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Validity of an Executive Function Model of ADHD Symptoms, Reading Difficulty and Substance Abuse in Adults

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The current study presents an integrated executive function model that explains the interrelationships among deficits in two executive functions (working memory and inhibition), ADHD symptoms, and two conditions (reading difficulty and substance abuse) that are commonly comorbid with ADHD in adulthood. The validity of the integrated model was tested using structural equation modeling with an adult sample consisting of consecutive referrals to a university-based research project. The final model suggested that working memory problems directly contribute to reading difficulty as well as to inattentive symptoms in adults. Direct contributions of inhibition to ADHD symptoms were not supported. However, ADHD symptoms had direct and indirect contributions to substance abuse symptoms. Current findings suggest the crucial role of working memory in the manifestation of ADHD symptoms and comorbid reading problems and also suggest ADHD symptoms' contribution to the development of adult substance abuse. These findings were interpreted as generally supporting the hypothesis of the developmental heterogeneity of executive function profiles associated with the manifestation of ADHD symptoms and comorbidities in adulthood.

Key words : ADHD, executive function, reading difficulty, substance abuse, working memory, inhibition

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Attention Deficit Hyperactivity Disorder (ADHD) is currently viewed as a chronic disorder, originating in childhood but with symptoms frequently continuing into adult life, causing distress and psychiatric comorbidities (Fischer, Barkley, Smallish, & Fletcher, 2005; Wasserstein, Wolfe, & LeFever, 2001). ADHD symptoms 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). 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). The diagnosis of ADHD in adults continues to be an area of controversy (Farone, Biederman, Feighner, & Monuteaux, 2000). It has been suggested that the way in which ADHD symptoms manifest may change over the course of development and, therefore, the application of childhood characteristics in the diagnosis of ADHD in adulthood may not be appropriate (Wender, Wolf, & Wasserstein, 2001). Related to potential differences in adulthood, Barkley (2006) reported that major complaints of adults seeking assessment for ADHD included difficulty with job placement and maintenance, inability to perform to capability, lack of organization, and low self esteem, as well as more typical complaints of forgetfulness and difficulty concentrating.

Increasing numbers of studies in the past decade have examined neuropsychological correlates of ADHD and found that children and adults with ADHD tend to perform more poorly than normal controls on various measures of neurocognitive functioning, especially those of executive function (EF). EF is generally defined as neurocognitive processes that adopt and maintain an appropriate problem solving set to attain a future goal (Welsh & Pennington, 1988). Given that EF is considered as an "umbrella" term for the complex cognitive processes that serve ongoing, goal-directed behaviors (Meltzer, 2007), many researchers have attempted to find if there are specific EF domains that are implicated in the manifestation of ADHD symptoms (Pennington & Ozonoff, 1996; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005). In this line of research, working memory and inhibition have received most theoretical and empirical attention in relation to ADHD and the role of working memory and inhibition in ADHD is well incorporated into the most comprehensive theoretical model of ADHD by Russell Barkley (Barkley, 1997, 2006).

According to Barkley (2006), the core deficit in ADHD lies in behavioral inhibition. Barkley (2006)'s disinhibition model further proposed that an inability to suppress prepotent responses to stimuli (i.e., disinhibition) interferes with the development and execution of other EFs such as

working memory, self-regulation, and reconstitution. The causal influence of disinhibition on these EFs is postulated to account for the impulsive/hyperactive behavior exhibited by individuals with ADHD. There is a substantial body of empirical findings evidencing poorer performance on purported inhibition tasks among children with ADHD compared to normal controls (for meta-analytic reviews, see Pennington & Ozonoff, 1996; Willcutt et al., 2005). Nigg (2001) reported that children with ADHD had less consistent difficulty with certain types of executive inhibition, including interference control, cognitive inhibition, and occulomotor inhibition as well as motivational inhibition and other automatic inhibition, than with behavioral/motor inhibition.

