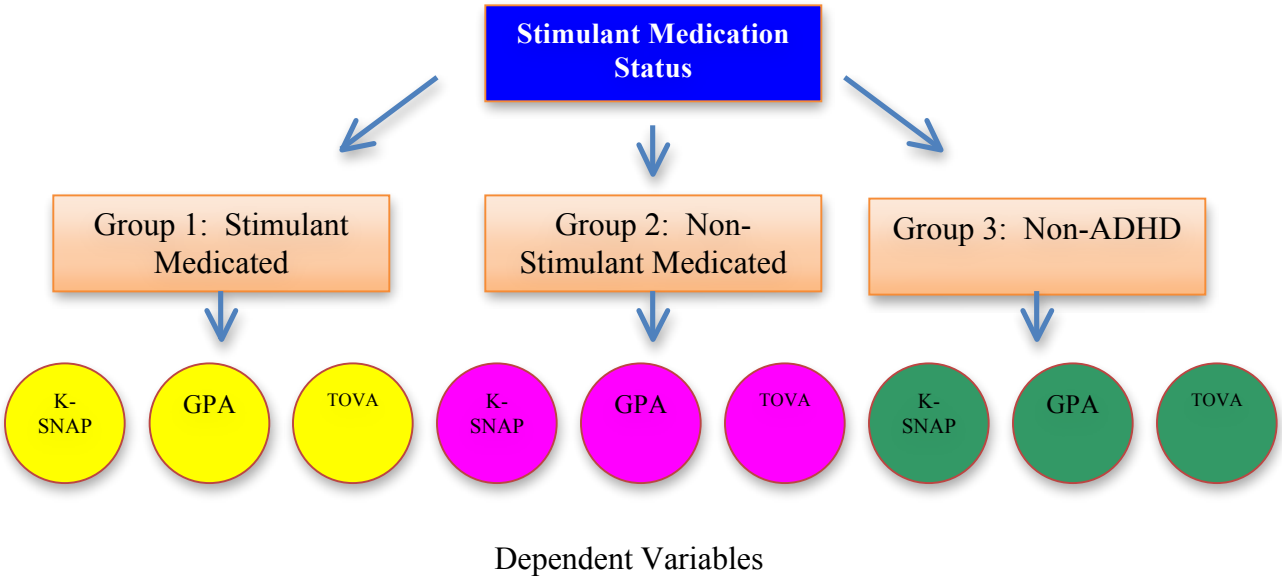

Figure 3.2. Study Design

Instrumentation and Materials

Measures of Executive Function

The Kauffman Short Neuropsychological Assessment Procedure.

In order to measure executive function, the pencil and paper based K-SNAP was used and administered to both the experimental and control groups. According to the *Kaufman Short Neuropsycholgical Assessment Procedure Manual*, the K-SNAP provides a general cognitive profile to assess attention-orientation, simple memory and perceptual skills, and complex intellectual functioning and planning ability (Kaufman & Kaufman, 1994). The measure was normed using representative standardization sample of 2000 people ranging from 11-85 years of age and older. All items were checked for cultural and intellectual ability bias as well and standardization scores are provided across a wide range of ability levels which allow for reliable assessment of persons with moderate to severe intellectual disabilities (Kaufman & Kaufman, 1994).

The K-SNAP yields three subtest scores and two composite scores, requiring a total of 30 minutes to administer. Subtest scores for attention-orientation, simple memory and perception, and complex intellectual functioning are evaluated using age-based norms as well as an impairment index (Kaufman & Kaufman, 1994). The two composite scores yield standard scores, percentile ranks, and descriptive categories of performance (Kaufman & Kaufman, 1994). These scores are then used to determine K-SNAP composite score and impairment index. To establish a frame for participant selection, 1988 census data was used. The K-SNAP was normed using a sample of 2000 subjects from 27 states and 60 communities across 13 age groups (Kaufman & Kaufman, 1994). To establish reliability, two measures were used. Internal consistency was established using split-half reliability for each age category and subtest. The following are reliability coefficients for Gestalt Closure, Number Recall, and Four Letter Words, and K-SNAP composite respectively: .82, .83, .84, and .89. Stability of the assessment was determined using the test-retest design. Reliability coefficients for each sub-test ranged from r = .64-.84 (Kaufman & Kaufman, 1994)

In this study, academic functioning was be measured by participant self-report of cumulative G.P.A. Each participant completed the ADHD Indicator Survey/Questionnaire (see Appendix A) consisting of 10-15 questions regarding G.P.A., gender, grade level/year in college, age, age of ADHD diagnosis, stimulant medication status, etc. The questionnaire was created by the primary investigator and reviewed by the clinical psychologist overseeing the study. This measure served to gather both general and academic needed for the study. Questionnaires were completed prior to administration of the K-SNAP and T.O.V.A. tests.

Test of Variables of Attention.

The second visual neuropsychological measure to assess EF was the computer-based Test of Variable Attention (T.O.V.A.). The T.O.V.A. was used and administered to all three participant groups. According to the *T.O.V.A. Professional Manual* (2007) "The T.O.V.A. was developed to measure attention and impulse control processes in four areas: response time variability, response time, impulse control (commission errors), and inattention (omission errors) (p. 2)." The assessments are designed to measure attention using visual and auditory tests and have been normed on populations aging from 4-80 years. In addition, norms have been differentiated by both age and gender of participants.

The T.O.V.A. consists of two parts and takes 21.8 minutes to administer. The first part of the exam consists of boring, fatiguing tasks that require sustained attention (Test of Variable Attention [T.O.V.A.], 2009). The second exam requires inhibitory skills in addition to sustained attention to respond to specific stimuli (*T.O.V.A. professional manual,* 2011). The visual and auditory T.O.V.A. have a reliability coefficients of r = .88 and .99, respectively (*T.O.V.A. professional manual,* 2011). Validity of the T.O.V.A. is based on the exams' ability to discriminate ADHD subjects from non-ADHD subjects using measures of attention. The T.O.V.A. is a valid measure considering the following validity coefficients: .80 for the visual exam and .86 for auditory measures (*T.O.V.A. professional manual*, 2011). Both auditory and visual components of the T.O.V.A. are designed to assess executive functions such as attention, inhibition, and working memory. The clinical version of the T.O.V.A. is used by licensed clinicians, such as psychologists, physicians, and social workers to aid in the diagnosis and treatment of attention disorders (T.*O.V.A. professional manual*, 2011). On the other hand, the screening T.O.V.A. can be used in non-clinical settings such as schools and rehabilitation

programs (*T.O.V.A. professional manual*, 2011). Therefore, appropriate licensure and training is needed depending on which form of the exam would be administered. In this study, the clinical version was given to research participants. All subjects took the exam in a room equipped with a computer and T.O.V.A. program. Each participant was under the supervision of the primary investigator. Upon completion of the exam, the software generated a score report based on participant performance with scoring details and interpretation in the form of an Attention Performance Index (A.P.I) which ranged from a score of -10 to +10, with lower scores being indicative of a possible attention problem (*T.O.V.A. professional manual*, 2011).

Experimental Procedures

The design of this study was correlational (Cresswell, 2009) in order to test for a potential relationship between stimulant medication status and executive and academic functioning of adults with ADHD. Subjects were not assigned to a treatment, so volunteers were not randomly assigned to a group. Based on voluntary disclosure of stimulant medication status, group association of participants was based on their responses to medication-related questions. Those taking stimulant medication for ADHD were automatically considered part of the experimental group, while those not taking stimulant medication were part of the control group. Both groups received the same academic and executive function measures but data was disaggregated by medication status to make a comparison between groups. Therefore, group assignment was strictly for data analysis purposes and not for experimental purposes. It is important to note that this study did not alter one's current medication status in any way whatsoever and served only to establish a possible correlation between stimulant medication status and executive and academic functioning of adults with ADHD.

Upon completion of the academic questionnaire, subjects will be asked to create a personal identification number (PIN) consisting of two letters and two numbers of their choice. Once chosen, the primary investigator will link this PIN to his/her group assignment based on self-disclosed stimulant medication status. Most importantly, the primary investigator will use these numbers for identification purposes rather than using subjects' names, allowing for complete privacy and anonymity for all participants. These numbers will also serve as identification for the K-SNAP testing/scoring. The PIN allows for participant identification without individual disclosure of identifying information at any point in the study.

Threats to Validity

Internal Threats to Validity.

