

ASSOCIATION BETWEEN COGNITION AND DEPRESSION: A CROSSSECTIONAL
AND LONGITUDINAL STUDY OF INDIVIDUALS WITH LEARNING DISABILITIES

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Dissertation Prepared for the Degree of
DOCTOR OF PHILOSOPHY

UNIVERSITY OF NORTH TEXAS

August 2003

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Schraufnagel, Caitlin D., Association between cognition and depression: a cross-sectional and longitudinal study of individuals with learning disabilities. Doctor of Philosophy (Clinical Psychology), August 2003, 114 pp., 13 tables, 2 figures, references, 128 titles.

Over the past twenty years the number of children identified with learning disabilities has risen drastically. In addition, 26 - 40% of these children also experience depression. While cognitive functioning has been demonstrated to be associated with depression, it is unclear whether the mood, vegetative, or cognitive symptoms of depression predict particular cognitive processes and vice versa. The purpose of this study was to determine which particular cognitive processes were associated with specific depressive symptoms and depression as a whole. Structural equation modeling (SEM) was conducted to test a model which examined how three cognitive processing factors (verbal & visual reasoning, and attention/working memory) were associated with three depressive symptom factors (disturbances in mood, vegetative, and cognitive functioning). The data for SEM came from a large data set of children with learning disabilities (n=227). Model fit results supported the proposed model, and a significant association was found between the attention/working memory factor and the depression symptom factor reflecting disturbances in cognitive functioning. Less robust relationships were observed between verbal reasoning and cognitive depressive symptoms and an approach toward the conventional level of significance was noted between visual reasoning and cognitive depressive symptoms. Using a sub-sample of original

participants who were re-evaluated 20-25 years later (n=40), longitudinal analyses were conducted to determine the predictive power of cognitive functioning and depression over time. There was some indication for the predictive power of visual reasoning performance in childhood on mood symptoms of depression in adulthood. The most robust association at both time 1 and time 2 was between attention/working memory performance and cognitive symptoms of depression. However, the association appeared to be time specific and not predictive.

ACKNOWLEDGMENT

Warren A. Weinberg, M.D. (1934 – 2002), Professor of Pediatrics and Neurology at the University of Texas Southwestern Medical Center at Dallas is greatly acknowledged for his generous contribution consisting of the data utilized in this study as well as for his guidance and support throughout this project.

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CHAPTER 1

INTRODUCTION

Learning disabilities and depression

Many researchers have cited high comorbidity rates of learning disabilities (LD) and depression in children. Generally, learning disabilities occur in approximately 2-10 % of school-age children and depression is present in approximately 2-8% of children (APA, 1994; Hammen & Rudolph, 1996). However, recent reported rates of depression in children with learning disabilities range from 26- 40% (Wright-Strawderman & Watson, 1992; Rock, Fessler, & Church, 1997). Some earlier studies (e.g., Stevenson & Romney, 1984; Kovacs, 1981) reported lower rates ranging from 10-14%; however, other early studies reported incidence rates as high as 58% (Weinberg, Rutman, Sullivan, Penick, & Dietz, 1973) and 54% (Colbert, Newman, Ney, & Young, 1982) in children referred for evaluation due to learning difficulties. Thus, despite some variability, children and adolescents with LD do appear to be at increased risk for depression (Huntington & Bender, 1993) and rates of depression are much lower in both non-learning disabled children and the general population. Rates as low as 1.8% have been reported in non-learning disabled populations (Wright-Strawderman & Watson, 1992). Epidemiological studies of 6 month – 1 year prevalence rates in the general population have reported depression to occur in approximately 2-3% of children ages 6-11 and 6% - 8% of adolescents (Hammen & Rudolph, 1996).

Learning disabilities are most commonly diagnosed in childhood, though there is a general consensus that these disorders persist throughout the lifetime (Raskind, Goldberg, Higgins, & Herman, 1999; Shaywitz, Fletcher, Holahan, Shneider, Marchione, Stuebing, Francis, Pugh, & Shaywitz, 1999; Spekman, Goldberg, & Herman, 1992). However, rates of comorbidity for depression and learning disabilities in adulthood are unclear. Adult one-year prevalence rates of depression based on large-scale epidemiologic studies (i.e., Epidemiologic Catchment Area study, National Comorbidity Survey) of the general population range from 3.7% – 10.3% (Maxmen & Ward, 1995). In contrast, Raskind et al. (1999) in a twenty-year longitudinal study of individuals with learning disabilities (mean age = 32.1 years), cited a rate of 42% for adult psychological disturbance (depression, alcohol abuse, anxiety disorders, etc.). While Werner (1993), in another long-term longitudinal study of individuals with learning disabilities, noted high comorbid rates of psychological disturbance in adolescence (32%), but in the subjects' early thirties, the rate had decreased to less than 10%.

Given the frequent co-occurrence of learning disabilities and depression, it is not surprising that theories of comorbidity exist. In an effort to understand the comorbidity of depression and difficulties in learning and cognition, Livingston (1985) posed several pertinent questions: (1) "Does depression cause or worsen learning difficulties? (2) Do learning difficulties put children at risk for major depressive disorder? (3) Is there some particular brain dysfunction that puts children at risk for both major depression and particular learning difficulties?" In sum, there is an overarching question of whether one of these conditions is primary. There seems to be at least three possibilities in this regard:

(a) depression is the primary condition, (b) learning disabilities cause depression, and (c) there is a shared etiology between the disorders. In addition, another possibility is the disorders are separate and unrelated, but co-occur by chance.

Livingston (1985) suggested the confirmation of two hypotheses would be necessary to provide evidence of the first possibility (1) "there is a temporal relationship between the onset of depression and the onset or worsening of impaired learning, and (2) the resolution or successful treatment of depression is associated with improvement in school performance" (Livingston, 1985, p. 518). Support for these hypotheses has been given by research indicating children first identified as depressed also had cognitive and achievement scores that classified them as learning disabled (Colbert et al., 1982; Weinberg, McLean, Snider, Nuckols, Rintelman, Erwin, & Brumback, 1989a). Also, the influence of depression on school performance is evidenced by the inclusion of impaired school performance in an established diagnostic criteria for depression (Weinberg et al., 1973) and the recognition of poor school performance as a symptom of masked depression (Hollon, 1970). Furthermore, Brumback (1988) noted significant improvement of neuropsychological test scores and graphic abilities as a result of treatment of depression. On the other hand, despite similar spelling and handwriting performance by adolescents who committed suicide and learning disabled adolescents, McBride and Siegel (1997) purported that depression does not specifically affect school skills. Rather, in support of the contention that depression is a primary condition, the authors contend that the established effects of depression upon areas of cognitive

functioning including free recall, attention, memory, and verbal functioning negatively affect achievement abilities (McBride & Siegel, 1997).

The second possibility (learning disabilities as a primary condition which leads to depression) could be supported by evidence that depression is more frequent in children with previously diagnosed learning disabilities than in controls or in children with other disorders (Livingston, 1985). Many studies of children first identified as learning disabled have reported higher rates of depression than in non-learning disabled children (e.g., Hall & Haws, 1989, Weinberg et al., 1989a). It may be that younger children with LD show more depressive symptoms, yet as they learn to cope with the condition, the symptoms of depression decrease (Hall & Haws 1989). However, some researchers question whether some referrals for evaluation for learning disability are initiated due to behavioral problems. Thus, if such children were evaluated for depression at the time of learning disability diagnosis, the number of concurrent diagnoses of learning disabilities and depression would be higher. Furthermore, the diagnosis of learning disability may be more palatable and thus more likely to be initially diagnosed (Colbert et al., 1982).

The third possibility proposes shared underlying brain dysfunction that puts children at greater risk for both depression and learning disabilities. This possibility appears to have been of most interest to researchers and is a focus of the current study. This latter hypothesis is based on research that highlights an association between right posterior hemisphere dysfunction and depression in adults (e.g., Yozawitz, Bruder, Sutton, Sharpe, Gurland, Fleiss, & Costa, 1979; Silberman, Weingartner, Stillman, Chen, & Post, 1983) and children (Brumback, Staton, & Wilson, 1980, Brumback & Staton,

1983; Weinberg, Harper, & Brumback, 1995). In addition, recent research has also implicated specific left anterior hemisphere dysfunction in depression (Heller & Nitschke, 1997). In an effort to review the literature on this subject, research regarding neuropsychological functioning in depression, depression and right hemisphere dysfunction, and depression and left hemisphere dysfunction will be reviewed below.

Depression and neuropsychological functioning

It is widely believed that there are many cognitive deficits associated with depression (Christensen & Segal, 2001; King & Caine, 1996). The study of cognition and depression was largely influenced by the identification of “pseudodementia” in the elderly. It was deemed important to separate “progressive neurodegenerative diseases (so-called “organic” conditions) from psychiatric (“functional”) syndromes that would respond to treatment” (King & Caine, 1996, p. 200). Moreover, researchers observed that treatment of psychiatric syndromes could result in the alleviation of mental status deficits and neurological signs (Freeman, Galaburda, Cabal, & Geschwind, 1985).

Despite significant concern regarding the identification of dementia versus depression, there were few controlled studies investigating cognitive deficits associated with depression during the early and mid 1980s (King & Caine, 1996). However, methodologically sound studies of the neuropsychology of depression in adults have increased substantially over the past 15 years. Unfortunately, many unresolved issues regarding cognitive abnormalities and depression remain. For example, controversies about the extent, pattern, association with age and medical comorbidity, and the rate of occurrence of cognitive dysfunction across severity of depression still remain (King &

Caine, 1996). Moreover, researchers have been more apt to focus on the elderly as well as younger adults. Studies investigating the relationship between depression and cognition in children have been less frequent.

One of the early ideas of the pseudodementia studies was the assumption that cognitive dysfunction associated with depression was reversible. However, there has been evidence to the contrary, especially in persons with severe depression (King & Caine, 1996). For example, King, Caine, Conwell, & Cox (1991) found that cognitive deficits (i.e., dysnomia) in late-life depression did not improve following effective treatment. This result suggests that there may be “markers of subtle brain dysfunction that influence the onset or course of depressive illness” (King & Caine, 1996, p. 206). Longitudinal studies should help to address this issue of persistence of cognitive deficits associated with depression.

Although depressed individuals frequently complain of attention/concentration and memory difficulties (APA, 1994), studies have tended to focus on the effects of depression on memory (Lezak, 1995). Findings regarding memory deficits in depressed persons have been equivocal. King & Caine (1996) offer that the severity of the depression may be a significant factor given that memory deficits are less likely in individuals with mild to moderate depression. In regards to types of deficits, it has been demonstrated that depressed persons show difficulty with long-term memory and explicit memory. Explicit memory tasks include direct tests of recall and recognition of which the subject is aware of being tested. Conversely, implicit memory tasks are based on

unconscious activation of an item and do not rely upon the subject's awareness (e.g., word-stem completion tasks) (King & Caine, 1996).

Some research suggests that working memory and implicit memory appears to be intact in depressed persons (Christensen & Segal, 2001). Likewise, an effect size analysis of neurocognitive function in patients with depression, found minimal effect sizes for tests of working memory. On the other hand, some studies have demonstrated depressed persons to have deficits in working memory (e.g., Geva, 2002; Keller, 2000; Landro, Stiles, & Sletvold, 2001; Nebes, Butters, Mulsant, Pollock, Zmuda, Houck, & Reynolds, 2000;)

In regard to explicit memory tasks, recall appears to be more impaired than recognition in depressed persons (Calev & Erwin, 1985). Researchers have hypothesized that these individuals are able to encode information effectively but struggle with search and retrieval processes (Ilsley, Moffoot, & O'Carroll, 1995). Others have contended that depressed persons use weak or incomplete encoding strategies resulting in memory dysfunction (Weingartner, Cohen, Murphy, Martello, & Gerdt, 1981). Thus, memory deficits could also stem from problems in attention (i.e., encoding of material for storage), which have also been demonstrated in depression (Christensen & Segal, 2001). Moreover, depressed persons do not have difficulty learning but rather require a cue or recognition format to retrieve learned information (Lezak, 1996; King & Caine, 1996). Thus, these individuals are less likely to employ learning strategies spontaneously but benefit from stimuli which produce a structured format. For example, when semantic clustering cues are available, performance does not appear to differ from controls. Such

findings have been hypothesized to occur due to reduced resources for conscious memory processes as a result of depressive ruminations and possibly difficulty filtering out irrelevant stimuli (King & Caine, 1996).

Extensive research has shown that memory performance in depressed individuals is impacted by the emotional valence of material presented. Specifically, when the affective valence of stimuli are congruent with one's mood, such stimuli are better encoded and retrieved than when the stimuli differs in valence (Blaney, 1986). Termed "mood-congruent memory," this has been a major area of research in cognitive psychology (King & Caine, 1996). Matt, Vazquez, & Campbell (1992), conducted a meta-analysis of 15 studies and found that recall was associated with negatively-valenced material in individuals with clinical depression and positively-valenced material in normal subjects. Such recall appears to be specific toward mood or personally relevant descriptive words (e.g., hopeless, guilty, worthless). In addition, mood-manipulation in non-depressed subjects has produced similar results. When depressed mood is temporarily induced in non-depressed individuals, preferential recall for negatively valenced stimuli occurs, though the effect is not as robust as that demonstrated by depressed persons (Matt et al., 1992).

As noted above, depressed persons frequently report attentional problems (APA, 1994; Christensen & Segal, 2001). Studies have shown that depression can temporarily reduce attentional capacity in otherwise neurologically intact adults (Lezak, 1995; Christensen & Segal, 2001). Depressed persons show slower reaction times on visual

attention tasks such as the Covert Orientation of Visual Attention (COVAT) than controls (Smith, Brebion, Banquet, & Cohen, 1995).