Compared to inhibition, studies on working memory function in ADHD often have yielded inconsistent results (e.g., Barnett et al., 2001; Karatekin, 2004; Stevens, Quittner, Zuckerman, & Moore, 2002; Willcutt et al., 2001) and suggested that only certain types of working memory may be particularly deficient in individuals with ADHD and the relation between working memory deficits and ADHD may be mediated by comorbid conditions such as reading disability. Two meta-analytic studies (Martinussen, Hayden, Hogg-Johnson, & Tannock, 2005; Willcutt et al., 2005) addressed these possible confounds and provided evidence of working memory deficits in children with ADHD relative to typically developing controls, even after controlling for comorbid language learning disorders and ADHD-related deficits in overall intelligence and reading achievement. These studies also reported larger effect sizes for the visuospatial (nonverbal) relative to phonological (verbal) working memory.

Although less is known regarding the neurocognitive characteristics of adults with ADHD and the extent to which the presence of comorbid disorders are associated with particular cognitive deficits, published studies have reported that adults with ADHD are significantly impaired, relative to normal controls, on purported measures of motor, response, and cognitive inhibition (Corbett & Stanczak, 1999; Hervey, Epstein, & Curry, 2004; Lovejoy et al., 1999; Ossmann & Mulligan, 2003), verbal and nonverbal memory (Hervey et al., 2004; Johnson et al., 2001), working memory (McLean et al., 2004; Walker, Shores, Trollor, Lee, & Sachdev, 2000), planning (McLean et al., 2004) and psychomotor speed (Johnson et al., 2001; Walker et al., 2000). In a meta-analytic review (Schoechlin & Engel, 2005), the highest effect sizes for adults were found for verbal memory, focused and sustained attention, and abstract verbal problem solving with working memory.

While Barkley's (2006) theory of ADHD posits working memory as one of several EFs undermined by poorly regulated behavioral inhibition processes in individuals with ADHD, some other authors consider it a central core component or a candidate endophenotype

(Castellanos & Tannock, 2002; Rapport, Chung, Shore, & Isaacs, 2001; Rapport, Kofler, Alderson, & Raiker, 2008). Intact working memory (i.e., an ability to hold and manipulate information held in temporary storage) is considered essential to successful inhibitory control (Pennington, Bennetto, McAleer, & Roberts, 1996) and working memory processes must be invoked to evaluate stimuli including situational cues prior to the initiation of the inhibitory process (Rapport et al., 2008).

Rapport and colleagues (Rapport et al., 2001, 2008) have recently proposed a multi-layered, developmental model of ADHD. Their conceptual model ("working memory model") attempts to link etiological factors to putative brain differences or abnormalities to candidate endophenotypes (e.g., working memory deficits) to cognitive/behavioral outcomes. More specifically, cortical underarousal in frontal and pre-frontal regions resulted from catecholaminergic dysregulation leads to an increased autonomic arousal to help compensate for such an underarousal during the tasks that tax working memory processes. According to Rapport et al. (2001), failure of working memory leads to inattentiveness and disorganized behaviors, and 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 hyperactivity/impulsivity (H/I) is a causal byproduct of inattentiveness and working memory deficits lead to hyperactive/impulsive behaviors via inattention. With regard to the role of other executive functions such as inhibition, the working memory model postulated that inhibitory control is secondary to working memory and inhibition 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 (Rapport et al, 2001).

The present authors extended the predictions from the working memory model (Rapport et al., 2001, 2008) and the disinhibitionn model (Barkley, 2006) to two comorbid conditions with ADHD in adults (i.e., reading difficulty and substance abuse) to construct an integrated model. The proposed model is shown in Figure 1.