Threats to internal validity are potential errors that can occur during experimental procedures, treatment, and participant experiences, which jeopardize the researcher's ability to make correct inferences from data collected during the study (Cresswell, 2009). According to Cresswell (2009) internal validity must be strengthened by considering the following factors: History, maturation, regression, selection, mortality, diffusion of treatment, compensatory/resentful demoralization, compensatory rivalry, testing, and instrumentation. Because this study involved data collection from ADHD individuals privately over the course of one to two days, a number of these threats are eliminated. Maturation or mortality were not concerns in this study due to the fact that participants were needed for a maximum of one hour to complete academic and EF measures. Diffusion of treatment was the primary limitation here in that subjects in the stimulant-medicated group were not taking the same medications, at the same time, using the same dosage. Stimulant medication status was altered in order to avoid interfering with medically prescribed treatment. In addition, both the experimental and control

group members completed the same measures once and only once, decreasing threats to history, regression, testing, instrumentation, diffusion of treatment, compensatory rivalry and demoralization. Because participation was voluntary and participants were not randomly assigned, primary threats to internal validity arose in the area of selection. Those who volunteered to participate may have had intelligence quotients that surpassed those of the average ADHD adult not enrolled in a university. These students may also have developed compensatory strategies over time, or been dishonest when completing the questionnaire. To compensate for this, confidentiality was ensured. In order to encourage honest responses the call for participants was structured to gather as many subjects as possible to allow for a heterogeneous sample.

External Threats to Validity.

Threats to external validity occur when inferences made by the researcher are erroneously are generalized to alternative populations, settings, or situations (Cresswell, 2009). Interaction of selection and treatment, setting and treatment, and history and treatment constitute three primary threats to external validity. The primary threat to external validity involved the taking of stimulant medications themselves. Due to the fact that participants were taking a variety of stimulant medications at different dosages and times of administration, generalizability to *all* stimulant medication takers and non-takers should be cautioned. In other words, the type and timing of stimulant medication usage was not controlled for in this study; therefore correlation rather than causation was analyzed. In addition, because this study took place at a two highly selective universities, generalization to other contexts and populations was limited. Subjects who volunteered to participate were enrolled at two prestigious universities and are therefore highly intelligent individuals who most likely developed internal and external compensatory

strategies to cope with ADHD. As a result, participant make-up created limitations concerning generalizeablility within this study. To address these threats, a limitations section in chapter 5 will be included to emphasize these concerns.

Procedure

All experimental sessions were conducted individually. Participants were greeted by the experimenter, an advanced doctoral student in educational psychology to explain the purpose of the study and given consent for participation. Upon receiving consent for participation, subjects completed the ADHD Indicator Survey/Questionnaire. When the survey was completed, the answers were reviewed by the primary investigator, group assignment was determined, and a 3-digit identification number was chose for identification purposes. Participants then spent approximately 30-40 minutes taking the K-SNAP administered by the experimenter. Upon completion of the K-SNAP, subjects completed the twenty minute T.O.V.A. test on a computer with the primary investigator observing the exam session. Experimenter contact information was

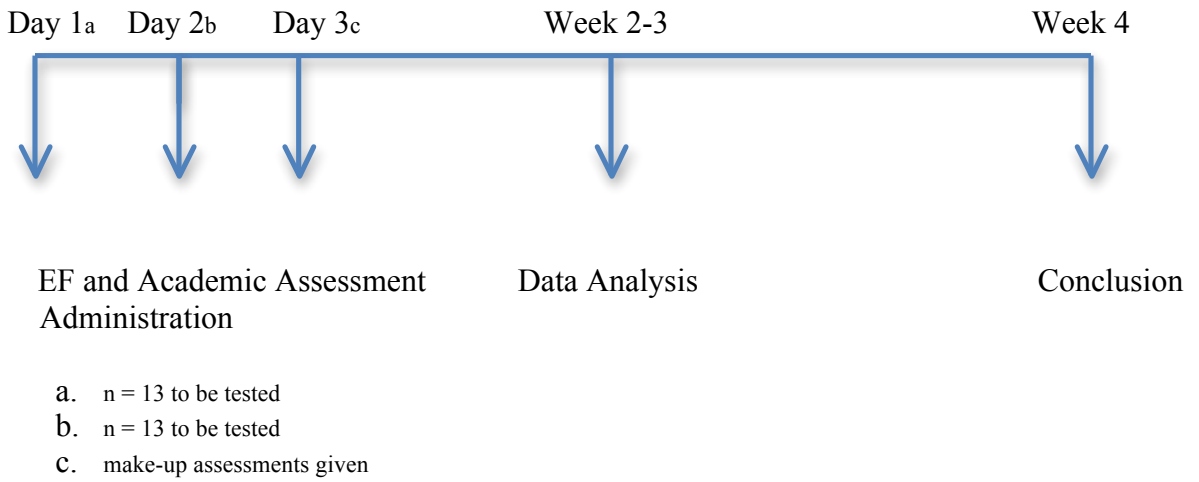

Figure 3.3. Procedure Timeline

given to all participants to answer additional questions if needed.

From start of finish, data were compiled for each individual in approximately 60 minutes, with complete sample data collection being complete over the course of three days at each university. This allowed for the assessment of approximate 13 subjects on Day 1, 13 subjects on Day 2, and Day 3 served as a "make-up" days for anyone unable able to attend the first or second testing day. When all subjects in both groups were finished being assessed, the results were calculated by hand according to directions outlined by the K-SNAP testing manual. T.O.V.A. test results were generated by the T.O.V.A. software, which produced an Attention Performance Index (A.P.I.) for each subject. After K-SNAP and T.O.V.A. results were determined, the experimental group's scores were compared to the control group to analyze the executive functioning of both groups. Mean G.P.A. for both the experimental and control groups were calculated as well using responses given on the ADHD Indicator Survey/Questionnaire. Statistical analyses were used test for significant differences in the executive and academic functioning of stimulant medicated (experimental) and non-stimulant medicated (control) groups (see Table 3.1).

In order to analyze data collected in the study, the statistical software SAS was used (SAS Institute Inc., 2009). To compare the academic and executive functioning of adults with ADHD on and off of stimulant medication, a two-tailed t-test will be used to test research hypotheses at the α .05 level of significance. t-testing will allow for comparison of the following for stimulant-medicated ADHD adults, non-stimulant medicated ADHD adults, and non-ADHD controls: a) medication status and executive functioning, b) medication status and academic functioning (G.P.A.), and c) G.P.A. and executive functioning. Additional t-tests will be used to

compare mean G.P.A.'s between groups. These tests will allow for a comparison of all groups on multiple measures of executive and academic functioning.

Table 3.1

Statistical Tests for Data Analysis

Research Question Number	Method	Alpha Level	Analysis	Decision Rule
1	Two-sample t	0.05	Between groups comparison (ADHD medicated and non-medicated) on EF measures.	Reject null if p < 0.05
2	Two-sample t	0.05	Between groups comparison (ADHD stimulant-medicated and non-stimulant medicated on academic measure (G.P.A.)	Reject null if p < 0.05
3	A. Cochran-Armitage Trend Test. B. Least Squares Regression	0.05	Relationship between EF and GPA	A. Reject null if p < 0.05 B. Correlation coefficient

Summary

ADHD in adults has not received the attention from researchers that childhood ADHD has garnered. The ADHD brain appears to differ from that of controls in structure and function, potentially resulting in executive dysfunctions such as inhibitory, attention, and working memory deficits. Such deficits are thought to contribute to difficulty with self-regulation, a cognitive construct thought to be a component of executive function and academic success. Neuropsychological measures attempt to measure EF deficits among adults with ADHD, but possess limitations and have yielded inconsistent findings. Because stimulant medication is often used to treat ADHD, the role these medications play in adult executive and academic function is important, yet remains unknown. In summary, very little is known about adult ADHD. There is even less of an understanding of this population's neuropsychological and

academic functioning under the influence of stimulant medication. As a result, additional research is required to understand how stimulants affect the executive and academic functioning of adults possessing ADHD at the college level.

Chapter 4: Results

Findings for this study will be presented in two sections. The first section discusses descriptive data of participants, their medical history, and academic performance. Data discussed within this section were collected via the ADHD Indicator Survey/Questionnaire distributed to the participants prior to administration of the K-SNAP and T.O.V.A tests (for information on the validity and reliability of the K-SNAP and T.O.V.A. assessments, see Chapter 3). The second section addresses results derived from the between groups comparison using statistical analysis on neuropsychological and academic measures used. Results in the second section are discussed to address the following research questions in order: *1. Is there a significant difference between stimulant medicated and non-medicated ADHD students' executive functioning (EF) as measured by scores on neuropsychological assessments such as the T.O.V.A. and K-SNAP? 2. Do ADHD students with higher EF scores have on average a higher G.P.A. than those with lower EF scores? 3. Do stimulant medicated ADHD students have a higher G.P.A. than non-medicated controls*? It was hypothesized that stimulant-medicated students would outperform non-stimulant medicated students on assessments of executive function (the T.O.V.A. and K-SNAP) and also have a higher cumulative G.P.A. It was also hypothesized that EF scores would be positively correlated with G.P.A. The threshold for all statistical tests was α 0.05 for between groups comparisons.