Despite some research demonstrating intact working memory in depressed persons, some studies have demonstrated both deficits in working memory (Landro et al., Stiles, 2001; Keller, 2000; Nebes et al., 2000; Geva, 2002) as well as proposed that attention and working memory are related or overlapping constructs. For example, some researchers have used tasks involving working memory in effort to evaluate attentional resources (Channon, Baker, & Robertson, 1993) or have considered working memory an attentional system with controlled processes (de Ribaupierre, 2000).

In line with the above discussion of mood congruent memory, depressed individuals tend to selectively attend to negative information. Studies of depressed persons performance on dichotic listening tasks, visual dot probes, and emotional Stroop tasks have shown that the emotional valence of the stimuli affects performance (Christensen & Segal, 2001). For example, negative auditory information presented in dichotic listening tasks is more distracting to depressed individuals than controls (McCabe & Gotlib, 1993). Likewise, in a probe detection task, depressed individuals showed an attentional bias to negative content words (Mogg, Bradley & Williams, 1995). Also, other research (e.g., Sutton & Davidson, 1997; Davidson, 1992) suggests depressed persons show more absence of bias for positively valenced information. Thus, decreased positive affect in depression could also be involved in affecting their cognitive functioning.

Depressed persons' color-naming on a modified Stroop color word task is also significantly affected by negative semantic stimuli. Depressed individuals were slower to name the color of depressed-content words than neutral- or manic-content words. In contrast, non-depressed individuals performance across word types did not differ (Gotlib & McCann, 1984). Interestingly, similar to findings in mood congruent memory research, negative, personally relevant words are most distracting to depressed individuals when performing the Stroop color word task (Christensen & Segal, 2001). In contrast to other findings regarding mood induction (Matt et al., 1992), Gotlib & McCann (1984), did not find differences in performance between non-depressed persons and non-depressed persons in whom depressed mood was induced. The authors suggest their results support the contention that negative cognition precedes instead of follows depressed affect. Also, Williams, Matthews, & MacLeod (1996), in a review of the emotional stroop task and psychopathology, noted that attentional deficits have been shown to disappear after successful treatment.

In addition to memory and attentional deficits, impairment in inhibition has been observed in individuals with depression. Specifically, persons with depression show impairment in the ability to inhibit personal, negatively valenced information. For example, on cognitive inhibition tasks (e.g., the Prose Distraction Task and the Fan Effect Task) depressed individuals showed greater difficulty inhibiting negative distractors than non-depressed individuals (Christensen & Segal 2001).

Over the past decade, researchers have begun to focus on the “neurobiological substrates of major depression by defining specific correlates of its cognitive features”

(King & Caine, 1996, p. 207). Positron-emission tomography (PET) and magnetic resonance imaging (MRI) studies have indicated physiological abnormalities of the basal ganglia in depressed persons (King & Caine, 1996). In addition, a computed tomographic (CT) study showed greater ventricular-to-brain ratio in elderly patients with depression related dementia and alzheimer's patients when compared to controls (Pearlson, Rabins, Kim, Speedie, Moberg, Burns, & Bascom, 1989). Decreased blood flow in the left anterior medial prefrontal cortex and increased blood flow in the cerebellar vermis has also been observed in elderly depressives with reversible cognitive impairment in comparison to elderly depressives without cognitive impairment (Dolan, Bench, Brown, Scott, Friston, & Frackowiak, 1992). Also, the dorsolateral prefrontal cortex, particularly in the right hemisphere, has been identified as a key brain structure in the interaction between emotion and cognition. This region has been stated to be a crucial "convergence zone" given its role in sustained attention and limbic-cortical influences (Liotti & Mayberg, 2001).

Neurochemical systems have also been implicated in the neuropsychological performance of persons with depression. Research, though equivocal, shows some evidence that hypothalamic-pituitary-adrenal (HPA) activity might be associated with the ability to selectively attend to information (i.e., to distinguish relevant from irrelevant information). This association could be through the effect of cortisol on the hippocampus (King & Caine, 1996). Given the wide diversity of physiological findings, King and Caine (1996) contend there is little evidence tying "specific types of neuropsychological functioning to focal brain regions." They hypothesize that such focal findings may not be

possible as mood disturbances “are likely the result of disordered neurochemical systems rather than any focal or discrete abnormality.” (King & Caine, 1996, p. 208). Likewise, Mayberg (1997) proposed a model of depression that implicated failure of the coordinated interactions of a distributed network system of limbic-cortical pathways. The hypothesis that depression is a result of disordered systems is consistent with attention problems in depression, given that attention is also thought to involve a widely distributed neural system (Posner & Petersen, 1990).

However, other investigators contend “depressed people are characterized by deficits in performance on tasks that depend on regions of the brain that are less active in depression” (Heller & Nitschke, 1997, p. 637). Also, there is a significant body of research suggesting lateralized cerebral deficits in depression (King & Caine, 1996) and as stated above, depression has been associated with right posterior hemisphere dysfunction. For instance, EEG, ERP, and blood flow studies have indicated less right posterior activity in depressed persons as compared to non-depressed persons (Heller & Nitschke, 1997). Consistent with the neurophysiological studies, “a left hemimotor syndrome (Brumback & Weinberg, 1990; Levy, Harper, & Weinberg, 1992; Weinberg & Brumback, 1992) is often detected in depressed children and adolescents. This syndrome is characterized by spooning (dishing) of the extended left hand, difficulty with rapid alternating movements in the left hand, mild left-sided tremor, occasional left Babinski reflex, and, in the supine position, external rotation of the left leg” (Emslie, Weinberg, & Kowatch, 1998, p. 374). Finally, deficits on tasks associated with cognitive functions of

the right posterior regions of the brain have been observed in depressed individuals (Heller & Nitschke, 1997).

Left hemisphere-related cognitive dysfunction has been associated with depression less frequently. However, strokes occurring on the left side are more frequently associated with depressed symptoms than strokes to the right side. Depression may be the result of hypoactivity of the left hemisphere or hyperactivity (i.e., disinhibition) of the nonaffected hemisphere. Thus, a problem in one hemisphere of the brain may result in the contralateral part to act abnormally, resulting in depression (Emslie et al., 1998). Recent research has suggested a relationship between left anterior dysfunction and depression (Heller & Nitschke, 1997).

In sum, cognitive problems including memory (i.e., long-term and explicit memory difficulties) and attentional dysfunction are associated with depression. On the other hand, depressed individuals have shown a tendency toward better recall and attention to information congruent with their negative mood. While research continues in this area, there appears to be reasonable support for lateralized cerebral deficits in depression and, right hemisphere-related cognitive dysfunction has been most often associated with depression.

Depression and right hemisphere-related cognitive dysfunction

The right cerebral hemisphere is thought to be the locus of visual-motor/visual-spatial integration, right-left discrimination, timing and time perception, temporal sequencing/planning, music appreciation, emotionality, and arousal (Livingston, 1985; Brumback, 1988). The relationship between depression and right brain dysfunction was

first suggested by Bruce (1895) in his description of a Welsh sailor with manic-depressive illness. When depressed, the sailor was completely left-handed, remained relatively immobile, appeared confused, and spoke incoherently in and understood only Welsh. In contrast, when in a manic state, he spoke and understood both Welsh and English, “was restless, destructive, thievish, and constantly playing practical jokes on his fellow patients” (Bruce, 1895, p. 60). In addition, in such states, he was completely right-handed. Similar cases have also been described (e.g., Robinson, 1976, Freeman et al., 1985). Right hemisphere dysfunction associated with depression, as evaluated by neuropsychological testing, was first suggested by Flor-Henry (1976). Due to growing evidence for their relationship, the association of depression and right hemisphere cognitive dysfunction has been reasonably well-established (Heller & Nitschke, 1997). However, there is not a general consensus regarding lateralizing variables (Livingston, 1985).

Specifically, depression has been most often associated with right posterior dysfunction. Furthermore, literature suggests the processing of emotional material is modulated by posterior right hemisphere regions. As stated above, there is research suggesting individuals with depression have difficulty understanding the global concept (gist) of stories. These individuals tend to pay greater attention to stimuli features, especially negative, personally relevant information (Heller & Nitschke, 1997). The posterior right hemisphere is suited for processing contextual, relational, and global information while the left hemisphere is geared toward the systematic processing of

details. Furthermore, studies have found depressed subjects display impaired recognition of facial affect, a right posterior function (Heller & Nitschke, 1997).

Several studies, in adults and children, have utilized standardized intelligence tests (i.e., Wechsler scales) to compare right hemisphere and left hemisphere cognitive abilities. Performance IQ tasks are considered to include predominantly non-verbal (right hemisphere) tasks while Verbal IQ tasks are considered to measure predominantly verbal (left hemisphere) abilities. It has been demonstrated that depressed individuals show deficits in performance IQ (e.g., Brumback, 1985; Flor-Henry, 1990; Sackeim, Freeman, McElhiney, Coleman, Prudic, & Devanand, 1992). However, as Emslie et al. (1998) noted, it is unclear whether such results reflect right-hemisphere dysfunction or general problems with attention and concentration due to psychomotor retardation. On the other hand, attention, concentration, and regulation of psychomotor activity are generally largely regulated by right hemisphere functions.

In contrast to the research cited above, some studies have not found deficits in performance IQ in depressed persons (e.g., Mokros, Poznanski, & Merrick, 1989; Kashani, McGee, Clarkson, Anderson, Walton, Williams, Silva, Robins, Cytryn, & McKnew, 1983; Stevenson & Romney, 1984). It may be that only a subgroup of depressed persons, as opposed to all individuals with depression, evidence right-cerebral hemisphere dysfunction (Brumback, 1988). Thus, only this subgroup would evidence performance IQ deficits. In addition, some researchers believe that many “non-verbal,” “right hemisphere” performance tasks also involve some degree of verbal abilities. As a result, using a deficit in performance IQ as an indicator of right hemisphere cognitive

dysfunction may be inaccurate (Weinberg 2001, personal communication). Another explanation could be that verbal (left hemisphere) tasks are more over-learned and thus less likely to be disrupted by depression. As such, discrepancies in cognitive performance can be suggestive of lateralized cerebral dysfunction, however it is important to understand that specific measures may be more or less indicative of right (or left) hemisphere dysfunction.

In the discussion that follows, performance on specific Wechsler subtests (e.g., digit symbol) and other neuropsychological measures/batteries are used to assess right and left hemisphere-related cognitive abilities. Researchers have also stressed the importance of paying careful attention to the psychometric properties of employed tests as well as subject selection (Miller, Fujioka, Chapman, & Chapman, 1995). The research presented below is considered representative of this developing area of study and supportive of the association between depression and impairments in right hemisphere tasks. However, it should be noted that the methods used in this area of research (i.e., types of measures employed) are variable and thus the discussion shifts rapidly.

Silberman, Weingartner, and Post (1983) found that on discrimination learning problems, depressed individuals had difficulty narrowing down a set of possibilities and often perseverated on disconfirmed hypotheses. The authors observed similar performance in patients with right hemisphere lesions. Miller et al. (1995) reported that patients with depression showed significantly poorer performance on a visual-spatial functioning (dot-localization task) than on a verbal (word finding) task. Berndt and Berndt (1980) used subjects' performance on four right hemisphere tasks (paired easy

associates, digit symbol test, writing speed task, and Necker Cube reversals) to successfully identify mildly depressed versus non-depressed college students. The authors concluded their results evidenced an association between mild depression and a deficit in energy during initial perceptual processing and in the organization and execution of psychomotor tasks.

Other cognitive and affective processes have been measured to assess for impaired right hemisphere functioning. Lowered ability to recognize emotionality (prosody) in others has been associated with depression. Researchers have observed impaired recognition of facial affect in depressed individuals (e.g., Rubinow & Post, 1992). In a study of dichotic perception, patients with affective psychosis displayed abnormal auditory perceptual asymmetries. Specifically, their ear asymmetries were similar to those of right temporal-lesioned controls (Yozawitz et al., 1979).

Research investigating an association between cognition and depression in children has lagged behind that of adults. Nonetheless, “investigators have accepted a variety of evidence for right-brain dysfunction including dyscalculia, motor development delays, poor handwriting, poor sequencing, verbal IQ greater than performance IQ, relatively minor abnormalities of hand and foot posture, and patterns of performance on dichotic listening tasks” (Livingston, 1985, p. 519). Brumback and Staton (1982) were among the first to examine the association between right hemisphere cognitive dysfunction and depression in children. As briefly reviewed above, early studies focused on IQ scores for comparison of right and left hemisphere abilities. In addition, adverse sensorimotor changes have been observed in individuals with depression (e.g., Bruce

1895; Robinson, 1976; Freeman et al., 1985; Cutler, Post, Rey, & Bunney 1981). Brumback and colleagues (1988) have described a right hemisphere hemisindrome linking depression, right hemisphere learning disabilities, and neurological motor abnormalities. Staton, Wilson, & Brumback (1981) reported that over 75% of their sample of depressed children showed specific postural, movement, and reflex asymmetries involving the left side of the body. The authors concluded that along with neuropsychological asymmetries, their motor asymmetries provided evidence for a “hemisindrome of dysfunction of the right cerebral hemisphere.” Notably, “the most significant problems for the child with right-cerebral-hemispheric dysfunction are: (1) difficulty in expressing nonverbal social cues (including happiness or anger) and in responding correctly to those cues expressed by others, and (2) difficulty in expressing and understanding the prosodic portions of language (the emotional and tonal inflections of speech” (Brumback, 1988, p. 484).