It has been reported that various EF deficits are found in many neurodevelopmental, psychiatric, behavioral, and learning disorders as well as in ADHD (for review, see Meltzer, 2007; Pennington & Ozonoff, 1996; Welsh, 2002). However, there is relatively little understanding of how different EF deficits specifically contribute to these problems including ADHD. To complicate the issue, many individuals with ADHD tend to have one or

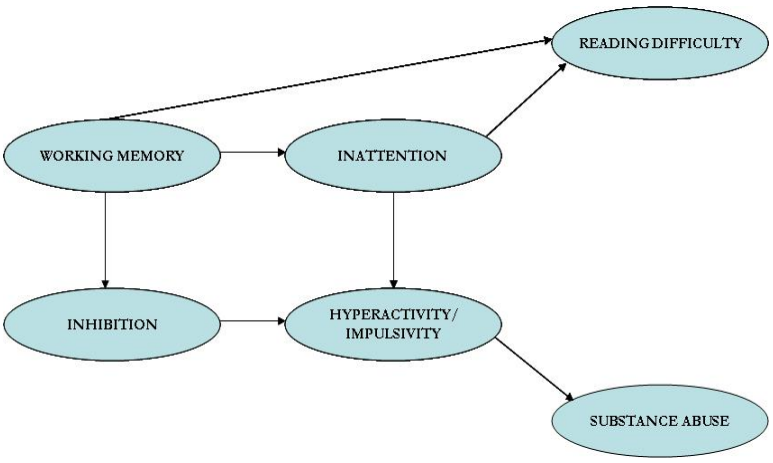

Figure 1. An integrated EF model of ADHD and comorbidities

more comorbid conditions. Hence, the understanding of how major EFs, such as working memory and inhibition, interplay each other in the manifestation of ADHD symptoms and its comorbid conditions would be very relevant to a more refined conceptualization of ADHD and its comorbid conditions from a neuropsychological standpoint.

While there is a substantial body of research findings evidencing disinhibition and working memory deficits among individuals (especially children) with ADHD, extant conceptual models of ADHD (i.e., Barkley, 2006; Rapport et al., 2008) do not address its comorbid conditions that are particularly relevant to adults with ADHD, such as reading difficulty and substance abuse, as part of their conceptual models. Although Rapport et al. (2001, 2008) indicated that the myriad behavioral and cognitive characteristic associated with ADHD are peripheral variables partially dependent on working memory processes, comorbid conditions frequently observed in individuals with ADHD have not been specified in their model. The integrated model (Figure 1) is an endeavor to generate a series of predictions regarding specific developmental and dynamic interrelationship among two EFs (i.e., working memory and inhibition), ADHD symptoms, reading difficulty and substance abuse in adults. The integrated model adopts major predictions from the working memory model (Rapport et al., 2001, 2008), but it also incorporates testable predictions from Barkley 's theory (Barkley, 2006).

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 extant theoretical accounts of prefrontal cognitive processes by working memory and inhibition (Kimberg & Farah, 1993; Pennington, 1997,

Roberts, Hager, & Heron, 1994; Roberts & Pennington, 1996), the integrated model posits that working memory deficits causally lead to disinhibition. It is postulated that behavioral inhibition is downstream of working memory processes, consistent with Rapport et al. (2008). It is further posited that two symptom dimensions of ADHD (i.e., inattention and H/I) are accounted for by two underlying, interrelated neurocognitive deficits in working memory and inhibition. That is, whereas the working memory deficit directly leads to inattention/distractibility symptoms (consistent with the working memory model), disinhibition has a direct impact on the motor control system, i.e., impulsive/hyperactive behaviors (as postulated by Barkley's model). These symptom-specific hypotheses originated from several 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, 2004). The integrated model also posits that poor working memory should contribute to H/I symptoms but this effect is posited to be mediated by disinhibition given the supposed primary role of working memory deficit in the manifestation of ADHD symptoms. The inattention/distractibility symptom and inhibition may be correlated according to our model, but the correlation between these two components is presumed to exist due to an underlying working memory deficit common to these two cognitive components. Finally, the integrated model specifies the nature of the interrelationship of working memory and inhibition components with two comorbid conditions, I. e., reading difficulty and substance abuse. Several studies on comorbidity in ADHD by subtype or symptom (e.g., Eiraldi, Power, & Nezu, 1997; Gadow et al., 2000; Morgan, Hynd, Riccio, & Hall, 1996; Willcutt & Pennington, 2000) 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 associated with delinquency, aggression, oppositional behavior, and substance abuse. Thus, based on these research findings and the differential EF hypotheses by two core ADHD symptoms as described above, reading difficulty is hypothesized as primarily related to working memory deficits and inattention, whereas substance abuse symptoms are primarily linked to a disinhibition -H/I path (See Figure 1).

