TESTING EXECUTIVE FUNCTION MODELS OF ADHD AND ITS COMORBID CONDITIONS: A LATENT VARIABLE APPROACH

A Dissertation

by

DONG HYUNG LEE

Submitted to the Office of Graduate Studies of
Texas A&M University
in partial fulfillment of the requirements for the degree of

DOCTOR OF PHILOSOPHY

August 2004

Major Subject: School Psychology

TESTING EXECUTIVE FUNCTION MODELS OF ADHD AND ITS COMORBID CONDITIONS: A LATENT VARIABLE APPROACH

A Dissertation

by

DONG HYUNG LEE

Submitted to Texas A&M University
in partial fulfillment of the requirements
for the degree of

DOCTOR OF PHILOSOPHY

Approved as to style and content by:

Cynthia A. Riccio (Chair of Committee)	Victor L. Willson (Member)
Michael J. Ash (Member)	William A. Rae (Member)
Emily S. Davidson (Member)	Victor L. Willson (Head of Department)

August 2004

Major Subject: School Psychology

ABSTRACT

Testing Executive Function Models of ADHD and Its Comorbid Conditions:

A Latent Variable Approach. (August 2004)

Dong Hyung Lee,

B.A.; M.A., Chungnam National University, South Korea

Chair of Advisory Committee: Dr. Cynthia A. Riccio

Current theoretical models of ADHD (i.e., Disinhibition Model: Barkley, 1997; Working Memory Model: Rapport et al., 2001) conceptualize ADHD as the disorder of executive function (EF) with some variation in their emphases on particular components of the broadly-defined EF (e.g., working memory vs. inhibition) and in their postulated relationships with ADHD symptoms. Although these models provide systematic accounts of the manifestation of ADHD, they have not been extensively tested from an empirical standpoint. Moreover, despite the fact that ADHD is highly comorbid with other additional conditions such as learning and behavioral problems and EF deficits are found in individuals with these conditions as well as in those with ADHD, current EF models have not specified the developmental relationship between ADHD and its comorbid conditions. This study was: (1) to examine the extent to which two current models of ADHD are supported in a sample of 102 adults; (2) to present an "integrated" model by combining two current models of ADHD and linking them to recent research findings on two common comorbid conditions with ADHD (i.e., reading difficulty and substance abuse); and (3) to test and revise such an integrated model in the light of data using a latent variable analysis. Major findings provided a strong support for the Working Memory Model with a lesser

degree of support for the Disinhibition Model. Preliminary evidence of working memory as the primary deficit in ADHD was also obtained in the present sample. Finally, the integrated EF model and its revised model (final model) demonstrated a very good fit to the data. These findings suggest that the integrated model provides a unified account of how EF deficits contribute to the manifestation of ADHD symptoms and comorbid conditions with ADHD. Given some limitations (e.g., sample size and scope) of the present study, current findings need to be replicated.

DEDICATION

To my parents, Sangsoon & Kiboon Lee

ACKNOWLEDGMENTS

This work would have been impossible without the help of many people. First and foremost, I give my sincere thanks to Dr. Cynthia Riccio, chair of my committee, for her guidance, support, and encouragement throughout this research project and my doctoral training. She introduced me to an exciting field of study of executive function and ADHD early in my doctoral training and has since offered numerous opportunities to expand my ideas with the best resources and information available. I appreciate her invaluable impact on my professional growth over the years.

I extend my thanks to my committee members: Dr. Victor Willson, Dr. Mike Ash, Dr. William Rae, and Dr. Emily Davidson. Their expertise, comments and suggestions have always been helpful and made me a better thinker and researcher. I am also grateful to other faculty members in the School Psychology Program at Texas A&M University – Dr. Cecil Reynolds, Dr. Hector Ochoa, Dr. Douglas Palmer, and Dr. Jan Hughes - for the excellent research and clinical training I have received.

My special thanks go to the Memory, Attention, and Planning Study (MAPS) team members who were involved in data collection. As a team member, I found collecting individual evaluation data extremely time-consuming and laborsome; however, our teamwork, under the leadership of Dr. Cynthia Riccio, has turned out to be an extremely positive experience. I also extend my appreciation to all research participants who received evaluation through the MAPS team.

I wish to thank SEM netizens who offered invaluable help through SEMNET listserv (semnet@bama.ua.edu). They graciously helped me out with useful suggestions,

comments, and tips when I was confronted with technical problems running statistical analyses with this research project.

I am greatly indebted to my parents and siblings for their love and support throughout my life. Their unfailing love and sacrifice mean more than I can put into words. I thank my wife, Kyunghee, for her love and prayer; she believed in me with great patience and endurance even when I was in doubt with myself. I also thank my two children, Jedidiah and Joyanne, for continuing to be my joy in life. Finally, I thank the Lord for always being my rock; His faithfulness has taught me to acknowledge Him in all my ways.

TABLE OF CONTENTS

LIST OF FIGURES

LIST OF TABLES

CHAPTER I

INTRODUCTION

Attention Deficit Hyperactivity Disorder (ADHD) is a psychiatric condition characterized by three major symptoms of inattention, hyperactivity, and impulsivity. ADHD is usually first diagnosed during elementary school years when school adjustment is compromised (American Psychiatric Association, 1994). ADHD originates in childhood, but with symptoms frequently continuing into adult life, causing distress and psychiatric comorbidity (Marks, Newcorn, & Halperin, 2001).

With an estimated prevalence of 3-5% in school-age children (American Psychiatric Association, 1994), ADHD is one of the most frequently diagnosed disorders in pediatric populations. The results of research to date suggest that problems with ADHD symptoms specifically continue for 10 to 60% of these children, depending on the study and the methods used to determine the presence of the diagnosis, as they achieve adulthood (Mannuzza, Klein, Bessler, Malloy, & LaPadula, 1993; Weiss & Hechtman, 1993). Also, the diagnosis of ADHD conveys a significant risk for other coexisting psychiatric disorders. Up to 44% of children with ADHD may have at least one other psychiatric disorder, 32% have two others, and 11% have at least three other disorders (Szatmari, Offord, & Boyle, 1989). Unsurprisingly, individuals who continue to have significant symptoms in adulthood are at greater risk for academic, vocational, and social impairments, and other clinical conditions such as substance abuse, anxiety, and mood disorders.

This dissertation follows the style and format of *Journal of Abnormal Psychology*.

In spite of its high prevalence, high comorbidity, and relatively chronic nature, ADHD has been one of the most misunderstood disorders (Bender, 1997) and probably one of the most controversial child medical diagnoses (Wolraich, 1999). Historically, this confusion and disarray about ADHD is partly due to the lack of comprehensive psychological theories on the nature of this disorder.

A more systematic account for the developmental manifestation of ADHD symptoms and the nature of functional deficits of this disorder was provided recently by Russell Barkley (1997). He made a monumental attempt to provide a comprehensive model of this disorder. Barkley (1997) constructed his model (called the Disinhibition Model) based on most seminal research on ADHD of the last half century. He dealt with the nature and the development of self-regulation as relevant to ADHD and the manifestation of myriad cognitive deficits associated with this disorder. The key concept in his model is "behavioral inhibition". Within the broader context of neuropsychological theories and research, inhibition is a concept that is often considered as one of the major functions of the frontal lobe, often called executive function. Although executive function deficits often had been postulated in individuals with ADHD, Barkley was the first to systematically incorporate this concept into the understanding ADHD.

According to the Disinhibition Model (Barkley, 1997), the core deficit in ADHD lies in behavioral inhibition. The deficit in behavior inhibition (e.g., disinhibition) has subsequent detrimental effects on the development of four other executive functions that are critical for self-regulation and self-control. These executive functions include (1) nonverbal working memory, (2) self regulation of affect, arousal and motivation, (3) internalization of speech (verbal working memory), and (4) reconstitution of behavior. Since

the advancement of the Barkley's theory in 1997, it has been considered as the major theory in the field.

Another theoretical model comparable to Barkley's was proposed by Mark Rapport and his colleagues (Denny & Rapport, 2000; Rapport, Chung, Shore, & Issacs, 2001). This model is relatively new, less known, and less comprehensive than Barkley's model. As in Disinhibition Model, the importance of executive function is clearly implicated in the nature of ADHD. However, the relative importance of different executive functions and their roles in the development and manifestation of ADHD are hypothesized in different ways. This model (called the "Working Memory Model") posits that deficits in working memory are more critical in ADHD than any other cognitive deficits. Numerous behavioral and cognitive characteristics associated with ADHD are actually peripheral variables that are partially dependent on the core deficit in working memory processes. Because the working memory representation fades so quickly in individuals with ADHD due to their working memory deficits, their attention is often easily distracted and redirected to other stimuli in their environments; this distraction is observed by others as hyperactivity and impulsivity. While disinhibition is a characteristic of individuals with ADHD, it is viewed as a byproduct of a more critical, underlying deficit in working memory (Rapport et al., 2001).

Statement of the Problem

There have been an increasing number of studies that investigated various hypotheses proposed by the Disinhibition Model for the last several years. Most of these studies tested a few particular predictions made by this model and reported both positive (e.g., Dietlein, 2001; Houghton et al., 1999; Stevens, Quittner, Zuckerman, & Moore, 2002) and negative (e.g., Hanford, 2001; Pluth, 2001; Sarkari, 2003) findings. However, because of

the model's complexity and comprehensiveness, the current empirical status of this model is still to be established. Moreover, while the Disinibition Model suggests various causal paths, no studies have investigated the validity of these paths in the model.

There have been fewer studies about working memory deficits in ADHD and those studies have provided mixed results depending on the presence of other variables such as different types of tasks and comorbid conditions (e.g., Dietlein, 2001; Lee, Riccio, & Hynd, in press; Stevens et al., 2002). Further, specific hypotheses premised by the Working Memory Model rarely have been tested, and causal paths indicated in the model have not been tested. This is not surprising considering that this model is relatively very new in the field.

Apparently, there are conflictive predictions made by current executive function models of ADHD. However, these two models have not been compared with each other on an empirical basis. For example, although two models have a different theoretical position with regard to the primary deficit in ADHD (i.e., inhibition versus working memory), very few studies (e.g., Lee et al., in press) have addressed this issue.

It should be noted that, despite considerable disagreement between two models, they are complimentary in addressing particular relationships between some components in their models. Moreover, neither of these two models specifies how executive functions and ADHD symptoms are related to comorbid disorders with ADHD. Considering these observations, it would be very important to evaluate the extent to which these models are supported and make an attempt to improve current models by incorporating comorbid conditions with ADHD; however, this attempt has not been made previously. This study can be considered one of the first such endeavors.

Purpose of the Study

This study has two purposes. The first purpose is to test two contemporary executive function models of ADHD with adults using structural equation modeling (SEM). Given that these two models posit particular causal relationships among model components, a causal modeling technique is well suited to directly test these models. Moreover, the latent variable approach incorporated in the SEM methodology can be a very effective way to partially resolve task impurity and reliability problems that have plagued the measurement of executive functions (see Chapters II and III for more discussion). It should be noted that neither of these models has been tested using this methodology, although there are some published research and dissertation studies that tested a few particular hypotheses generated by these two models. In the process of testing models, an attempt also will be made to determine which of the two executive functions (i.e., working memory and inhibition) can be considered as the primary deficit in ADHD.

The second purpose of this study is to present and test an integrated model that incorporates these two models into one and extends predictions to two common comorbid conditions with ADHD. The integrative model is to be developed from existing executive function models of ADHD, but new predictions proposed by the model will reflect recent empirical findings regarding comorbidity, ADHD subtypes, and executive function profiles in ADHD. Again, a latent variable analysis using SEM will be used to determine the extent to which the integrated executive function model of ADHD and its comorbid conditions is valid. The final model that best fits to the data will be presented.

Significance of the Study

It is often necessary to develop a good theory that is rooted in empirical data to understand the nature of a clinical disorder and to draw meaningful implications from the theory for the most effective intervention; ADHD is not an exception. Especially, given that ADHD is often misunderstood and considered as one of the most controversial medical diagnoses, developing an empirically-supported theoretical model appears to have the utmost importance in the advancement of the field. This study will not only evaluate the validity of currently available models of ADHD but also attempt to improve current models by addressing comorbid conditions with ADHD. Moreover, given that current models have been developed primarily to be applied to children, this study also will be one of the first attempts to investigate the extent to which these models are supported in an adult sample. The final model is expected to provide a systematic account of how executive functions are linked ADHD and its comorbid conditions.

Definition of Terms

The brief definitions of major terms used in this study – ADHD, executive function, inhibition, and working memory - are offered (see Chapter II for more complete descriptions). ADHD is a persistent pattern of inattention and/or hyperactivity-impulsivity that is more frequent and severe than is typically observed in individuals at a comparable level of development (American Psychiatric Association, 1994).

This study adopts the definition of executive function (EF) provided by Welsh and Pennington (1988). EF is defined as "the ability to adopt and maintain an appropriate problem-solving set for attainment of a future goal (p. 101)." As a very broad term, EF is comprised of the three major components or dimensions – (a) an intention to inhibit a

response or to defer it to a later more appropriate time; (b) a strategic plan of action sequences; and (c) a mental representation of the task including the relevant stimulus information encoded into memory and the desired future goal-state. As the two major components of executive function, working memory is defined as the maintenance of transient information over brief temporal intervals to direct future-oriented activity, and inhibition is the ability to engage in the appropriate response instead of the more likely, albeit maladaptive, response (Roberts, Hager, & Heron, 1994).

Summary

The conceptualization of this study and its potential contribution to the field has been articulated in this chapter. Chapter II provides a more comprehensive review of the literature and introduces the integrated model. The methodology to be used is presented in Chapter III. Results and discussions are provided in Chapter IV and V.

CHAPTER II

LITERATURE REVIEW

Executive function (EF) is considered by many scientists as one of the crowning achievements of human development (Eslinger, 1996), the hallmark of intelligence (Belmont, 1978), and the most appealing, yet least understood, aspect of cognition (Weinert & Kluwe, 1987). EF underlies many seemingly unique realms of adaptive human behavior, including wonders of being able to think about ourselves, our social relationships, and what the future may bring. It allows us to be guided by our personal goals and to act despite long delays in rewards (Eslinger, 1996). As Borkowski and Burke (1996) argued, this marvelous function appears to have the potential for explaining the maintenance and generalization of behaviors across time and settings.

From a developmental perspective, Denckla (1996) succinctly argued that the difference between a child and an adult resides in the unfolding of executive function. Increasing independence, maturation of self-regulation, and development of self-generated productivity from childhood to adulthood can be viewed as the unfolding of executive function. From a clinical standpoint, the understanding of executive function also is expected to have crucial implications since executive dysfunction have been found in a broad range of neuropsychiatric and developmental disorders (for review, see Ozonoff, 1997; Pennington, 1997; Pennington & Ozonoff, 1996; Welsh, 2002).

Conceptualization of EF

There has been significantly increased interest in EF over the past 10-15 years in research in clinical neuropsychology (Lyon & Krasnegor, 1996) and the importance of

executive function for human development and adaptation has been more recognized. However, there is incredibly little understanding of what this function is and what fosters its development (Eslinger,1996). The conceptualization of executive function has been confounded significantly by variations in definition. EF has been investigated from different theoretical point of views, leading to a wide variety of definitions. Lyon and Krasnegor (1996) identified three major paradigms with which executive functions have been investigated; neuropsychological, information-processing, and behavior analytic perspectives.

The neuropsychological paradigm is anchored in the following three contexts or models to conceptualize the EF: (1) the historic linkage of executive function to the prefrontal cortex and its interconnected subcortical regions; (2) the diverse clinical presentations of executive dysfunction in child and adult neurological disorders; and (3) the critical role of executive processes and its associated neural circuitry in psychological development from childhood to adulthood (Denckla, 1996). Some researchers have defined the concept of executive function simply as the actions of the frontal lobes. But, importantly, this is neither wholly correct nor helpful. Although it is undeniably a historical fact that EF has a more concrete neuroanatomical context than a purely theoretical one, there are functions of the prefrontal lobes that extend well beyond the list of cognitive capabilities for which EF is an umbrella (Denckla, 1996) and the neural substrate of EF includes but is not limited to the functions of the prefrontal cortex. Therefore, the neural substrate of executive function is more accurately conceptualized as a neural network that involves synchronized activation of multiple cerebral regions.

Researchers in the information-processing paradigm rely on cognitive psychological theories to help define executive function. For example, Torgesen (1994) considered

executive function as equivalent to metacognition, more specifically "metacognitive behavior", that includes monitoring, planning, organizing, coordinating, and adapting knowledge and strategic resources as well as self-regulation. Butterfield and Albertson (1995) have defined it further as the "coordinator" of cognitive and metacognitive processes that acts through monitoring and controlling the use of knowledge and strategies in accordance with metacognitive processes.

The behavior analytical approach is functionally oriented and is interested in the vital connection between verbal abilities and behavioral regulation within specific contexts (Hayes, Gifford, & Ruckstuhl, 1996). For example, Hayes et al. (1996) conceptualized executive function as a subset of rule-governed behavior, noting that the key process underlying many executive function tasks is the flexibility and effectiveness of verbal self-regulation.

It also should be noted that the term of EF often has been confused with other cognitive processes such as attention and memory and used interchangeably with other similar concepts such as self-regulation or other mental control process (Eslinger, 1996). Moreover, there has been considerable disagreement among researchers on what components or parameters should be included in the EF. For example, Lezak (1995) conceptualized the EF into four distinct components including volition, planning, purposive action, and effective performance. More comprehensively, Barkley (2000) provided a long list of components of EF: (1) volition, planning, and purposive, goal-directed, or intentional action; (2) inhibition and resistance to distraction; (3) problem-solving and strategy development, selection, and monitoring; (4) flexible shifting of actions

to meet the task demands; (5) maintenance of persistence toward attaining a goal; and (6) self-awareness across time.

Given this significant variability in theoretical orientations and conceptualization, the next steps required for conceptual advancement would be to identify a commonality among various working definitions of EF. Currently, there seems to be a general agreement among researchers that EF is composed of multiple components (i.e., a cluster of skills) and all executive processes are goal-directed and future-oriented. Among many definitions available, Welsh and Pennington (1988) provided a relatively concise working definition of EF: "the ability adopt and maintain an appropriate problem-solving set for attainment of a future goal" (p 101). Welsh and Pennington (1988) also identified three major mechanisms underlying many executive functions: (a) an intention to inhibit a response or to defer it to a later more appropriate time; (b) a strategic plan of action sequences; and (c) a mental representation of the task including the relevant stimulus information encoded into memory and the desired future goal-state.