Descriptive Data: Demographic Information

Sample Composition.

Participants in the study were volunteers responding to a university-wide call for participants at two highly selective 4-year universities. University A was private, located in an urban area of California with over 36,000 students. Participants were also recruited from

University B a public, rural 4-year institution with approximately 18,000 attendees. 40.7% of the ADHD subjects attended University A (*n* = 11) while 59.3% attended University B (*n* =16). 16% of Non-ADHD (control group) participants attended University A (*n* = 1), while the remaining 84% attended University B (*n* = 5). Controls volunteered their time for the study. There was a greater interest to contribute to research without financial compensation among students attending University B than University A. Participants ranged in age from 18-45, with the average age being 23.4 years. The sample was almost equally split between genders as shown in Table 4.1 below. 48% of the participants were male, while 51% were female. In terms of ethnicity, almost 70% of participants in the study were Caucasian (*n*=23), with 12.1% being Latino/a (*n*=4), 6% Asian (*n*=2), 3% African American (*n*=1) and Persian (*n* = 1), and 6% (*n*=2) describing themselves as mixed and thus classifying themselves as 'Other' as indicated by Table 4.2.

Table 4.1

Descriptive Results: Student Demographic Data by Gender & Age

Gender	Number	Percent
Male	16	48.48
Female	17	51.51
Mean Age of Sample	23.4	Range: 18-45

Table 4.2

Descriptive Results: Student Demographic Data by Ethnicity

Ethnicity	Number	Percent	Ethnicity	Number	Percent	Ethnicity	Number	Percent
Asian	2	6%	African American	1	3%	Persian	1	3%
Caucasian	23	69.6%	Latino/a	4	12.1%	Other	2	6%

Table 4.3

Descriptive Results: Sample Composition by Year in School

Year in School	Year in College	Percent
Freshman	5	15.2%
Sophomore	8	24.24%
Junior	3	9%
Senior	8	24.2%
Graduate	9	27.27%

Table 4.4

Descriptive Results: Sample Composition by Major

Major	Number of Participants	Percent
STEM	11	33.33%
Agriculture	2	6%
Political Science/Law	2	6%
Business	7	21.2%
Visual/Performing Arts	3	9.1%
Liberal Arts	3	9.1%
Social Sciences	5	15.2%

Table 4.5

Descriptive Results: Male and Female Two Sample t Test Comparing Executive Function

	TOVA Mean	K-SNAP Mean	TOVA σ	K-SNAP σ
Males	2.07	106.85	3.3727	7.8935
Females	1.18	99.57	2.3581	9.5088
P-Value	p= 0.9148	p = 0.0399		

Table 4.6

Descriptive Results: Male and Female Two Sample t Test of G.P.A.

	Mean Cum GPA	St. Dev	P-Value	Percentage
Males	3.0	0.4782		> 3.0 =66.6%
Females	2.86	0.5690	0.2900	< 3.0 =33.4%

Gender Comparisons on Study Measures.

Data analyses of scores on the T.O.V.A. and K-SNAP were analyzed for between group differences of males and females using a two-sample t test. Table 4.5 displays results of these tests. The average T.O.V.A. A.P.I. for males was 2.07, while that of the females was 1.18.

Analysis yielded a p-value of 0.9148 suggesting that T.O.V.A. scores between genders did not differ significantly. This was contrary to the K-SNAP analysis. The mean K-SNAP score for males was 106.85 and 99.57 for females. A p-value of 0.0399 indicates that there was a significant difference between male and female scores on this EF measure.

Using self-reported cumulative G.P.A. as an academic performance measure, a t test was used to determine if there was a significant difference between male and female G.P.A. With the mean being 2.86 for females and 3.0 for males, a p-value of 0.2900 suggests that there was not a significant difference between male and female cumulative G.P.A. (see 4. 6).

Academic and Medical History

Academic History.

Representation by year in college was fairly well distributed with the exception of participants of Junior class standing. As indicated by Table 4.3, only 9% of subjects were Juniors at the time of the study. Freshman, Sophomores, Seniors, and Graduate students made up 15%, 24%, 24% and 27% of the sample respectively. This provided for a heterogeneous sample in terms of academic standing based on year in school.

As referenced in Table 4.4, over half of the study's participants were majoring in an area of STEM or business. 15% of participants were majoring in the social sciences, while approximately 18% of subjects were specializing in Visual and Performing or Liberal Arts. Agriculture and Political Science majors completed the sample, with 2% coming from each area. Overall, the cumulative self-reported G.P.A. for participants was rather high. With controls excluded, 63% of ADHD participants taking part in the study reported having a G.P.A. of over 3.0. 18.5% claimed to have an overall G.P.A of 2.5-2.9 with the remaining 18.5% maintaining a

2.0-2.49 (see 4. 5). Control G.P.A. data is shown in 4. 6, indicating that 66.6% of controls had over a 3.0 and 33.4% maintain between a 2.5-2.9 G.P.A.

There was an almost even split between participants with ADHD who reported using student support services at school as those who were not. With regard to the use of such services, Table 4.9 shows the number of participants who had academic accommodations at the college level. Of those with ADHD, 48.1% (n=13) stated that they took advantage of student support services to assist them with their coursework. Approximately 51.9% (n=14) of the participants with the disorder did not use these services. Of the participants with comorbid conditions, 3 out of the 4 participants possessing a learning disability had academic accommodations but neither of the students (n=2) who suffered from a Traumatic Brain Injury (TBI) in childhood felt they needed support services and therefore, chose not to use them in college. The participant possessing Generalized Anxiety Disorder reported using support services as well. Additional information regarding participants with comorbid conditions is summarized in 4. 9.

Medical History.

At the time of ADHD diagnosis, most participants were diagnosed either really early in life or really late in their academic life. As indicated by Table 4.11, 33.3% of participants received their diagnosis as an adult (over 17 years old) and 22.2% of participants were between the ages of 5-8. The remaining 18.8% were in either middle or high school when diagnosed. Though ADHD diagnosis is coupled with a subtype, many participants were unaware of which subtype their medical practitioner diagnosed them with. 26% of subjects reported to have been diagnosed with the hyperactive subtype of ADHD, 22.2% claimed to have the combined form and 3.7% had the inattentive type. Surprisingly, 41% did not know which subtype of ADHD

they possessed. Data indicated that most participants were diagnosed with ADHD by a clinical psychologist or psychiatrist (*n*=19), making up 77.7% of the sample tested. 18.5% (*n*=5) received their ADHD diagnosis from a general practitioner or pediatrician and one participant was unsure as to who diagnosed him with the condition because he was in elementary school at the time.

Stimulant medication status was an important aspect of the study in that group assignment was determined by whether or not a subject was taking stimulant medication on a regular (daily) basis. Out of 27 participants, 13 took stimulant medication for ADHD on a daily basis. This comprised 48.2% of the ADHD participants. 14 subjects stated that they did not take stimulant medication on a regular basis for ADHD, making up the remaining 51.8% of participants. Of the non-medicated group, 4 students admitted that they did use a stimulant on an irregular or 'as needed' basis. However, these subjects were not medicated at the time of testing. Participants were considered 'Stimulant Medicated' if they had taken medication for ADHD within 24 hours of testing and had been taking this medication on a regular basis. Medication status is summarized in Table 4.12.

Overall a total of 7 participants (21.2%) possessed conditions in addition to ADHD (see Table 4.9). 12% (*n*=4) claimed to have a learning disability and 6% (n=2) suffered from a Traumatic Brain Injury (TBI) as a young child. One subject expressed that he had been diagnosed with Generalized Anxiety Disorder in addition to ADHD.