Children with non-verbal learning disabilities (LDs characterized primarily by social difficulties) share similar primary characteristics to those of the right hemisindrome described above (Heller & Nitschke, 1997). Moreover, children with non-verbal learning disabilities have been found to have an increased risk for depression and suicide (Rourke, Young, & Leenaars, 1989). Unanswered questions relating to the study of cognitive dysfunction in depressed children include those posed by Emslie et al. (1998) regarding whether the consequences of depression are related to specific periods of development and/or the duration of depression. Also, Livingston (1985, p. 519) questioned whether “children who only have disabilities associated with right-brain

dysfunction (e.g., visual-motor or arithmetic problems) develop depression more frequently than children with other learning problems.” There are few studies that have examined the relationship between depressive disorders and the development of cognitive abilities, social competence, and academic achievement (Emslie et al., 1998). In one such study, Kovacs (1989) suggested that cognitive development is delayed in depressed children and as a result there may be interference with the acquisition of verbal skills. Similarly, van Os, Jones, Lewis, Wadsworth, & Murray (1997) concluded that their findings support the notion that early-onset affective disorder is preceded by impaired neurodevelopment and cognitive delays. Longitudinal studies should help in addressing many of the above questions.

Another method of evaluating the link between right hemisphere dysfunction and depression is to examine the effect of treatment of depression on cognitive performance. Several studies have indicated that depressed individuals’ performance on neuropsychological measures improve after drug therapy. For instance, depressed patients’ performance on Halstead-Reitan tasks involving visuo-spatial processes and retention of both nonverbal and verbal information was impaired pre-treatment. However their performance did not significantly differ from that of the control group, post-treatment (Fromm & Schopflocher, 1984). Savard, Rey, & Post (1980) found that the performance on the Halstead-Reitan Category Test of patients with unipolar depression improved substantially following effective treatment of depression. However, as stated earlier, some research has documented that some cognitive skills remain impaired following treatment of depression (King & Caine, 1996). For example, Sackeim et al.

(1992) observed that performance IQ deficits in depressed persons did not improve significantly post treatment.

Improvement in cognitive functioning following treatment of depression has also been observed in children. For example, in an early study, Rapoport (1965) observed improved school skills (i.e., handwriting, reading, arithmetic) in depressed, learning-disabled children following treatment with imipramine. Staton et al. (1981) found that successful treatment of major depression with tricyclic antidepressants was associated with improvement on several cognitive measures. Children's performance post-treatment significantly improved on the WISC-R subtests of Similarities, Comprehension, Block Design and Coding as well as overall Verbal and Performance IQ. A significant improvement in performance was also evidenced on the Halstead Categories Test, the Visual Reception subtest of the Illinois Test of Psycholinguistic Abilities, and in response latencies on the Matching Familiar Figures test. In other studies, investigators (Brumback, 1985; Wilson, Staton, & Brumback, 1982 as cited in Brumback, 1988) found that improvement in depression was specific to children with performance IQ deficits versus verbal IQ deficits. The children with performance IQ deficits demonstrated improvement in alertness, visual sequencing, visual perception, long-term visual retention, perceptual organization and auditory information processing as well as improvement in depressive symptoms following antidepressant treatment. However, depressed children who evidenced an initial Verbal IQ deficit showed only slight to moderate improvement in verbal comprehension, reading achievement, practical

reasoning, and visual –motor coordination. Moreover, the Verbal IQ deficit group showed minimal change in depressive symptoms.

Kronfol, Hamsher, Digre, & Waziri (1978) found that unilateral (delivered to the right or left hemisphere) ECT treatment improved right hemisphere functions when depression was ameliorated. However, Goldstein, Filskov, Weaver, & Ives (1977) compared pre- and post-treatment performance on the Halstead-Reitan neuropsychological battery in patients receiving ECT and found a higher number of impaired right hemisphere functions post-treatment. Warneke (1975) observed improved performance WISC IQ scores following ECT treatment.

In sum, the association of depression and right hemisphere (specifically right posterior) cognitive dysfunction is reasonably well-established. There have been varied findings regarding performance IQ deficits in depressed persons. However, research does appear to support the use of subtests of the Wechsler scales for comparison of right and left hemisphere cognitive abilities. In addition, there may be a subgroup of depressed individuals that is more likely to evidence performance IQ deficits. The research reviewed indicates depressed individuals often show impairment on right hemisphere functions including visual-spatial skills, perceptual processing, psychomotor skills, ability to recognize emotionality in others, and auditory perception. Tasks employed to evaluate such functions include dot-localization, paired easy associates, writing speed task, Neckar Cube reversals, Halstead-Reitan Category Test, Visual Reception Test of the Illinois Test of Psycholinguistic Abilities, matching Familial figures Test, and examination for postural, movement, and reflex asymmetries. Moreover, the absence of a

pattern of global cognitive impairment in depressed patients argues against motivational factors as the primary cause of such impairments.

In contrast to right hemisphere-related cognitive dysfunction, the association of left hemisphere-related cognitive dysfunction and depression is less clear.

Depression and left hemisphere-related dysfunction

The left cerebral hemisphere is thought to be the locus for the comprehension and expression of oral and written language (Livingston, 1985; Brumback, 1988). Left cerebral dysfunction is much less commonly associated with depression (Brumback, 1988; Heller & Nitschke, 1997). Brumback (1988) contends that left hemisphere dysfunction (coined the “left hemisyndrome”) involves “varying degrees of disturbed language function.” For example, children with a severe left cerebral hemispheric hemisyndrome may evidence delayed development of language, have a poor vocabulary and relative lack of spoken language.

While left hemisphere dysfunction is not commonly associated with depression, there is evidence that anterior cortical dysfunction may or may not involve the left more than the right hemisphere. There is significant literature that supports the contention that anterior asymmetries are associated with emotional valence. “More left than right anterior cortical activity has been consistently associated with pleasant affect and happy mood states, whereas the converse has been found for unpleasant affect and sad mood states” (Heller & Nitschke, 1997, p. 643). Furthermore, research pertaining to memory and attention deficits indicates a cognitive bias in depressed persons toward unpleasant stimuli. There is also evidence this bias remits following treatment of depression.

Cognitive bias in depression fits well with the association of less left than right anterior activity for unpleasant emotional valence (Heller & Nitschke, 1997).

Individuals with depression have been shown to have impairment in anterior, frontal or “executive functions.” These functions include “judgment, planning, abstract thinking, metacognition (i.e., “thinking about thinking”), cognitive flexibility (i.e., flexibility in strategy use), ability to generate alternate strategies, verbal fluency, initiative, and motivation” (Heller & Nitschke, 1997, p. 644). The anterior regions of the brain have been shown to specialize for these behaviors. Moreover, “there are differences between the left and right anterior regions. For example, the left is more involved in verbal fluency and sequencing, whereas the right is more involved in design fluency and recency judgments” (Heller & Nitschke, 1997, p. 644). However, there are inconsistencies in related literature. For example, some contend that sequencing is a function of the right (inferior parietal lobule, supramarginal gyrus) hemisphere (Weinberg, et al., 1995).

In EEG alpha and PET studies, depression has been associated with less left than right anterior activity. However, illustrative of the inconsistencies in the literature, significant and near significant reductions in right anterior brain activity in depressed persons has been observed in blood flow studies (Heller & Nitschke, 1997).

Depressed people tend to display deficits in many of the “executive functions” or activities dependent upon anterior functioning. For example, depression has been associated with poor problem-solving, explicit memory, general learning, reading, inaccurate predictions regarding performance and feedback, poor use of organizing strategies, and poor initiative to use memory and problem-solving strategies (Heller &

Nitschke, 1997). Studies also suggest that unpleasant affect is associated with a decrease in the cognitive processes associated with frontal lobe function (Heller & Nitschke, 1997). Furthermore, there is indication that pleasant affect facilitates better use of cognitive abilities (i.e., creative problem-solving, more flexible thinking, and a more integrated organization of cognitive material). Still, Heller & Nitschke (1997) conclude that the relative contribution of the left versus the right hemisphere to the executive functions in depression is unclear. They note that decreases in bilateral anterior activity, decreased left anterior activity alone, or less left than right asymmetric activity may be related to compromised executive functions in depression. Nonetheless, Heller & Nitschke (1997) contend that there is compelling evidence regarding reduction in left anterior and right posterior activity in depression. They purport that deficits in persons with depression would be present for both hemispheres. However, the left hemisphere would be primarily anterior functions and the right hemisphere primarily posterior functions.

Overall, left hemisphere-related dysfunction is much less commonly associated with depression. However, it does appear that anterior dysfunction (specifically, deficits in executive functions) is related to depression. While research is equivocal, the left anterior region is considered to be more involved in verbal fluency and sequencing. In addition, the “cognitive bias” in depression appears to fit with research finding less left than right anterior activity for unpleasant emotional valence. Overall, while the literature on the subject is not yet conclusive, there is some support for the assertion that the anterior

dysfunction associated with depression may be more attributable to the left hemisphere versus the right hemisphere.

Symptoms of depression and cognition

Few studies have examined the relationship between specific symptoms or diagnostic subtypes of depression and cognition. Bruder, Quitkin, Stewart, Martin, Voglmaier, & Harrison (1989) examined differences in perceptual asymmetry among diagnostic subtypes. The authors found that patients with melancholia had abnormal perceptual asymmetry for dichotic nonsense syllable and complex tone tasks. However, patients who did not evidence melancholia but were diagnosed with an “atypical depression” (defined as reactivity of mood with preserved pleasure capacity and associated features), performed similarly to controls on these tasks. Patients with atypical depression did show an increased incidence of left-handedness.

Brumback (1985) and Wilson, et al. (1982, as cited in Brumback, 1988) found that children with performance IQ deficits demonstrated improvement in alertness, visual sequencing, visual perception, long-term visual retention, perceptual organization and auditory information processing as well as improvement in depressive symptoms following antidepressant treatment of depression. In contrast, depressed children with Verbal IQ deficits showed only slight to moderate improvement in neuropsychological skills (i.e., verbal comprehension, reading achievement, practical reasoning, and visual – motor coordination) and minimal change in their level of depression. According to Brumback (1988, p. 489), a possible explanation could be that depression is distinguishable into two types: “(1) a depression associated with right-cerebral

hemisphere deficits, that responds to antidepressant therapy and that also improves the associated right cerebral dysfunction and (2) a depressive disorder without evidence of right hemisphere deficit, which is poorly responsive or unresponsive to conventional antidepressant treatment.”

The symptoms of depression can be separated into psychological symptoms and vegetative symptoms (Brumback & Weinberg, 1990). Psychological symptoms include dysphoric mood, cognitive negatives (e.g., negative feelings about self, relationships, or the future), irritability, social withdrawal, guilt, anhedonia, and preoccupations with death. Somatic (vegetative) symptoms include sleep disturbance, physical complaints, appetite disturbance, weight change, fatigue, psychomotor disturbance, inconsolability, and diurnal variation in moods and symptoms (Brumback, 1988, Brumback & Weinberg, 1990). Some researchers propose that a physiological depression (depression marked by physiological symptoms) could be more strongly associated with right hemisphere cognitive dysfunction and amenable to treatment. In contrast, a depression marked by psychological symptoms (cognitive negatives, etc.) may be less associated with right hemisphere dysfunction and less amenable to treatment (Brumback, 1988).

However, others contend that it is not possible to separate symptomatology and distinguish depression into a “biological” and “psychological” depression in this manner. Moreover, it is currently held that depression is not caused by a single factor, but by an interaction among biological (e.g., serotonin receptors), cognitive (e.g., negative thinking style), and social (e.g., negative events, poor social support) factors. While some individuals may have a biological predisposition to depression, it is thought that such

individuals will not become depressed unless faced with a life stressor, or hold overly negative views of themselves, their world and the future (Arean, McQuaid & Munoz, 1997). At the same time, individuals who do not have increased genetic risk for depression may be at increased risk for depression if they display negative cognitive styles. Also, such individuals will likely show disruptions in biological functions (e.g., sleep and cortisol regulation) that are related to depression when facing negative life events. Relatedly, there appears to be an increase in the rate of depressive disorders since WW-II (Arean et al., 1997), which cannot be explained by genetic factors alone. As such, a biopsychosocial model appears the most appropriate for explaining the nature and causes of depression. However, the relationship between specific symptoms of depression and cognitive features is unclear and is an area in need of further investigation.

Differences in cognition may be dependent upon the symptom presentation of depression. It may be possible to link particular symptom categories (e.g., mood, cognitive, and vegetative symptoms) to specific cognitive processes. On the other hand, it may not be possible to categorize depression in this manner. A related question posed by the current study is whether cognitive problems predict depressive symptoms and/or depression over time or vice versa. There is little research addressing this question directly. However, while also a relatively sparse area of study, review of longitudinal studies addressing learning disabilities and depression may help to provide insight into the relationship of learning abilities, cognition, and depression over time.

Longitudinal literature regarding learning disabilities and depression

Raskind et al (1999) conducted 10- and 20-year follow-up studies of previous students from the Frostig Center (a private school for children with learning disabilities). They noted that none of the 41 subjects examined at follow-up had been diagnosed with severe emotional disturbance when they entered the Frostig center. However, they recognized that participants may not have had serious emotional disturbances but may have had psychological problems contributing to their needs for special education. The authors noted that this information was not accessible as more detailed psychological information from the initial data gathering was not available. Still, it was reported that 42% of the participants met criteria for DSM-IV psychological difficulties (e.g., depression, schizophrenia, alcohol abuse, drug abuse, social phobias, etc.) at follow-up. Most disorders emerged between years 10 and 20 of data gathering.