Welsh and Pennington (1988)'s definition nicely fits with a model of prefrontal function proposed by Fuster (1989). He suggested that the three functions were mediated by different loci within the prefrontal cortex: a temporally retrospective function of working memory, a temporally prospective function of anticipatory set, and an interference-control mechanism that suppresses behavior incompatible with the current goal. In the same vein with this view, more recent theoretical and computational accounts of EF have further proposed that particularly two cognitive processes, working memory and inhibition, may be sufficient to characterize the entire domain of EF (e.g., Cohen & Servan-Schreiber, 1992; Dehaene & Changeux, 1991; Diamond, 1991; Kimberg & Farah, 1993; Pennington, 1994;

Pennington, Benneto, McAleer, & Roberts, 1996; Roberts, Hager, & Heron, 1994; Roberts & Pennington, 1996). For example, Roberts and colleagues (Roberts et al., 1994; Roberts & Pennington, 1996) have argued that many of commonly used EF tasks appear to require both working memory and inhibition, and these two components parsimoniously represent executive processes mediated by the frontal cortex. As defined by Roberts et al. (1994), working memory is the maintenance of transient information over brief temporal intervals to direct future-oriented activity; inhibition is the ability to engage in the appropriate repose instead of the more likely, albeit maladaptive, response. Using an experimental paradigm, Roberts et al. (1994) examined nonpatient subjects on a task that is sensitive to prefrontal dysfunction - the antisaccade task - in conditions that made varying demands on working memory. They found that the highest working memory load produced inhibitory errors comparable to those found with patients with prefrontal dysfunction. Most recently, Welsh (2002) also argued that a relatively high convergence can be achieved in explaining normal development and clinical variations in EF by replacing the current broad EF concept with a working memory/inhibition metaphor.

Measurement of EF

A review of research on EF reveals at least 60 different EF tasks that have been used in the literature as measures of some aspects of EF (Pennington & Ozonoff, 1996). In spite of this abundance of measures, the measurement of EF is far from being straightforward. For example, there is significant overlap and interrelatedness of attention, memory, and EF at a measurement level as well as a conceptual level. There is no easy way to separate attention from other perceptual processes or cognitive processes. One could not encode information into memory without adequate attention or without an adequate

strategy (i.e., executive function). Similarly, executive function would not be able to emerge if memory systems could not operate to register, store, and make available diverse forms of knowledge and experience (Eslinger, 1996). Morris (1996) researched six well-respected journals that frequently publish articles related to learning disabilities to look at what measures are commonly used for attention, memory, and executive functions. He found 20 different measures of EF, 25 attention measures, and 15 memory measures. Interestingly, many of the EF measures were found listed as measures of attention within the same journal. These include the Trail Making Test, the Stroop test, continuous performance tests (CPT), event-related potential (ERP) measures, and a number of other less common measures. There was also overlap between measures of attention and memory. For example, the Digit Span subtest from the Wechsler scale was frequently used as a measure of both attention and memory.

As Borkowski and Burke (1996) pointed out, EF is elusive to define operationally because it can be observed only as changes in low-level cognitive functions or behaviors. As such, there are no unambiguous or direct indicators or measures of executive dysfunction. In other words, EF likely always will be inferred as a remote, mediational process rather than observed directly. Current EF tasks often involve the performance of actions, usually instructed or self-instructed, that conflict systematically with immediate and well-established nonverbal and verbal sources of behavior control. One important limitation of these measures is that they are not theoretically well-specified and do not allow us to identify component processes (Pennigton, Bennetto, McAleer, & Roberts, 1996). To the extent that an EF task is a complex, molar task that assesses many interacting component processes, performance on it can be disrupted in many different ways. It is not surprising that many

popular EF measures (e.g., Wisconsin Card Sorting Test, Trail Making Test, Stroop Color and Word Test, Continuous Performance Test, Towers of Hanoi or London, etc.) have shown generally unsatisfactory psychometric properties, given that most common EF measures are molar tasks tapping several different executive functions and possibly some non-executive functions.

Fortunately, some noticeable efforts have recently been made both in cognitive psychology (Miyake et al., 2000) and neuropsychology (Lehto, Juujarvi, Kooistra, & Pulkkinen, 2003; Pennington, 1997) to partially resolve these measurement and conceptual issues by employing latent variable approaches. Miyake and colleagues (2000) confirmed the existence of three often-postulated executive functions (i.e., shifting of mental sets, updating and monitoring of working memory representations, and inhibition of prepotent responses) with an adult sample by employing a set of simple experimental tasks, and found that frequently-used EF tasks tap one or more of these components. Based on factor analytical studies of a wide variety of common EF tasks, Pennington (1997) concluded that at least three major dimensions of executive functions - verbal working memory, cognitive flexibility or set shifting, and motor inhibition - appeared to possess some validity in both normal and abnormal populations. Most recently, using a children sample, Lehto et al. (2003) identified three basic dimensions of executive function that are very similar to those from Miyake et al. (2000). It should be noted that working memory and inhibition factors were consistently identified across these studies, supporting Welsh and colleagues' view (Welsh, 2002; Welsh & Pennington, 1988) of working memory and inhibition as core components of the broad EF concept.

Current EF Models of ADHD

Attention Deficit Hyperactivity Disorder (ADHD) is a pattern of deficits in behavioral control and self-regulation characterized by poor sustained attention, impulsivity, and hyperactivity (Barkley, 1997). With an estimated prevalence of 3-5% in school-age children (American Psychiatric Association, 1994), ADHD is one of the most frequently diagnosed disorders in pediatric populations.

It has long been hypothesized that frontal lobe dysfunction, and subsequent executive impairments, are involved in the pathophysiology of ADHD. Exploration of executive-type deficits in children with ADHD also has been a very active topic of research investigation (Ozonoff, 1997) and recent theoretical development in ADHD (e.g., Barkley, 1997; Quay, 1997). Particularly, two components of EF (i.e., working memory and inhibition) have been incorporated into contemporary theories of ADHD such as the Disinhibition Model (Barkley, 1997) and the Working Memory Model (Denny & Rapport, 2000; Rapport, Chung, Shore, & Issacs, 2001). In both models, working memory and/or inhibition are posited to play pivotal roles in the manifestation of ADHD symptoms, but their relative importance and the exact mechanisms by which those symptoms manifest have been postulated differently.

Barkley (2000) asserted that executive functions allow for the developmental shift from external controls and cues to internal, mental representations, and self-control of one's behavior. In explaining EF in relation to ADHD, Barkley (1997) posited that the core deficit in ADHD is that of behavioral inhibition. This is consistent with Quay's idea that an under-responsive behavioral inhibition system (BIS) located in the septo-hippocampal system of the brain, having connections to the frontal cortex, is responsible for ADHD

(Quay, 1997). According to Barkley, behavioral inhibition refers to three interrelated processes: (1) inhibiting the initial prepotent response to an event; (2) stopping an ongoing response or response pattern, thereby permitting a delay in the decision to respond or continue stopping; and (3) protecting this period of delay and the self-directed responses that occur within it from disruption by competing events and responses (Barkley, 1997). In addition to behavioral inhibition, he further identified key executive function processes including reconstitution, self-regulation, internalized speech, and working memory. The central executive in Barkley's model is that of time awareness including the ability of the individual to use hindsight and foresight in the problem-solving process. He argued that the inability to suppress prepotent responses to stimuli (i.e., disinhibition) interferes with the development and execution of other executive functions such as working memory, self-regulation, and reconsititution. The causal influence of disinhibition on these executive functions is postulated to account for the impulsive/hyperactive behavior exhibited by individuals with ADHD. The hypothesized interrelationship between the two major executive functions (i.e., working memory and inhibition) and core ADHD symptoms in Barkley's model is shown in Figure 1.

The Disinhibition Model (Barkley, 1997) articulates that disinhibition has a direct causal influence on the motor (behavior) control systems that are comparable to impulsive/hyperactive response. Also, disinhibition has an indirect effect on hyperactivity via working memory deficit. The relationship between inhibition and working memory, however, is somewhat ambiguous in this model. Barkley (1997) posited that behavioral inhibition merely sets the occasion for the performance of working memory and protects that performance from interference. In other words, the behavioral inhibition system plays a

supportive and protective role with regard to the other executive functions including working memory (Barkley, 1997). Although effective inhibition appears prerequisite to working memory processes, inhibition is *not* posited to directly cause working memory to occur in this model. A bi-directional arrow between inhibition and working memory in Figure 1 reflects this "unanalyzed" association (i.e., correlation).

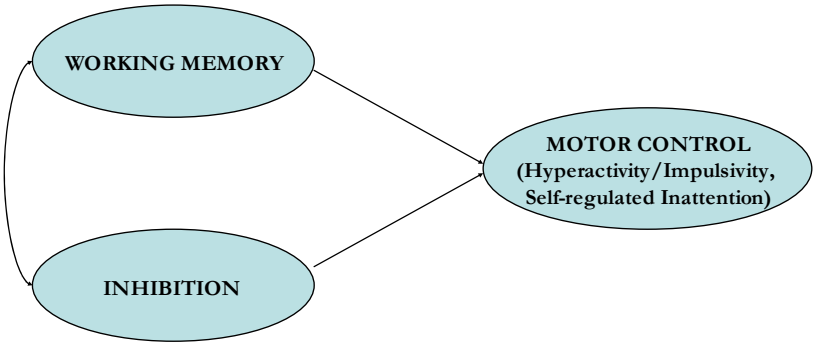

Figure 1. Interrelationship of Working Memory, Inhibition, and Motor Control in the Disinhibition Model.

Barkley's model is confined to ADHD combined type (ADHD-C). Barkley has argued that attention problems experienced by individuals with ADHD-predominantly inattentive type (i.e., selective/focused attention) are qualitatively different from those of ADHD-C (i.e., self-regulation and goal-directed persistence). Moreover, the lack of persistence or inattention observed in ADHD-C is conceptualized as a secondary symptom

resulting from the interaction of the behavioral inhibition system with other executive functions that provide for the control of behavior by internally represented information (i.e., a consequence of the impairment that poor behavioral inhibition and interference control create in the executive control). Thus, this model implicates that the self-regulated form of inattention arises via the same mechanism as the motor control component as shown in Figure 1.

Whereas the Disinhibition Model posits a central role of behavioral inhibition, the Working Memory Model (Denny & Rapport, 2000; Rapport et al., 2001) suggests that the core deficit in ADHD is that of working memory. The relationship of working memory, inhibition, and ADHD symptoms in Working Memory Model is depicted in Figure 2.

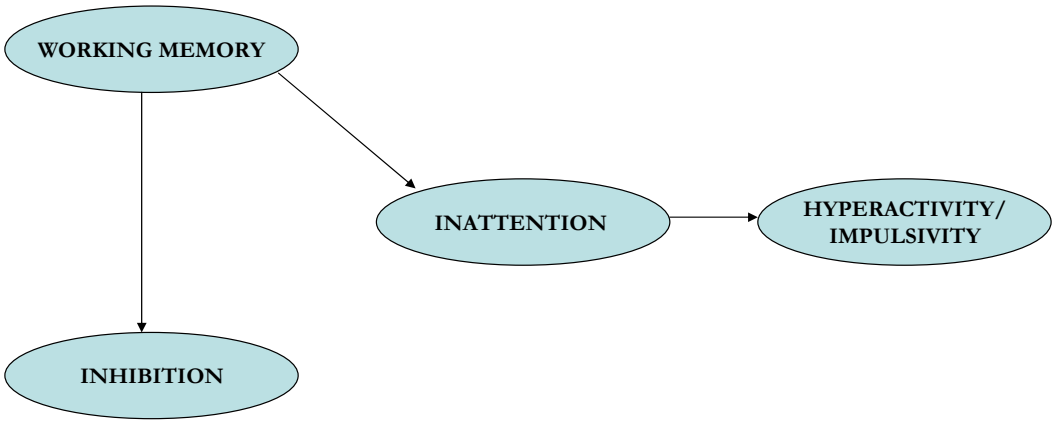

Figure 2. Relationship of Working memory, Inhibition, and ADHD symptoms in the Working Memory Model (Rapport et al., 2001).

As seen in Figure 2, the impulsivity/hyperactivity associated with ADHD is the result of the deficiencies in working memory. Rapport and colleagues (Denny & Rapport, 2000; Rapport et al., 2001) have asserted that working memory includes those processes needed to construct, maintain, and manipulate incoming information – in other words, working memory is needed for problem solving and for the execution of an organized plan of action or behavioral sequence. Thus, rather than deficits in memory being viewed from the perspective of an "effect" of deficits in executive function, working memory is seen as having the pivotal role in determining the individual's behavior. More specifically, the inability to maintain working memory representations leads to behavior that increases the rate at which input is delivered to working memory so as to compensate for the rapid rate at which representations fade. In other words, failure of working memory not only leads to disorganized behavior, but also motivates individuals to redirect their attention to other stimuli in the environment. This redirection of attention, or stimulation seeking behavior, is conceptualized as a form of escape from monotonous or high task demand conditions, maintained by a negative reinforcement principle, and observed by others as hyperactivity and impulsivity. These postulations imply that impulsivity/hyperactivity is a causal byproduct of inattention, and that working memory deficits lead to hyperactive/impulsive behaviors via inattention (see Figure 2). Clearly, the role of disinhibition is secondary to working memory in this model. However, the mechanism by which disinhibition contributes to ADHD symptoms has not been specified in this model. As Rapport et al. (2001) argued, disinhibition is more parsimoniously viewed as a product of working memory process rather than a cause thereof, with working memory playing a primary

controlling influence on inhibition. This assertion is expressed in Figure 2 with a directional arrow from working memory to inhibition.

Then, to what extent have these two models been empirically supported? There is a dearth of studies that directly test the predicted relationships by these two models. This is not surprising given that these EF model were recently developed. However, there is a substantial body of empirical findings evidencing poorer performance on purported inhibition tasks among children with ADHD compared to normal controls. In a meta-analytic study covering 18 studies, Pennington and Ozonoff (1996) concluded that ADHD children demonstrated very consistent difficulty with a relatively large effect size on many purer measures of behavioral (motor) inhibition such as Go-No-Go, Stopping, Antisaccade, Conflict Motor task, and NEPSY inhibition. In a more recent and extensive review, Nigg (2001) reached the same conclusion. He demonstrated that ADHD children had less consistent difficulty with certain types of executive inhibition, including interference control, cognitive inhibition, and oculomotor inhibition as well as motivational inhibition and other automatic inhibition, than with behavioral/motor inhibition.

There are relatively fewer studies specifically exploring working memory function in ADHD and apparently those studies have reported mixed findings. For example, Stevens (2001) reported that children with ADHD had deficits in working memory as well as inhibitory control and short-term memory relative to children without ADHD. Dietlein (2001) found that children with ADHD performed significantly worse on the verbal and spatial working memory tasks than did non-ADHD children. In contrast, Vaughn (1998) found that although ADHD-I children tended to perform poorest, there were no significant differences across ADHD-C, ADHD-I, and normal control children on various working

memory tasks. In a study with a nonreferred sample of twins, Willcutt et al. (2001) contrasted the performance of individuals with reading disability (RD; n=93), ADHD (n=52), and RD+ADHD (n=48), and neither RD nor ADHD (n=121) showed deficits on measures of phoneme awareness and executive function. Interestingly, ADHD was associated with inhibition deficits, whereas RD was associated with significant deficits on measures of phoneme awareness and "verbal working memory"; the RD+ADHD group was most impaired on virtually all measures. Finally, Barnett et al. (2001) found that, compared with the controls (n=26), performance in children with ADHD who were not on medication was significantly worse on a "spatial" working memory task. There was no difference in performance on this task between the children with ADHD who were on medication and the controls. Given all these findings, the relationship between the working memory component of EFs and ADHD appears to be far from straightforward presently. Only certain types of working memory, such as spatial/non-verbal, may be particularly deficient in individuals with ADHD. Also, the relation between working memory deficits and ADHD may be mediated by the comorbid conditions such as RD.

Comorbidity in Adult ADHD

ADHD is currently viewed as a chronic disorder, originating in childhood but with symptoms frequently continuing into adult life, causing distress and psychiatric comorbidity (Rosca-Rebaudengo, Durst, & Dickman, 2000). Researchers have historically adopted three competing views regarding the longitudinal course of ADHD (Marks, Newcorn, & Halperin, 2001). Early investigators believed that ADHD would dissipate with maturation, most commonly around the time of adolescence. Others have hypothesized that the core features of the disorder would persist into adulthood accompanied by demoralization and academic

deficits. Finally, some researchers have conceptualized ADHD as a risk factor for serious forms of psychopathology that emerge later in development including delinquency and antisocial behavior.

Follow-up studies of ADHD children partially support the accuracy of all these views (Marks et al., 2001). ADHD behaviors have been shown to persist into adulthood in 10 to 60 % of cases with documented childhood onset (Mannuzza, Klein, Bessler, Malloy, & LaPadula, 1993; Weiss, Hechtman, Milroy, & Perlman, 1985). Specifically, it has been found that ADHD relatively persists through adolescence and apparently decreases in early adulthood (Mannuzza et al., 1993). Also, those who continue to have the disorder as adolescents and adults have been found to be at greater risk for antisocial and substance abuse disorders as well as academic and vocational problems (Mannuzza et al., 1993; Satterfield & Schell, 1997).

Despite the fact that an extensive literature exists regarding patterns of neuropsychological dysfunction among children with ADHD, far less is known regarding the neuropsychological profiles of adults with ADHD and the extent to which the presence of comorbid disorders are associated with unique patterns of cognitive deficits (Marks et al., 2001). The relatively few published studies have reported that adults with ADHD are significantly impaired, relative to normal controls, on purported measures of inhibition (Corbett & Stanczak, 1999; Lovejoy et al., 1999), verbal and nonverbal memory (Johnson et al., 2001), working memory (Walker, Shores, Troller, Lee, & Sachdev, 2000), and psychomotor speed (Johnson et al., 2001; Walker et al., 2000). Although more studies are needed with adults with ADHD to replicate these findings, an EF hypothesis of ADHD appears to be applicable to adults at this point.

An Integrated EF Model of ADHD and Its Comorbid Conditions

This new model was expanded and modified from two existing models of ADHD. There are important rationales for an integrated model. First, while current EF models of ADHD are convergent in postulating executive dysfunction as a core neurocognitive deficit in ADHD, hypotheses on the relative importance of different EF components and their roles in the development and manifestation of ADHD symptoms are posited in a different way, but not necessarily, in an incompatible way. Because some specific relationships among EF components and core ADHD symptoms have been specified in one model but not in the other, these two models could be complementary to each other. For example, whereas the nature of relations between inhibition and working memory is somewhat ambiguous in the Disinhibition Model, the Working Memory Model clearly conceptualizes inhibition as a product of deficient working memory processes. In contrast, whereas the Working Memory Model underspecifies the nature of the inhibition-hyperactivity/impulsivity link, the causal influence of inhibition on hyperactivity is a critical component in the Disinhibition Model. Also, whereas the exact relationship between (self-regulated) inattention/distractibility and motor control systems are not well specified in Barkley's model, hyperactivity/impulsivity represents the byproducts of inattentiveness by means of negative reinforcement principles in Rapport et al.'s model.