Table 4.7

Descriptive Results: Cumulative Self-Reported Grade Point Average of ADHD Sample

G.P.A.	**2.0-2.5**	**2.6-2.9**	**3.0-3.5**	**3.6-3.9**	**4.0+**
Number	5	7	14	6	1
Percentage	15%	21%	42%	18%	3%

Table 4.8

Descriptive Results: Cumulative Self-Reported Grade Point Average by Group

Medication Status	Cumulative G.P.A.	Number	Percent
Stimulant	2.0-2.5	2	6%
Stimulant	2.6-2.9	4	12.1%
Stimulant	3.0-3.49	5	15.1%
Stimulant	3.5-4.0	2	6%
Stimulant	4.0 and above	0	0%
Non-Stimulant	2.0-2.5	3	9%
Non-Stimulant	2.6-2.9	1	3%
Non-Stimulant	3.0-3.49	6	18.1%
Non-Stimulant	3.5-4.0	3	9%
Non-Stimulant	4.0 and above	1	3%
Control	2.0-2.5	2	6%
Control	2.6-2.9	0	0%
Control	3.0-3.49	3	9%
Control	3.5-4.0	1	3%
Control	4.0 and above	0	0%

Table 4.9

Descriptive Results: Support Service Use and Comorbid Disorders

Medication Status	Yes	Percentage	No	Percentage
Stimulant Medicated	7	25.9	5	18.5
Non-Stimulant Medicated	6	22.2	9	33.3
Learning Disabled	3		1	
Traumatic Brain Injury	0		2	
TOTALS	**13**	**48.1**	**14**	**51.8**

Table 4.10

Descriptive Results: ADHD Diagnostician

Clinical Psych/Psychiatrist	Pediatrician/G.P.	Unknown
21	5	1
77.7%	18.5%	3.7%

Table 4.11

Descriptive Results: Age of ADHD Diagnosis

	5-8 Years	9-11 Years	12-13 Years	14-16 Years	17+ Years
Number	6	3	3	3	11
Percentage	22.2	11.1	11.1	11.1	40.7

Table 4.12

Descriptive Results: Medication Status of Subjects

Group	Number	Percentage
Stimulant-Medicated	13	48.2
Non-Stimulant Medicated[a]	14	51.8
[a]Irregularly Medicated	4	

Note. a = 'Irregularly Medicated' subjects were assigned to 'Non-Stimulant Medicated'group due to medication status at time of testing.

Statistical Analysis by Research Question

Research Question #1

The first research question, *Is there a significant difference between stimulant medicated and non-stimulant medicated ADHD students' executive functioning as measured by scores on neuropsychological assessments such as the T.O.VA. and K-SNAP*?, was addressed using the following tests: goodness-of-fit test (Kolmogorov-Smirnov test) for normality and a two-sample t test. Tests for equal and unequal variances were also conducted. The t test was used to compare K-SNAP and T.O.V.A. scores between ADHD subjects.

Executive function data is based on T.O.V.A. and K-SNAP scores and listed below in Table 4.12 and 4.13 using simple summary statistics. According to the *T.O.V.A. manual* (2011) The T.O.V.A. produces an Attention Performance Index (A.P.I.), which is based on subject response time, commission errors, and omission errors. Commission errors are though of as impulsivity. Omission errors occur when the participant should initiate a response while commission errors are inappropriate responses. The score range for the T.O.V.A. A.P.I. is -10 to +10, with lower scores suggestive of an attention problem. K-SNAP data are listed in the form of a composite, standard score. These scores are based on subject performance on the Gestalt Closure, Number Recall, and Four-Letter Word tasks. Average scores range from 91-100, with

scores above 111 being considered above average (Kaufman & Kaufman, 1994). Characteristics of the T.O.V.A. and K-SNAP norming sample can be found in Chapter 3.

Simple summary statistics of T.O.V.A. and K-SNAP for the two populations being compared (stimulant-medicated and non-medicated ADHD students) are displayed in Table 4.12 and 13. The sample size (*n*), mean, standard deviation, and minimum and maximum values are also presented. The results of the goodness-of-fit test (Kolmogorov-Smirnov Test) for normality indicate that these data are normally distributed. A two-sample t-test was used to determine whether the mean K-SNAP for stimulant medicated ADHD students differed significantly from the mean K-SNAP for non-stimulant medicated ADHD students. Another two-sample t test was used to determine whether the mean T.O.V.A. for stimulant medicated ADHD students differed significantly from the mean T.O.V.A. for non-stimulant medicated ADHD students. As shown in Table 4.15, the Method column denotes which test is being used for that row (pooled: assuming equal variances for the two groups, Satterthwaite: assuming unequal variances). The pooled test assumes that the two populations have equal variances and uses degrees of freedom n1 + n2 - 2, where n1 and n2 are the sample sizes for the two populations. The other test does not assume that the populations have equal variances. The Satterthwaite test uses the Satterthwaite approximation for degrees of freedom. The test statistics, associated degrees of freedom, 95% confidence intervals for the mean differences and p-values are displayed.

Table 4.13

Simple Summary Statistics: ADHD Participant Neuropsychological Descriptive Data

EF Scores	Group	N	Mean	Standard Deviation	Minimum	Maximum
K-SNAP	stimulant	13	100.07	10.59	80	117
	non-stimulant	14	105.86	7.39	93	117
T.O.V.A.	stimulant	13	1.2	2.7	-3.25	4.8
	non-stimulant	14	1.36	3.1	-7.09	4.92

Table 4.14

Simple Summary Statistics: Control Group Data-Academic and Executive Functioning

K-SNAP St. Score	TOVA API	G.P.A.
99	3.95	3.0-3.5
96	3.48	3.0-3.5
105	0.26	3.0-3.5
117	3.93	3.5-3.9
88	3.9	2.5-3.0
101	3.2	2.5-3.0
Mean: 101	Mean: 3.12	Mean: 3.0
St. Deviation: 9.69	St. Deviation: 1.43	St. Deviation: 2.5-3.9
Min: 88 Max: 117	Min: 0.26 Max: 3.95	Min: 2.5 Max: 3.9

To test for equality of variances (test if the two groups have the same variances), a folded F test is used. Let s^2_{group1} and s^2_{group2} be the standard deviation estimates of the EF score (such as K-SNAP or T.O.V.A.) for group 1 and group 2. The folded form of the F statistic F', tests the hypothesis that the variances are equal, where

$$F' = \frac{\max(s^2_{group1}, s^2_{group2})}{\min(s^2_{group1}, s^2_{group2})}.$$

A test of F' is a two-tailed test because which variance expected to be larger is not specified. The p-value gives the probability of a greater F value under the null hypothesis that the two variances are equal. Table 4.16 shows the testing result of equal variances. The pooled test revealed insufficient evidence of unequal variances (the Folded F statistic=2.05, with p-value = 0.2128 for K-SNAP, and the Folded F statistic=1.32, with p-value = 0.6380 for T.O.V.A.). Using a one-tailed hypothesis, post-hoc power analysis yielded a p-value of 0.3961 given a sample size of 33 given an α of 0.05 using a medium effect size of 0.5. Thus, assuming equal variances (Method: pooled), the two t tests did not result in significant values (the t-statistic = 1.66 with p-value = 0.1103 for K-SNAP, and the t statistic = 0.15 with p-value = 0.8853 for T.O.V.A.), indicating that there are no statistically significant differences between stimulant medicated and non-

medicated ADHD students' executive functioning as measured by scores on neuropsychological assessments such as the T.O.V.A. and K-SNAP.

Table 4.15

Two-sample t-tests for K-SNAP and T.O.V.A.

EF Measure	Method	Mean Difference	DF	t	p-value	95% Confidence Interval
K-SNAP	Pooled	5.7802	25	1.66	0.1103	(-1.4112, 12.9716)
	Satterthwaite	5.7802	21.29	1.63	0.1171	(-1.5724, 13.1328)
T.O.V.A.	Pooled	0.1638	25	0.15	0.8853	(-2.1512, 2.4788)
	Satterthwaite	0.1638	24.91	0.15	0.8847	(-2.1394, 2.4670)

Table 4.16

Test for Equality of Variances: Folded F Test

EF Score	Method	Num DF	Den DF	F value	P-Value
K-SNAP	Folded F	12	13	2.05	0.2128
TOVA	Folded F	13	12	1.32	0.6380

Table 4.17

Descriptive Statistics for ADHD Groups on T.O.V.A. and K-SNAP

EF Score	N	Mean	Standard Dev	Minimum	Maximum
K-SNAP	27	103.27	9.36	80	117.00
TOVA	27	1.28	2.86	-7.09	4.92

Table 4.18

Correlation Using Least Squares Regression: Relationship Between Executive Function and G.P.A.

ADHD TOVA and GPA	Coefficient	St. Error	t-value	r^2
Variable				
Intercept	-6.5132	2.6826	2.428	r = 0.2588
ADHDTo	2.644	0.895	2.9542	
ADHD K-SNAP and GPA				
Variable				
Intercept	2.2472	1.2092	1.8585	
ADHDK-Sn	0.0068	0.0117	0.582	r = 0.0134
Control TOVA and GPA				
Variable				
Intercept	2.9243	0.4443	6.5824	
ConTOVA	-0.0024	0.1313	0.0185	r = 0.0001
Control K-SNAP and GPA				
Variable				
Intercept	-0.1993	1.1946	0.1668	
ConK-Sn	0.0309	0.0118	2.6184	r = 0.6315

Note: ADHDTo = ADHD T.O.V.A. scores; ADHDK-Sn = ADHD K-SNAP scores, ConTOVA = Control T.O.V.A. scores, and ConK-Sn = Control K-SNAP scores.