The authors sought to identify predictors of success and identified 6 success attributes: self-awareness, proactivity, perseverance, emotional stability, appropriate goal setting, and the presence and use of support systems. The attributes were operationalized to facilitate the study. Surprisingly, mood disturbances at follow-up were evenly distributed among successful and unsuccessful participants. Furthermore, the researchers did not find higher levels of substance abuse and criminal behavior in their population in comparison to the general population.

Esser, Schmidt, & Woerner (1990), conducted a large-scale study to evaluate the epidemiology and course of psychiatric disorders in German children. Children were examined at age 8 and 13 years and several attributes that predicted psychiatric disorders

at age 13 years were noted: psychiatric disorders at age 8 years, number of life events between ages 8 and 13 (e.g., loss of parents, change in school), and specific learning disabilities at age 8 years.

Prior, Smart, Sanson, & Oberklaid (1999) examined relationships between learning difficulties and psychological problems in preadolescent children using a longitudinal design. The researchers compared a clinical group of children with behavior problems to a comparison group without significant behavioral problems. They reported that over half of the children in the clinical group also met criteria for a learning disability, while less than 20% of the children in the comparison group evidenced learning disabilities. A learning disability was defined as an IQ of at least 80 and scores on standardized spelling and/or arithmetic below 30%. Numeracy difficulties were associated with DSM diagnoses in both boys and girls. In addition, externalizing problems tended to co-occur with a combination of learning problems. Children identified with learning disabilities at ages 7-8 years persisted with literacy difficulties (over 80%) and arithmetic problems (57%). The authors concluded that early behavior difficulties predicted the development of learning difficulties rather than the reverse. In contrast, others have suggested the reverse. LDs (i.e., reading delay) may predispose children to subsequent increases in behavior problems (Hinshaw, 1992).

In a study of the clinical significance of neurocognitive impairments among children with psychiatric disorders, Szatmari, Offord, Siegel, Finlayson, & Tuff (1990) reported differences between children with externalizing (i.e., ADHD and/or conduct disorders) vs. internalizing disorders (i.e., pure anxiety or affective disorders). Children

with externalizing disorders generally performed more poorly on neuropsychological tests than children with pure internalizing disorders. Similarly, McKinney (1989), in a 3-year longitudinal study of behavioral characteristics of children with learning disabilities, found that children with attention and conduct problems had poorer academic outcomes than children with either withdrawn/dependent behavior or normal behavior.

The school-related difficulty of children with externalizing problems do not appear to be limited to children with learning disabilities (Hinshaw, 1992). Nonetheless, in contrast to the findings of Prior et al. (1999) a reading delay may predispose children to subsequent increases in externalizing behavior (Hinshaw, 1992). Hinshaw (1992) describes a developmental progression citing a link between hyperactivity-inattention and underachievement with aggression in early and middle childhood. Aggression is believed to overlap with learning problems during these years chiefly through comorbidity with inattention-hyperactivity. As children progress to adolescence clear links have emerged between frankly antisocial behavior and variables related to verbal deficits and underachievement. Aggression at age 8 years predicted low achievement in adulthood (Huesmann, Eron, & Yarmel, 1987). Thus, while not focused exclusively on children with LD, there appears to be a clear connection between externalizing disorders and cognitive functioning, particularly left hemisphere-related verbal abilities.

Comorbid behavior and or attention problems may effect school achievement in children with learning disabilities. Children with learning disabilities who also have attention problems (low task orientation and high distractibility), and those who have behavior problems, tended to decline in achievement longitudinally (over 5 years)

compared to those without attention and behavior problems (Mckinney, Osborne, & Schulte, 1993).

In a large-scale study of developmental precursors of affective illness, van Os et al. (1997) concluded that low childhood cognitive ability predicted both childhood and adult affective disorder, the latter independent of the former. Thus, cognitive impairment was noted to be a risk factor for affective disorder and early onset of affective disturbance was demonstrated to predict a more severe, persistent disease.

There is some evidence that the psychological health of individuals with learning disabilities improves in adulthood. In a longitudinal study of 22 participants with learning disabilities, Werner (1993) found that 32% of participants demonstrated serious mental health problems in adolescence and 27% evidenced delinquency records while none of the controls evidenced such problems. However, by age 32, less than 10% had a record of persistent mental health problems. Furthermore, less than 10% had criminal records as adults. Of the 4 individuals with persistent psychiatric problems, 2 women evidenced such problems at ages 10 and 18. Two men who were offspring of alcoholics evidenced persistent criminal records.

In sum, few studies have examined the longitudinal relationship of learning disabilities and depression. However, there appears to be a high number of psychological problems in individuals with learning disabilities followed in a longitudinal manner. Furthermore, studies have supported the contention that learning disabilities predict psychiatric disorders with aging. Also, low childhood cognitive ability has been shown to predict both childhood and adult affective disorders, independently.

On the other hand, some contend that behavior difficulties predict learning disabilities. Specifically, there appears to be a connection between externalizing problems (e.g., Attention Deficit Hyperactivity Disorder, conduct disorders) and cognitive functioning (i.e., verbal deficits, academic underachievement) in children with and without learning disabilities. Furthermore, children with co-occurring externalizing disorders and learning disabilities may be more likely to have poorer academic achievement than children with co-occurring internalizing disorders (withdrawn/depressed behavior).

The life trajectory of persons with learning disabilities and mood disturbances is clearly in need of further study. There is some evidence for equal levels of success in adulthood for learning-disabled persons with and without mood disturbance. In addition, mental health problems in persons with LD may reach a peak in adolescence and decline in adulthood. On the other hand, mood disturbance may persist into adulthood.

Structural Equation Modeling (SEM)

Structural equation modeling (SEM) was used for analysis of the cross-sectional data and a general description of SEM is provided below. SEM has also been termed causal modeling, covariance structure analysis, path analysis, and linear structural relations and is a method of analysis that tests the structural theories of a phenomenon (Neumann, 1994). “A structural theory is intended to represent the “causal” process that gives rise to the correlations among the observed variables” (Bentler, 1988, p.317). Furthermore, the intent of causal modeling is to rule out implausible causal connections. As methods for testing theories are not well-developed in quasi-experimental and

nonexperimental research, causal modeling has been stated to have great potential (Bentler, 1980).

With SEM a distinction between measured (manifest) (MVs) and latent variables (LVs) is made. An LV represents a theoretical construct (e.g., right hemisphere-related cognitive function) and represent MVs (e.g., scores on right hemisphere-related measures). The correlations among the observed variables (MVs) are hypothesized to be attributable to the LVs (Bentler, 1980). Thus, LV models can represent the common factor variance separate from the error/unique variance in a set of MVs that represent a particular theoretical construct. In contrast, in MV causal models bias can result due to the level of measurement error contained in each variable (Bentler, 1980).

A two-tiered process to performing SEM has been suggested (Anderson & Gerbing, 1988). First, the measurement model, a confirmatory factor model, is used to specify the relations of the observed measures (MVs) to their posited theoretical construct (LVs). This allows confirmation of the observed variables used to measure each latent construct. Once it is ensured that the measured variables are good representatives of the latent constructs (LVs), hypothesized causal relational pathways between latent constructs are included in the model (termed the structural model). Thus, the structural model specifies the causal relations among the constructs, as proposed by a particular theory.

In SEM, the primary statistical problem is to estimate the unknown parameter values of the model as well as determine the goodness of fit of the model to the sample data of measured variables (Bentler, 1980). Goodness of fit indices (e.g., chi-square,

normed-fit-index, etc.) indicate how likely it is that the proposed model of LVs represent the causal structure of the MVs. If there is a good fit, the model is a plausible representation of the hypothesized causal structure of the observed variables. For more information regarding how SEM was applied to this study, see the results section below.

Statement of Problem

The frequent comorbidity of depression and learning disabilities may be due to a shared underlying brain dysfunction that puts individuals at greater risk for both disorders. Moreover, given that cortical functions appear to be implicated in learning disabilities, the study of the association between cognition and depression in individuals with learning disabilities has the potential to reveal valuable information. Cognitive problems associated with depression include impaired memory, attentional dysfunction and increased ability to recall and attend to information congruent with negative mood. Moreover, an association between right posterior hemisphere dysfunction and depression has been consistently shown in the literature. Depressed individuals have shown impairment on right hemisphere functions including impairment on non-verbal Wechsler IQ subtests, visual-spatial skills, perceptual processing, psychomotor skills, ability to recognize emotionality in others, and auditory perception.

Research has also implicated anterior dysfunction (deficits in executive functions) in depression. While such research is equivocal, the left anterior region is considered to be more involved in verbal fluency and sequencing and less left than right anterior activity for unpleasant emotional valence has been observed. Also, absence of positive affect has been associated with depression and left anterior dysfunction. There is some

support for the assertion that the anterior dysfunction associated with depression may be more attributable to the left hemisphere versus the right hemisphere.

The relationship of particular symptoms of depression and cognition is unclear. Specific symptoms of depression may predict cognitive functioning and vice versa. Thus, symptom categories (e.g., mood, cognitive, and vegetative symptoms) may be linked to specific cognitive impairments. However, it is also possible that cognition may only be related to the global syndrome of depression.

While research appears to lean toward the contention that treatment of depression improves cognitive functions, there is not yet a general consensus regarding this matter. Furthermore, it is unclear whether these cognitive problems predispose a person to depression. Some studies have supported cognitive dysfunction and learning disabilities as predictors for mood disturbance. Others contend that mood difficulties predict cognitive dysfunction. Several studies have cited a connection between externalizing disorders and poor cognitive functioning over time in individuals with and without learning disabilities. In addition, internalizing disorders (e.g., depression) may have less negative impact over time. Therefore, further investigation of this developmental trajectory is needed.

Hypotheses/Research Questions

Hypotheses of the study are as follows: (1) Using structural equation modeling (SEM) to examine associations, performance on right hemisphere-related cognitive measures of perceptual organization will show significant inverse associations with symptoms of depression; (2) Using SEM to examine associations, left hemisphere-related

cognitive dysfunction (not including specific left anterior dysfunction) will be minimally related to depression; (3) Using SEM to examine associations, deficits in processing speed/attention will be inversely associated with symptoms of depression. See below for a listing of specific dependent measures to be used for the particular cognitive domains discussed in these hypotheses.

Longitudinal research questions of the proposed study are as follows: (1) Do symptoms of depression in childhood predict cognitive functioning in adulthood?; (2) Does cognitive dysfunction in childhood predict symptoms of depression in adulthood?; (3) Do cognitive difficulties associated with depressive symptoms and/or depression (i.e., based upon past literature and evaluation of time 1. data) persist over time? (4) Do these associated cognitive problems occur only in those who have persisted with depression as adults?

CHAPTER 2.

METHOD

Participants

Time 1. 227 students were admitted to the Winston School of Dallas between 1975 and 1980. In the fall of 1975 the Winston School of Dallas, Texas was established to study and educate children of at least average intelligence with severe specific learning disabilities. Prior to admission, each individual underwent an extensive battery of tests (see below). All 227 students who entered the Winston School during its first five years (1975-1980) of existence participated in the study. At entrance, the students ranged in age from 6 years, 0 months to 15 years, 11 months. The student population was 82% male and 18% female, with a racial mixture of 94% Caucasian, 4% African American, and 2% Hispanic. The socioeconomic status of the families of the students was determined by the Hollingshead Index of Social Position with 68% of the families being in the upper and upper-middle class (Weinberg, McLean, Snider, Rintelmann, & Brumback, 1989b).

Ten percent of enrolled students were adopted. Seventy-five percent had failed in other private or public schools. This population was determined to have severe, multiple learning disabilities as determined by Public Law 94-142. Most common was difficulty with oral expression, basic reading and written expression. Many students had difficulties with mathematical calculations and reading comprehension. Less common was difficulty in listening comprehension and least common was difficulty with mathematical reasoning. Seventy-one percent of the enrolled student body met established criteria for

depression (Weinberg et al., 1973). An additional 15% had other forms of behavioral disturbance (e.g., hyperactivity). Early analysis of the data suggested a relationship between family and personal history for affective illness and learning disability with student school failure (Weinberg & Rehmet, 1983; Weinberg et al., 1989b).

Time 2. Twenty-three (10 %) of the 227 students were randomly selected. An additional 17 non-randomized, local ex-students also participated. These participants were recruited due to the convenience of their geographic location. The subjects underwent an 8-10 hour battery of testing consisting of clinical interview, general and neurological examinations including evaluation of higher cortical functions, standardized intelligence and achievement testing and subjective surveys (Learning Disabilities of America survey and Satisfaction with Life Scale). Subjects were assigned DSM-IV diagnoses based upon responses to interview and surveys. Standardized tests were chosen to be most comparable to those used in 1975-1980. Evaluations were completed between February 1999 and February 2000.

Materials

Time 1. Measures of Depression. Comprehensive semi-structured interviews of primary caretakers and their children were conducted by the medical director of the Winston School, a pediatric behavioral neurologist. The interviewer, an established clinician with many years of experience interviewing children and their caretakers, was the first to establish criteria for depression in children (Weinberg et al., 1973). Interviews included detailed evaluation of mood and affect according to the Symbol Language Communication Battery (SLCB) (Weinberg, Harper, & Brumback, 1998) and established

diagnostic criteria for depression (Weinberg et al., 1973; Weinberg & Rehmet, 1983; Weinberg et al., 1998). History of any depressive symptoms, including onset, duration, frequency and severity was obtained. Family history of any mood difficulty was obtained along with general developmental history. General pediatric and neurological examinations were also conducted. Based upon interview as well as parental questionnaires (see below), diagnoses were made and specific symptoms identified by the examiner.