Finally, although Rapport et al. (2001) indicated that the myriad behavioral and cognitive characteristics associated with ADHD are peripheral variables partially dependent on working memory processes, comorbid conditions frequently observed in individuals with ADHD generally have been overlooked in their model, as well as Barkley's. Considering that EF deficits also are observed in many neurodevelopmental, psychiatric,

behavioral, and learning disorders other than ADHD (for review, see Pennington, 1997; Pennington & Ozonoff, 1996; Welsh, 2002) and that many individuals with ADHD tend to have one or more comorbid disorders, comorbidity with ADHD would need to be adequately addressed for better understanding of current EF accounts of ADHD. Thus, any integrated EF model of ADHD should specify the developmental or dynamic relationship between ADHD and its comorbid disorders.

An integrated EF model of ADHD was developed based on these rationales, current EF models of ADHD, and existing literature on comorbidity in ADHD. This model is intended to incorporate major predictions of the existing EF models and specify the interrelationship of major ADHD symptoms, as well as the two EF components with two common comorbid conditions with ADHD. This model is shown in Figure 3.

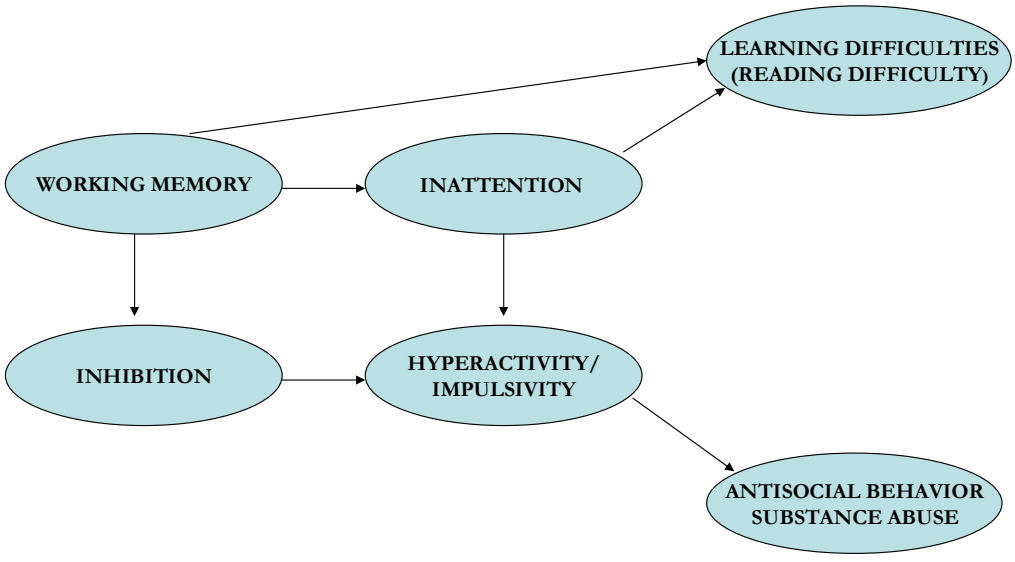

Figure 3. An Integrated EF Model of ADHD and Its Comorbid Conditions.

Major characteristics and predictions of the model are summarized as follows: First, based on experimental evidence that high working-memory load produces more inhibitory errors, and recent theoretical accounts of prefrontal cognitive processes by working memory and inhibition (Cohen & Servan-Schreiber, 1992; Dehaene & Changeux, 1991; Kimberg & Farah, 1993;Levine & Prueitt, 1989; Pennington, 1994; Roberts, Hager, & Heron, 1994; Roberts & Pennington, 1996), this integrative model posits that working memory deficits causally lead to disinhibition, consistent with Rapport et al. (2001)'s prediction. Secondly, this model postulates that inattention/distractibility and hyperactivity/impulsivity comprise two separate but interrelated dimensions of ADHD symptoms, consistent with current diagnostic criteria (DSM-IV; American Psychiatric Association, 1994) and many confirmatory and exploratory factor analytic studies (e.g., Burns et al., 1997a; Burns, Walsh, Owen, & Snell, 1997b; Conners, Sitarenios, Parker, & Epstein, 1998a, 1998b; DuPaul et al., 1997, 1998). It is further posited that those two symptoms are accounted for by two underlying, interrelated neurocognitive deficits in working memory and inhibition. That is, whereas the working memory deficit directly leads to poor sustained attention and distractibility symptoms (consistent with the Working Memory Model), disinhibition has a direct influence on impulsive/hyperactive behaviors (consistent with the Disinhibition Model). This symptom-specific hypothesis was generated from recent studies on differential EF profiles in children with ADHD by subtype (Houghton et al., 1999) and by symptom (Chhabildas, Pennington, & Willcutt, 2001; Lee, Riccio, & Hynd, in press). Importantly, this model further posits that poor working memory may contribute to impulsive/hyperactive behavior (as Barkley has suggested), but this effect is predicted to be mediated by disinhibition. Likewise, working memory also may result in

hyperactive/impulsive behaviors (as Barkley has suggested), but the relationship is mediated by inattention/distractibility symptoms (as Rapport et al. have suggested).

The inattention/distractibility symptom and inhibition may be correlated according to this model, but the correlation between these two components reflects a spurious effect due to an underlying working memory deficit common to these two cognitive components. Finally, this model specifies the nature of the interrelationship of EF components with two broadly-defined comorbid conditions, i.e., learning difficulties and antisocial behavior/substance abuse. Several studies on comorbidity in ADHD by subtype or symptom (e.g., Eiraldi, Power, & Nezu, 1997; Gadow et al., 2000; Lahey, et al., 1998; Lahey & Willcutt, 1998; Morgan, Hynd, Riccio, & Hall, 1996; Willcutt & Pennington, 2000; Willcutt, Pennington, Chhabildas, Friedman, & Alexander, 1999) have suggested that primarily inattentive features of children with ADHD are more likely to be associated with comorbid learning and/or internalizing disorders, while hyperactive and/or combined subtypes are more frequently related with delinquency, aggression, oppositional behavior, and substance abuse. Thus, based on these research findings and the differential EF hypotheses by ADHD symptoms as described above, learning difficulties are hypothesized as primarily related to working memory deficits and inattention, whereas antisocial behavior/substance abuse are primarily linked to a disinhibition-hyperactivity/impulsivity path (See Figure 3).

A substantial body of research has suggested working memory deficits in children with learning problems including reading disorder and arithmetic difficulties (e.g., de Jong, 1998; Hitch & McAuley, 1991; Siegel & Linder, 1984; Siegel & Ryan, 1989; Swanson, 1993, 1994). It is has been suggested that phonological awareness is a primary determinant of

early reading acquisition and a cause of reading disabilities (Wagner & Torgesen, 1987), and working memory capacity is essential in effective performance on phonological awareness tasks because those tasks often require the storage and manipulation of phonemes (Tunmer & Hoover, 1992). Two studies with adults (Isaki & Plante, 1997; Swanson, 1994) also suggest that adults with learning disabilities demonstrate poorer performance on working memory tasks. Thus, in the integrative model, it is hypothesized that poor working memory directly contributes learning difficulties as well as inattention. Learning difficulties could be correlated with inhibition (Passolunghi & Siegel, 2001), but this correlation is predicted to be generated by the same underlying working memory deficit. In addition to the direct effect, the working memory deficit further is hypothesized to have an indirect influence on academic difficulties through inattention/distractibility.

Children with ADHD are at risk for comorbid conduct disorder in childhood and antisocial personality and substance-use disorders in childhood and adulthood (Sullivan & Rudnik-Levin, 2001). Untreated ADHD has been found to be a significant risk factor for substance-use disorder, even after controlling for conduct disorder (Biederman et al., 1999). Recent research also has suggested that the presence of ADHD may mediate the course of substance-use disorder (Biederman et al., 1997). There may be a variety of reasons why individuals with ADHD preferentially seek out drugs. Vulnerabilities particular to this population may include impulsivity, poor choice in peer groups, impaired occupational and social functioning, and the desire to get high, as well as efforts at self-medication (Sullivan & Rudnik-Levin, 2001).

Notably, EF deficits also have been found to be associated with antisocial behavior. A meta-analytic review (Morgan & Lilienfeld, 2000) of 39 published studies on the

relationship between antisocial behavior and a variety of EF measures indicated medium to large effect sizes with an average weighted mean effect size of 0.62. However, because only one study that examined the presence of ADHD was included in their meta-analysis, whether there is evidence of deficits when comorbid ADHD is excluded was unclear. In another meta-analysis of 9 published studies of EFs in conduct disorder (CD), Pennington and Ozonoff (1996) found that samples of individuals with CD demonstrate fairly consistent EF deficits, but only when comorbid ADHD has not been removed. Moreover, they found that non-EF measures, such as verbal measures and behavioral impulsivity, are often more sensitive to CD than are EF measures. Finally, a recent meta-analytic study (Oosterlaan, Logan, & Sergeant, 1998) suggests that response inhibition may be a particularly impaired EF component in children with CD as well as children with ADHD. Based on these empirical findings, the integrative model hypothesizes that hyperactivity/impulsivity of ADHD has a direct influence on the development of antisocial behavior, and that the relation between EF deficit (specifically, disinhibition) and antisocial behavior is mediated by the presence of hyperactivity/impulsivity symptoms. Given that ADHD behaviors appear during childhood and usually precede the development of substance abuse problems, the integrative model assumes the same pattern of relationship among inhibition, impulsivity, and substance abuse with a direct influence of impulsivity on substance abuse and the mediating role of impulsivity in the relation between inhibition and substance abuse.

Present Study

The major purpose of present study is to test current EF models of ADHD (Barkley, 1997; Rapport et al., 2001) and a new integrated EF model of ADHD and its

comorbid conditions. Although disinhibition and working memory deficits have been identified as comprising neurocognitive dysfunction related to the manifestation of ADHD symptoms in children, few studies have attempted to directly test these two models. Moreover, because current EF models of ADHD have been developed primarily to be applied to children with ADHD, the extent to which they are applicable to adults with ADHD has completely unknown. This study will examine which of two current models provides the better fit with data and determine the extent to which each model's predictions are supported in an adult sample. The integrated model also will be tested and modified as needed to improve the model. The final model will be presented.

Methodologically, this study employs a latent variable approach including confirmatory factor analyses and structural equation modeling (SEM). While the SEM is considered to be one of the most advanced and sophisticated statistical procedures in psychology and other social sciences, only a few studies (e.g., Lehto et al., 2003; Miyake et al., 2000) in the field of executive function and ADHD have employed this procedure. Moreover, path models such as current EF models of ADHD never have been tested using this procedure. A latent variable approach (compared to a manifest variable approach) is considered as very adequate for studying executive functions, particularly given that many common EF tasks frequently have questionable psychometric properties and intrinsic task impurity problems. These problems can be alleviated, at least partially, by using multiple tasks for each target EF and aggregating the results to extract what is common among those tasks selected to tap a putative EF and use that "purer" latent variable factor to examine how different EFs relate to each other and other constructs in the model. The methodology

is covered in more detail in Chapter III. Results are provided in Chapter IV. The results and implications are presented in Chapter V.

CHAPTER III

METHODOLOGY

Participants

Participants in this study consisted of 102 adults who were consecutive referrals to a research project, Memory, Attention and Planning Study (MAPS), led by Dr. Cynthia Riccio at Texas A&M University. Participants were recruited through the use of announcements distributed in the local community to physicians, local support groups for individuals with Attention Deficit Hyperactivity Disorder, a community-based counseling center, on local bulletin boards, and in the local newspaper. Each participant was given a comprehensive assessment report of the results, along with recommendations, following completion of the evaluation.

The sample was made up of predominantly college students or graduates of college with a mean educational level of 14.50 years (*SD*=1.27). Age of participants ranged from 18.25 to 33.75 years (*M*=21.96, *SD*=3.47). For self-reported ethnicity, 86 (84.3%) were white non-Hispanic, 10 (9.8%) were Hispanic, 4 (3.9%) were Asian, 1 (1.0%) was African American, and 1 (1.0%) was bi-racial. For gender, 54 (52.9%) were male and 48 (47.1%) were female. For the total sample, 43 (42.2%) participants indicated a history of one or more previous psychiatric diagnoses. Of these 43, 20 participants (32.6%) were previously diagnosed with ADHD; the other 23 participants were diagnosed with other diagnoses including major depressive disorder (n=9), learning disorders (n=5), dysthymic disorder (n=3), generalized anxiety disorder (n=2), conduct disorder (n=1), and others (n=3). Prior diagnosis of schizophrenia or history of severe head injury was established as exclusionary

criteria for the MAPS data. Participants also had to have obtained an IQ greater than or equal to 80 and had to speak and read English to be included. Full Scale IQ of the participants in this study ranged from 85 to 147 (*M*=111.93, *SD*=13.63) and total achievement score of the sample (measured with Woodcock Johnson III) ranged from 78 to 137 (*M*= 102.74, *SD*=11.97).

Based on current diagnostic considerations (see diagnostic decision making), 31 (30.4 %) constituted a no-diagnosis control group; the remaining participants were found to meet criteria for a diagnosis of ADHD (n= 32, 31.4%) with or without comorbid disorders, or to meet criteria for psychiatric disorder(s) other than ADHD (n=39, 38.2%). Of the adults in the other diagnoses group (n=39, 38.2%), the diagnoses included mood disorders, conduct disorder, anxiety disorders, learning disorders, substance use disorders, and schizoaffective disorder. Of the adults in the ADHD group (n=32), 14 met criteria for Predominantly Inattentive (PI) type and 18 met criteria for Combined (C) type. Of those 32 individuals diagnosed with ADHD, 14 had a previous diagnosis of ADHD, 10 had a current prescription for medication (e.g., Ritalin®, Concerta®, Adderall®), and 19 were diagnosed with an additional disorder (e.g., learning disorders, anxiety disorders, mood disorders). Current Global Assessment of Functioning (GAF) scores ranged from 50 to 88 (*M*=66.78, *SD*=8.12). Demographics and descriptive data on selected variables are provided in Table 1.

Table 1

Participants: Demographics and Descriptive Data (N=102)

GENDER:	
Males	54
Female	48
ETHNICITY:	
White, non-Hispanic	86
African-American	1
Hispanic	10
Asian	4
Other/Biracial	1
PREVIOUS DIAGNOSES:	
ADHD	20
Other Psychiatric Disorders	23
None	59
CURRENT DIAGNOSES:	
ADHD	32
Other Psychiatric Disorders	39
None	31
MEAN AGE (SD) – years	21.96 (3.47)
MEAN EDUCATIONAL LEVEL (SD) – years	14.5 (1.27)
MEAN FULL SCALE IQ (SD)	111.93 (13.63)
MEAN WJ-III ACHIEVEMENT SCORE (SD)	102.74 (11.97)
MEAN CURRENT GAF (SD)	66.78 (8.12)

Procedures

All participants received a comprehensive individual evaluation in the Counseling and Assessment Clinic at Texas A&M University. The evaluation included the areas of intelligence, language, executive function, memory, achievement, behavior, and social and emotional functioning. Table 2 lists all the measures that were used for adult participants in the MAPS project.

Table 2

Measures Used for Adults in the MAPS Project

History	History Form and Interview Questionnaire
Semi-structured Diagnostic Interview	• Structured Clinical Interview for DSM-IV Axis I Disorders (SCID: First, Gibbon, Williams, & Spitzer, 1997)
Cognitive Ability	• Wechsler Adult Intelligence Scale – Third Edition (WAIS-III: Wechsler, 1997a)
Language	• Peabody Picture Vocabulary Test -3 (Dunn & Dunn, 1997) • Expressive Vocabulary Test(Williams, 1997)
Executive Functions	• Gordon Diagnostic System (GDS: Gordon, 1983) • Conners Continuous Performance Test – II (CCPT-II; Conners, 1999) • Trail Making Test (TMT: Reitan, 1992) • Wisconsin Card Sorting Test (WCST: Heaton, Chelune, Talley, Kay, & Curtiss, 1993) • Stroop Color and Word Test (Golden, 1978; Golden & Freshwater, 2002) • Tower of London – Drexel Edition (TOL^{DX}: Culbertson & Zillmer, 2000) • Clock Face (Cohen, Riccio, Kibby, & Edmonds, 2000) • Comprehensive Complex Figure Test (Reynolds, Bigler, & Riccio, 2004) • Controlled Oral Word Association Test (FAS: Benton & Hamsher, 1978; Benton, Hamsher, & Sivan, 1994) • Torrance Figural Fluency (Torrance, 1962) • It's About Time (adapted from Barkley, 1998)
Memory	• Wechsler Memory Scale – Third Edition (WMS-III: Wechsler, 1997b)
Achievement	• Woodcock Johnson Test of Achievement – Third Edition (WJ-III: Woodcock, McGrew, & Mather, 2001)

Table 2 (Continued)

History	History Form and Interview Questionnaire
Behavioral, Social, & Emotional Functioning	• Conners Adult ADHD Rating Scale (CAARS; Conners, Erhardt, & Sparrow, 1997) • Beck Depression Inventory-2 (BDI-II: Beck, Steer, & Brown, 1996) • State Trait Anxiety Inventory (STAI: Spielberger, 1983) • Substance Abuse Subtle Screening Inventory-3 (SASSI-3: Miller & Lazowski, 1999)

These measures were administered according to standardized procedures by advanced doctoral students supervised by a licensed psychologist, or by a licensed psychologist. The order of administration was random; however, the order of two continuous performance tests (i.e., GDS and CCPT-II) was controlled to ensure equal proportions of subjects received each one first. The length of each testing session varied depending on the availability of the individual being assessed. Two to three testing sessions were typically needed to complete testing procedures. Participants who were currently taking stimulant medication (n=10) were asked to consult with their physician regarding the possibility of omitting medication on those days they were being evaluated. However, those who were taking other types of medications (e.g., antidepressants, anxiolytics; n=9) continued on the medication as prescribed without interruption.

Diagnostic Decision-Making

Diagnostic decisions were made after at least two individuals (advanced doctoral students and at least one licensed psychologist) independently reviewed history, intelligence

and achievement test results, self-reported behavioral and emotional functioning, and structured clinical interview data. In making diagnoses, these independent reviewers were blind to results from other measures such as those of memory and executive function. Inter-diagnostician agreement was determined using Cohen's Kappa and ranged from .93 when considering multiple Axis I categories to .97 when limited to the three groups of No Diagnosis, ADHD, and Other Psychiatric. Any disagreements in diagnostic decision were resolved through discussion and further review of information pertinent to the Diagnostic and Statistical Manual – the Fourth Edition (DSM-IV) criteria.

Selection of Indicators

For the purpose of present study, two or three representative tasks or indicators were selected for each target latent variable based on literature review. Latent variables in this study include working memory, inhibition, inattention, and hyperactivity/impulsivity. Reading difficulty and substance abuse symptoms were measured with single indicators.