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). 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). Some 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 to reading difficulty as well as inattention. Reading difficulty could be correlated with inhibition (e.g., 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 and distractibility symptoms.

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, Wilens, Mick, Spencer, & Farone., 1999). ADHD itself appears to be a risk factor for later substance abuse disorders and the presence of ADHD may mediate the course of substance-use disorder (Biederman, Wilens, & Mick, 1997). There may be a variety of reasons why individuals with ADHD preferentially seek out drugs. Sullivan and Rudnik-Levin (2001) have hypothesized that 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. Neuropsychological deficits, such as response inhibition, motor speed, vigilance, and verbal learning, in substance abusers have been consistently documented in studies of adults with ADHD (Wilen, 2006). Based on these research findings, the integrated model hypothesizes that H/I of ADHD has a direct influence on the development of substance abuse symptoms in adults, and that the relation between disinhibition and substance abuse symptoms is mediated by the severity of H/I symptoms. That is, given that ADHD symptoms first appear during childhood and precede the development of substance abuse problems, the integrated model presumes a direct devel opmental influence of impulsivity on substance abuse and the mediating role of impulsivity in the relation between inhibition and substance abuse.

The purpose of present study is to examine the validity of the proposed integrated model that addresses interrelationships of working memory deficits, disinhibition, ADHD symptoms, reading difficulty, and substance abuse in adults. The proposed model will be revised as needed in the light of data. Our integrated model was evolved from extant EF models of ADHD and extended predictions to comorbid conditions

especially relevant to adults. Given that contemporary EF theories have been primarily developed to address ADHD in children, as opposed to adults, our study will help determine the applicability of current EF accounts of ADHD and its comorbidities to adults. Moreover, considering some differences in the manifestation of ADHD symptoms in adulthood in the literature (e.g., Barkley, 2006), adults who experience ADHD symptoms may not necessarily demonstrate the same pattern of impairment on neuropsychological measures with children.

Methodologically, the present study employs a latent variable approach including confirmatory factor analyses (CFA) and structural equation modeling (SEM). An increasing number of studies in the field of EF (e.g., Lehto et al., 2003; Miyake et al., 2000; Miyake, Friedman, Rettinger, Shah, & Hegarty, 2001) have employed this procedure as a sound methodological alternative to a manifest variable approach, given that popular EF tests frequently have questionable psychometric properties and intrinsic task impurity problems (Miyake et al., 2000, 2001). By using multiple tasks for each target variable and aggregating the results to extract what is common among those tasks, "purer" latent variables are expected to be generated to examine how these constructs relate to each other and other constructs in the model.

Method

Participants

Participants in this study consisted of 102 adults who were consecutive referrals to a university-based research project in the southwest in the United States. Participants were recruited through the use of announcements distributed in the local community to physicians, local support groups for individuals with ADHD, a community-based counseling center, on local bulletin boards, and in the local newspaper. The announcement indicated that the research study focused on memory, attention, planning, and problem solving. Participation was voluntary with informed consent obtained from each participant. Participants were 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). Of the participants, only 3 were not currently enrolled in a university, college or community college program. 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 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 this study. Participants had to obtain an IQ greater than or equal to 80 and had to speak and read English to be included. Full Scale IQ scores of the participants in this study ranged from 85 to 147 (M=111.93, SD=13.63) and Total Achievement scores measured with Woodcock-Johnson Test of Achievement - Ⅲ (WJ-III: Woodcock, McGrew, & Mather, 2001) ranged from 78 to 137 (M=102.74, SD= 11.97).

Diagnostic decisions were made after at least two individuals (advanced doctoral students and at least one licensed psychologist) independently reviewed results from comprehensive individual evaluations. Thirty-one participants (30.4%) constituted a no-diagnosis 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).