Primary caretakers completed mood and behavior questionnaires including the Affective Behavior Questionnaire (ABQ) and Child Development Study (CDS) regarding their children. The questionnaires were both developed by Warren A. Weinberg, M. D. and are based on clinical experience with referred and non-referred populations. The Affective Behavior Questionnaire contains 46 items and uses a likert scale response format consisting of the following: 0 = no problem, 1 = mini problem, 2 = past problem, now absent, 3 = present problem, first episode, 4 = present problem, 2nd or more episode, and 5 = present problem, longstanding. Respondents identified which rating best described their child in relation to the behavior or trait presented. Items include many mood symptoms of depression (e.g., statements or appearance of sadness, loneliness, unhappiness, and/or pessimism, socially withdrawing), cognitive symptoms of depression (e.g., feelings of being worthless, useless, dumb, etc.) Vegetative symptoms of depression presented include such items as “trouble falling to sleep,” “difficulty awakening in the morning,” and “stomach aches,” etc.

The CDS contains 108 items and also employs a likert scale response format: 0 = no problem or trait, 1 = problem or trait in past only, 2 = problem or trait which is improving, 3 = problem or trait which is persisting, 4 = problem or trait which is becoming worse. Similarly to the ABQ, many items relate specifically to depression. For example, mood items include “unhappy, sad, depressed,” moody, sad one day and happy the next without cause,” etc. Cognitive symptoms include “feels he is not as good as others,” “poor memory,” etc. Lastly, vegetative symptom items such as “trouble in sleeping,” “trouble around eating,” and “bed wetting” are included.

Of note, these questionnaires are not standardized and norming data has not been developed. The idea that depression could exist in children was not widely accepted (Weinberg et al., 1973) at the time of the initial study. Thus, there was a lack of normed questionnaires for depression at that time.

Select items of the Affective Behavior Questionnaire (ABQ) and the Child Development Study (CDS) questionnaires were used to create scales in order to examine specific depressive symptom factors. Previous factor analytic work with various personality measures (Byrne, 1988; Marsh, 1994; Marsh, Smith & Barnes, 1985; Marsh & O’Neill, 1984), as well as other instruments (Bagozzi & Heatherton, 1994; Greenbaum & Dedrick, 1998), has relied upon item composites, sometimes referred to as parcels, rather than all single items as indicators for latent variables (LVs). The use of parcels, versus single items as indicators for LVs, has been used in other CFA studies because parcels: (a) tend to be more reliable and valid indicators of LVs, (b) are less skewed than individual items, and (c) reduce the number of parameters that have to be estimated, thus

improving the ratio of the number of estimated parameters to the number of subjects (Bagozzi & Heatherton, 1994; Marsh, 1994).

ABQ and CDS items pertaining to depressive mood (e.g., sadness, dysphoria, anhedonia) were selected to form parcels and served as MVs for the mood depression factor. Likewise, items pertaining to vegetative symptoms (e.g., sleep problems, appetite problems, low energy, somatic complaints) were included in composites and served as MVs for the vegetative depression factor while items pertaining to cognitive symptoms of depression (e.g., low self-esteem, poor concentration) were selected for parcels and MVs for the cognitive depression factor. In order to maximize the number of subjects entered in the model, means of 3 to 8 items were utilized to form the parcels. See Appendix A, Table 2. for scale items, mean number of items per scale, and alpha coefficients.

Time 2. Measures of Depression. A similar interview including detailed history of mood symptoms, developmental history, family history and administration of the SLCB was obtained. The clinician who performed the interviews at time 2 was thoroughly (over a 2 – year time period) trained by the time original Winston School medical director to conduct interviews. Brief neurological examinations and interviews regarding current mood status were conducted by a trained neurologist. The Diagnostic Interview Schedule for the DSM-IV (DIS-IV) was administered by graduate students in clinical psychology. The original Diagnostic Interview Schedule was developed by Helzer, J.E. & Robins, L.N. The most current version (DIS-IV) was developed by Robins, Cottler, Bucholz, and Compton (1995). The DIS-IV is a structured interview consisting of questions relating to specific criteria for DSM-IV disorders. Thus, it allows

for differential diagnosis of such disorders as major depressive disorder and dysthymia. Questions also provide information regarding the number, length and duration of episodes of depression. While there are insufficient studies regarding the reliability of the individual symptoms of the DIS-IV, the reliability of diagnosis and lifetime diagnosis is average or comparable to other diagnostic interviews. Diagnostic validity is also average while convergent validity is weaker than other measures. However, the validity of longitudinal measures such as course of the disorder is stronger than other diagnostic interviews (Rogers, 1995). Consensus diagnosis of each patient was made regarding each patient by the examiners.

The Personality Assessment Inventory (PAI) was administered to participants who were followed-up. The PAI was developed by Leslie C. Morey, Ph.D. and is an objective, self-report measure of personality consisting of 344 items and 22 non-overlapping full scales (Morey, 1991). The PAI includes 4 validity scales, 11 clinical scales, 5 treatment scales, and 2 interpersonal scales. The PAI, in contrast to measures such as the Minnesota Multiphasic Personality Inventory (MMPI), is a theory-based measure geared toward the identification of psychopathology. The scale most pertinent to this study is the depression scale that consists of 24 items and focuses on symptoms and phenomenology of depressive disorders. This scale includes 3 subscales with 8 items each. The subscales are cognitive depression, affective depression, and physiological depression (Morey, 1991).

Internal consistency alphas for the scales included on the PAI are high with median alphas for the full scales of .81, .86, and .82 for the normative, clinical, and

college samples, respectively. In regards to the depression scale, alpha coefficients are .87, .93, and .87 for the normative, clinical, and college samples, respectively. Subscale alpha coefficients for cognitive depression are .74, .84, and .78; for affective depression, .80, .88, and .79; for physiological depression .71, .80, and .64, for the normative, clinical, and college samples, respectively. Test-retest reliability of the PAI is good with a median correlation over time of the clinical scale configuration of .83 (Morey, 1991). Numerous validity studies included the use of 5 validation samples (one clinical, two community, and two college student) and several validation measures (e.g., NEO Personality Inventory, MMPI clinical and content scales, personality disorder scales, etc.). Results of validity studies indicate the PAI has good validity (Morey, 1991).

The Weinberg Depression Scale for Children and Adolescents (WDSCA) was administered as a quick measure of depression. It was developed by the initial investigator of this study (Weinberg, W.A.). Though norm-referenced for children and adolescents ages 7-0 through 18-11, it has good reliability and validity and was considered appropriate as a conjunctive measure of depression (Weinberg, Harper, & Emslie, 1998). The WDSCA includes 56 yes/no items and matches questions to two sets of established criteria for depression: the Weinberg criteria and DSM-IV criteria.

The Hamilton Depression Inventory (HDI), another short measure of depression, is described as a screening measure of depression. The HDI has been shown to be useful in identifying depression in adults (Beckham & Leber, 1995).

Time 1. Cognitive Measures. Wechsler Intelligence Scale for Children – Revised (WISC-R). The Wechsler scales were at the time of initial study, and continue to be, the

most widely used tests of intellectual ability (Sattler, 1992). The WISC-R provides norms for children ages 6-6 to 16-11 years and has outstanding reliability and good validity (Wechsler, 1974). It provides an overall measure of intellectual ability (full scale IQ) as well as indication of how well an examinee reasons with words (verbal IQ) and without words (performance IQ). IQ raw scores are converted to standard scores consisting of a mean of 100 and standard deviation of 15. Subtest raw scores are converted to standard scores with a mean of 10 and standard deviation of 3.

Specific verbal subtests include Information (range of knowledge), Similarities (verbal comprehension), Arithmetic (numerical reasoning/concentration), Vocabulary (language development), Comprehension (social judgment), and Digit Span (short-term memory). Performance subtests include Picture Completion (perceptual organization), Picture Arrangement (nonverbal reasoning/sequencing), Block Design (spatial visualization), Object Assembly (spatial relations), Coding (visual motor coordination), and Mazes (visual-motor control).

The Wide Range Achievement Test (WRAT) was initially developed by Joseph Jastak in the 1930s (Wilkinson, 1993). This version was utilized during time 1 data collection. The WRAT consists of 3 subtests: reading, spelling, and arithmetic. The reading subtest includes recognizing and naming letters and pronouncing words out of context. Spelling involves writing names, letters and words to dictation while the arithmetic subtest consists of counting, reading number symbols, solving oral problems, and performing written computations.

The Peabody Individual Achievement Test (PIAT) was developed in 1970 by authors Dunn & Markwardt. It is an individually administered achievement test consisting of 5 content areas: General Information, Reading Recognition, Reading Comprehension, Mathematics, and Spelling. The PIAT differs from other achievement tests (i.e., WRAT) in that two subtests (Spelling, Mathematics) utilize a multiple choice format in order to evaluate examinee's recognition skills.

The Gilmore Oral Reading Test was developed by Gilmore & Gilmore (1968). It is an individually administered test of oral reading and reading comprehension. The Gilmore includes two equivalent forms, form C and form D. Form C was utilized in this study. The test includes ten oral reading paragraphs that are commensurate with respective 1-10 grade levels. Performance ratings for accuracy, comprehension, and rate are provided. The test was standardized in 1967 on 4455 students in six school systems. Validity studies demonstrated high correlations between the Gilmore and other reading tests (e.g., Gray Oral Reading Test and WRAT reading). Test-retest reliability as well as alternate forms reliabilities were reported to be good (Gilmore & Gilmore, 1968).

Participants were asked to draw a clock stating "10 minutes until 2 O'clock." In addition, participants were asked to "draw a person but not a stick figure." These measures were utilized as indicators of visual-spatial skills.

The Developmental Test of Visual-Motor Integration (VMI) consists of 24 geometric forms to be copied with pencil and paper by children ages 2-15 years. The forms are presented in order of increasing difficulty. Validity of the measure is reported

to be good (Beery, 1967) and correlations as high as .89 have been cited between VMI performance and mental age.

Time 2. Cognitive Measures.

The Wechsler Adult Intelligence Scale, 3rd Edition (WAIS-III), the adult version of the WISC, is presently the most widely used test of intellectual ability. In line with the WISC, the WAIS-III has excellent reliability and validity and provides a measure of both verbal and performance IQ (Wechsler, 1997). The subtests are the same as those included in the WISC-R, excepting the performance subtest Mazes. Also, the subtest Object Assembly, while required on the WISC-R, is an optional subtest on the WAIS-III and does not factor into overall intellectual quotients. The performance subtest Matrix Reasoning, is not included on the children's version of the Wechsler scales.

The WRAT-3 is the most current version of the WRAT. As with the WRAT, subtests include reading, spelling, and arithmetic. The standardization process of the WRAT-3 included the testing of nearly 5,000 people and the test is an age-normed referenced test for individuals ranging from age 5 to 74 years, 11 months. Raw scores are converted to standard scores with a mean of 100 and standard deviation of 15. According to the test manual (Wilkinson, 1993) reliability and validity of the WRAT-3 is good.

The PIAT-Revised (Markwardt, 1989) differs from the original PIAT in that a 6th subtest, Written Expression was added and norms were updated. In addition, more items were added as well as more contemporary item content. Correlation studies of the PIAT and PIAT-R reflect that PIAT-R subtests tend to correlate most highly with its PIAT counterpart. The four methods utilized to estimate reliability of the PIAT-R were split-

half, Kuder-Richardson, test-retest, and item response theory. According to studies utilizing these methods, reliability of the PIAT-R subtests and composite scores is good. Validity of this measure was also reported to be good (Markwardt, 1989).

The same version of the Gilmore Oral Reading Test utilized at time 1 was administered at time 2. This test has not been revised since the original version published in 1968. See above for further information.

As at time 1, participants were asked to draw a clock stating “10 minutes until 2 O’clock.” In addition, the Draw-a-Person test was administered.

Procedure

Time 1. As part of the evaluation model, the medical director of the Winston School Evaluation Center, a pediatric behavioral neurologist, clinically evaluated each prospective student. The comprehensive evaluation included detailed developmental, medical, educational, and family histories, complete pediatric and neurological examinations and evaluation of higher brain functions using the Symbol Language and Communication Battery (Weinberg, et al., 1998). Special attention was given to each student’s personal and family history for affective illness and diagnostic specific learning disabilities. Psychometric/ educational evaluations using multiple standardized, individually administered measures of cognitive and school achievement functioning (see above) were administered by trained school diagnosticians. A detailed evaluation of the child’s mood and feelings, volition, diligence, activity level, ability to maintain attention, social skills, social communication and character was conducted by the medical director.

Unique, as part of this comprehensive evaluation, was the detailed evaluation for affective illness in each child and the child's family history for affective illness. The Weinberg Criteria for Depression (Weinberg et al., 1973) and Mania (Weinberg & Brumback, 1976) were used. These criteria were developed in parallel to the Research Diagnostic Criteria (RDC) for psychiatric illnesses in adults.

The mean Wechsler Intelligence Scale for Children, Revised (WISC-R) Full Scale IQ was 97 with a standard deviation of +/- 14.4. The Severe Learning Discrepancy Model (significant disparity between achievement and intellectual ability with achievement at or below 50% of expected grade level achievement) was utilized to determine learning disability diagnosis on 224 of the 227 students (incomplete information was available on 3 of the students). "As defined by P.L. 94-142, the seven basic school skills (basic reading skills, reading comprehension, written expression, oral expression, listening comprehension, mathematical calculation, and mathematical reasoning) were assessed using standardized achievement tests (see above). One or more learning discrepancies were present in 212 (95%) students, with 169 (76%) students having multiple discrepancies. The average number of discrepancies per student was 2.6, and only 12 (5%) students did not qualify as having a severe learning discrepancy. Most of the severe learning discrepancies occurred in basic reading skills (58%), written expression (57%), and oral expression (57%), while severe learning discrepancy for mathematical reasoning occurred infrequently (7%)" (Weinberg, et al., 1989b).