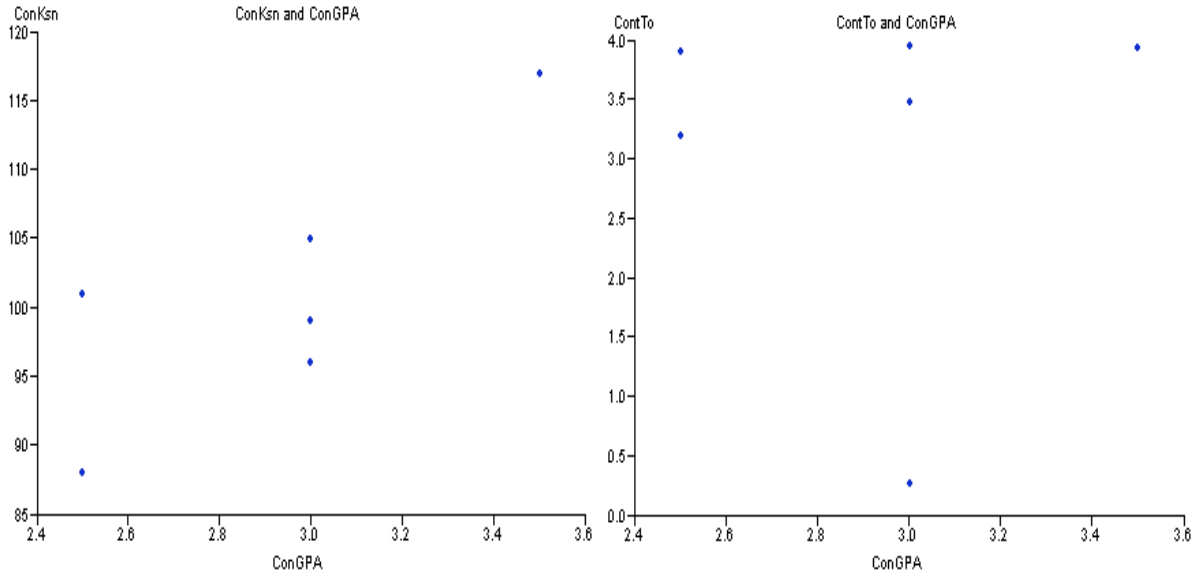

Figure 4.1 Relationships Between Control Subjects' Executive Functioning and G.P.A.

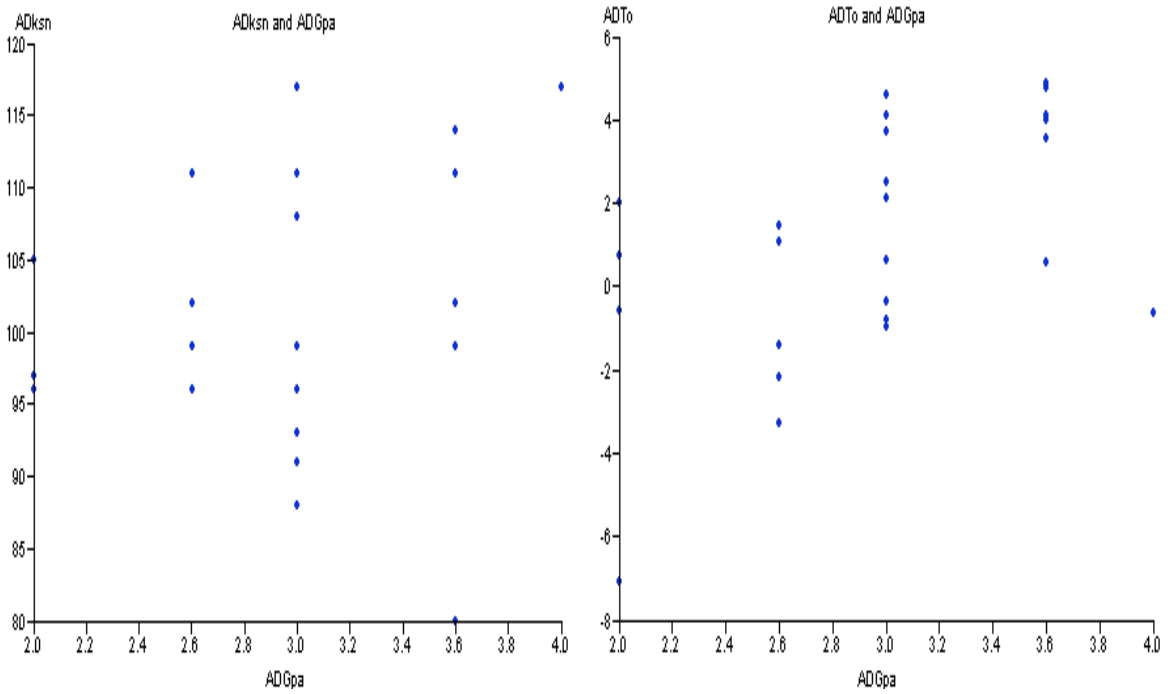

Figure 4.2 Relationships between ADHD Subjects' Executive Functioning and GPA

Research Question #2

In order to answer the research question, *Do ADHD students with higher EF scores have on average a higher GPA than those with lower EF scores*?, tests for independence, an exact test, and a trend test were conducted. Table 4.17 shows the descriptive statistics for T.O.V.A. and K-SNAP for ADHD students. ADHD students scored higher than the mean value of K-SNAP (or T.O.V.A.) are considered as having a higher EF score in terms of K-SNAP (or T.O.V.A.), and students scored below than the mean value of K-SNAP (or T.O.V.A.) are considered as having a lower EF score in terms of K-SNAP (or T.O.V.A.). To evaluate if GPA depends on EF scores, χ^2 test statistic for independence, Fisher's exact test, and Cochran-Armitage trend test (Cochran, 1954; Agresti, 2002; Armitage, 1955).

In general, if the sample size is large, χ^2 test statistic for test of independence is appropriate for when the sample size is small, alternative methods use exact small-sample distributions rather than large-sample approximations. Therefore, in addition to χ^2 test, Fisher's Exact Test (Agresti, 2002) is also used for testing independence (Agresti, 2002). The associated null and alternative hypotheses are: H_0 : GPA and variable X are independent, H_a : GPA and variable X are not independent, variable X is type of students (2 categories: high EF score and low EF score, EF score is measured in terms of K-SNAP or T.O.V.A.).

Table 4.18 is the general form of the two-way contingency table for variable X (high and low) and GPA using the *Cochran-Armitage Trend Test*. n_{0i} and n_{1i} are the counts of students with high EF score (K-SNAP or T.O.V.A.) and low EF score at each level of GPA, respectively. $i = 1, \ldots, 5$. n_{0+} is the total number of students with high EF score and n_{1+} is the total number of students with low EF score. n_{++} is the sum of n_{0+} and n_{1+}. n_{i+} is the number of ADHD students in the ith level of GPA. When a series of proportions occurring in groups which fall into some

natural order, the question often asked is whether the proportions show a significant trend, upwards or downwards, with the ordering of the groups (Armitage, 1955; Cochran, 1954). The null and alternative hypotheses are:

H_0 : there is no linear trend in proportions of types of students across the level of GPA,

H_a : there is a linear trend in proportions of types of students across the level of GPA.

GPA and K-SNAP Analysis

The underlying null and alternative hypotheses for testing if there is a relationship between GPA and types of students (high K-SNAP and low K-SNAP) are:

H_0 Level of GPA and types of students (high K-SNAP and low K-SNAP are independent.

H_1 : Level of GPA and types of students (high K-SNAP and low K-SNAP are not independent.

Tables 4.18 and 4.20 summarize the level of GPA for students with high K-SNAP and low K-SNAP. The χ^2 test statistic (DF = 4, N = 27) = 1.9232 with p-value = 0.7499 and the p-value for Fisher's exact test 0.9171 indicate that the level of GPA does not depend on types of students (high K-SNAP and low K-SNAP). The Cochran-Armitage trend test statistic $z = 0.1487$ (right-sided p-value = 0.4409) also supports this argument. In addition, least squares regression was used to calculate a correlation coefficient to analyze the relationship between GPA and K-SNAP scores. Table 4.18 indicates that the correlation between K-SNAP and G.P.A. for ADHD subjects was $r = 0.0134$, Hence, the conclusion was that, based on the data, ADHD students with higher EF scores (measured by K-SNAP) did not have on average a higher GPA than those with lower EF scores (measured by K-SNAP). On the other hand, control K-SNAP scores were correlated to G.P.A. There was a

correlation of r = 0.6315, which indicated that there was a strong positive relationship between K-SNAP scores among non-ADHD students and cumulative G.P.A. (see Table 4.18).