Procedures

All participants received a comprehensive individual evaluation in a university-based assessment clinic. The evaluation included a variety of measures in the areas of intelligence, language, EFs, memory, achievement, behavior, and social and emotional functioning. 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 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.

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 H/I. Reading difficulty and substance abuse symptoms were measured with single indicators.

Working Memory

Three subtests from the Wechsler Memory Scale-Third Edition (WMS-Ⅲ: Wechsler, 1997b) were used as 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. In addition, Digit Span Backward (i.e., longest digit span backward) was also used as the third indicator of working memory Digit Span Forward was excluded because it is rather viewed as a short term memory task and measures only the storage component of working memory, while deemphasizing the manipulation of information (Reynolds, 1997). 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 functional 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 EF equally and cannot be clearly differentiated (Miyake et al., 2001).

Inhibition

As two indicators of inhibition, the Interference score from Stroop Color and Word Test (Golden & Freshwater, 2002) and the time on Part B to time on Part A ratio on the Trail-Making Test (TMT: 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). As a standard measure of interference, the Interference score from the Stroop 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, which 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).

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, McClure, & Post, 1986) 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). 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 errors of omission (EO) in CCPT-Ⅱ 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. The EO, compared to errors of commission (EC), is expected to correlate with ADHD inattention symptoms (Epstein, Conners, Sitarenios, & Erhardt, 1998). Thus, the EO was selected for the second indicator of inattention.

Hyperactivity/Impulsivity (H/I)

A 5-item subscale ("Hyperactivity/Restlessness") in the Conners Adult ADHD Rating Scales: Self-Report Short Version (CAARS-SSV: Conners, Erhardt, & Sparrow, 1997) and the number of items endorsed in the DSM-IV H/I diagnostic criteria (0-9) were used as two indicators of H/I 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. 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-3 (SASSI-3: Miller, Roberts, Brooks, & 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 addictions (Miller et al., 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 data trimming/screening procedures recommended by Kline (1998) were used as follows:.

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 1).

Descriptive statistics including mean, standard deviation, skewness, and kurtosis for these

Table 1. Descriptive Statistics for All Manifest Variables (N=102)

Variables	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 ratio 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 score.

[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.

variables are provided in Table 1. 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.

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; 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) (3) the Non-Normed Fit Index (NNFI) (4) the Comparative Fit Index (CFI); (5) the Root Mean Square Error of Approximation (RMSEA); (6) the Bayes Information Criteria (BIC); and (7) the Expected value of the Cross-Validation Index (ECVI).

Results

Correlations Among All Manifest Variables

Table 2 presents zero-order correlations among all manifest variables employed in the study. The correlation matrix shows that 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 EF 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, 2001). Relatively low intercorrelations are in part due to the complex nature of EF measures (i.e., task impurity issue) and a great deal of error variance involved in measurement and suggest that the latent variable approach is more suited for investigating the nature and role of EF than the manifest variable analysis.

Bivariate correlations and squared multiple correlations between each variable and all the rest indicate that none of the first-order correlations were above .55; the highest squared multiple correlation was .41. indicating that multicollinearity would not be a problem with the data for further analysis at the latent variable level.

Validity of the Integrated Model of ADHD and Comorbidities

Measurement Model

A confirmatory factor analysis (CFA) was conducted to develop a measurement model that possessed an acceptable fit with the data. The measurement model contains 6 structural variables and 11 manifest variables. Working memory was comprised of 3 indicators and inhibition, inattention, and H/I were measured with two indicators each. Reading difficulty and substance abuse were measured with single indicators. The resulting measurement model was a "nonstandard" model (Hatcher, 1994) in which two structural variables were measured with single indicators.

A CFA with this nonstandard model indicated a very good overall fit to the data as follows: a nonsignificant χ^2 value, $\chi^2(31, N=102)= 26.12/31=.84$, $p>.10$, GFI=.96, NNFI=1.06,

Table 2. Correlation Matrix for All Manifest Variables

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 Difficulty	**.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.