As stated above, all students were examined for mood disturbance including depression, mania, hyperactivity, conduct disorder, thought disturbance, and other

behavioral and emotional problems. The following behavioral diagnostic categories were used for students entering the school. The number of children in each category is shown in parentheses following the category name: depression without hyperactivity (82), depression and hyperactivity (79), hyperactivity without depression (34), and “mini” depression or no mood disturbance (32).

Time 2. Forty adults from the original 227 students were studied from February 1999 through February 2000. Participants were initially recruited by telephone by one of the sub-investigators at which time a brief description of the research (e.g., “a longitudinal study of Winston School students is being conducted to determine how students are doing following their education and as adults”) was given. Potential subjects were then asked if they were interested in hearing more about the study and details regarding participation. The caller next described the procedure (where the testing would take place, what kinds of testing to expect, length of testing, incentives) and a time for testing was scheduled. Prior to testing, subjects were read the consent form (see Appendix A). Testing began following signed consent by the subject.

All testing was completed at the Winston School Evaluation and Testing Center. Participants usually completed the 8-10 hours of testing in one day. However, two days of testing were scheduled if needed due to scheduling constraints or the need for additional time. Standardized intellectual and achievement measures were administered to each subject by one of two Master’s level trained educational diagnosticians. Next, the semi-structured interview (similar to the interview conducted at time 1) was conducted by a clinician with a bachelor’s degree in psychology. This clinician had undergone two

years of training with the original medical director of the study. The Personality Assessment Inventory (PAI), Hamilton Depression Inventory (HDI), and the Weinberg Depression Scale for Children and Adolescents (WDSCA) were administered by the same clinician. Each participant underwent a brief neurological examination by a trained pediatric neurologist. In addition, participants were administered the Diagnostic Interview for the DSM-IV (DIS-IV) by either a Clinical Psychology graduate student with a master's degree in psychology or by a graduate student in Clinical Psychology. Both these clinicians were trained in the use of diagnostic interviews and were highly familiar with the administration process of the DIS-IV. While the order that the intellectual and IQ tests were administered remained consistent (i.e., WAIS-III, WRAT-3 Reading Recognition, Spelling, PIAT, and WRAT-3 Math Calculation), the order the semi-structured interview, administration of mood and personality measures, neurological examination, and administration of the DIS-IV, varied at times due to the time availability of the clinicians involved. Following completion of testing, participants each received a check for \$100.00 and a gift bag containing pens, mints, and photographs of the subjects as students at the Winston School.

Right Hemisphere Manifest Variables

As reviewed above, depressed individuals have shown impairment in Wechsler non-verbal (performance) IQ subtests. Based on extensive factor analytic research, the subtests Block Design and Object Assembly appear to be most representative of right hemisphere perceptual organization skills (Wechsler, 1997). These variables consisted of

a right hemisphere factor that was utilized to examine the association of depression and right hemisphere cognitive skills.

The inclusion of a “processing speed” factor was initially proposed to include subtest Symbol Search in addition to Coding. However, the WISC-R does not include a Symbol Search subtest. Further review of the literature regarding WISC-R/WAIS-III factors (Allen & Thorndike, 1995) supported the use of a composite consisting of Digit Span and Coding. The Digit Span inclusion has altered the composite to reflect attention/working memory.

Left Hemisphere Manifest Variables

Verbal comprehension is generally considered to be a left hemisphere function. The Wechsler subtests Similarities and Comprehension are representative of verbal abilities, specifically verbal comprehension. While Information, Vocabulary, and Arithmetic also represent verbal abilities, these subtests are likely to be directly affected by learning disabilities. Thus, Similarities and Comprehension composed the left hemisphere factor.

Manifest Variables of Depressive Symptoms

In order to evaluate whether symptoms of depression can predict cognitive functioning and vice versa, symptoms of depression were divided as follows: mood symptoms, cognitive symptoms, and vegetative symptoms. Symptoms were divided in this manner in order to explore the proposition that depression can be differentiated as a primarily “physiological” (vegetative symptoms) and “psychological” (mood symptoms) depression. Cognitive symptoms may also prove to be a meaningful psychological

category. The Affective Behavior Questionnaire (ABQ), the Child Development Study (CDS) questionnaire, and record of symptoms/diagnosis of depression based on clinical interview were utilized to represent these variable domains.

Data Analysis

SEM is a method of causal modeling analysis. Using SEM, the researcher is able to determine the plausibility of proposed theoretical models within a particular population. Specifically, structural equation modeling tests the specified causal structure among a set of constructs. EQS, developed by Bentler (1989), uses generalized least squares (GLS) procedures or maximum likelihood procedures to examine patterns of relationships among latent variables. The SEM analyses for this study were performed using the EQS Structural Equations Program published by Multivariate Software, Inc. EQS incorporates a mathematical and statistical approach to the analysis of linear structural relationships using matrix algebra and includes parameter estimates and several goodness of fit indices. EQS generates a matrix specification and designates independent and dependent variables in accordance with the representation system of Bentler and Weeks (1980).

EQS provides several goodness of fit indices. These fit indices assess the degree of “fit” of a proposed model to the sample data. The chi-square test statistic provides a test of the null hypothesis. Thus, model fitting is a matter of “how robustly one can accept the null hypothesis.” A small chi-square and large p value would indicate that the null hypothesis is correct and the model is a good representation of the causal structure of the observed data. Unfortunately, the chi-square test is affected by sample size.

Therefore, with larger samples, non-meaningful discrepancies between the model and data may result in false positive rejection of the model. Due to such limitations, Bentler (1989) suggests the use of other goodness of fit indices.

Thus, three additional indices were also used to address goodness of fit. These indices are the normed fit index (NFI; Bentler & Bonnett, 1980), the nonnormed fit index (NNFI; Bentler & Bonnett, 1980) and the comparative fit index (CFI; Bentler, 1980). These fit indices tend to provide an accurate assessment of how well the model fits the data, regardless of sample size. Values over .9 are viewed as desirable as they indicate good fit. Small values (<.70) indicate poor fit of the model to the data.

The Root Mean Square Residual (RMR) and the Root Mean Square Error of Approximation (RMSEA) are also available estimates of model fit. The RMR is the square root of the average squared difference between each sample and estimated covariance divided by all of the variables in the covariance matrix (Ullman, 1996). Small RMR values are associated with better fit. However, RMSEA provides an estimation of the lack of fit of the model to the estimated population covariance matrix (Browne & Cudeck, 1993). Therefore a value of zero would indicate that the model fits exactly and values of 0.05 or less would indicate a close fit of the model.

Maximum Likelihood (ML) is the predominant procedure used for parameter estimation (Anderson & Gerbing, 1988). The ML procedure has been shown to be robust over normality violations (Hu & Bentler, 1998; Satorra & Bentler, 1986). Nonetheless, violations of multinormality may affect the standard error (SE) estimates of parameters, as well as the chi-square estimates (Anderson & Gerbing, 1988). Thus, robust estimates

of parameters and their SEs provided by the EQS program (Bentler, 1995) were used to determine parameter significance.

CHAPTER 3.

RESULTS

Sample Characteristics

Two hundred twenty seven students were entered into the study upon admission to the Winston School during 1975-1980. Data was available for 212 of the students. At entrance, the students ranged in age from 6 years, 3 months to 16 years. The study population is 83% male and 17% female. Specific ethnicity and class information by subject was not available on the 212 students. However, the total population (N=227) has been cited (Weinberg et al. 1989b) as predominantly Caucasian (94%) with 4% of the students African American and 2% Hispanic. Furthermore, the socioeconomic status of the families of the students was stated to be middle to upper class with 68% falling in the upper and upper-middle class range. It is likely that the present study population (N=212) is similar to that for the population of 227 students. The gender, age, time 1 behavior subtype, time 2 mood diagnosis, and learning disability subtype for both time 1 and time 2 study populations are presented in Appendix A, Table 1.

Affective Behavior (ABQ) & Child Development Study (CDS) Questionnaires

Given the lack of normative data regarding these measures, it was necessary to develop a method of organizing item responses in order to examine depressive symptom categories. Thus, select items of these questionnaires were used to create mood, cognitive, and vegetative symptom scales. Internal consistency of selected scale items

was examined and alpha coefficients for the scales range from .72 - .84. See Appendix A, Table 2. for included items and scale reliability coefficients.

Cognitive Composites

Cognitive composite variables were defined as follows:

VERBAL1: Mean of WISC-R Similarities & Comprehension

VERBAL2: Mean of WAIS-III Similarities & Comprehension

VISUAL1: Mean of WISC-R Block Design & Object Assembly

VISUAL2: Mean of WAIS-III Block Design & Object Assembly

Attention/Memory 1 (ATTN/MEM1): Mean of WISC-R Coding & Digit Span

Attention/Memory 2 (ATTN/MEM2): Mean of WAIS-III Coding & Digit Span

Initially, supplementary analyses including a learning disability factor were proposed. However, it was deemed appropriate to examine a model including expanded factors. The expanded composite variables intentionally include Wechsler subtests likely to be implicated by learning disabilities (LD) (i.e., information, vocabulary, and arithmetic), in order to examine the presence of possible LD effects. The expanded cognitive composite variables were defined as follows:

Verbal Expanded 1 (VERBALE1): Mean of WISC-R Information, Vocabulary, Similarities & Comprehension

Verbal Expanded 2 (VERBALE2): Mean of WAIS-III Information, Vocabulary, Similarities & Comprehension

Visual Expanded 1 (VISUALE1): Mean of WISC-R Picture Completion, Picture Arrangement, Block Design & Object Assembly

Visual Expanded 2 (VISUALE2): Mean of WAIS-III Picture Completion, Picture Arrangement,

Block Design & Object Assembly

Attention/Memory Expanded 1 (ATTN/MEME1): Mean of WISC-R Coding, Digit Span, & Arithmetic

Attention/Memory Expanded 2 (ATTN/MEME2): Mean of WAIS-III Coding, Digit Span, & Arithmetic

Mood Composites

Mood composites were defined as follows:

Mood Depression 1 (MD1): Mean of mood depression scales of ABQ & CDS

Cognitive Depression 1 (CD1): Mean of cognitive depression scales of ABQ & CDS

Vegetative Depression 1 (VD1): Mean of vegetative depression scales of ABQ & CDS

Personality Assessment Inventory Affective (PAIA): Personality Assessment Inventory affective depression subscale t score

Personality Assessment Inventory Cognitive (PAIC): Personality Assessment Inventory cognitive depression subscale t score

Personality Assessment Inventory Physical (PAIP): Personality Assessment Inventory physical depression subscale t score

Personality Assessment Inventory Depression (PAID): Personality Assessment Inventory depression scale t score

Descriptive Analyses

Descriptive statistics from the present study are provided, including mean and standard deviations for the cognitive composites (see Appendix A, Table 3) and mood composites (Appendix A, Table 4). In addition, the distributional properties of each measured variable (including those variable merged to form composites) were examined for normality for both univariate and multivariate analysis. Only one variable, the ABQ vegetative depression scale was noted to be minimally kurtotic. Likewise, multivariate kurtosis was in the acceptable range for Model 1 variables and minimally kurtotic for Model 2 variables. All other variables appeared to be in acceptable normal distribution range.

Results of the study will be presented in the following manner. First, correlation results will be outlined. Next, results of structural equation modeling (SEM) and the testing of the time 1 hypotheses will follow. Lastly, the results of time 2 data analyses including examination of research questions will be presented.

Correlation matrices were run to examine the relationships between measured variables. As would be expected, time 1 cognitive composite variables were highly correlated with time 2 cognitive composite variables (i.e., VERBAL1 and VERBAL2, $r = .577$, $p = .000$.; VISUAL1 and VISUAL2, $r = .627$, $p = .000$; ATTN/MEM1 and ATTN/MEM2, $r = .439$, $p = .006$; VERBALE1 and VERBALE2, $r = .646$, $p = .000$; VISUALE1 and VISUALE2 $r = .760$, $p = .000$; ATTN/MEME1 and ATTN/MEME2, $r = .547$, $p = .000$). Likewise, significant relationships were noted between the following time 1 and time 2 mood composite variables: MD1 and PAIC, $r = .395$, $p = .025$; CD1 and PAIC, $r = .474$, $p = .006$; MD1 and PAIP, $r = .419$, $p = .017$; CD1 and PAIP, $r =$

.402, $p = .022$; MD1 and PAID, $r = .416$, $p = .018$; CD1 and PAID, $r = .465$, $p = .007$ (tables 5-6). No significant correlations between time 1 cognitive composites and time 1 mood composites or between time 2 cognitive composites and time 2 mood composites. However, when the composite consisting of cognitive depression was restricted to consist of items that did not reflect attention problems (e.g., attention deficit hyperactivity disorder), a significant correlation was observed between cognitive symptoms of depression and the attention/working memory expanded composite (CD1 (without attention problem symptoms) and ATTN/MEME1 $r = -.194$, $p = .016$).