Table 4.19

GPA & T.O.V.A. Comparison

	G.P.A.				
	2.0-2.5	**2.6-2.9**	**3.0-3.5**	**3.6-3.9**	**4.0 +**
Score					
High K-SNAP	2 (50.00)	2 (40.00)	6 (54.55)	2 (33.33)	1 (100.00)
Low K-SNAP	2 (50.00)	3 (60.00)	3 (60.00)	4 (66.67)	0 (0)

G.P.A. and T.O.V.A. Analysis

The underlying null and alternative hypothesis for testing if there is a relationship between GPA and types of students (high T.O.V.A. and low T.O.V.A.) are:

$H_{0:}$ Level of GPA and types of students (high TOVA and low TOVA) are independent.

$H_{1:}$ Level of GPA and types of students (high TOVA and low TOVA) are not independent.

Tables 4.19 and 4.20 show the level of GPA for students with high T.O.V.A. and low T.O.V.A.. The χ^2 test statistic (DF = 4, N = 27) = 7.2578 with p-value = 0.1229 and the p-value for Fisher's exact test 0.1297 indicate that the level of GPA did not depend on types of students (high T.O.V.A. and low T.O.V.A.). Taking advantage of the ordinal classifications (level of GPA: 2.0-2.5, 2.6-2.9, 3.0-3.5, 3.6-3.9, 4.0+), the Cochran-Armitage trend test statistic z = 1.6761 (right-sided p-value = 0.0469) indicates that the probability of the row 1 level ("type of student" = "high T.O.V.A.") increases as level of GPA increases or, equivalently, that the probability of the row 2 level ("type of student" = "low T.O.V.A.") decreases as level of GPA increases. $p_{0|i}$ (the observed sample proportion of students with high T.O.V.A. given the ith level of GPA) and $p_{1\ i}$ (the observed sample

proportion of students with low T.O.V.A. given the ith level of GPA) summarized as the column percentages in Table 4.20 also show this decreasing trend. In addition to the trend test, least squares regression yielded a correlation coefficient of $r = 0.2588$. This denoted a weak positive relationship between the two variables. Therefore, based on the data, the Cochran-Armitage trend test results and least squares regression, ADHD students with higher EF scores (measured by T.O.V.A.) had on average a higher GPA than those with lower EF scores (measured by T.O.V.A.). In contrast, non-ADHD subject scores on the T.O.V.A. were not correlated with G.P.A. A correlation of $r = 0.0001$ suggested that there was no relationship between control T.O.V.A. scores and control cumulative G.P.A

Table 4.20

A 2-way frequency and contingency table for GPA and types of students

	G.P.A.				
	2.0-2.5	**2.6-2.9**	**3.0-3.5**	**3.6-3.9**	**4.0 +**
Score					
High T.O.V.A.	1 (25.00)	1 (20.00)	7 (63.64)	5 (83.33)	0 (0)
Low T.O.V.A.	3 (75.00)	4 (80.00)	4 (6.36)	1 (16.67)	1 (100.00)

Table 4.21

Fisher's Exact Test Comparing T.O.V.A., K-SNAP, & G.P.A

Fisher's Exact Test	**Test Statistic**	**P-Value**
TOVA	1.6761	0.0469
K-SNAP	1.9232	0.7499

Research Question #3

In order to determine if there is a relationship between medication status and G.P.A. the underlying null and alternative hypotheses for testing if there is a relationship between GPA and types of ADHD students (stimulant and non-stimulant) were:

$H_{0:}$ Level of GPA and types of ADHD students (stimulant and non-stimulant) are independent.

$H_{1:}$ Level of GPA and types of ADHD students (stimulant and non-stimulant) are not independent.

Tables 4.21 and 4.22 summarize the level of GPA for stimulant and non-stimulant medicated ADHD students. The χ^2 test statistic (DF = 4, N = 27) = 3.8592 with *p*-value = 0.4254 and the *p*-value for Fisher's exact test 0.4461 indicate that the level of GPA did not depend on types of ADHD students (stimulant and non-stimulant). The Cochran-Armitage trend test statistic $z = 0.2163$ (right-sided *p*-value = 0.4144) also support this argument. Hence, the conclusion is that, stimulant medicated ADHD students do not have a higher GPA than non-stimulant medicated ADHD students.

Table 4.22

GPA and Medication Status Frequency and Contingency Table for ADHD Students

GPA	**2.0-2.5**	**2.6-2.9**	**3.0-3.5**	**3.6-3.9**	**4.0+**
Non-Stimulant	3 (75.00)	1 (20.00)	6 (54.55)	3 (50.00)	1 (100.00)
Low TOVA	1 (25.00)	4 (80.00)	5 (45.45)	3 (50.00)	0 (0.00)

Table 4.23

Between Groups Comparison of G.P.A. using Fisher's Exact Test

Stimulant	**G.P.A.**			
2.0-2.5	2.6-2.9	3.0-3.5	3.6-3.9	4.0+
2	4	5	2	0
15%	31%	38%	15%	0%
Non-Stimulant	**G.P.A.**			
2.0-2.5	2.6-2.9	3.0-3.5	3.6-3.9	4.0+
3	1	6	3	1
21%	7%	43%	21%	7%
Df = 4	N = 27	Mean = 3.8592	p = 0.4254	

Summary

Statistical analysis indicated that there was not a significant difference between the neuropsychological functioning of stimulant-medicated and non-medicated ADHD students as assessed by the T.O.V.A. and K-SNAP. An observable difference in mean G.P.A. also was not evident. Though K-SNAP scores were not correlated with cumulative G.P.A., T.O.V.A. scores were. This indicated that students with higher T.O.V.A. A.P.I.'s tended to have higher G.P.A.'s than students with lower T.O.V.A. scores.

In light of these results, it can be concluded that stimulant-medicated and non-stimulant medicated college students with ADHD performed at similar levels on measures of executive and academic function. In depth conclusions focused on the sample characteristics, the measures administered, and the overall heterogeneity of ADHD as a disorder.

Chapter 5: Discussion

The area of adult ADHD is not as well researched as adolescent ADHD in terms of its persistence beyond the K-12 years (Doyle, 2006; Ossmann & Mulligan, 2003). With regard to the neuropsychological effects of the disorder, even less research among adults with the condition exists (Hervey, Epstein, & Curry, 2004). How medicated adults with ADHD function compared to non-medicated persons also remains in question. This study attempted to contribute to knowledge gaps within the following areas: 1) Adult ADHD as a disorder, 2) The neuropsycholgical correlates stimulant-medicated and non-stimulant medicated adults with ADHD, 3) The academic functioning of stimulant-medicated and non-stimulant medicated ADHD adults.

Hypotheses were formulated based on Barkley's (1997) *Inhibitory Hypothesis of ADHD* and the *Dopamine (DA) Hypothesis of ADHD* (Wender, 1971; Levy, 1990). It was hypothesized that stimulant-medicated adult college students with ADHD would outperform non-stimulant medicated students on neuropsycholgical and academic assessments. As a result, the following hypotheses were formulated in an attempt to answer the following research questions using the T.O.V.A. Test, K-SNAP, and student cumulative G.P.A.:

Research Question #1: Is there a significant difference between the executive functioning of stimulant-medicated ADHD college students compared to non-medicated ADHD college students as measured by neuropsychological assessments? *Hypothesis #1: Those taking stimulant medications will out- perform non-medicated controls on tests of executive function.* Research Question #2: Do students with higher EF scores have on average a higher G.P.A. than those with lower EF scores? Hypothesis #2: *G.P.A. is positively correlated with EF scores.* Research Question #3: Do stimulant medicated ADHD students have a higher G.P.A. than non-

medicated controls? Hypothesis #3: *It is hypothesized that students taking stimulant medication will have higher G.P.A.'s than non-medicated controls.*

Conclusions based on Data

The following section discusses the study's conclusions in the order research questions and subsequent hypotheses are presented. Discussion is based on the results of statistical tests and how these conclusions are consistent or inconsistent with current ADHD research in the area. Notable observations are also made as they pertain to the outcomes of the study. All observations took place during the data collection processes. Additional comments are made regarding the executive and academic functioning of participants involved in the study.

Research Question and Hypothesis #1.