CFI=1.0, and RMSEA=.00 (0-.06). 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.

Structural Model

The tested structural equation model for the integrated model, along with all standardized parameters estimated, is displayed in Figure 2. Solid arrows indicate statistically significant paths ($p<.05$), while dashed ones indicates non-significant paths ($p>.05$) The fit indices for this initial model indicated that this SEM model had a good fit to the data (see Table 3): nonsignificant χ^2 value, $\chi^2(39, N=102)=33.97/39=.87$, $p>.10$, GFI=.94, NNFI=1.05, CFI=1.0, and RMSEA=.00 (0-.06). As shown in Figure 2, however, not all standardized path coefficients were statistically significant; two path coefficients were not significant and the other two paths were only marginally significant (all $ps>.05$). Thus, these statistically 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.

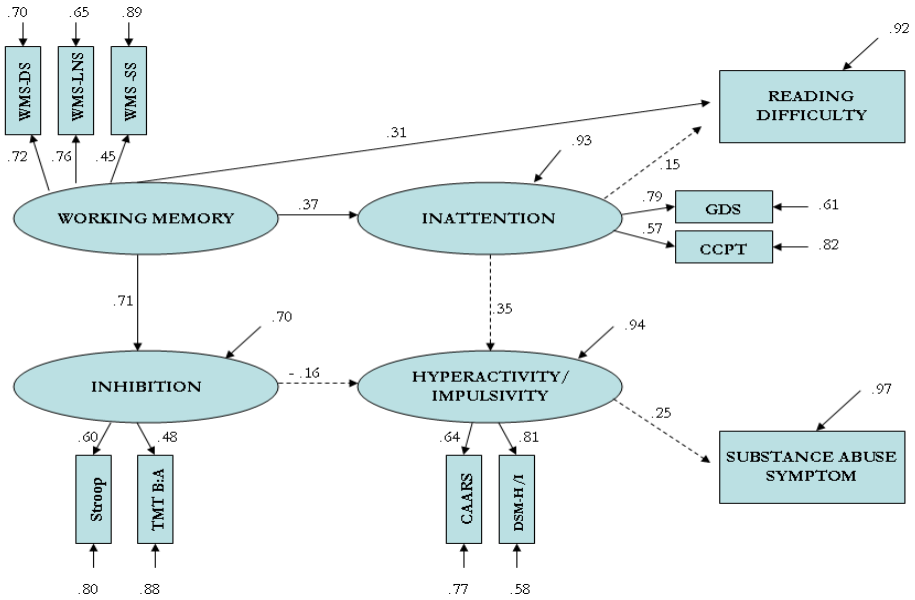

Figure 2. SEM for the integrated model of ADHD and comorbidites

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 model hypothesizes that there are direct effects of inhibition (on H/I) and inattention (on reading difficulty), the elimination of these effects appeared 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 3 presents the revised SEM model for the integrated model. For simplicity, the measures that were used to construct the latent variables

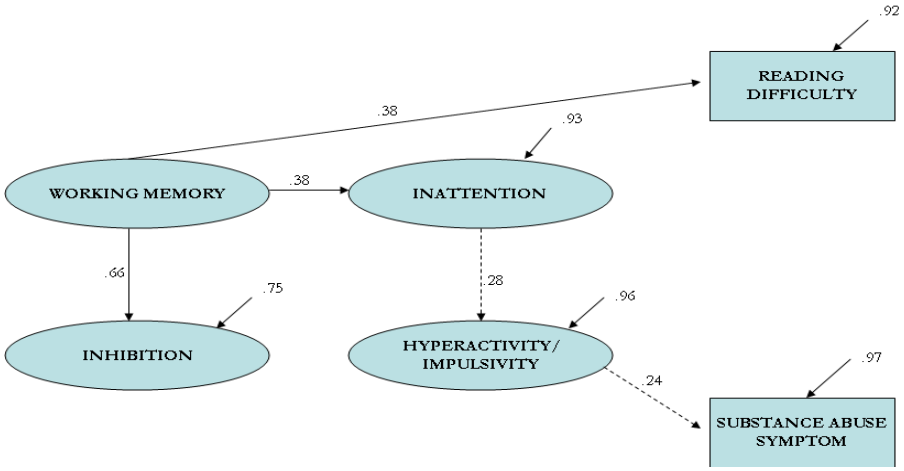

Figure 3. A more parsimonious integrated model of ADHD and comorbidities(final model)

Table 3. 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 (p>.05).

are not shown in the figure.