No significant correlations were found between time 1 cognitive composites and time 2 mood composites or time 1 mood composites and time 2 cognitive composites (N=40) when examining the study population. This finding may have been due to the manifest variables and the resulting bias that can occur due to measurement error. Subpopulation analyses were conducted by comparing correlation matrices and relationships of interest were noted. For males (N = 32) not females (N = 8), moderate relationships were observed between PAI depression subscales and time 2 WAIS-III subtests Similarities and Comprehension (PAIP and VERBAL2, $r = -.439$, $p = .012$; PAID and VERBAL2, $r = -.368$, $p = .038$). When expanded left cognitive subtests (VERBALE2) were examined this relationship was not of significant strength. For females (N=7), but not males (N= 32), a strong relationship was observed between time 2 attention working memory and time 1 mood depressive symptoms (ATTN/MEM2/ ATTN/MEME2 and MD1, $r = -.766$, $p = .044$; $r = -.791$, $p = .034$ respectively).

However, this result should be interpreted with caution due to the very low number of females.

In subjects who received diagnoses of depression (with or without hyperactivity, N=29), a moderate relationship between left cognitive subtests and PAI scales (i.e., VERBAL1 and PAIC, $r = -.385$, $p = .039$; VERBAL1 and PAIA, $r = -.415$, $p = .025$; and VERBAL1 and PAID, $r = -.394$, $p = .035$) was observed. These relationships were not observed in subjects with depression only (N = 20), in subjects without depression nor with the expanded left cognitive subtests (VERBALE1). For those with hyperactivity (with or without depression, N = 12) a strong relationship was noted between VERBAL1 and PAIC ($r = -.714$, $p = .009$) as well as ATTN/MEM1 and PAIA ($r = -.744$, $p = .006$). However, given the low N of this group, results will be interpreted with caution. The following relationships were observed in children without behavior problems (N = 26) (VISUAL1/VISUALE1 and MD1, $r = -.471$, $p = .015$; $r = -.513$, $p = .007$, respectively). A relationship between attention/working memory and depressive symptoms was also observed in children without behavior problems (N = 23) (ATTN/MEM1/ATTN/MEME1 and MD1, $r = -.556$, $p = .006$; $r = -.546$, $p = .007$; ATTN/MEM1 and CD1, $r = -.450$, $p = .031$).

Hypothesis Testing and Examination of Research Questions

Structural equation modeling. Structural equation modeling (SEM) was utilized with the time 1 data of 154 children (58 children had incomplete data) to test the proposed model (model 1, figure 1) and model 2 (figure 2). Demographics of the 154 children did not differ from demographics of the 212 total study population. The

Wechsler verbal IQ subtests Similarities and Comprehension were MVs for the construct of left hemisphere-related cognitive functioning. The Wechsler performance IQ subtests Block Design and Object Assembly were indicators (MV) for the construct of right hemisphere-related (perceptual organization) cognitive functioning. The Wechsler performance IQ subtests Digit Symbol Coding and Digit Span were MVs for the attention/working memory factor.

As stated above, a second model including expanded cognitive factors was also examined. The verbal IQ subtests Information, Vocabulary, Similarities, and Comprehension were MVs for the expanded construct of left hemisphere-related cognitive functioning. The performance IQ subtests Block Design, Object Assembly, Picture Completion, and Picture Arrangement were MVs for the expanded construct of right hemisphere-related cognitive functioning. The Wechsler performance IQ subtests Digit Symbol Coding, Digit Span, and Arithmetic were MVs for the attention/working memory factor (see figure 2).

For both models the ABQ mood depression scale and the CDS mood depression scale were MVs for the mood depression factor. Likewise, the ABQ cognitive depression scale and CDS cognitive depression scale were MVs for the cognitive depression factor. Also, the ABQ vegetative depression scale and the CDS vegetative depression scale were MVs for the vegetative depression factor. It should be noted that the model was also tested utilizing ABQ cognitive depression scale items and CDS cognitive depression scale items that did not include items reflective of Attention Hyperactivity Deficit

Disorder (ADHD) (e.g., attention problems, organization difficulties) and results were consistent with the presented models.

The overall fit of the models was assessed using the Chi-Square criterion, NFI, NNFI, CFI, the RMSEA, and the SRMR Overall, the fit indices indicate good model fit for both models with all fit indices within desirable ranges. See Appendix A, Table 7 for model fit results. Good model fit indicates that the specified relations between the MVs and the LVs (see figures 1-2) are adequate representations of the observed data.

Standardized parameter latent variable and error loadings for Model 1 and 2 are presented in tables 8 and 9. Review of these tables reveals that all loadings are relatively high and statistically significant (p 's $<.05$ - $.01$). The loading for ABQ Cognitive (Model 1 = $.53$, Model 2 = $.52$) is likely to be lower due to the lack of specificity of items 3-5 (e.g., "many incomplete assignments"). Also, Coding (Model 1 = $.58$, Model 2 = $.56$) may be lower than Digit Span and Arithmetic due to the fact that the task requires writing, whereas the others do not.

Hypothesis 1. In order to determine whether performance on right hemisphere-related cognitive measures of perceptual organization showed significant inverse associations with symptoms of depression (hypothesis 1), latent variable intercorrelation values were examined. Results indicated an approach toward the conventional level of significance between the Model 1 right cognitive (perceptual organization) factor and cognitive depression ($-.15$, $p=.08$). Likewise an approach toward the conventional level of significance existed between the Model 2 right cognitive (perceptual organization) expanded factor and cognitive depression ($-.15$, p 's $<.05$).

Hypothesis 2. This hypothesis stated that left hemisphere-related cognitive dysfunction (not including specific left anterior dysfunction) would be minimally related to depression. Surprisingly, a mildly significant inverse association was observed between the left cognitive factors (both Model 1 and 2) and cognitive depression (-.18, -.16, respectively).

Hypothesis 3. It was hypothesized that deficits in processing speed/attention would be inversely associated with symptoms of depression. As stated above, this factor was altered and renamed “attention/working memory” due to the addition of the Wechsler subtest Digit Span. A significant inverse association between the attention working memory factors (both Model 1 and 2) and cognitive depression (-.27, -.34, respectively) was observed. Approaches toward the conventional level of significance were also noted between Models 1 and 2 attention working memory factors and mood depression (-.15, -.18, respectively) and vegetative depression (-.20, -.15, respectively). See Tables 10 and 11 for all latent variable intercorrelations.

Time 2. Data Analysis.

Research questions 1 - 3. Stepwise multiple regression analyses were performed in order to test whether symptoms of depression, cognitive functioning, in childhood, and gender could predict cognitive functioning in adulthood (see tables 12 – 13). It was determined how well the variables included in time 1 factors utilized in SEM [left hemisphere-related cognitive dysfunction (VERBAL1, VERBALE1), (right hemisphere-related cognitive dysfunction (VISUAL1, VISUALE1), attention/working memory (ATTN/MEM1, ATTN/MEME1), mood symptoms of depression (MD1), cognitive

symptoms of depression (CD1), and vegetative symptoms of depression (VD1)] predicted time 2 factor variables. Separate regressions were run when using the expanded cognitive variables.

The time 2 factors consisted of similar manifest variables as those described for time 1 data. Time 2 cognitive composites are described above and consist of: VERBAL2, VERBALE2, VISUAL2, VISUALE2, ATTN/MEM2, and ATTN/MEME2. As the ABQ and the CDS questionnaires were not utilized at time 2, other measures of depression were utilized to represent the variable domains of mood, cognitive, and vegetative symptoms of depression. The Personality Assessment Inventory (PAI) provides subscales of these depressive symptom domains. The 8-item depression subscales (affective (PAIA), cognitive (PAIC), and physiological PAIP), and summative depression scale (PAID) (Morey, 1991) as well as record/diagnosis of depression based on clinical interview were utilized. Regressions were run utilizing all variables (i.e., both time 1 and time 2 cognitive variables and time 1 mood variables were entered as potential predictors for time 2 mood variables; both time 1 and time 2 mood variables and time 1 cognitive variables were entered as potential predictors for time 2 cognitive variables).

In addition to gender, the time 1 visual (VISUAL1) composite predicted the Personality Assessment Inventory affective depression subscale (PAIA) (Beta = .506, .358, respectively; Adj R2 = .281, F = 6.461, $p < .006$) (see Appendix A, Table 12). Interestingly, when utilizing the expanded (model 2) composite variables in the regression analyses, VISUALE2 predicted, in addition to gender, PAIA (Beta = .357, .556, respectively; Adj R2 = .272, F = 6.218, $p < .007$). Time 2 attention/memory

expanded (ATTN/MEME2) along with cognitive depression at time 1 (CD1) and gender, predicted PAI cognitive depression subscale (PAIC) (Beta = .348, .483, .515, respectively; Adj R2 = .465, F = 9.126, $p < .001$) (see Appendix A, Table 13). Note that when regressions were run with only time 1 variables as potential predictors for time 2 variables, similar results were obtained.

Stepwise multiple regressions were also performed with specific WAIS-III subtests entered as dependent variables. The mood depression composite (MD1) predicted performance on WAIS-III Coding (Beta = -.464, Adj R2 = .186, F = 7.408, $p = .011$). In addition, PAIA, along with attention/memory at time 1 and gender, predicted performance on WAIS-III Digit Span (Beta = .341, .580, -.384, respectively; Adj R2 = .398, F = 7.179, $p < .002$).

In comparing SEM results to regression results, an approach toward the conventional level of significance was noted for the association between VISUAL1 and the cognitive depression composite (CD1), but not for the mood depression subscale (MD1). Similarly to regression results, CFA demonstrated ATTN/MEM1 and ATTN/MEME1 were significantly correlated with time 1 cognitive depression (CD1). Lastly, while significant intercorrelations between the verbal composites (VERBAL1 & VERBALE1) and cognitive depression (CD1) were noted with CFA, regression results demonstrated VERBAL1 and VERBALE1 to only predict VERBAL2 and VERBALE2, respectively.

Research question 4. In order to examine whether associated cognitive problems occur only in those who have persisted with depression as adults subpopulation multiple

regressions were performed. VERBALE1, but not VERBAL1, predicted the PAI depression scale for subjects with depression or bipolar disorder (N=17) but did not predict the PAI depression scale or subscales in subjects without mood disorders (N=10). Both ATTN/MEM2 and ATTN/MEME2 predicted the PAI affective depression subscale in subjects without mood disorders. For individuals with depression or bipolar disorder who received psychotropic medication treatment in the past or present (N=15), no significant predictions between cognitive and depressive symptom factors were noted. However, it should be noted that these results should be interpreted with caution due to the low N.

CHAPTER 4.

DISCUSSION

Summary and Integration of Results.

Structural Equation Modeling.

Model 1. Results indicate good model fit to the data as well as high latent variable loadings. Thus, it can be concluded that the specified manifest variables adequately represent the latent constructs. Specifically, WISC-R subtests Similarities and Comprehension represent left hemisphere-related cognitive functioning/verbal reasoning; Block Design and Object Assembly represent right hemisphere-related cognitive functioning/visual reasoning; and Digit Symbol Coding and Digit Span represent attention/memory. In addition, ABQ and CDS cognitive scales represent the latent construct of cognitive depression; ABQ and CDS mood items represent the latent construct of mood depression; and ABQ and CDS vegetative items represent the construct of vegetative depression (see figure 1.)

Previous factor analyses of the WISC-R have demonstrated a robust and reliable three-factor structure consisting of similar factors to those utilized in this study. Allen & Thorndike (1995) used cross-validation of covariance structure models to examine the standardization sample data provided in both the WISC-III and WAIS-R manuals. Specifically, Verbal Comprehension (WISC-R subtests Vocabulary, Information, Comprehension, & Similarities), Perceptual Organization (Picture Completion, Block Design, Object Assembly, & Picture Arrangement) and Freedom from Distractibility

(Digit Span, Arithmetic, & Digit Symbol Coding) comprise the three factors. The authors noted the instability of the Freedom from Distractibility factor to have caused the most debate in factor analyses of the Wechsler scales. Of note, in the WISC-III, Symbol Search was added as an attempt to strengthen the Freedom from Distractibility factor yet resulted in adding a Processing Speed factor consisting of Digit Symbol Coding and Symbol Search. In order to remain consistent with the WISC-R factor composition, Symbol Search was excluded from their analyses. Thus, there is certainly literature support for the alteration made to the proposed “processing speed” factor resulting in an attention/memory factor consisting of Digit Symbol Coding and Digit Span.

Model 1. differs from the 3 factor structure identified by previous research in that fewer subtests were included in each factor. Given that the participants were all diagnosed with some form of learning disability it was considered appropriate to exclude the WISC-R subtests likely to be directly affected by learning disabilities (i.e., Information, Vocabulary, and Arithmetic). Also, the subtests Picture Arrangement and Picture Completion were considered less representative of right hemisphere-related cognitive functioning than Block Design and Object Assembly. Of note, despite these differences the latent variable loadings were high and goodness of fit indices indicated good fit. Thus, it can be concluded that WISC-R scores for this population of children with learning disabilities fit a 3-factor structure, despite the inclusion of fewer subtests.

Furthermore, the ABQ and CDS scales were representative of their underlying depressive symptom constructs. One scale, ABQ cognitive, showed the lowest loading of the examined variables. This result is likely due to the generalized questions included in

the scale (i.e., incomplete homework/classroom assignments) that may be attributed to factors other than attention and self-esteem. However, the loading was adequate and it can be concluded that all scales represented the specified constructs.