Working Memory

Three subtests from the WMS-III (Wechsler, 1997b) were used the indicators of working memory: Letter-Number Sequencing, Spatial Span, and Digit Span Backward (longest digit span backward). Letter-Number Sequencing and Spatial Span comprise the Working Memory Index score in WMS-III. Letter-Number Sequencing is considered as a measure of auditory working memory and requires the participant to order sequentially a series of numbers and letters orally presented in a specified random order. Spatial Span is a visual analogue of the Digit Span subtest and taps the participant's ability to hold a visual-spatial sequence of events in the working memory (Psychological Corporation, 1997). In addition to these two subtests, Digit Span Backward (i.e., longest digit span backward) was

also used as the third indicator of working memory. It has been suggested that Digit Span Forward and Digit Span Backward may tap different functions (Reynolds, 1997). Digit Span Forward was excluded in this study because it is viewed as a short term memory task as opposed to a working memory task; simple span tasks such as Digit Span Forward measure only the storage component of working memory, while deemphasizing the manipulation of information (Turner & Engel, 1989). It should be noted that while there are also forward and backward procedures in Spatial Span, the distinction between these two was not made because functioning differences between forward and backward on Spatial Span have not been proven yet. Moreover, there is proven research evidence that, in the visuospatial domain, short term memory and working memory span tasks are related to executive function equally and cannot be clearly differentiated (Miyake, Friedman, Rettinger, Shah, & Hegarty, 2001)

Inhibition

As two indicators of inhibition, the Interference score from Stroop Color and Word Test (Golden, 1978) and the time on Part B to time on Part A ratio on the Trail-Making Test (Reitan,1992) were used. The Stroop task is considered as a prototypal inhibition task (Miyake et al., 2000), particularly tapping "interference control" (Barkley, 1997; Nigg, 2001). The Stroop task has three parts (i.e., Word, Color, and Color-Word). In Color-Word procedure, the participant is required to inhibit or override the tendency to produce a more dominant or automatic response (i.e., name the color word). Several functioning brain imaging studies have established that, at least in adults, performing Stroop task activates the anterior cingulated cortex, a region of the frontal cortex associated with the frontal executive networks (Cabeza & Nyberg 1997; Posner & DiGirolamo, 1998). The

Interference score is the difference between Color-Word and a predicted Color-Word score that is based on the subject's raw Word and Color scores. As a standard measure of interference, the Interference score provides a good measure of pure interference corrected for speed factors; it was found stable in impaired populations as well as normal population (Golden & Freshwater, 2002).

The other indicator of inhibition chosen was the time on TMT-B to time on TMT-A Ratio. TMT-A requires the subject to connect a series of numbered circles distributed arbitrarily on a page; TMT-B comprises circles that contain letters or numbers scattered randomly across the page and requires the subject to alternate connecting numbers and letters in ascending order until the end of the sequences. The time on TMT-B to time on TMT-A Ratio is believed to reflect an ability to inhibit the prepotent response set and successfully switch to the correct one that is adjusted for other basic cognitive abilities such as visual scanning ability or motor speed (Arbuthnott & Frank, 2000). In an exploratory factor analytic study of a battery of measures of attention with a sample of 154 normal adult subjects (Robertson, Ward, Ridgeway, & Nimmo-Smith, 1996), Trail B was loaded on the same factor with Stroop interference score. Reinterpreting this factor analytic study in the context of executive function, Barkley (1997) considered this factor as reflecting the executive function of resistance to distraction (i.e., interference control) and the larger construct of behavioral inhibition.

Inattention

Two indicators of inattention were chosen from two continuous performance test (CPT) variables: Correct Responses (CR) score from the Vigilance task in Gordon

Diagnostic System (GDS: Gordon, 1983) and Omission Error (OE) score from Conners Continuous Performance Test – II (CCPT-II: Conners, 1999). The CPTs are the most popular laboratory measures of sustained attention and vigilance (DuPaul, Anastopoulos, Shelton, Guevremont, & Metevia, 1992). There is increasing evidence that adults with ADHD demonstrate impairment on the CPT and the CPT is sensitive to CNS dysfunction (for review, see Riccio & Reynolds, 2001). Reviewing research on the sensitivity and specificity of various CPTs, Riccio and Reynolds (2001) concluded that CPTs may be useful tools in ruling out ADHD because normal performance on most CPTs by an individual with ADHD would be unusual, but they are not useful in ruling in ADHD or in differentiating ADHD from other clinically significant CNS dysfunction.

The GDS is the most researched of the commercially available CPTs (Riccio & Reynolds, 2001) and is administered via a stand-alone microprocessor. The GDS Vigilance task yields data regarding the individual's ability to focus attention on a task and to maintain this attention over time in the absence of reinforcement. Due to concerns for possible ceiling effects, the longer, 9-minute version with 1-9 sequence was used as opposed to the shorter 6-minute adult version. The CR score reflects the subject's level of "vigilance" or ability to focus the attentional processes in a goal-directed manner and to maintain this investment of attention over time (Gordon, McClure, & Post, 1986).

The CCPT-II is a 14-min visual CPT completed on an IBM-compatiable desktop computer. In CCPT-II, the subject is required to respond to each stimuli presented except when the letter "X" appears. There are several variables available in CCPT-II. For example, the errors of omission (EO) occur when subjects fail to depress the space bar on trials

where letters other than "X" are present, thus reflecting instances in which the subject is not attending to the situation sufficiently to respond to the "X" stimuli. In contrast, the errors of commission (EC) occur when the subject depresses the space bar on trails when the letter "X" was present. The EO, rather than EC, is expected to correlate with ADHD inattention symptoms (Epstein, Conners, Sitarenios, & Erhardt, 1998). Thus, the OE was selected the second indicator of inattention.

Hyperactivity/Impulsivity

The Hyperactivity/Restlessness subscale (5 items) in the Conners Adult ADHD Rating Scales – Self-Report Short Version (CAARS-S:S; Conners, Erhardt, & Sparrow, 1997) and the number of items endorsed in the DSM-IV hyperactivity/impulsivity diagnostic criteria (0-9) were used as two indicators of hyperactivity/impulsivity factor. The 5-item Impulsivity/Emotional Lability subscale in the CAARS-S:S was judged inadequate as an indicator of hyperactivity/impulsivity based on the review of items. This scale contains more items that appear to tap emotional lability (e.g., "My moods are unpredictable.", "Many things set me off easily.", "I have a short fuse/hot temper", "I still throw tantrums."); obviously, these items are far from hyperactive/impulsive behaviors currently described by the DSM-IV. Instead, the number of items endorsed in the DSM-IV ADHD hyperactivity-impulsivity criteria was used as the other indicator of Hyperactivity/Impulsivity factor.

Reading Difficulty

Reading difficulty was measured by a single manifest variable. Reading difficulty in this study was operationalized by the WJ-III (Woodcock, McGrew, & Mather, 2001) Broad

Reading Cluster score *after* controlling for the WAIS-III (Wechsler, 1997a) Verbal Comprehension Index (VCI) score. This residualized reading achievement score on verbal comprehension score is conceptually analogous to the current discrepancy model of reading disorder. The Broad Reading Cluster is a combination of three reading achievement tests (i.e., Letter-Word Identification, Reading Fluency, and Passage Comprehension) and provides a comprehensive measure of reading achievement including reading decoding, reading speed, and the ability to comprehend connected discourse while reading (Mather & Woodcock, 2001). For statistical control, the VCI, instead of Verbal IQ (VIQ), on WAIS-III was chosen because a subtest (i.e., Digit Span) comprising VIQ was used as an indicator of working memory construct.

Substance Abuse

Substance abuse was measured by a single indicator: Symptoms (SYM) subscale on Substance Abuse Subtle Screening Inventory (SASSI-3: Miller & Lazowski, 1999). The SASSI-3 is a screening measure that helps identify individuals who have a high probability of having a substance abuse disorder. The SYM scale is an 11-item true/false measure of the extent to which the subject acknowledges specific problems associated with substance misuse and a pattern and history of serious substance misuse, including negative consequences and being part of a family system that is affected by additions (Miller, Roberts, Brooks, & Lazowski, 1999).

Data Preparation and Screening

Across 11 manifest variables with 102 subjects, there were eight missing observations in total; one missing observation for each of six variables and two missing observations for one variable. Considering the very small percentage of missing values,

missing observations on a particular variable were substituted with the overall sample average for that variable. Because the multivariate techniques used in this study – Confirmatory Factor Analysis (CFA) and Structural Equation Modeling (SEM) - assume multivariate normal distributions and are sensitive to extreme outliers, the following data trimming/screening procedures as recommended in Kline (1998) were used:

For each variable, any observations with values that exceeded three standard deviations from the mean were set to values that were three standard deviations from the mean. This is a fairly conservative trimming procedure that retains extreme observations without those observations having adverse effects on the distributions or undue influence on the covariances. For the 11 manifest variables used in the CFA and SEM analyses, this trimming procedure affected only eight observations across all 11 variables (.007%). To ensure univariate normality, skewness and kurtosis for each variable were calculated. Absolute values of the univariate skew indexes greater than 3.0 were considered as extremely skewed; absolute values of the univariate kurtosis index greater than 8.0 were considered as indicating extreme kurtosis (Kline, 1998). Based on these criteria, two CPT variables were found to have extreme skewness and kurtosis. These variables were transformed to achieve normality by applying logarithmic or inverse functions; these transformations pulled outlying scores closer to the center of the distributions. This trimming procedure resulted in satisfactory distributions for all 11 variables used in the CFA and SEM models (See Table 3).

Descriptive statistics including mean, standard deviation, skewness, and kurtosis for these variables are provided in Table 3. Note that 4 variables were adjusted for all analyses so that larger numbers always indicated higher functioning or better performance. In

addition, two CPT variables are not on their original scales due to transformation to correct severe non-normality.

Table 3

Descriptive Statistics for All Manifest Variables (N=102)

	Mean	SD	Skewness	Kurtosis
WMS-DS	5.61	1.46	-0.11	-0.79
WMS-LNS	11.29	2.59	0.24	0.11
WMS-SS	11.38	2.36	-0.01	0.05
Stroop	52.79	7.87	-0.56	0.61
TMT B:A[1]	4.29	1.01	-1.47	2.72
GDS[2]	1.91	0.33	-0.95	-0.04
CCPT[2]	2.02	0.38	-1.52	2.96
CAARS[1]	22.11	9.47	-0.01	-0.37
DSM-H/I[1]	6.60	2.27	-0.48	-0.42
Reading Diff.	0	10.21	0.38	0.06
SASSI[1]	6.77	1.91	-0.78	0.52

Note. WMS-DS=WMS longest Digit Span backward; WMS-LNS=WMS Letter-Number Sequence; WMS-SS=WMS Spatial Span; Stroop=Stroop Interference Score; TMT B:A= the ration of TMT time on Part B to time on Part A; GDS=GDS Vigilance Correct Responses; CCPT=CCPT Errors of Omission; CAARS=CAARS Hyperactivity/Restlessness Scale T score; DSM-H/I= the number of criteria endorsed with DSM-IV ADHD hyperactivity/impulsivity diagnosis; Reading Diff.= WJ-III Broad Reading Cluster score after controlling for WAIS VCI score; SASSI=SASSI Symptoms subscale raw.

[1]: The directionality of these measures were adjusted so that larger numbers always indicate higher functioning or better performance across all 11 variables.

[2]: These variables were transformed by applying log or inverse conversions to correct extreme non-normal distributions.

In addition, Mardia's (1970) normalized test for multivariate kurtosis, which follows an approximate standard normal distribution, turned out to be .54 for all 11 variables (p >.10), indicating adequate multivariate normality. Finally, the data were also checked for multicollinearity. Bivariate correlations and squared multiple correlations between each variable and all the rest were examined; none of the first-order correlations were above .55 (see Table 4); the highest squared multiple correlation was .41, indicating that multicollinearity is not a problem with these data at the measurement level (Kline, 1998).

Table 4

Correlation Matrix for All Manifest Variables (N=102)

Variables	1	2	3	4	5	6	7	8	9	10	11
1. WMS-DS	---										
2. WMS-LNS	**.546**	---									
3. WMS-SS	**.294**	**.381**	---								
4. Stroop	**.253**	**.323**	**.301**	---							
5. TMT B:A	**.281**	.171	.184	**.306**	---						
6. GDS	**.219**	**.232**	.132	.115	.137	---					
7. CCPT	.174	.130	.057	.112	**.224**	**.448**	---				
8. CAARS	-.042	-.057	.045	.045	-.176	.141	.111	---			
9. DSM-H/I	-.033	-.065	-.020	.073	.026	**.197**	.188	**.518**	---		
10. Reading Diff.	**.318**	**.286**	-.024	**.215**	.078	**.222**	.146	.007	.001	---	
11. SASSI	-.135	-.152	.017	.003	-.058	.057	.010	.152	**.207**	-.189	---

Note. Significant correlations ($p < .05$) are in bold.

As the Table 4 shows, the 11 measures tended to correlate with one another, with the pairs of measures chosen to tap the same latent variable generally showing higher

correlations. Zero-order correlations among different executive function measures or CPT variables are generally low (often .30 or less); however, it should be noted that correlations of this magnitude have been typically reported in other studies on EF measures (e.g., Lehto et al., 2003; Miyake et al., 2000; Miyake et al., 2001). As discussed in Chapter II, relatively low intercorrelations are in part due to complex nature of EF measures (i.e., task impurity issue) and a great deal of error variance involved in measurement. Because of this unique challenge, the latent variable approach is particularly suited for investigating the nature and role of EF.

Statistical Analysis and Procedure

All of the CFA and SEM analyses were performed with the SAS/CALIS procedure (SAS 8.01; SAS Institute, 1999) using maximum likelihood estimates derived from the covariance matrix. A two-step procedure recommended by Anderson and Gerbing (1988) was used. In the first step, CFA was used to develop a measurement model that demonstrated an acceptable fit to the data. In step two, the measurement model was modified so that it came to represent the structural (causal) model of interest. This structural model was then tested and revised until a theoretically meaningful and statistically acceptable model was found.

Because there is no clear consensus as to the best fit indices for the evaluation of measurement and structural models (see Bollen, 1989; Hoyle & Panter, 1995; Hu & Bentler, 1995), multiple fit indices were used to evaluate and compare the models: (1) the χ^2 and χ^2/df statistics; (2) the Goodness of Fit Index (GFI; Joreskog & Sorbom, 1981); (3) the Non-Normed Fit Index (NNFI; Bentler & Bonett, 1980); (4) the Comparative Fit Index

(CFI; Bentler, 1990); (5) the Root Mean Square Error of Approximation (RMSEA; Steiger, 1990); (6) the Bayes Information Criteria (BIC; Raftery, 1993; Schwarz, 1978); and (7) the Expected value of the Cross-Validation Index (ECVI; Browne & Cudeck, 1989, 1993).

The χ^2 is the most common fit index that measures the "badness of fit" of the model compared with a saturated model. Because the χ^2 statistic can be statistically significant even though differences between observed and model-implied covariances are slight as sample sizes increase (Kline, 1998), χ^2/df statistic is preferred with $\chi^2/df < 2$ indicating a good fit (Byrne, 1989). GFI indicates the proportion of the observed covariances explained by the model-implied covariances. NNFI and CFI are incremental fit indexes indicating the proportion of the improvement of the overall fit of the researcher's model relative to a null (independence) model. CFI is less affected by sample size and NNFI includes a correction for model complexity. For GFI, NNFI, and CFI, higher values are desired, with values above .90 indicating good model fit. RMSEA provides a measure of the discrepancy between elements of the model fitted to the sample and the model fitted to the population covariance matrix. Although exact fit to the model would be indicated by RMSEA=0, values less than .08 indicate reasonable model fit, and values less than .05 indicate a close fit in relation to degrees of freedom (Browne & Cudeck, 1993). BIC and ECVI are used for model comparison, with lower values indicating better fit. A BIC difference of 5 points provides strong evidence of model fit in favor of the model with the lower BIC value (Raftery, 1993). ECVI is useful for comparison of alternative models, especially when the sample size is not large, providing an indication of which model yields a solution with greatest generalizability (MacCallum & Austin, 2000). Model comparisons

were also made with the χ^2 difference test when one model was nested within another. Results of these analyses are presented in Chapter IV. Implications are then discussed in Chapter V.

CHAPTER IV

RESULTS

As stated earlier in Chapters I and II, multiple models of EF have been posited for ADHD in the literature. A third model, that integrates two of these models and expands into two common comorbid conditions with ADHD, was offered in Chapter II as well. The purpose of this study was to determine the extent to which these EF models fit to the data generated by an adult sample. Results on existing EF models of ADHD are presented first, followed by results on the new integrated EF model of ADHD and its comorbid conditions.

Testing Current EF Models of ADHD

The Measurement Model: CFA

CFA was conducted to develop a measurement model that possessed an acceptable fit with the data. A measurement model describes the nature of the relationship between a number of latent variables, or factors, and the manifest indicator variables that measure those latent variables. Once a measurement model that displays an acceptable fit is developed, theoretical causal models (i.e., current EF models of ADHD) can be evaluated against the measurement model. Four latent variables corresponding to the four constructs of the current EF models of ADHD were working memory, inhibition, inattention, and hyperactivity/impulsivity. Working memory was measured by three manifest indicator variables; all the other latent variables were measured by two indicators.

The first step was to estimate the full, four-factor model that assumes some degree of separability among all four latent variables. The estimated model is illustrated in Figure 4.

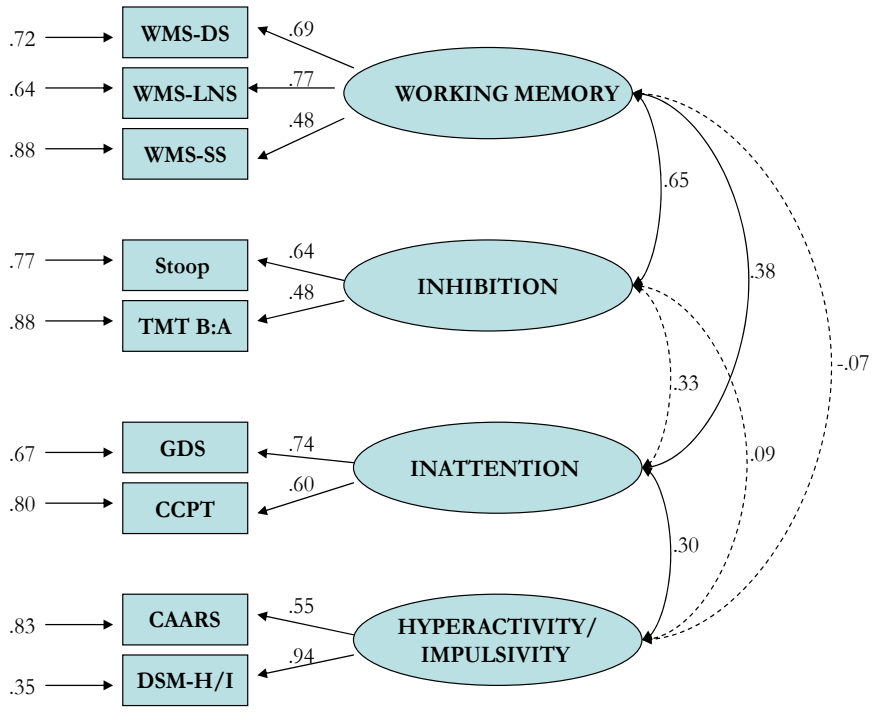

Figure 4. The Estimated Four-factor Model for Two Executive Functions and ADHD Symptoms.