The reduced SEM model (Figure 3) demonstrated a good fit to the data as indicated by overall fit indices (see Table 3): nonsignificant χ^2 value, $\chi^2(10, N=102)=36.11/41=.88$, p>.10, 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 6) and the measurement model indicated no significant difference, $\chi^2(10, N=102)=9.99$, p>.10, meaning that the revised integrated was successful in accounting for the observed relations between the six structural variables. In short, these findings support the validity of the revised integrated model. Table 3 provides a comparison of fit indices between the original model and the revised model.

Discussion

The present study proposed an integrated

model, derived from two major executive function theories of ADHD (Barkley, 2006; Rapport et al., 2001, 2008) and recent research findings on comorbid conditions with ADHD, that addresses interrelationship of two executive function (i.e., working memory and inhibition) deficits, ADHD symptoms, and two common comorbid conditions with ADHD in adulthood (i.e., reading difficulty and substance abuse). The validity of the integrated model was tested using structural equation modeling techniques and the model was revised in the light of the data.

The nonstandard CFA model for the integrated model in which two constructs (i.e., reading difficulty and substance abuse) were measured with single manifest variables and four constructs with multiple manifest variables (working memory, inhibition, inattention, and H/I) demonstrated a very good overall fit to the data. Therefore, this measurement model was used as the basis for subsequent SEM analysis.

Estimated parameters for the integrated model and the overall fit indices indicated that the structural model successfully accounted for the observed covariances among six variables in the model. While the proposed integrated model (Figure 1 and Figure 2) indicated a very good overall fit to the data, it also had multiple non-significant paths. Therefore, the model was "trimmed" in the light of theoretical/logical as well as statistical grounds to yield a more parsimonious model that is theoretically sound and statistically well-fit to the data (Figure 3).

According to this final model (Figure 3), working memory is the underlying deficit of inattention and disinhibition, consistent with the prediction from the working memory model (Rapport et al., 2001, 2008). 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 a plausible explanation for why many individuals with ADHD also have comorbid reading problems and illustrates a common etiology hypothesis of the comorbidity between ADHD and reading disability, characterized by common genetic influences and at least some common neuropsychological deficits (Willcutt, Pennington, Olson, Chhabildas, & Hulslander, 2005). Our findings are consistent with previous studies in which 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; Lee et al., 2004 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 Willcutt et al., 2005).

According to our final model (Figure 3), the

direct contribution of inhibition to H/I or inattention symptoms was not supported. However, working memory deficit directly contributed to inattention symptoms and indirectly contributed to H/I through inattention symptoms. This pattern of relationship is again well supportive of major predictions from Rapport and his colleagues (2001, 2008) and appears to challenge Barkley's current conceptualization of ADHD as a disorder of a primarily inhibitory control(Barkley, 2006). An important caveat of such an interpretation is that inhibition measures employed in this study reflect only a particular type of inhibition (i.e., cognitive inhibition or interference control) and therefore limits its generalizability. Alternative explanation is that the more important role of working memory in ADHD and its comorbid conditions in current study may in fact reflect a developmental shift from childhood to adulthood in terms of neurocognitive characteristics. Given that ADHD in adults is understood as a continuation of a developmental disorder, the cognitive profile would be expected to show not only some similarity to that in childhood but also some differences due to maturation of underlying brain substrates. The evidence that ADHD is associated with significant weaknesses in several key EF domains rather than a single primary neuropsychological cause (Willcutt et al., 2005) would further support the hypothesis of the developmental heterogeneity of neuropsychological profiles associated with the manifestation of ADHD symptoms and comorbidities in adulthood. This interpretation may be somewhat premature but it is worthwhile to further explore in subsequent studies.