In order to answer the question of whether there was a significant difference between stimulant-medicated and non-stimulant medicated adult college students with ADHD, it was hypothesized that stimulant-medicated students would outperform those who were not. As indicated by scores on the K-SNAP and T.O.V.A. that there was no difference between the groups. However, based strictly on mean T.O.V.A. and K-SNAP scores, non-stimulant medicated students had slightly higher scores on the assessments than stimulant-medicated groups. Though this was not a randomized, double-blind experiment where participants are assigned to a treatment group consisting of a medication regimen or placebo, these findings are inconsistent with previous research indicating that medicated subjects' neuropsychological functioning was better than non-medicated subjects (Wilson, Cox, Merkel, Moore, & Coghill, 2006). Multiple studies evaluating inhibitory response and attention of medicated and non-medicated participants concluded too that medicated individuals outperformed non-medication takers (Bedard, Ickowicz, Logan, Hogg-Johnson, Schachar, & Tannock, 2003; Biederman et al.,

2008, Boonstra et al., 2005; Vaida et al., 1998, Vaidya et al., 1998; Wilson, et al., 2006). However, results from this study are consistent with those of Rhodes, Coghill, & Matthews (2006) who failed to observe a significant difference on measures of inhibition (like the T.O.V.A. in this study) between medicated and non-medicated participants. Also like Biederman et al (2008) and Gathercole & Alloway's (2006) findings, limited effects of stimulants were observed on WM processes required for the number recall subtest of the K-SNAP in that stimulant medicated subjects did not outscore non-medicated participants on neuropsychological assessments. Rather, non-medicated subjects scored on average slightly *higher* on these measures than their medicated counterparts though not significantly different.

Research Question and Hypothesis #2.

It was originally hypothesized that EF is positively correlated with student G.P.A. Therefore, it was suspected that students with a higher G.P.A. would have on average higher scores on EF assessments. Interestingly, T.O.V.A. scores were correlated with student cumulative G.P.A. but K-SNAP scores were not. Students with higher T.O.V.A. scores had on average a higher G.P.A. than students with lower T.O.V.A. scores. However, a relationship could not be established between G.P.A. and the K-SNAP composite scores. Though (Biederman et al., 2004; DuPaul et al., 2009; Proctor & Prevatt, 2009) found that children and young adults with the disorder have lower grades, more academic struggles in general, and were more likely to be on academic probation than non-ADHD youths, the same conclusions could not be made in this study. Participants in both groups had a relatively high cumulative G.P.A. and as a result, a significant difference between stimulant medicated and non-medicated participants was not observed.

Research Question and Hypothesis #3.

Originally it was hypothesized that stimulant-medicated students would have higher cumulative G.P.A.'s than those who were non-stimulant medicated. Based on statistical analysis of the data, this hypothesis could not be supported. There appeared to be no significant difference between the groups. These conclusions are in contrast to research in this area by Meaux et al., 2006 & Powers, Marks, Miller, Newcorn, & Halperin (2008) who observed stimulant-medicated students as slightly outperforming non-medicated adolescents with ADHD. However, T.O.V.A. performance was correlated with student G.P.A. Students with higher T.O.V.A. scores tended to have a higher G.P.A. as well. This is possibility suggestive that the attention skills required to do well on the T.O.V.A. test would also serve one well while sitting in class, taking exams, and studying.

Notable Observations

Overview

Initially participants were to be drawn from the urban university only, but the failure to recruit a large number of subjects forced the primary investigator to seek approval to recruit ADHD subjects from another university as well. It was a challenge to recruit male participants early in the study. The majority of respondents were females, resulting in some being turned away in order to maintain a balanced sample. Additionally participants were only to be admitted to the study if they possessed a childhood diagnosis of ADHD. This had to be revised to include adult-diagnosed participants with the development of the DSM-V.

An interesting finding was that a significant number of participants were STEM or business majors. This was an interesting finding in that many would consider STEM to be a major less suited to a student with ADHD. Few participants were majoring in the arts,

education, or liberal studies at the time data were collected. It is important to note that the two universities participants were attending were considered to be renowned Engineering schools.

Another notable finding was that most of the participants had some experience with stimulant-medication use. A significant number of students within the non-stimulant medicated group admitted to taking medication on an as-needed basis. Though these participants were not taking medication regularly (daily) nor were they medicated the day of testing, students did take medication during academically critical times. Few participants avoided medication all together.

Executive Function Discussion.

Though significant differences were not observed between the groups on executive and academic performance assessments, average scores on the neuropsychological measures survey results indicated that stimulant medicated students reported having more moderate to severe symptoms than non-medicated subjects. Many of these participants had been taking medication since elementary school. Because of the heterogeneity of ADHD, those taking medication may have been suffering from more severe form of the disorder. Non-medicated participants tended to have an adult diagnosis, indicating that an early need for identification and support service delivery in K-12 setting may have been unnecessary.

Because this study fails to provide evidence that there is a significant difference between the EF of medicated and non-medicated students with ADHD, ADHD as a disorder of executive dysfunction is called into question. However, if ADHD does contribute to EF deficits, typical measures designed to capture EF may be inadequate. Not one subject (even those who had suffered a TBI during childhood) exhibited a neuropsychological impairment according to the K-SNAP. It may be that ADHD does contribute to EF deficits but deficits may not be significant enough to be detected by certain neuropsychological measures. Though ADHD is currently

diagnosed by behaviors using patient history, rating scales, etc., a battery of neuropsychological assessments could be needed to detect executive dysfunction associated with ADHD if these measures are to be used for diagnostic purposes.

Academic Function Discussion.

Failure to observe a difference between stimulant-medicated and non-medicated subjects should also be discussed in conjunction with the rather high G.P.A. of *all* participants. The two universities from which subjects were recruited are highly sought-after, selective 4-year schools. In order to receive an offer of admission and maintain status as a student, grades must remain high. It is possible that these students have developed compensation strategies that other students with ADHD have not, thus allowing them to do very well academically.

In addition to having a high G.P.A., survey results indicated that a significant number of students utilized academic support services at school. Students indicated that accommodations such as extended time and testing in an alternative setting provided them with additional support. Such supports can be especially helpful for testing. The study was conducted during mid-terms at University B and many participants stated that they were using accommodations to assist them with test-taking that week. Because of the strong emphasis on exam grades at the undergraduate level, the use of student supports may have contributed to a higher G.P.A. for these participants.

Implications for Practice

From an educational psychology perspective, this study contributes to a knowledge gap in the area of EF and academic functioning of adults with ADHD who use and choose not to use medication for their condition. Therefore, adult students with the condition have additional information to consider when weighing the options of medication. Though this was a quantitative study, casual conversation with participants suggested that students either could not

imagine their academic life without medication or could not imagine life with medication *on a regular basis*. A regular basis is emphasized because many participants stated that they did use medication occasionally for test taking, studying, or whenever they felt they *really* needed to focus. However, side effects according to some subjects made it too difficult to take daily. These individuals stated that if they took their medication, they would be up all night, unable to sleep, or became nauseous. Others claimed that medication was a part of their normal routine and taking a pill daily for ADHD was not an issue. It may be that medication provides more benefits to some and fewer to others and is not a "cure-all" for the condition as a whole.

This study's primary implications for educators and psychologists centers on the academic performance of these students. Because participants maintained a high G.P.A. in spite of possessing ADHD it is necessary to emphasize that medication is only part of the ADHD puzzle. School officials should approach the subject of medication with students carefully. Based on student reports, the severity of ADHD differed from person to person. This must be factored into decisions regarding whether or not to take stimulant medication for ADHD. It is important to emphasize that it is possible to be academically successfully on and off stimulant medication if one possesses ADHD. It is not, however a panacea for the disorder.

Directions for Further Research

There is much room for additional research in the area of adult EF and academic function among medicated and non-medicated ADHD adults. In order to examine the role of compensation strategies on academic performance, a qualitative component to a similar study is recommended. This would provide additional insight as to which strategies have helped students with such high G.P.A.'s succeed at the college level. Secondly, there was an initial concern that the sample would be overwhelmingly male. Since this was not the case and the female response

with overwhelming, an additional study of women at this level with ADHD would be interesting. Because females often fail to manifest the hyperactivity component as a child, it may be that many women are not receiving an ADHD diagnosis until later in life. In addition to studying females, replication of the study at the junior college level would also be recommended. This allows for a more academically diverse population to be evaluated, as strict admissions requirements would not be in place. Lastly, because medication status was not altered by the experimenter in this study, it is recommended that the study be conducted using a double-blind, randomized design where subjects are assigned to a treatment group. This would control for variation between participants in terms of medication type, dosage, and regimen.