The single-headed arrows (←) have standardized factor loadings next to them. The loadings are standardized regression coefficients estimated with maximum-likelihood estimation. All factor loadings in this baseline model (Figure 4) were statistically significant at the .05 level, indicating the convergent validity of the indicators. The numbers at the ends of single-headed arrows (→) are squared error terms, that give estimates of variance for each task that is not accounted for by the latent construct. The curved, double-headed arrows have correlation coefficients next to them and indicate estimated intercorrelations between the four latent variables. Solid arrows indicate significant correlations ($p < .05$), while dashed ones indicate non-significant correlations ($p > .05$).

As shown in Table 5, in addition to a nonsignificant chi-square, χ^2 (21, N=102) = 16.57, $p > .10$, and a small χ^2/df value, various fit indices for this baseline model (Model B) suggest a superior fit of this model to the data (see Table 5). Additionally, the distribution of asymptotically standardized residuals was satisfactory; the distribution was approximately symmetrical and centered on zero, and no standardized residuals were considered to be large (all less than 2.21 in absolute value). An examination of correlations between the latent variables reveals that not all variables significantly correlate with one another; this will be further discussed in detail when addressing the primary deficit in ADHD later. A moderately high correlation between two EF factors (r=.65) appears to be consistent with current conceptualization of these concepts; a relatively high correlation is well expected given that working memory and inhibition are conceptualized as two interrelated, but separable constructs under the umbrella concept of executive function. However, this result also raises a question as to whether these two constructs are truly separable or should be considered to be measuring the same construct. This question can be answered by demonstrating the discriminant validity of the two factors. Two procedures discussed by Hatcher (1994) were used to address this issue: a confidence interval (CI) test and chi-square difference tests. The 95% CI for the correlation between working memory and inhibition was .36 to .94, which does not contain 1.0, meaning that it is very unlikely that the actual population correlation between these two factors is 1.0. Thus, these factors are separable according to the result from the CI test.

Alternatively, a reduced three-factor model (Model A1) was created by constraining the correlation between working memory and inhibition to 1.0 and was compared against

the four-factor baseline model (Model B). Overall fit indices for Model A1 are shown in Table 5.

Table 5

Fit Indices for the Baseline CFA Model and Alternative Reduced Models for EF and ADHD Symptom Constructs

Model	χ^2/df	GFI	NNFI	CFI	RMSEA *(90% CI)*	BIC	ECVI *(90% CI)*
Model B	16.57/21	.97	1.06	1.0	0 (0-.06)	-80.55	.69 (0-.82)
Model A1	20.32/22	.96	1.02	1.0	0 (0-.08)	-81.43	.71 (0-.86)
Model A2	**34.67**/22	.93	.84	.90	.08 (.01-.12)	-67.08	.85 (0-1.06)
Model A3	24.41/22	.95	.97	.98	.03 (0-.09)	-77.34	.75 (0-.91)
Model A4	**38.16**/24	.93	.84	.89	.08 (.02-.12)	-72.84	.84 (0-1.05)

Note. χ^2 values in bold are significant ($p < .05$). Model B is the baseline 4-factor model. Model A1 is a 3-factor model with working memory-inhibition correlation constrained to 1. Model A2 is a 3-factor model with working memory-inattention correlation constrained to 1. Model A3 is a 3 factor model with inhibition-inattention correlation constrained to 1. Model A4 is a 2 factor model with intercorrelations among working memory, inhibition, and inattention constrained to 1.

All fit indices were indicative of a superior fit of the Model A1 to the data and a minimal statistical difference between Model B vs. Model A1. In fact, although the BIC value difference between the two models (B, A1) was very small (less than 5), the BIC that imposes greater penalty for model complexity than other competitive indexes of the same kind prefers Model A1 to Model B. A chi-square difference test shows that Model A1 is not significantly worse (or marginally worse) than Model B, χ^2 (1, N=102) = 3.75, $p < .10$, and

indicates that Model A1 is a more parsimonious model than Model B. Thus, the discriminant validity of working memory and inhibition constructs received mixed support across two different procedures.

In addition to Model A1, other alternative reduced models were created and evaluated against the full baseline model (Model B) to determine a "final" measurement model. Models A2 and A3 are 3-factor models where correlations between inattention and working memory (Model A2) or inhibition (Model A3) are fixed to 1. These two alternative models are worthwhile to examine given significant confusion in the literature of using measures of executive function, working memory, and attention interchangeably (e.g., Morris, 1996). Fit indexes for Model A2 indicate only a marginal fit with the data and a chi-square difference test shows that Model A2 is significantly worse than Model B, χ^2 (1, N=102) = 18.1, $p < .001$. Model A3 demonstrates a relatively good fit; however, a chi-square difference test again indicates that this model is significantly worse than Model B, χ^2 (1, N=102) = 7.84, $p < .01$. Finally, a 2-factor model where intercorrelations among 3 constructs sharing the same measurement method were constrained to 1, was created and evaluated against the Model B. The rationale for this 2-factor model was that hyperactivity/impulsivity was measured only by behavior ratings, while all the others were measured by performance-based cognitive tests. That is, this model is to examine if the factor structure in Model B can be explained by this same method factor. Fit indexes for Model A4 show that the model fit is not satisfactory; a chi-square difference test also suggests a significantly worse fit than Model B, χ^2 (3, N=102) = 21.59, $p < .001$.

These results indicate that the baseline four-factor model (Model B) is the best fit with the data; overall, the model demonstrated a superior fit with the data and, although somewhat mixed, the validity of the constructs and their indicators also was evidenced. It should be noted that Model A1 is a more parsimonious model that is statistically no worse (or marginally worse) than the full model. However, the original 4-factor model is more consistent with the current conceptualization of EF (Miyake et al., 2000; Pennington, 1997; Welsh & Pennington, 1988) and is more suitable for the subsequent SEM analyses in this study. Based on these reasons, the 4-factor model (Model B) was determined to be retained as the final measurement model for subsequent SEM analyses.

Testing the Disinhibition Model

There are two considerations in testing the Disinhibition Model (see Figure 1). First, as shown in Figure 1, the model is a saturated (i.e., just-identified) path model, meaning that three structural variables are related to one another by causal paths. If the structural portion of the model is saturated, it is not possible to test just the structural portion of the model for goodness of fit, although it is possible to estimate the model and test the overall model for goodness of fit. Therefore, after evaluating the overall model fit, attempts were made to identify parameters that could be dropped from the model without significantly hurting the model's fit. Secondly, the Disinhibition model doesn't specify the relationship between inattention and hyperactivity/impulsivity (H/I); instead, a self-regulated form of attention is seen as arising via the same mechanism with hyperactivity/impulsivity. Therefore, inattention and H/I were considered separately for SEM analyses, resulting in two 3-factor models.

The Disinhibition Models are depicted in Figure 5, along with standardized parameter estimates (path coefficients). For simplicity, the individual indicators used to construct the latent variables are omitted in Figure 5 (see Figure 4 for factor loadings and error terms).

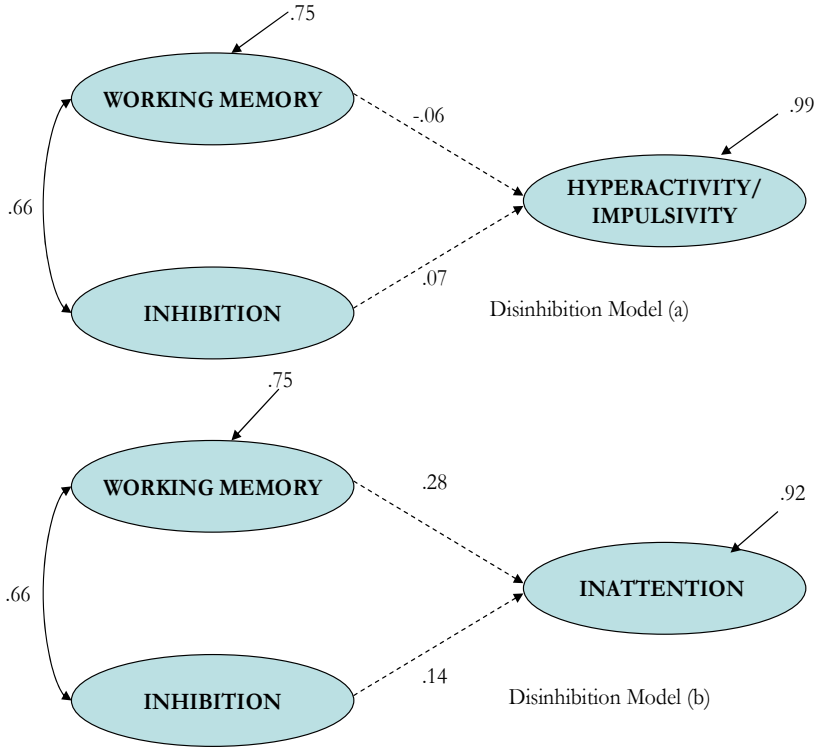

Figure 5. SEM for the Disinhibition Model.

The Disinhibition Model (a) for H/I indicated a good fit with the data as evidenced by χ^2/df=11.45/11=1.04, GFI=.97, NNFI=.99, CFI=.99, and RMSEA=.02 (0-.10). Disinhibition Model (b) for inattention also demonstrated a good fit with the data: χ^2/df=8.53/11=.76, GFI=.98, NNFI=1.05, CFI=1.0, and RMSEA=0.0 (0-.08). Again, it should be remembered that because model (a) and model (b) are saturated path models,

these overall fit indexes do not inform anything about the structural portion of the models. Also, it should be noted that the path coefficients (unique contribution) to inattention and H/I are not significant (see Figure 5).

For the model (a), the modification index (Wald test) suggested that two paths (i.e., working memory→H/I, inhibition→ H/I) could be dropped (these two free parameters could be fixed to zero) without a significant increase in chi-square (i.e., without hurting the model's fit). For Model (b), however, the Wald test indicated that only inhibition→inattention could be dropped without hurting the model fit. Based on the results from the Wald test, a reduced model (b') where the inhibition-inattention path coefficient was fixed to zero was created and tested against the original model (b), Disinhibition Model. A chi-square difference test indicated that this reduced model (b') was not significantly worse than the original model (b), χ^2 (1, N=102) = 0.21, $p > .10$. This revised Disinhibition Model (b'), a more parsimonious model, is presented in Figure 6.

This model shows that the path coefficient from working memory to inattention is significant. Table 6 also provides a comparison between the original Disinhibition Model [Model (b) in Figure 5.] and the revised Disinhibition Model [Model (b') in Figure 6] in terms of goodness-of-fit statistics. In fact, the BIC value and ECVI for the revised model was lower than for the original model, providing evidence of better fit for the revised model. Finally, it should be pointed out that this revised model is statistically equivalent with a path model where working memory has direct effects on inhibition and inattention and there is no direct relationship between disinhibition and inattention, which is presumed in the Working Memory Model that will be tested next.

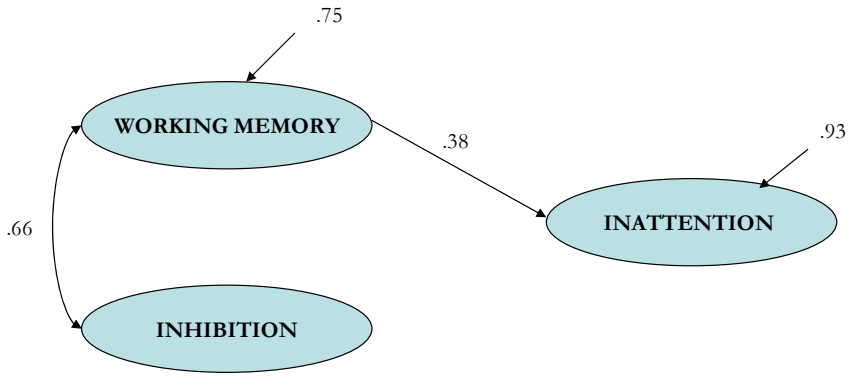

Figure 6. A Revised (Parsimonious) Disinhibition Model (b').

Table 6

Fit Indices for the Disinhibition Model and a More Parsimonious Revised Disinhibition Model

Model	χ^2/df	GFI	NNFI	CFI	RMSEA *(90% CI)*	BIC	ECVI *(90% CI)*
Original (b)	8.53/11	.98	1.05	1.0	0 (0-.08)	-42.34	.45(0-.56)
Revised (b')	8.74/12	.98	1.06	1.0	0 (0-.08)	-46.76	.43 (0-.53)

Note. No χ^2 values were significant.

Testing the Working Memory Model

Parameters for the Working Memory Model were estimated. The tested structural equation model for the Working Memory Model, along with standardized parameter

estimates, is presented in Figure 7. Note that, although for simplicity the tasks that were used to construct the latent variables are omitted in the figure, they are present in the models tested (see Figure 4).

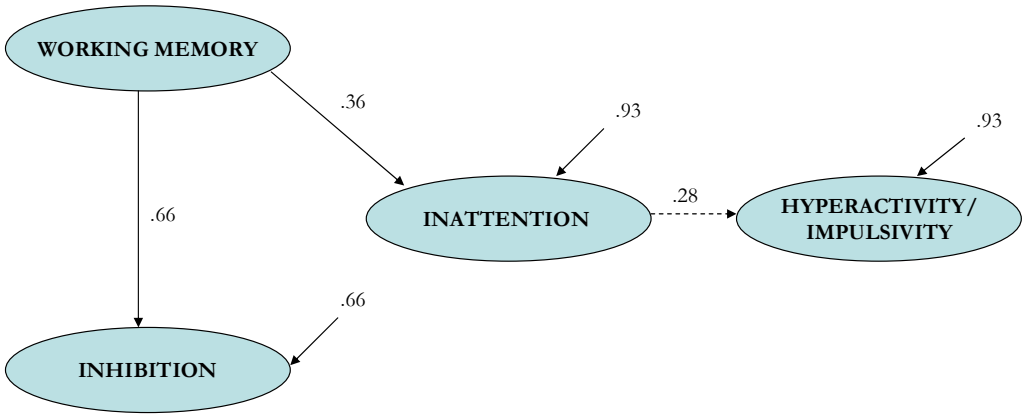

Figure 7. SEM for the Working Memory Model.

The fit indices indicated that this SEM model has a good fit to the data: nonsignficant χ^2 value, χ^2/df=19.62/24=.82, GFI=.96, NNFI=1.05, CFI=1.0, and RMSEA=.00 (0-.06). Path coefficients, except for inattention-H/I path, were significantly different from zero (all *ps* < .05). A Wald test estimated that this non-significant path could be dropped, but dropping the inattention-H/I path is acceptable only if it does not result in a significant increase in model chi-square (Hatcher, 1994). A reduced model where inattention-H/I path was fixed to zero resulted in a chi-square value of 24.13 with 25 *df.*

The chi-square difference test demonstrated a significant increase in chi-square, χ^2 (1, N=102) = 4.51, $p < .05$, meaning that dropping the inattention-H/I path significantly reduces the model fit with the data. The inattention-H/I path was therefore retained.

A Lagrange multiplier test also was used to see if there was any path that could be added to increase the model fit; there was no path to be added that is theoretically or logically justifiable. The distribution of asymptotically standardized residuals was approximately symmetrical and centered on zero, and no standardized residuals were large (all less than 1.99 in absolute value). Finally, a chi-square difference test was conducted to see if there was no significant difference between the fit of the structural model (Figure 7) and the measurement model (Figure 4), χ^2 (3, N=102) = 3.05, $p > .10$. This non-significant difference between the chi-square for the theoretical and measurement models shows that the theoretical model successfully accounts for the observed covariances between the latent variables in the structural portion of the model. Combined, these findings generally provide support for the Working Memory Model.

Inhibition or Working Memory: Which Is the Primary Deficit in ADHD?

As described in Chapters I and II, the Disinibition Model and the Working Memory Model are not incompatible. However, a major difference lies in the theoretical position of the relative role of working memory and inhibition in the manifestation of ADHD symptoms. Whereas the Disinhibition Model argues that the primary deficit of ADHD is in that of behavioral inhibition (i.e., disinhibition), the Working Memory Model posits that disinibition is a by-product of deficient working memory and working memory is in the core of myriad behavioral and cognitive characteristics associated with ADHD. Because

multiple measures of working memory and inhibition were included in this study, it appears that this theoretical question can be answered in part by examining correlations of these two latent variables with ADHD symptom variables and chi-square difference tests.

The correlations among the four latent variables can be found in Figure 4. As seen in Figure 4, neither working memory nor inhibition significantly correlates with H/I, meaning that the primary deficit hypothesis is not testable with H/I in this study. However, working memory significantly correlates with inhibition and inattention, while inhibition displays a non-significant correlation with inattention. Further, when the correlation between inhibition and inattention was fixed to zero in the 3-factor measurement model (working memory, inhibition, and inattention), the reduced model did not display a significant increase in chi-square value, $\Delta\chi^2$ (1, N=102) = 2.96, $p > .05$. In contrast, when the correlation between working memory and inattention was fixed to zero, the model fit decreases significantly, $\Delta\chi^2$ (1, N=102) = 6.05, $p < .05$. This result appears to be in favor of working memory against inhibition as a primary deficit. However, a more rigorous and accurate test would be to look at the amount of unique contribution of each variable to inattention. As seen in Figure 5, the direct effect of working memory on inattention is not significant when controlling for the effect of inhibition; likewise, the direct effect of inhibition on inattention is not significant either when the effect of working memory is controlled for. This result indicates a significant amount of shared variance between working memory and inhibition in predicting inattention and no convincing evidence of inhibition or working memory as a primary deficit.

The same conclusion is reached with a chi-square difference test. If inhibition is the primary deficit, the overall fit of the model in Figure 5 should be significantly decreased (i.e., increase in chi-square value) when the inhibition-inattention path is fixed to zero. However, as discussed in a previous section, the elimination of the inhibition-inattention path did not result in a significant change in chi-square value, χ^2 (1, N=102) = 0.21, p >.10. Likewise, if working memory is the primary deficit, the overall model fit should be significantly decreased when the working memory-inattention path is removed. Again, dropping the working memory path did not cause a significant decrease in the model fit, χ^2 (1, N=102) = 0.81, p > .10. Taken together, although working memory appears to be "primary" in that it shares more variance with inattention than inhibition does, more rigorous tests of the primary deficit hypotheses failed to provide convincing evidence of working memory as the primary deficit in ADHD. Thus, it appears reasonable that working memory as a primary deficit in ADHD is considered to be tentative at the most.