The mediating role of H/I symptom in the relation between inhibition and substance abuse was not supported in the present study. However, the direct contribution of H/I symptoms and the indirect contribution of inattention symptoms to substance abuse were supported, which is overall consistent with existing literature demonstrating the role of H/I in the development of substance use problems in adulthood (e.g., Biederman et al., 1997; Sullivan & Rudnik-Levin, 2001). With the limitation that ADHD symptoms and substance abuse symptoms were measured concurrently in this study in mind, this finding suggests that the presence of significant ADHD symptoms in adulthood may increase the risk of problems with substa nce use. Additionally, 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 our study did not include these variables; thus, it is possible that a relatively small correlation between H/I and substance abuse observed in our sample might have been reduced by the presence of

these additional factors.

This study has limitations. First, the sample size was relatively small considering that SEM is inherently a large sample technique (Kline, 1998). However, it is very likely that relatively reliable parameter estimates and fit indices have been obtained from current sample, given a Monte Carlo simulation study (Jackson, 2001) in which the number of observations per estimated parameter did *not* account for an appreciable amount of the variation in parameter estimates and values of summary fit indices beyond what was explained by the effects of sample size, indicator reliability, and the number of indicators per factor. Another limitation related to the sample is its representativeness. Given that the sample was made up of predominantly college students and graduates, adults with ADHD in this study may not represent the majority of adults with ADHD in the population, limiting the scope of generalizability.

The present study is one of a very few studies that employed a latent variable approach with multiple neuropsycholgical measures to examine the relationship of working memory and inhibition to adult ADHD symptoms and comorbidities. Our findings contribute to the understanding the nature of ADHD in adults and provide a neuropsychological explanation of why adults with significant ADHD symptoms often experience other problems such as reading difficulty and substance abuse problems. One particular implication would be that the crucial role of working memory in the manifestation of adult ADHD symptoms and reading problems probably should be more recognized and integrated in the development of any treatment program such as compensatory or remedial cognitive skills training and environmental modifications and accommodations. Given some limitations discussed above, however, current findings should be replicated and further examined with different samples.

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성인 ADHD, 난독성향 및 약물남용 증후에 대한 집행기능 모형의 타당성 검증

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본 연구는, 성인기 집행기능(작업기억과 억제)의 결함, ADHD 증상, 난독성향 및 약물남용 증후의 상호관련성을 설명하는 통합 모형을 제시하고 그러한 통합 모형의 타당성을 구조방정식 모형의 기법을 적용하여 검증하고자 하였다. 최종적으로 검증된 통합 모형에 따르면, 작업기억이 ADHD의 부주의 증상 뿐 아니라 난독성향에 직접적으로 기여하는 것으로 나타났다. 원래의 모형에서 제안된 바와 달리, 억제는 본 연구의 성인 표본에서 ADHD의 두 핵심 증상에 직접적인 영향을 미치지 않는 것으로 나타났다. 하지만, ADHD 증상은 성인기의 약물남용 증후에 유의한 직접 및 간접 효과를 보였다. 이러한 결과는, 성인기의 ADHD 증상 및 이에 동반되는 난독성향의 발현에 있어서 작업기억의 핵심적 역할을 시사하며, ADHD 증상이 약물남용 성향의 발달에 기여할 수 있음을 시사한다. 본 연구 결과는, 성인기의 ADHD 및 동반 증후 발현과 관련되는 집행기능 프로파일이, 아동기와는 다른 발달적 이질성을 보인다는 주장과 전반적으로 일치하는 것으로 해석되었다. 마지막으로 본 연구의 제한점 및 후속 연구의 필요성이 논의되었다.

주요어 : ADHD, 작업기억, 억제, 집행기능, 난독성향, 약물남용