Limitations

The most significant limitation of this study is the failure to control medication status. Participants were taking a variety of stimulant medications, at different times, and in various dosages. With a double-blind, randomized design stronger conclusions regarding the influence of medication on EF and academics could be made. Another limitation at the time of data collection had to do with the time at which the T.O.V.A. test was given to some of the participants. The T.O.V.A. should be given in the mornings; however, student class schedules and mid-term exams prevented many from taking the T.O.V.A. before 1:00 pm. As suggested by the T.O.V.A. manual, this was adjusted for by administering the test during the same time frame for those unable to take the exam in the morning.

An additional limitation had to do with the battery of EF assessments given to participants. In order to capture complex constructs associated with EF, a larger battery of assessments would be ideal. Such assessments should also be given in conjunction with an I.Q. test to provide an additional baseline for intelligence. Due to restrictions on time and funding,

this study utilized only two EF measures. Greater differences in EF may have been observed using a variety of neuropsychological assessments.

Lastly, the sample size in this study was small. Having a larger *n* would have added depth to the study. This is true for both ADHD and non-ADHD controls. Because this study was not funded by a grant or sponsor, participant compensation, assessment purchases, and all other expenses associated with the study was funded by the primary investigator. This limited the number of subjects accepted to participate in the study though many more were desired.

The primary delimitation for this study was the disproportionate number of control participants from University B compared to University A. Because controls were volunteers, there was more interest in participating for the sake of contributing to research rather than for compensation alone among students at University B. Potential control subjects at University A lost interest in participating unless they could be paid.

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Appendix A

ADHD Indicator Survey/Questionnaire

1. Please create a personal identification number for yourself, which includes TWO letters and TWO numbers. EX: NN51
2. Gender: Male Female Transgender
3. Age: _____
4. Ethnicity:
   a. African American c. Caucasian e. Persian
   b. Asian d. Latino/a f. Other:_____
5. Year in College:
   a. Freshman c. Junior e. Graduate
   b. Sophomore d. Senior
6. What is your current cumulative G.P.A. ? If you are a first-time freshman or transfer student, please circle your cumulative G.P.A. exiting high school or junior college.
   a. 2.0-2.5 c. 3.0-3.5
   b. 2.6-2.9 d. 3.6-3.9 e. 4.0 or above
7. Do you use student support services at your school to provide you with accommodations in your classes?
   a. Yes b. No

8. When were you diagnosed with ADHD?
   a. Early Elementary School (Ages 5-8)
   b. Late Elementary School (Ages 9-11)
   c. Middle School (Ages 12-13)
   d. High School (Ages 14-16)
   e. *As and adult (> 17 years of age)
      i. (*If you circle "e", please stop here and see primary investigator).
9. What ADHD sub-type were you diagnosed as having?
   a. Hyperactive
   b. Impulsive
   c. Combined (Hyperactive/Impulsive)
   d. I do not know

10. How did you receive a formal diagnosis of ADHD?
    a. From a general practitioner or pediatrician.
    b. Clinical Psychologist or Psychiatrist
    c. Other:_____

11. In addition to ADHD have you been formally diagnosed with a learning disability or ever suffered from a traumatic brain injury?
    a. YES b. NO

12. Do you take stimulant medication as part of a treatment regimen for ADHD?
    a. YES b. NO (If no, skip #9 & #10)

13. If yes, do you take this medication on a regular basis (as prescribed by your physician, psychiatrist, etc.?

a. Yes, I take my medication regularly.
b. No, I do not take medication regularly.

14. How long have you been taking stimulant medication on a regular basis to treat your ADHD?
    a. 5 months or less
    b. 6 months – 1 year
    c. 1 – 2 years
    d. 3-5 years
    e. 5 + years
    f. I do not take medication for my ADHD

15. Prior to being medicated for ADHD, how would you describe your symptoms:
    a. My symptoms were severe
    b. My symptoms were moderate
    c. My symptoms were mild
    d. I do not remember

Appendix B

IRB INFORMATION SHEET

Dear potential participant,

My name is Nicole Nicholson, and I am a doctoral candidate in the Rossier School of Education at University of Southern California. I am conducting a research study as part of my dissertation, focusing on the executive and academic functioning of stimulant-medicated and non-stimulant medicated ADHD college students. Academic functioning will consist of you recording your cumulative G.P.A. in a survey. Thus, If you are an adult 18 years or older and have been diagnosed with ADHD, you are eligible to participate in a research study.

The study procedures include completing a questionnaire and two short assessments designed to measure attention, memory, and critical thinking skills. The assessments are anticipated to take no more than 30 minutes each to complete. The study procedures are not clinical or intended as clinical diagnosis. Your participation is anticipated to last no more than 1.5 hours.

Participation in this study is voluntary. Your identity as a participant will remain confidential at all times during and after the study. Your relationship with USC or your doctor(s) will not be affected whether or not you participate in this study. You will be compensated for your time.

If you have questions or would like to participate, please contact me at 805-235-5677 or by email at nlnichol@usc.edu

Thank you.

Nicole Lee Nicholson

University of Southern California

Study ID: UP-11-00419 Valid From: 11/13/2011

Appendix C

INFORMED CONSENT TO PARTICIPATE IN:

The Executive and Academic Functioning of Stimulant Medicated and Non-Stimulant Medicated ADHD College Students.

A research project on Attention Deficit Hyperactivity Disorder is being conducted by Nicole Nicholson in the Department of Education at the University of Southern California, Los Angeles. The purpose of the study to compare the results of academic and executive functioning measures between students who claim to be medicated for ADHD, non-medicated with ADHD, and controls (non-ADHD) participants. Medication status is NOT being altered in any way during this study or by the primary investigator.

If you agree to participate, you will be asked to do the following:

1. Complete a survey (ADHD Indicator Survey) requesting your age, ethnicity, year in school, cumulative G.P.A., gender, age of ADHD diagnosis, ADHD subtype (ex: Hyperactive, impulsive, or combined types), type of diagnosis (ex: Diagnosed by physician, psychiatrist, psychologist, or other mental health professional), and stimulant medication status.

2. Complete the Kauffman Short Neuropsychological Assessment Profile (K-SNAP)

3. Take the computer-based Test of Variable Attention (TOVA).

The K-SNAP is a brief pencil-paper test that assesses aspects of the mental functioning of adolescents and adults, ages 11 to over 85 years. Included are four subtests, organized in three levels of cognitive complexity:

- Simple memory and perceptual skills (Number Recall and Gestalt Closure or ink blot test)
- Complex intellectual functioning and planning ability (Four-Letter Words)

The TOVA is a computer based test that measures response time, impulsivity, and inattention.

The tests are not diagnostic. The data are collected for the purposes of this research study only. Test results will not be given to you, nor will a diagnosis be made by the researcher.

You do not have to participate in this study if you don't want to. You may discontinue your participation at any time without penalty. You may also choose not to answer any questions that you would prefer not to answer. Your participation will take approximately 1 hour during a time that is convenient for you.

The possible risks associated with participation in this study include minor fatigue due to sitting for a period of about 1 hour. If you should experience any discomfort whatsoever, please

be aware that you may inform the primary researcher, Nicole Nicholson and you will be provided with assistance immediately.

Your confidentiality will be protected by the researcher. You will create a Personal Identification Number which will be used to identify your data. Your name will NOT be attached to any documents, and it will not be used in any reports of this research.

Potential benefits associated with the study include a better understanding of adult ADHD, how college students with ADHD perform on academic and executive functioning measures, and contributions to reduce the current knowledge gap within ADHD research.

If you have questions regarding this study or would like to be informed of the results when the study is completed, please feel free to contact:

Principal Investigator: Nicole Nicholson
Phone: 805-235-5677
Email: nlnichol@usc.edu
Address: Waite Phillips Hall 3470 Trousdale Parkway Los Angeles, CA 90089

Faculty Advisor: Dr. Patricia Tobey
Phone: (213) 740-0776
Email: tobey@usc.edu
Address: Waite Phillips Hall 3470 Trousdale Parkway Los Angeles, CA 90089

If you have concerns regarding the manner in which the study is conducted, you may contact Dr. Steve Davis, Chair of the Cal Poly Human Subjects Committee, at (805) 756-2754, sdavis@calpoly.edu, or Dr. Susan Opava, Dean of Research and Graduate Programs, at (805) 756-1508, sopava@calpoly.edu. You may also contact the University of Southern California's Institutional Review Board: University Park IRB, Office of the Vice Provost for Research Advancement, Stonier Hall, Room 224a, Los Angeles, CA 90089-1146, (213) 821-5272 or upirb@usc.edu.

If you agree to voluntarily participate in this research project as described, please indicate your agreement by signing below. Please keep one copy of this form for your reference, and thank you for your participation in this research.

_____ _____
Signature of Volunteer Date

_____ _____
Signature of Researcher Date