Testing the Integrated EF Model of ADHD and Its Comorbid Conditions

The Measurement Model: CFA

The first step to testing the Integrated EF Model is to create a measurement model (a CFA model) that contains six structural variables; working memory, inhibition, inattention, hyperactivity/impulsivity, reading difficulty, and substance abuse. This measurement model is equivalent to the previous measurement model displayed in Figure 4 except that two manifest variables – i.e., reading difficulty and substance abuse – are newly added. Please note that these two variables were measured with single indicators, resulting in what is called a "nonstandard" model (Hatcher, 1994).

A confirmatory factor analysis with the nonstandard model indicated a very good overall fit to the data as follows: nonsignificant χ^2 value, χ^2/df=26.12/31=.84, GFI=.96, NNFI=1.06, CFI=1.0, and RMSEA=.00 (0-.06). Because the estimated parameters including correlations among latent factors, factor loadings, and error terms are almost equivalent to those displayed in Figure 4, they are not presented again (also see Figure 7). The distribution of asymptotically standardized residuals was approximately symmetrical and no standardized residuals were considered to be large (all less than 2.42 in absolute value). Based on these supporting results, this measurement model was used as the basis for subsequent SEM analyses.

The Structural Model: The Integrated EF Model

The tested structural equation model for the Integrated EF Model, along with all standardized parameters estimated, is presented in Figure 8. The fit indices for this initial model indicated that this SEM model had a good fit to the data (see Table 7): nonsignficant χ^2 value, χ^2/df=33.97/39=.87, GFI=.94, NNFI=1.05, CFI=1.0, and RMSEA=.00 (0-.06). As shown in Figure 8, however, not all standardized path coefficients were statistically significant; four path coefficients were not significantly different from zero (*ps* > .05). Thus, these nonsignificant paths were further examined to see if they could be eliminated. The Wald test and the chi-square difference test were used for this purpose. The Wald test estimated that all these paths could be deleted without significantly hurting the model's fit. Only one parameter was fixed at zero at a time and then the model was re-estimated.

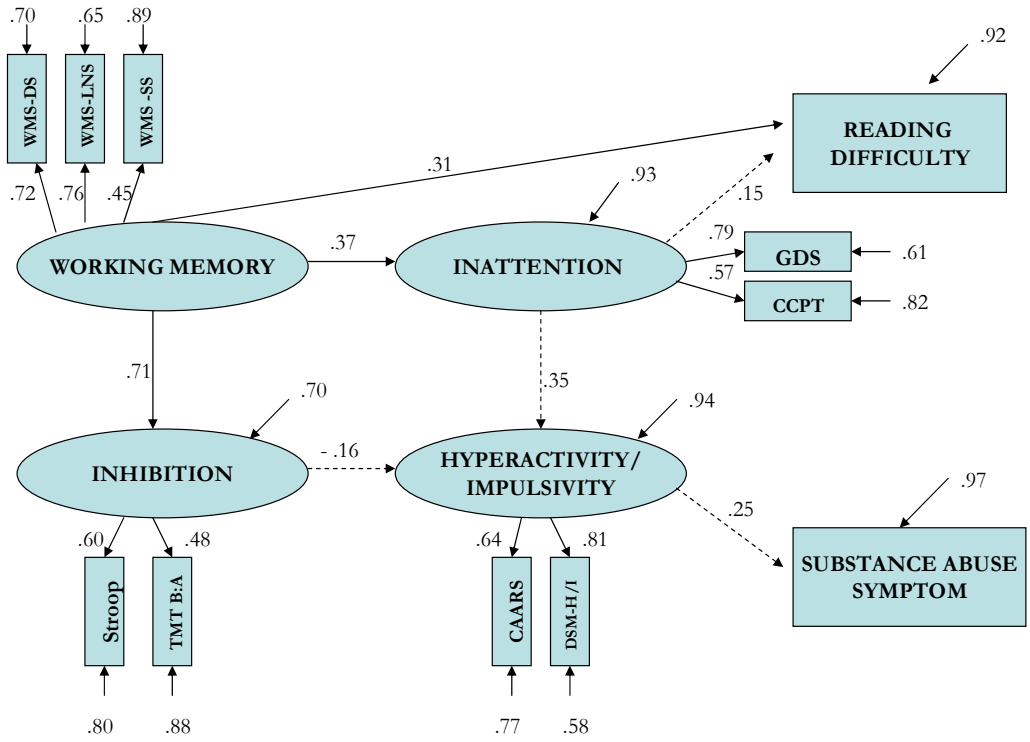

Figure 8. SEM for the Integrated EF Model of ADHD and Its Comorbid Conditions.

When the inhibition-H/I path was fixed at zero, the resulting χ^2 was 34.95 with *df*=40. A subsequent chi-square test indicated that dropping this path does not cause a significant decrease in model chi-square, χ^2 (1, *N*=102) = .068, $p > .10$. Therefore, this path was eliminated from the model. The same chi-square difference tests were used to determine if the other three paths indicated by the Wald test (i.e., inattention-reading difficulty, inattention-H/I, and H/I-substance abuse) should be dropped. The elimination of the inattention-reading difficulty path did not cause a significant decrease in model fit, χ^2 (1, *N*=102) = 1.45, $p > .10$. Thus, this path also was dropped from the model. Dropping these two paths was re-examined on theoretical and logical grounds. Although the

Integrated EF Model hypothesized that there were direct effects of inhibition (on H/I) and inattention (on reading difficulty), the elimination of these effects appears to be justifiable.

The next step was to fix the inattention-H/I path at zero; unlike the first two paths, the elimination of this path significantly affected the model fit, χ^2 (1, N=102) = 4.39, p<.05. Finally, when the H/I-substance abuse path was fixed at zero, the resulting chi-square value indicated that the elimination of this path significantly would hurt the model chi-square, χ^2 (1, N=102) = 4.63, p < .05. These two paths were therefore retained for the revised model. Figure 9 presents the revised SEM model for the integrated EF model. For simplicity, the measures that were used to construct the latent variables are not shown in the figure.

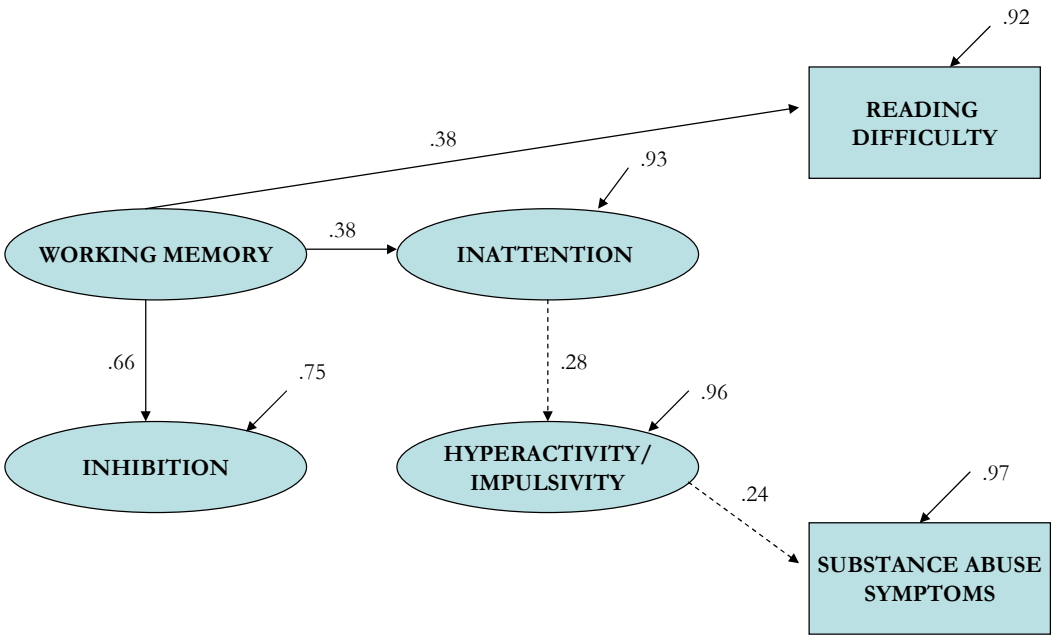

Figure 9. A More Parsimonious Integrative EF Model of ADHD and Its Comorbid Conditions. (Final Model)

The reduced SEM model (Figure 9) demonstrated a good fit to the data as indicated by overall fit indices (see Table 7): nonsignificant χ^2 value, χ^2/df=36.11/41=.88, GFI=.94, NNFI=1.05, CFI=1.0, and RMSEA=0 (0-.06). Asymptotically standardized residuals were distributed approximately symmetrically and centered on zero; no standardized residuals were considered large (all less than 2.56 in absolute value). Further, a chi-square difference test between the structural model (Figure 9) and the measurement model indicated no significant difference, χ^2 (10, N=102) = 9.99, $p > .10$, meaning that the revised integrated EF model was successful in accounting for the observed relations between the six structural variables. In short, these findings support the validity of the revised integrated EF model. Table 7 provides a comparison of fit indices between the original model and the revised model.

Table 7

Fit Indices for the Original Integrated Model vs. the Revised Integrated Model

Model	χ^2/df	GFI	NNFI	CFI	RMSEA *(90% CI)*	BIC	ECVI *(90% CI)*
Original	33.97/39	.94	1.05	1.0	0 (0-.06)	-146.40	.94 (0-1.12)
Revised	36.11/41	.94	1.05	1.0	0 (0-.06)	-153.52	.92 (0-1.10)

Note. No χ^2 values were significant.

Summary

Table 8 lists specific predictions made by the original integrated EF model and shows whether they have been supported by the revised integrated model.

Table 8

Specific Predictions Made by the Integrated EF Model and Their Support by the Data

Predictions	Support
• Working memory deficit leads to disinhibition.	Yes
• Working memory deficit directly contributes to inattention.	Yes
• Inhibition has a direct influence on H/I.	No
• Working memory deficit contributes to H/I, but this effect is mediated by inhibition.	No
• Working memory deficit contributes to H/I via inattention.	Yes
• Inattention and inhibition may be spuriously correlated due to their respective relationships with working memory	Yes
• Reading difficulty is primarily related to working memory deficit and inattention.	Yes (working memory); No (inattention)
• Substance abuse is primarily linked inhibition and hyperactivity.	No (inhibition); Yes (hyperactivity)
• Working memory deficit directly contributes to reading difficulty and inattention.	Yes
• Working memory also has an indirect effect on reading difficulty via inattention.	No
• Reading difficulty may be correlated with inhibition, but this correlation is generated by the same underlying working memory deficit.	Yes
• H/I has a direct effect on the development of substance abuse.	Yes
• The relation between inhibition and substance abuse is mediated by the presence of H/I.	No

CHAPTER V

SUMMARY AND DISCUSSION

The purpose of this study was to test executive function models of ADHD and its comorbid conditions using a sample of 102 adults. Two current executive function (EF) models of ADHD – i.e., the Disinhibition Model (Barkley, 1997) and the Working Memory Model (Rapport et al., 2001) – were evaluated to determine the extent to which they are supported by the data. Although there are some contradictory predictions between these two models, it was noted that these models are complementary as well in many aspects. An important limitation of current EF models is the lack of theoretical propositions that link ADHD to its comorbid conditions. Given that many individuals with ADHD develop one or more additional clinical problems, it is very important to address how EF and ADHD symptoms contribute to or interact with these coexisting disorders not only for the theoretical understanding of ADHD but for effective interventions with individuals with ADHD. Based on these rationales, this study presented an "integrated" model by combining current EF models of ADHD and linking them to current research findings on two common comorbid conditions with ADHD (i.e., reading difficulty and substance abuse). Like current EF models of ADHD, the Integrated Model was tested and revised in the light of data.

Methodologically, this study employed a latent variable approach including a series of CFA and SEM. A latent variable approach is considered well-suited to partially resolve measurement problems that have plagued EF researchers and investigate theoretical relationship between EF and ADHD and it comorbid conditions because the latent variable

analysis statistically extracts the common variance between the tasks chosen to tap a particular executive function, thereby separating the variance due to executive processes from the considerable variance due to nonexecutive task requirements and measurement errors. Major findings in this study are summarized and discussed as follows:

A CFA with the 4-factor measurement model of two EFs and two ADHD symptoms demonstrated a superior fit to the data. Although an alternative 3-factor model in which the working memory-inhibition correlation is fixed to 1 was also acceptable, a confidence interval test with the correlation between these two latent variables (r=.65) indicated that these EF constructs are not completely redundant; this finding suggests that inhibition and working memory should be considered as highly inter-related but separable constructs. This interpretation and the obtained magnitude of the correlation between inhibition and working memory are highly consistent with other CFA studies with adults (Miyake et al., 2000) and children (Lehto et al., 2003). For example, using different sets of measures for working memory and inhibition from this study, Lehto et al. (2003) obtained the correlation of .63 between two latent variables. Similarly, Miyake et al. (2000) reported a correlation of .63 between inhibition factor and "information updating and monitoring" factor (comparable to working memory) in their CFA study.

Because the Disinhibition Model (Barkley, 1997) is a "saturated" path model, it was not possible to test just the structural portion of the model for goodness of fit. Moreover, hyperactivity/impulsivity (H/I) symptoms did not significantly correlate with either working memory or inhibition in this study. However, it was evident that a more parsimonious model (Figure 6) in which a direct path from inhibition to inattention is dropped is statistically equivalent to (or no worse than) the original Barkley's Model (i.e., saturated

model). Whereas the original Disinhibition Model posits two direct paths from working memory and inhibition to inattention, this result implies that only one direct path (i.e., working memory – inattention) may be needed to explain inattention. In fact, this finding is consistent with the theoretical position of the Working Memory Model in which inhibition is considered as a byproduct of deficient working memory process. Estimated parameters for the Working Memory Model and the overall fit indexes indicated that the path model based on the Working Memory Model successfully accounted for the observed covariances between the latent variables in the structural portion of the model; the original Working Memory Model demonstrated a superior fit to the data without any revision. Although not directly comparable to each other, these results are more supportive of the working memory model than the disinhibition model.

In relation to current EF models of ADHD, this study attempted to determine whether inhibition or working memory is the primary deficit in ADHD. This inquiry is important given that EF deficits are not specific to ADHD; EF deficits are found in many other clinical disorders such as learning disability, antisocial behavior, autism, Tourette syndrome, Phenylketonuria (PKU), and traumatic brain injury. If EF is a multi-component concept, is there a more critical EF component underlying ADHD? In this study, neither working memory nor inhibition significantly correlated with the H/I symptoms; thus, it was not possible to test the primary deficit hypothesis with H/I. However, while inhibition showed a non-significant correlation with inattention, working memory did significantly correlate with inattention. Similarly, while the model chi-square did not significantly change when the inhibition-inattention correlation was fixed to zero, the model chi-square significantly increased when the correlation between working memory and inattention was

fixed to zero. These findings imply that working memory is more important than inhibition in explaining inattention. However, it also should be noted that neither of these two latent variables demonstrated a unique contribution to inattention after controlling for the other latent variable (i.e., nonsignificant path coefficients). Moreover, chi-square difference tests indicated no significant change in the model fit when eliminating the unique contribution of either working memory or inhibition. In short, whereas working memory shares more variance with inattention than inhibition does, more rigorous tests provided no definite evidence that working memory is primary.

Therefore, working memory deficit should be considered as the primary deficit only tentatively based the results from this study. On the one hand, these favorable findings for working memory are convergent with a previously reported study (Lee, Riccio, & Hynd, in press) in which only the working memory composite score had a unique contribution to the teacher-rated inattention symptoms over and above the effect of inhibition. On the other hand, current findings appear to be conflictive with the currently prevailing conceptualization of ADHD as a disinhibitory disorder (Barkley, 1997; Quay, 1997). Recently, Nigg (2001) argued that the definition of inhibition has been imprecisely distinguished across theories. Further, Nigg (2001) suggested that only certain types of inhibition (e.g., executive motor inhibition) might account for the core deficit of ADHD, particularly combined type ADHD. Given that the two indicators of inhibition in this study (i.e., Stroop and TMT B to A ratio) are considered as measures of cognitive inhibition or interference control, it would be reasonable to conclude based on current findings that working memory is primary only in comparison to "cognitive" inhibition in ADHD.

The non-standard CFA model for the Integrated EF Model in which two constructs were measured with single manifest variables and four constructs with multiple manifest variables demonstrated a very good overall fit to the data, suggesting that this model can be used as the measurement model against which the structural model (Figure 3) would be compared. The original Integrated EF Model of ADHD and Comorbid Conditions (Figure 3) indicated a very good overall fit to the data. However, the original model had multiple non-significant paths and thus was "trimmed" in the light of theoretical/logical as well as statistical criteria. These model trimming procedures led to a more parsimonious integrated model that is theoretically sound and statistically well-fit to the data. This revised model was presented as the final Integrated EF Model of ADHD and Its Comorbid Conditions (Figure 9).

According to this final model, working memory is the underlying deficit of inattention and disinhibition, consistent with the prediction of the Working Memory Model. Although the direct effect of inattention on reading difficulty was not supported, working memory problems directly contributed to reading difficulty as well as inattention symptoms. In other words, working memory deficits would be the underlying deficit that is common to inattention and reading difficulty. This finding provides an explanation for why many individuals with ADHD also have comorbid reading problems. This is also consistent with research findings that particularly inattentive features of ADHD are related to comorbid learning disorders in children (e.g., Eiraldi et al., 1997; Willcutt & Pennington, 2000). Further, this finding also bridges studies evidencing working memory deficit in ADHD (e.g., Barnett et al., 2001; Dietlein, 2001; Lee et al., in press; Sarkari, 2003; Stevens et al., 2002) to

those demonstrating the same working memory deficit in learning disabilities (e.g., Isaki & Plante, 1997; Swanson, 1994; Willcutt et al., 2001)

The direct contribution of inhibition to hyperactivity/impulsivity (H/I) symptoms was not evidenced in this study. Instead, working memory deficit indirectly contributed to H/I through inattention, but not through disinhibition. Again, this pattern of relationship is consistent with the Working Memory Model but not with the Disinhibition Model. According to the final integrated model, only H/I symptoms, but not inattention, predict substance abuse symptoms.

There was no significant direct or indirect effect of inhibition on substance abuse symptoms. Instead, inattention had an indirect effect on substance abuse via H/I symptoms. Although ADHD symptoms and substance abuse symptoms were measured concurrently in this study, significant direct and indirect effects of ADHD symptoms on substance abuse suggest that ADHD in adulthood may increase the risk of substance use disorders. However, this interpretation should be taken as preliminary because the etiological role of ADHD in the development of substance abuse appears to be dependent on other mediating or moderating variables such as presence of comorbid antisocial behavior/conduct disorder, gender, and ADHD subtypes (Lynskey & Hall, 2001; Modestine, Matutat, & Wurmle, 2001). It should be noted that this study did not take into account any of these variables; it is plausible that the correlation between H/I and substance abuse might have been reduced in this study by these variables.

In summary, this study provided a strong support for the Working Memory Model. The Disinhibition Model was supported to a lesser degree; a direct causal path from inhibition to inattention posited by this model was not needed once a direct path from

working memory to inattention was considered. A more parsimonious disinhibition model was equivalent to or no worse than the original model. Working memory as a primary deficit in ADHD was supported in this study, although this conclusion should be taken as preliminary. The Integrated EF model of ADHD and Its Comorbid Conditions also demonstrated a very good fit to the data. The final Integrated Model explains how inhibition and working memory are related to ADHD symptoms and how EF deficits and ADHD symptoms contribute to the development of comorbid problems with ADHD such as reading difficulty and substance abuse.

This study has strengths. First, this study is one of very few studies that employed a latent variable approach with multiple executive function measures to examine the relationship of executive function to ADHD. Moreover, this study is one of the pioneering attempts to test current executive function models of ADHD and expand these models by further addressing the relationships with comorbid conditions with ADHD. Finally, it should be noted that although inhibition and working memory have been indicated as underlying deficits of ADHD in the literature, this study is one of the first studies that examined the "relative" importance of working memory and inhibition in ADHD.

This study also has limitations and implications. A major limitation of this study is that the sample size (N=102) for this study was very small considering that SEM is inherently a large sample technique (Kline, 1998; Thompson, 2000). As the ratio of the number of cases to the number of parameters is smaller, the statistical stability of the estimates becomes more doubtful (Kline, 1998). As a rule of thumb, when the number of cases to the number of parameters ratio is less than 10:1, there may be a cause for concern (Kline, 1998). However, in a recent Monte Carlo simulation study, Jackson (2001) found

that, in the context of maximum likelihood confirmatory factor analysis, the number of observations per estimated parameter does *not* account for an appreciable amount of the variation in parameter estimates and values of summary fit indexes beyond what is explained by the effects of sample size, indicator reliability, and the number of indicators per factor. Mueller (1997) suggested that the ratio of the number of people to the number of measured (observed) variables should be at least 10:1. This study has a total of 11 observed variables for the integrated EF model; then, the sample size still falls short of, but is close to, the minimum number of people in terms of sample size. It is often difficult to get a large sample when data collection involves long individual testing time. Current CFA and SEM results based on a small sample should be considered as preliminary and should be replicated in larger samples in future studies for generalization.

Another limitation is that this study employed only two indicators to construct each latent variable except for working memory. Particularly, for inhibition, only two indicators that are tapping a certain type of inhibition (i.e., cognitive inhibition or interference control) were used. Given that other types of inhibition (i.e., motor and response inhibition) are more consistently found deficient in individuals with ADHD, results obtained in this study regarding the role of inhibition should be interpreted only in relation to cognitive inhibition and the future study should examine how other types of inhibition are related to ADHD. Current findings on the relative importance of working memory versus inhibition also should be interpreted with this limitation in mind. In short, although the latent variable analysis reduces measurement error and minimizes the effects of idiosyncratic aspects of individual tasks, the generalizability of the current results to a different set of tasks remains to be seen in future studies.

This study aimed to test current EF models of ADHD – i.e., Disinhibition Model and Working Memory Model; however, it should be emphasized that this study was not intended to test all aspects of these two models. Specifically, the Disinhibition Model is very comprehensive in its scope. It also should be remembered that while the Disinhibition Model applies only to ADHD-Combined type, this study made no distinction between ADHD subtypes under the assumption that inattention problems experienced by individuals with different subtypes are essentially homogenous. A recent finding (Collings, 2003) that a sustained attention deficit measured by CPT is found in ADHD-Combined type, but not in ADHD-Inattentive type, suggests that the subtype distinction should be used for a more rigorous testing of the Disinhibition Model in future studies.

While the final Integrated EF Model of ADHD and Its Comorbid Conditions demonstrated a very good fit to the data, there is a room for further expansion. For example, antisocial behavior/conduct disorder was not included in the model. Moreover, possible moderating or mediating role of antisocial behavior in the relationship between ADHD and substance abuse are not specified in the final model.

In conclusion, current findings on the relationship and relative importance of working memory and inhibition in ADHD and comorbid conditions (as summarized by the integrated EF model) provide a unified account how executive function deficits are related to the manifestation of ADHD symptoms and comorbid conditions with ADHD. However, given the complexity of the model and the small sample size in this study, current findings should be replicated and further examined with different samples including children with ADHD.

REFERENCES

American Psychiatric Association (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

Anderson, J. C., & Gerbing, D. W. (1988). Structural equation modeling in practice: A review and recommended two-step approach. *Psychological Bulletin, 103*, 411-423.

Arbuthnott, K., & Frank, J. (2000). Trail Making Test, Part B as a measure of executive control: Validation using a set-switching paradigm. *Journal of Clinical and Experimental Neuropsychology, 22*, 518-528.

Barkley, R. A. (1997). *ADHD and the nature of self-control.* New York: Guilford.

Barkley, R. A. (1998). *It is about time.* Unpublished manuscript, University of Massachusetts Medical Center.

Barkley, R. A. (2000). Genetics of childhood disorders: XVII. ADHD, Part 1: The executive functions and ADHD. *Journal of the American Academy of Child and Adolescent Psychiatry, 39,* 1064-1068.

Barnett, R., Maruff, P., Vance, A., Luk, E. S. L., Costin, J., Wood, C., & Pantelis, C. (2001). Abnormal execution function in attention deficit hyperactivity disorder: The effect of stimulant medication and age on spatial working memory. *Psychological Medicine, 31,* 1107-1115.

Beck, A. T., Steer, R. A., & Brown, G. K. (1996). *BDI-II manual.* San Antonio, TX: Psychological Corporation.

Belmont, J. M. (1978). Individual differences in memory: The cases of normal and retarded development. In M. M. Gruneberg & P. Morris (Eds.), *Aspects of memory* (pp. 153-185). London: Methuen.

Bender, W. D. (1997). *Understanding ADHD: A practical guide for teachers and parents*. Columbus, OH: Prentice Hall.

Bentler, P. M. (1990). Comparative fit indexes in structural models. *Psychological Bulletin*, *107*, 238-246.

Bentler, P. M., & Bonett, D. G. (1980). Significance tests and goodness of fit in the analysis of covariance structures. *Psychological Bulletin, 88,* 588-606.

Benton, A. L., & Hamsher, K. deS. (1978). *Manual for the multilingual aphasia examination* (Rev. ed.). Iowa City, IA: University of Iowa.

Benton, A. L., Hamsher, K. deS., & Sivan, A. B. (1994). *Multilingual aphasia examination: A manual of instruction* (3rd ed.). Iowa City, IA: AJA Associates, Inc.

Biederman, J., Wilens, T. E., & Mick E. (1997). Is ADHD a risk factor for psychoactive substance use disorders? Findings from a four-year prospective follow-up study. *Journal of the American Academy of Child and Adolescent Psychiatry, 36*, 21-29.

Biederman, J., Wilens, T., Mick, E., Spencer, T., & Faraone, S. V. (1999). Pharmacotherapy of attention-deficit/hyperactivity disorder reduces risk for substance use disorder. *Pediatrics, 104*. e20.

Bollen, K. A. (1989). *Structural equations with latent variables*. New York: Wiley.

Borkowski, J. G., & Burke, J. E. (1996). Theories, models, and measurement of executive functioning: An information processing perspective. In G. R. Lyon, & N. A.

Krasnegor (Eds.), *Attention, memory, and executive function* (pp. 235-262). Baltimore, MD: Paul H. Brookes.

Browne, M. W., & Cudeck, R. (1989). Single sample cross-validation indices for covariance structures. *Multivariate Behavioral Research, 24,* 445-455.

Browne, M. W., & Cudeck, R. (1993). Alternative ways of assessing model fit. In K. A. Bollen & J. S. Long (Eds.), *Testing structural equation models* (pp. 136-162). Newbury Park, CA: Sage.

Burns, G. L, Walsh, J. A., Patterson, D. R., Holte, C. S., Sommers-Flanagan, R., & Parker, C. M. (1997a). Internal validity of the disruptive behavior disorder symptoms: Implications from parents ratings for a dimensional approach to symptom validity. *Journal of Abnormal Child Psychology, 25,* 307-320.

Burns, G. L., Walsh, J. A., Owen, S. M., & Snell, J. (1997b). The internal validity of the DSM-III-R and DSM-IV disruptive behavior disorder symptoms: Implications from teacher ratings for a dimensional approach to symptom validity. *Journal of Child Clinical Psychology, 26,* 266-275.

Butterfield, E. C., & Albertson, L. R. (1995). On making cognitive theory more general and developmentally pertinent. In F. Weinert & W. Schneider (Eds.), *Research on memory development* (pp. 73-99). Hillsdale, NJ: Lawrence Erlbaum Associates.

Byrne, B. M. (1989). *A primer of LISREL: Basic applications and programming for confirmatory factor analytic models*. New York: Springer Verlag.

Cabeza, R., & Nyberg, L. (1997). Imaging cognition: An empirical review of PET studies with normal subjects. *Journal of Cognitive Neuroscience, 9*, 1-26.

Chhabildas, N., Pennington, B. F., & Willcutt, E. G. (2001). A comparison of the neuropsychological profiles of the DSM-IV subtypes of ADHD. *Journal of Abnormal Child Psychology, 29*, 529-540.

Cohen, J. D., & Servan-Schreiber, D. (1992). Context, cortex, and dopamine: A connectionist approach to behavior and biology in schizophrenia. *Psychological Review, 99,* 45-77.

Cohen, M. J., Riccio, C. A., Kibby, M. Y., & Edmonds, J. E. (2000). Developmental progression of clock face drawing in children. *Child Neuropsychology, 6*, 64-76.

Collings, R. D. (2003). Differences between ADHD inattentive and combined types on the CPT. *Journal of Psychopathology and Behavioral Assessment, 25*, 177-189.

Conners, C. K. (1999). *The Conners Continuous Performance Test - II*. North Tonawanda, NY: Multi-Health Systems.

Conners, C. K., Erhardt, D., & Sparrow, E. (1997). *Conners Adult ADHD Rating Scales (CAARS)*. North Tonawanda, NY: Multi-Health Systems, Inc.

Conners, C. K., Sitarenios, G., Parker, J. D., & Epstein, H. N. (1998a). Revision and restandardization of the Conners Parent Rating Scale-R (CPRS-R): Factor structure, reliability, and criterion validity, *Journal of Abnormal Child Psychology, 26*, 257-268.

Conners, C. K., Sitarenios, G., Parker, J. D., & Epstein, H. N. (1998b). Revision and restandardization of the Conners Teacher Rating Scale-R (CTRS-R): Factor structure, reliability, and criterion validity, *Journal of Abnormal Child Psychology, 26*, 279-291.

Corbett, B., & Stanczak, D. E. (1999). Neuropsychological performance of adults evidencing attention-deficit hyperactivity disorder. *Archives of Clinical Neuropsychology, 14,* 373-387.

Cullbertson, W. C., & Zillmer, E. A. (2000). *Tower of London, Drexel University, research version: Examiner's manual.* North Tonawanda, NY: Multi-Health Systems, Inc.

de Jong, P. F. (1998). Working memory deficits of reading disabled children. *Journal of Experimental Child Psychology, 70,* 75-96.

Dehaene, S. & Changeux, J. P. (1991). The Wisconsin Card Sorting Test: Theoretical analysis and modeling in a neuronal network, *Cerebral Cortex, 1*, 62-79.

Denckla, M. B. (1996). A theory and model of executive function: A neuropsychological perspective. In G. R. Lyon, & N. A. Krasnegor (Eds.), *Attention, memory, and executive function* (pp. 263-278). Baltimore, MD: Paul H. Brookes.

Denny, C. B., & Rapport, M. D. (2000). Cognitive pharmacology of stimulant drugs in AD/HD. In M. Solanto, A. F. T. Arnsten & F. X. Castellanos (Eds.), *Stimulant drugs and ADHD: Basic and clinical neuroscience* (pp. 283-302). New York; Oxford University Press.

Diamond, A. (1991). Guidelines for the study of brain-behavior relationships during development. In H. S. Levin, H. M., Eisenberg, & A. L. Benton. (Eds.), *Frontal lobe function and dysfunction* (pp. 339-378). New York: Oxford University Press.

Dietlein, N. R. (2001). Working memory and disinhibition in children with ADHD. *Dissertation Abstracts International: Section B: The Sciences & Engineering, 62,* 1074.

Dunn, L. M., & Dunn, L. M. (1997). *Peabody Picture Vocabulary Test-Third Edition (PPVT-III).* Circle Pines, MN: American Guidance Services, Inc.

DuPaul, G. J., Anastopoulos, A. D., McGoey, K. E., Power, T. J., Reid, R., & Ikeda, M. J. (1997). Teacher ratings of attention deficit hyperactivity disorder symptoms: Factor structure and normative data. *Psychological Assessment, 9,* 436-44.

DuPaul, G. J. Anastopoulos, A. D., Power, T. J., Reid, R., Ikeda, M. J., & McGoey, K. E. (1998). Parent ratings of attention deficit/hyperactivity disorder symptoms: Factor structure of normative data. *Journal of Psychopathology and Behavioral Assessment, 20,* 83-102.

DuPaul, G. J., Anastopoulos, A. D., Shelton, T. L., Guevremont, D. C., & Metevia, L. (1992). Multimethod assessment of attention deficit hyperactivity disorder: The diagnostic utility of clinic-based tests. *Journal of Clinical Child Psychology, 21*, 394-402.

Eiraldi, R. B., Power, T. J., & Nezu, C. M. (1997). Patterns of comorbidity associated with subtypes of attention deficit hyperactivity disorder among 6-to 12-year-old children. *Journal of the American Academy of Child and Adolescent Psychiatry, 36,* 503-514.

Epstein, J. N., Conners, C. K., Sitarenios, G., & Erhardt, D. (1998). Continuous performance test results of adults with attention deficit hyperactivity disorder. *The Clinical Neuropsychologist, 12*, 155-168.

Eslinger, P. J. (1996). Conceptualizing, describing, and measuring components of executive function: A summary. In G. R. Lyon, & N. A. Krasnegor (Eds.), *Attention, memory, and executive function* (pp. 367-396). Baltimore, MD: Paul H. Brookes.

First, M. B., Gibbon, M., Williams, J. B.W., & Spitzer, R. L. (1997). *SCID Screen Patient Questionnaire – Extended Version*. North Tonawada, NY: Multi-Health Systems, Inc.

Fuster, J. M. (1989). *The prefrontal cortex*. New York: Raven Press.

Gadow, K. D., Nolan, E. E., Litcher, L., Carlson, G. A., Panina, N., Golovakha, E., Sprafkin, J., & Broment, E. J. (2000). Comparison of attention deficit hyperactivity disorder symptoms subtypes in Ukrainian schoolchildren. *Journal of the American Academy of Child and Adolescent Psychiatry, 39,* 1520-1527.

Golden, C. J. (1978). *The Stroop Color and Word Test.* Chicago, IL: Stoelting.

Golden, C. J., & Freshwater, S. M. (2002). *The Stoop Color and Word Test: A manual for clinical and experimental uses.* Wood Dale, IL: Stoelting.

Gordon, M. (1983). *The Gordon Diagnostic System*. DeWitt, NY: Gordon Systems.

Gordon, M., McClure, F. D., & Post, E. M. (1986). *Interpretive guide to the Gordon Diagnostic System.* DeWitt, NY: Gordon Systems.

Hanford, R. B. (2001). Working memory and nonverbal affect recognition: Differentiation among ADHD subtypes in adults. *Dissertation Abstracts International: Section B: The Sciences & Engineering, 61*, 5564.

Hatcher, L. (1994). *A step-by-step approach to using the SAS system for factor analysis and structural equation modeling.* Cary, NC: SAS Institute Inc.

Hayes, S. C., Gifford, E. V., & Ruckstuhl, L. E. (1996). Relational frame theory and executive function: A behavioral approach. In G. R. Lyon, & N. A. Krasnegor (Eds.), *Attention, memory, and executive function* (pp. 279-305). Baltimore, MD: Paul H. Brookes.

Heaton, R. K., Chelune, G. J., Talley, J. L., Kay, G. G., & Curtiss, G. (1993). *Wisconsin Card Sorting Test: Revised and Expanded.* Odessa, FL: Psychological Assessment Resources.

Hitch, G. J. & McAuley, E. (1991). Working memory in children with specific arithmetical learning difficulties. *British Journal of Psychology, 82,* 375-386.

Houghton, S., Douglas, G., West, J., Whiting, K., Wall, M., Langsford, S., Powell, L., & Caroll, A. (1999). Differential patterns of executive function in children with attention deficit hyperactivity disorder according to gender and subtype. *Journal of Child Neurology, 14,* 801-805.

Hoyle, R. H., & Panter, A. T. (1995). Writing about structural equation models. In R. H. Hoyle (Ed.), *Structural equation modeling: Concepts, issues, and applications* (pp. 158-176). Thousand Oaks, CA: Sage.

Hu, L., & Bentler, P. M. (1995). Evaluating model fit. In R. H. Hoyle (Ed.), *Structural equation modeling: Concepts, issues, and applications* (pp. 76-99). Thousand Oaks, CA: Sage.

Isaki, E., & Plante, E. (1997). Short-term and working memory differences in language/learning disabled and normal adults. *Journal of Communication Disorders, 30,* 427-438.

Jackson, D. L. (2001). Sample size and number of parameter estimates in maximum likelihood confirmatory factor analysis: A Monte Carlo investigation. *Structural Equation Modeling, 8,* 205-223.

Johnson, D. E., Epstein, J. N., Waid, L. R., Latham, P. K., Voronin, K. E., & Anton, R. F. (2001). Neuropsychological performance deficits in adults with attention deficit/hyperactivity disorder. *Archives of Clinical Neuropsychology, 16*, 587-604.

Joreskog, K.G., & Sorbom, D. (1981). *LISREL V: Analysis of linear structural relationships by the method of maximum likelihood.* Chicago: National Educational Resources.

Kimberg, D, Y., & Farah, M. J. (1993). A unified account of cognitive impairments following frontal lobe damage: The role of working memory in complex, organized behavior. *Journal of Experimental Psychology: General, 122*, 411-428.

Kline, R. B. (1998). *Principles and practice of structural equation modeling*. New York: Guilford.

Lahey, B. B., & Willcutt, E. G. (1998, November). *Validity of the diagnosis and dimensions of attention-deficit/hyperactivity disorder*. Paper presented at the National Institutes of Health Consensus Development Conference on the Diagnosis and Treatment of Attention-Deficit/Hyperactivity Disorder, Washington, DC.

Lahey, B. B., Pelham, W. E., Stein, M. A., Loney, J., Trapani, C., Nugent, K., Kipp, H., Schmidt, E., Lee, S., Cale, M., Gold, E., Hartung, C. M., Willcutt, E. G., & Baumann, B. (1998). Validity of DSM-IV attention deficit hyperactivity disorder for young children. *Journal of the American Academy of Child and Adolescent Psychiatry, 37,* 695-702.

Lee, D., Riccio, C. A., & Hynd, G. W. (in press). The role of executive functions in attention deficit hyperactivity disorder: Testing predictions from two models. *Canadian Journal of School Psychology*.

Lehto, J. E., Juujarvi, P., Kooistra, L., & Pulkkinen, L. (2003). Dimensions of executive functioning: Evidence from children. *British Journal of Developmental Psychology, 21*, 59-80.

Levine, D. H., & Prueitt, P.S. (1989). Modeling some effects of frontal lobe damage: Novelty and perseveration. *Neural Networks, 2,* 103-116.

Lezak, M. D. (1995). *Neuropsychological assessment* (3rd ed.). New York: Oxford University Press.

Lovejoy, D. W., Ball, J. D., & Keats, M. (1999). Neuropsychological performance of adults with attention deficit hyperactivity disorder (ADHD): Diagnostic classification estimates for measures of frontal lobe/executive functioning. *Journal of the International Neuropsychology Society, 5*, 222-233.

Lynskey, M. T. & Hall, W. (2001). Attention deficit hyperactivity disorder and substance use disorders: Is there a causal link? *Addiction, 96,* 815-822.

Lyon, G. R., & Krasnegor, N. A. (Eds.). (1996). *Attention, memory, and executive function.* Baltimore, MD: Paul H. Brookes.

MacCallum, R. C. & Austin, J. T. (2000). Applications of structural equation modeling in psychological research. *Annual Review of Psychology, 51*, 201-226.

Mannuzza, S., Klein, R., Bessler, A., Malloy, P., & LaPadula, M. (1993). Adult outcome of hyperactive boys: Educational achievement, occupational rank, and psychiatric status. *Archives of General Psychiatry, 50*, 565-576.

Mardia, K. V. (1970). Measures of multivariate skewness and kurtosis with applications. *British Journal of Mathematical and Statistical Psychology, 28*, 205-214.

Marks, D. J., Newcorn, J. H., & Halperin, J. M. (2001). Comorbidity in adults with attention deficits hyperactivity disorder. In J. Wasserstein, L. E. Wolf, & F. F. Lefever (Eds.), *Adult attention deficit disorder: Brain mechanisms and life outcomes* (pp. 216-238). New York, NY: New York Academy of Sciences.

Mather, N., & Woodcock, R. W. (2001). *Examiner's manual: Woodcock-Johnson III Tests of Achievement.* Itasca, IL: Riverside Publishing.

Miller, F. G., & Lazowski, L. E. (1999). *The SASSI-3 manual.* Springville, IN: The SASSI Institute.

Miller, G. F., Roberts, J., Brooks, M. K., & Lazowski, L. E. (1999). *SASSI-3 user's guide: A quick reference for administration and scoring.* Bloomington, IN: Baugh Enterprises Inc.

Miyake, A., Friedman, N. P., Emerson, M. J., Witzki, A. H., Howerter, A., & Wager, T. D. (2000). The unity and diversity of executive functions and their contributions to

complex "frontal lobe" tasks: A latent variable analysis. *Cognitive Psychology, 41*, 49-100.

Miyake, A., Friedman, N. P., Rettinger, D. A., Shah, P., & Hegarty, M. (2001). How are visuospatial working memory, executive functioning and spatial abilities related? A latent variable analysis. *Journal of Experimental Psychology: General, 130*, 621-640.

Modestin, J. Matutat, B., & Wurmle, O. (2001). Antecedents of opioid dependence and personality disorder: attention-deficit/hyperactivity disorder and conduct disorder. *European Archives of Psychiatry and Clinical Neuroscience, 21*, 42-47.

Morgan, A. B., & Lilienfeld, S. O. (2000). A meta-analytic review of the relation between antisocial behavior and neuropsychological measures of executive function. *Clinical Psychology Review, 20,* 113-136.

Morgan, A. E., Hynd, G. W., Riccio, C. A., & Hall, J. (1996). Validity of DSM-IV ADHD predominantly inattentive and combined types: Relationship to previous DSM diagnoses/subtype differences. *Journal of the American Academy of Child and Adolescent Psychiatry, 35,* 325-333.

Morris, R. D. (1996). Relationships and distinctions among the concepts of attention, memory, and executive function: A developmental perspective. In G. R. Lyon, & N. A. Krasnegor (Eds.), *Attention, memory, and executive function* (pp. 11-16). Baltimore, MD: Paul H. Brookes.

Mueller, R. O. (1997). Structural equation modeling: Back to basics. *Structural Equation Modeling, 4,* 353-369.

Nigg, J. T. (2001). Is ADHD a disinhibitory disorder*? Psychological Bulletin, 127,* 571-598.

Oosterlaan, J., Logan, G. D., & Sergeant, J. A. (1998). Response inhibition in AD/HD, CD, comorbid AD/HD + CD, anxious, and control children: A meta-analysis of studies with the stop task. *Journal of Child Psychology and Psychiatry, 39*, 411-425.

Ozonoff, S. (1997). Components of executive function in autism and other disorders. In J. Russell (Ed.), *Autism as an executive disorder* (pp.179-214). New York, NY: Oxford University Press.

Passolunghi, M. C., & Siegel, L. S. (2001). Short-term memory, working memory, and inhibitory control in children with difficulties in arithmetic problem solving. *Journal of Experimental Child Psychology, 80,* 44-57.

Pennington, B. F. (1994). The working memory function of the prefrontal cortices: Implications for developmental and individual differences. In M. M. Haith, J. B. Bension, R. Roberts, & B. F. Pennington (Eds.), *The development of future-oriented processes* (pp. 248-289). Chicago: U. of Chicago Press.

Pennington, B. F. (1997). Dimensions of executive functions in normal and abnormal development. In N. A. Krasnegor, G. R. Lyon, & P. S. Goldman-Rakic (Eds.), *Development of the prefrontal cortex: Evolution, neurobiology, and behavior* (pp. 265-281). Baltimore, MD: Paul H. Brookes.

Pennington, B. F., & Ozonoff, S. (1996). Executive functions and developmental psychopathology. *Journal of Child Psychology and Psychiatry, 37,* 51-87.

Pennington, B. F., Bennetto, L, McAleer, O., & Roberts, R. J. (1996). Executive functions and working memory; theoretical and measurement issues. In G. R. Lyon and N. A. Krasnegor (Eds.), *Attention, memory, and executive function* (pp. 327-348). Baltimore, MD: Paul H. Brookes.

Pluth, S. D. (2001). The impaired responding theory of AD/HD: An examination of learning and memory function in adults with symptoms of AD/HD. *Dissertation Abstracts International: Section B: The Sciences & Engineering, 61 (8-B),* 4423.

Posner, M. I., & DiGirolamo, G. J. (1998). Executive attention: Conflict, target detection, and cognitive control. In R. Parasuraman (Ed.), *The attentive brain* (pp. 401-423). Cambridge, MA: MIT Press.

Psychological Corporation. (1997). *The WAIS-III/WMS-III technical manual.* San Antonio, TX: Author.

Quay, H. C. (1997). Inhibition and attention deficit hyperactivity disorder. *Journal of Abnormal Child Psychology, 25,* 7-13.

Raftery, A. E. (1993). Bayseian model selection in structural equation models. In K. A. Bollen & J. S. Long (Eds.), *Testing structural equation models* (pp. 163-180). Newbury Park, CA: Sage.

Rapport, M. D., Chung, K-M., Shore, G., & Isaacs, P. (2001). A conceptual model of child psychopathology: Implications for understanding attention deficit hyperactivity disorder and treatment efficacy. *Journal of Clinical Child Psychology, 30,* 48-58.

Reitan, R. M. (1992). *Trail Making Test: Manual for administration and scoring.* Tucson, AZ: Reitan Neuropsychological Laboratory.

Reynolds, C. R. (1997). Forward and backward memory span should not be combined for clinical analysis. *Archives of Clinical Neuropsychology, 12*, 29-40.

Reynolds, C. R., Bigler, E., & Riccio, C. A. (2004). *Comprehensive Complex Figure Test.* Unpublished manuscript.

Riccio, C. A., & Reynolds, C. R. (2001). Continuous performance tests are sensitive to ADHD in adults but lack specificity: A review and critique for differential diagnosis. In J. Wasserstein, L. E. Wolf, & F. F. Lefever (Eds.), *Adult attention deficit disorder: Brain mechanisms and life outcomes* (pp. 113-139). New York, NY: New York Academy of Sciences.

Roberts, R. J. & Pennington, B. F. (1996). An interactive framework for examining prefrontal cognitive processes. *Developmental Neuropsychology, 12,* 105-126.

Roberts, R. J., Hager, L. D. & Heron, C. (1994). Prefrontal cognitive processes: Working memory and inhibition in the antisaccade task. *Journal of Experimental Psychology: General, 123*, 374-393.

Robertson, I. H., Ward, T., Ridgeway, V., & Nimmo-Smith, I. (1996). The structure of normal human attention: The test of everyday attention. *Journal of the International Neuropsychological Society, 2,* 525-534.

Rosca-Rebaudengo, P., Durst, R., & Dickman, M. (2000). Adult attention deficit hyperactivity disorder and comorbidity. *International Journal of Psychiatry in Clinical Practice, 4,* 35-39.

Sarkari, S. (2003). Do verbal working memory and reconstitution differentiate children with AD/HD hyperactive-impulsive/combined type from children with AD/HD predominantly inattentive type and controls? *Dissertation Abstracts International: Section B: The Sciences & Engineering, 63*, 3483.

SAS Institute (1999). *SAS/STAT User's Guide, Version 8*. Gary, NC: SAS Institute Inc.

Satterfield, J. H., & Schell, A. (1997). A prospective study of hyperactive boys with conduct problems and normal boys: adolescent and adult criminality. *Journal of American Academy of Child and Adolescent Psychiatry, 36,* 1726-1735.

Schwarz, G. (1978). Estimating the dimension of a model, *Annals of Statistics, 6,* 461-464.

Siegel, L. S. & Linder, A. (1984). Short-term memory processes in children with reading and arithmetic disabilities. *Developmental Psychology, 20,* 200-207.

Siegel, L. S., & Ryan, E. B. (1989). The development of working memory in normally achieving and subtypes of learning disabled children. *Child Development, 60,* 973-980.

Spielberger, C. D. (1983). *State-Trait Anxiety Inventory for Adults*. Palo Alto, CA: Mind Garden, Inc.

Steiger, J. H. (1990). Structural model evaluation and modification: An interval estimation approach. *Multivariate Behavioral Research, 25,* 173-180.

Stevens, J. (2001). *Behavioral inhibition, self-regulation of motivation, and working memory in children with and without attention deficit hyperactivity disorder (ADHD).* Unpublished doctoral dissertation, Indiana University, Bloomington.

Stevens, J., Quittner, A. L., Zuckerman, J. B., & Moore, S. (2002). Behavioral inhibition, self-regulation of motivation, and working memory in children with attention deficit hyperactivity disorder. *Developmental Neuropsychology, 21,* 117-140.

Sullivan, M. A., & Rudnik-Levin, F. (2001). Attention deficit/hyperactivity disorder and substance abuse. In J. Wasserstein, L. E. Wolf, & F. F. Lefever (Eds.), *Adult attention deficit disorder: Brain mechanisms and life outcomes* (pp. 251-270*).* New York, NY: New York Academy of Sciences.

Swanson, H. L. (1993). Working memory in learning disability subgroups. *Journal of Experimental Child Psychology, 56,* 87-114.

Swanson, H. L. (1994). Short-term memory and working memory: Do both contribute to our understanding of academic achievement in children and adults with learning disabilities. *Journal of Learning Disabilities, 27,* 34-50.

Szatmari, P., Offord, D. R., & Boyle, M. H. (1989). Ontario Child Health Study: Prevalence of attention deficit disorder with hyperactivity. *Journal of Child Psychology and Psychiatry, 30*, 219-230.

Thompson, B. (2000). Ten commandments of structural equation modeling. In L. G. Grimm & P. R. Yarnold (Eds.), *Reading and understanding more multivariate statistics* (pp. 261-284). Washington, DC: American Psychological Association.

Torgesen, J. K. (1994). Issues in the assessment of executive function: An information-processing perspective. In G. R. Lyon (Ed.), *Frames of reference for the assessment of learning disabilities: New views on measurement issues* (pp. 143-162). Baltimore, MD: Paul H. Brookes.

Torrance, E. P. (1962). *Thinking creatively with pictures*. Bensenville, IL: Scholastic Testing Service.

Tunmer, W. E., & Hoover, W. A. (1992). Cognitive and linguistic factors in learning to read. In P. B. Gough, L. C. Ehri, & R. Treiman (Eds.), *Reading acquisition processes* (pp. 175-214). Hillsdale, NJ: Erlbaum.

Turner, M. L., & Engel, R. W. (1989). Is working memory capacity task dependent? *Journal of Memory and Language, 28*, 127-154.

Vaughn, M. L. (1998). *Working memory deficits in children with attention deficit hyperactivity disorder: An examination of an executive function component in Barkley's unifying theory of ADHD*. Unpublished doctoral dissertation, University of Georgia, Athens.

Wagner, R. K., & Torgesen, J. K. (1987). The nature of phonological processing and its causal role in the acquisition of reading skills. *Psychological Bulletin, 101*, 192-212.

Walker, A. J., Shores, E. A., Troller, J. N., Lee, T., & Sachdev, P. S. (2000). Neuropsychological functioning of adults with attention deficit hyperactivity disorder. *Journal of Clinical and Experimental Neuropsychology, 22,* 115-124.

Wechsler, D. (1997a). *WAIS-III administration and scoring manual.* San Antonio, TX: Psychological Corporation.

Wechsler, D. (1997b). *Wechsler Memory Scale – Third Edition.* San Antonio, TX: Psychological Corporation.

Weinert, F., & Kluwe, R. (1987). *Metacognition, motivation, and performance.* Hillsdale, NJ: Lawrence Erlbaum Associates.

Weiss, G., & Hechtman, L. (1993). *Hyperactive children grown up* (2nd ed.). New York: Guildford Press.

Weiss, G., Hechtman, L., Milroy, T., & Perlman, T. (1985). Psychiatric status of hyperactives as adults: A controlled perspective 15-year follow-up of 63 hyperactive children. *Journal of the American Academy of Child Psychiatry, 24*, 211-220.

Welsh, M. C. (2002). Developmental and clinical variations in executive functions. In D. L. Molfese, & V. J. Molfese (Eds.), *Developmental variations in learning: Applications to social, executive function, language, and reading skills* (pp.139-185). Mahwah, NJ: Lawrence Erlbaum Associates, Inc.

Welsh, M. C., & Pennington, B. F. (1988). Assessing frontal lobe functioning in children: Views from developmental psychology. *Developmental Neuropsychology, 4,* 199-230.

Willcutt, E. G., & Pennington, B. F. (2000). Comorbidity of reading disability and attention deficit/hyperactivity disorder: Differences by gender and subtype. *Journal of Learning Disabilities, 33*, 179-191.

Willcutt, E. G., Pennington, B. F., Boada, R., Ogline, J. S., Tunick, R. A., Chhabilda, N. A., & Olson, R. K. (2001). A comparison of the cognitive deficits in reading disability and attention-deficit/hyperactivity disorder. *Journal of Abnormal Psychology, 110,* 157-172.

Willcutt, E. G., Pennington, B. F., Chhabildas, N. A., Friedman, M. C., & Alexander, J. A. (1999). Psychiatric comorbidity associated with DSM-IV ADHD in a nonreferred sample of twins. *Journal of the American Academy of Child and Adolescent Psychiatry, 38,* 1455-1362.

Williams, K. T. (1997). *Expressive Vocabulary Test*. Circle Pines, MN: American Guidance Services, Inc.

Wolraich, M. L. (1999). Attention deficit hyperactivity disorder. *Mental Retardation and Developmental Disabilities Research Reviews, 5*, 163-168.

Woodcock, R. W., McGrew, K. S., & Mather, N. (2001). *Woodcock-Johnson III Tests of Achievement.* Itasca, IL: Riverside Publishing.

VITA

Dong Hyung Lee

Permanent Address
9515 W. Gulf Bank Rd. #200
Houston, TX 77040

Email Address
donhlee@hotmail.com

Education

Ph.D.	Aug. 2004	School Psychology	Texas A&M University (APA-accredited)
	2003-2004	Predoctoral Internship	Houston ISD, TX (APA-accredited)
M.A.	May 1996	Clinical Psychology	Chungnam National University, South Korea
B.A.	Mar. 1994	Psychology	Chungnam National University, South Korea

Publications

Lee, D., Riccio, C. A., & Hynd, G. W. (in press). The role of executive functions in attention deficit hyperactivity disorder: Testing predictions from two models. *Canadian Journal of School Psychology.*

Riccio, C. A., Wolfe, M., Davis, B., Romine, C., George, C., & **Lee, D.** (in press). Attention deficit hyperactivity disorder: Manifestation in adulthood. *Archives of Clinical Neuropsychology.*

Homack, S., & **Lee, D.** (in press). Test review: Delis-Kaplan Executive Function System. *Journal of Clinical and Experimental Neuropsychology.*

Lee, D. & Riccio, C. A. (in press). Cognitive retraining. In R. C. D'Amato, E. Fletcher-Janzen, & C. R. Reynolds (Eds.), *The Handbook of School Neuropsychology*. New York: Wiley.

Romine, C. B., **Lee, D.**, Wolfe, M. E., Homack, S., George, C., & Riccio, C. A. (in press). Wisconsin Card Sorting Test with children: A meta-analytic study of sensitivity and specificity. *Archives of Clinical Neuropsychology.*

Lee, D. (2003). Down syndrome. In E. Fletcher-Janzen, & C. R. Reynolds (Eds.), *Childhood Disorders Diagnostic Desk Reference* (pp. 188-189). New York: Wiley.

Lee, D., Reynolds, C. R., & Willson, V. L. (2003). Standardized test administration: Why bother? *Journal of Forensic Neuropsychology, 3*, 55-82.

Lee, D. & Kim, K.-H. (2000). Effects of hostility, social support, and task difficulty on cardiovascular reactivity. *Korean Journal of Health Psychology*, *5*, 209-222.

Lee, D. & Lim, J. (1997). Adaptation of Snyder's Hope Scale to Korean college students. *Journal of Student Guidance*, *24*, 85-108. Taejon, Korea: Chungnam National University.

Selected Presentations

Riccio, C., A., **Lee, D.**, Romine, C., Cash, D., & Davis, B. (2003, October). *Relation of memory and attention to academic achievement in adults.* Poster presented at the annual conference of the National Academy of Neuropsychology, Dallas, TX.

Lee, D., Romine, C., Wolfe, M., Wong, S., & Riccio, C. A. (2002, March). *Executive function: Everything school psychologists wanted to know about but were afraid to ask.* Paper presented at the annual conference of the National Association of School Psychologists, Chicago, IL.