Model 2. Results for model 2 also indicate good model fit as well as high latent variable loadings. Thus, it can be concluded that the specified manifest variables are also representative of the latent constructs. Model 2 differs from model 1 in that the manifest variables for the cognitive latent constructs were expanded and consist of the subtests identified by Allen & Thorndike, 1995. WISC-R subtests Information, Vocabulary, Similarities and Comprehension represent left hemisphere-related cognitive functioning/verbal reasoning; Picture Arrangement, Picture Completion, Block Design and Object Assembly represent right hemisphere-related cognitive functioning/visual reasoning; and Arithmetic, Digit Symbol Coding and Digit Span represent attention/memory. Again, fit indices indicated good model fit and latent variable loadings were good. However, lower loadings were noted with both ABQ cognitive and WISC Digit Symbol Coding. Digit Symbol Coding requires writing while the other two subtests included on the Attention/Memory factor (Digit Span & Arithmetic) do not. The fact that many of the children had difficulty with writing may have impacted this result (see figure 2.).

Performance on the subtests Information, Vocabulary, and Arithmetic has the potential to be impacted by learning disabilities. However, results indicate these subtests also fit a 3-factor WISC-R structure in this sample of children with learning disabilities.

Other factor analytic WISC-R studies with children with learning disabilities have reported similar findings (Fischer & Dean, 1987).

Hypothesis 1. It was hypothesized that performance on right hemisphere-related cognitive measures of perceptual organization would show significant inverse associations with symptoms of depression. Results indicate some support for this hypothesis as the association between perceptual organization and cognitive symptoms of depression approached the conventional level of significance for both models. Furthermore, there is evidence that performance on perceptual organization tasks predicts affective symptoms of depression in adulthood.

Given past research regarding cognitive functioning and depression, it is surprising that greater support for this hypothesis was not observed. There are many possible explanations for the fact that a more significant relationship was not observed. All children in the population had learning disabilities and many of the participants experienced hyperactivity in addition to depression. It may be that more robust findings would be observed in children with unipolar depression without learning disabilities. However, according to correlation subpopulation analyses, no significant inverse associations were observed between the right hemisphere-related cognitive composites and depressive symptoms in diagnostic subgroups. Moreover, a higher number of participants would be necessary to thoroughly examine this idea. In addition, use of measures with greater specificity for perceptual organization may have resulted in greater significance. Lastly, the influence of attention/working memory on other cognitive

abilities such as perceptual organization might minimize the direct relationship between perceptual organization and depression.

Hypothesis 2. Left hemisphere-related cognitive dysfunction (verbal reasoning) was hypothesized to be minimally related to depression. CFA results for both models indicated a mildly significant inverse relationship between verbal cognitive factors and cognitive depression. However, results did not indicate that verbal cognitive skills could predict symptoms of depression.

There is little support in the literature for an association between verbal cognitive functioning and depression. However, there is some support for an association between left anterior cognitive dysfunction and depression, namely the cognitive abilities of verbal fluency and sequencing. Furthermore, long term memory difficulties have been associated with depression and the WISC-R verbal subtests Information, Similarities, and Vocabulary tap into long term memory ability. Perhaps children with learning disabilities including difficulty with verbal fluency, sequencing, and long term recall are more likely to evidence cognitive symptoms of depression (e.g., low self-esteem and attention problems).

Hypothesis 3. As hypothesized, there was a significant inverse association between Model 1 and Model 2 attention/memory factors and symptoms of depression. Specifically, the association was most significant between attention/memory and cognitive symptoms of depression. In the present study, attention and memory has been defined by performance on tasks considered representative of short-term memory and

attention. Thus, findings are consistent with literature supporting an association between depression and difficulty with explicit/working memory and attention.

Attention is thought to involve a widely distributed neural system (Posner & Petersen, 1990). Likewise mood disturbances such as depression have been described as the result of disordered neurochemical systems (King & Caine, 1996). Davidson, Pizzagalli, Nitschke, & Putnam (2002) reviewed the circuitry underlying the representation and regulation of normal emotion and mood and data was used to construct a model of the ways in which affect can become disordered in depression. Specifically, research on the prefrontal cortex, anterior cingulate, hippocampus, and amygdala was reviewed and abnormalities in the structure and function of these different regions in depression were considered. They concluded their review “offers very strong support for the view that depression refers to a heterogeneous group of disorders.” Furthermore, they noted it was possible that depression-spectrum disorders can be produced by abnormalities in many different parts of the circuitry reviewed.” Results of this study are consistent with the complexity of depression as well as the idea that is a circuitous, systemic phenomenon.

Given the complexity involved in studying circuitous constructs, it is important to recognize the potential impact of cognitive skills on one another. To that end, it may be that the attention/memory problems often evident in depression contribute to other cognitive problems (i.e., Visual and/or Verbal) or that there is a systemic process to these cognitive factors and depression. Results are supportive of this contention because the correlation between the visual and verbal factors and attention/memory are high.

Consistent with findings, recent studies have examined the cognitive abilities of persons with unipolar major depression and found selective attention and working memory to be implicated (e.g., Geva, 2002; Keller, 2000; Landro et al., 2001; Nebes et al., 2000; Stiles & Sletvold, 2001). Moreover, this study examined the symptom characteristics of depression in relation to cognitive abilities. Results indicate that cognitive depressive symptoms specifically characterized by low self-esteem and problems with attention are associated with performance on attention/memory cognitive tasks. Thus, it may be that when persons with depression do not evidence these specific cognitive symptoms, their cognitive performance is less different from that of their non-depressed peers.

Research questions 1-3.

The predictive power of cognitive functioning and depression over time was explored. Cognitive performance at time 1 was predictive of cognitive performance at time 2. Likewise, time 1 depressive symptoms were predictive of time 2 depressive symptoms. Given the long follow-up period, these results are substantial.

The question of persistence of associations between cognitive difficulties and depression was examined. The proposed association between right hemisphere-related cognitive measures of visual reasoning and cognitive depressive symptoms proved to be minimal (approached conventional level of significance) and no association was noted between visual reasoning and mood depressive symptoms according to the structural equation modeling. However, in regards to the predictive power of cognitive functioning on depression, performance on right hemisphere-related cognitive measures at time 1

predicted the Personality Assessment Inventory Affective (PAIA) subscale at time 2. When examining Model 2 expanded variables, only performance on right hemisphere-related cognitive measures at time 2 predicted PAIA. Thus, it appears that subtests Block Design and Object Assembly may account for more variance over time than Picture Arrangement and Picture Completion.

CFA results indicated significant associations between verbal cognitive functioning and cognitive depression. However, left hemisphere related cognitive composites did not predict depressive symptoms nor did depressive symptoms predict performance on left hemisphere related cognitive tasks.

At both time 1 and time 2, significant associations between attention/memory cognitive tasks and cognitive depressive symptoms were evident. These associations were time sensitive, they only occurred at the same time. While attention/memory at time 1 was predictive of attention/memory at time 2, attention/memory at time 1 did not predict cognitive depression at time 2. Thus, while the association appears to be persistent between attention/memory cognitive task performance and cognitive depression, it does not appear that the former predicts the latter over time. Also, the prediction at time 2 only occurred with the expanded composite which included the subtests Arithmetic along with Digit Symbol Coding and Digit Span. The subtest Arithmetic may be of greater import in evaluating attention/memory in adults as opposed to children.

Research question 4. Whether or not associated cognitive problems occur only in those who have persisted with depression as adults was examined. The expanded verbal

cognitive functioning composite predicted the depression scale when only examining subjects who received diagnoses of depression or bipolar disorder as adults. In regards to time 2 predictions, attention/memory predicted the PAI affective depression subscale in individuals without adult diagnoses of a mood disorder. The lack of prediction in persons with mood disorders may be most attributed to the low number of subjects included in the subgroup comparison.

Overall, the number of subjects included in the regression analyses was low. However, there are few longitudinal studies that have examined the predictive power of cognitive functioning and depression. Therefore, while the regression results are limited, the findings offer directions for future research.

Implications of Findings & Future Directions

The purpose of this study was to determine which particular cognitive processes are associated with specific depressive symptoms and depression as a whole. Also, the predictive power of cognitive functioning and depression over time was examined. The most compelling and consistent finding of this study relates to attention/working memory. Results suggest that attention/working memory is most prominent in the association between cognition and depression. Furthermore, there is indication that the connection is most significant with depressive symptoms reflecting disturbances in cognitive functioning. Thus, it can be implied that without such depressive symptoms (e.g, low self-esteem, problems with attention), there may be less likelihood for significant implication in cognitive performance. However, given that there were high associations between attention/working memory factors and visual reasoning as well as

verbal reasoning, it could also be that attention/working memory problems contribute to other cognitive deficits found in depression. Moreover, the variability of findings regarding the relationship between cognition and depression in the literature could also be in part attributed to attention/working memory problems.

Furthermore, the association between attention/working memory and depression is consistent with the idea that a widely distributed network is involved in depression. Likewise, results corroborate with the idea that attention/working memory is part of a widely distributed neural system. Findings indicate the importance of recognizing the potential impact of attention/working memory on cognitive performance and other aspects of cognitive functioning when evaluating persons with depression.

Results of this study lend support to the proven effectiveness of cognitive behavior therapy in the treatment of depression. Given the approach, one assumes that there is indeed a relationship between cognition and depression. In this study it was the cognitive symptoms of depression that were most associated with cognition. It could be that these symptoms are primary in depression, meaning that alleviation of such symptoms (i.e., by use of cognitive behavior therapy techniques) leads to relief of mood and vegetative symptoms. On the other hand, the relationship between symptom subgroups could be bi-directional. Regardless, this study highlighted the importance of recognizing cognitive symptoms of depression and it can be extrapolated that relief of such symptoms can lead to amelioration of depression. Whether improved cognitive symptoms of depression could also lead to improved cognitive performance (i.e., working memory/attention) is an area in need of further research due to the equivocal findings

regarding the improvement of cognitive functioning following effective treatment of depression.

The less robust findings of the study imply a continued need for further research in the area of cognition and depression. In regards to the indication for the predictive ability of visual reasoning skills on mood symptoms, it could be implied that such cognitive problems predispose individuals to depression in later life. One explanation may be that when the region of the brain most often associated with visual reasoning deficits (right posterior) functions less well in childhood, the likelihood of such dysfunction leading to depression increases. There is literature support for cognitive impairment predicting affective disorder (vanOs et al., 1997). Likewise, research has demonstrated a link between verbal cognitive deficits and externalizing disorders (i.e., ADHD, aggression) (Hinshaw, 1992). However, the predictive ability of specific cognitive deficits on depressive symptoms is an area in need of further study.

The observed relationship between verbal reasoning and cognitive symptoms of depression was surprising. As stated earlier, it could be implied that when children have learning disabilities including difficulty with verbal fluency, sequencing, and long term recall learning disabilities, they are more likely to evidence cognitive symptoms of depression. However, it would be necessary to clearly identify such difficulties in further research in order to examine this idea. Another implication of this finding could be related to the possibility of comorbidity of externalizing problems, such as aggression. Likewise, the fact that the cognitive symptoms of depression include attention problems, there is a possibility of co-occurring ADHD. As stated above, there is literature support

for an association between verbal deficits and externalizing problems. In the future, sufficient numbers of individuals to allow subtype diagnostic comparison (i.e., depression only versus ADHD only, etc.) could help clarify this issue.

Also, though not a focus of this study, results supported past research that has demonstrated the predictive ability of depression in childhood for depression in adulthood. Likewise cognitive performance in childhood was highly predictive of cognitive performance in adulthood. This finding is important given the long amount of time (20-25 years) between follow-up as well as given the population of study. Research has demonstrated that learning disabilities generally persist over time. Likewise, results imply that levels of cognitive performance on standardized IQ tests are also consistent over time.

Given the complexity of constructs such as depression and cognition, continued research is needed to increase understanding of their relationship. This study supported the contention that specific depressive symptoms (e.g., cognitive) are associated with cognitive abilities (e.g., attention/working memory). However, more research is needed to corroborate this finding. Likewise, more precise measures of cognitive processes and depressive symptoms that can be used with both adults and children are needed. Lastly, longitudinal study is necessary to provide further information regarding the developmental trajectory of the association between cognition and depression.

Limitations

A limitation of this study was that the entire population consisted of individuals with learning disabilities. Given the increased prevalence of depression in persons with

learning disabilities as well as the inherent implication of cortical functions, the population appeared appropriate and useful for the study of the association between depression and cognition. However, the generalizability of the results of this study is unclear and in need of further research.

This study was also limited in that archival data was used in the SEM analyses. While the available data is considered thorough and detailed, the measures are limited in that they reflect the era of the testing (late 1970s) and were not chosen for the specific purposes of this study.

Recent research has identified the usefulness of comparing diagnostic subtypes to further clarify the relationship between mood and cognition. Specifically, studies have shown differences in cognitive performance between individuals diagnosed with unipolar depression versus bipolar disorder. Likewise, verbal cognitive deficits have been observed in individuals with externalizing (e.g., attention deficit hyperactivity disorder, conduct disorder) as opposed to internalizing disorders such as depression and anxiety. A higher number of subjects would have allowed for meaningful diagnostic subgroup comparison and is an area in need of further research.

Summary

This study used structural equation modeling to determine which particular cognitive processes were associated with specific depressive symptoms. The model examined how three cognitive processing factors (verbal & visual reasoning, and attention/working memory) were associated with three depressive symptom factors (disturbances in mood, vegetative, and cognitive functioning). Model fit results supported

the proposed model. Moreover, the most compelling finding was the observed association between attention/working memory and cognitive symptoms of depression.

Though less robust, a relationship between verbal reasoning and cognitive depressive symptoms was observed and an approach toward the conventional level of significance was noted between visual reasoning and cognitive symptoms of depression. Longitudinal evaluation of the data indicated some support for the predictive power of visual reasoning performance in childhood on mood symptoms of depression in adulthood. Lastly, the robust association observed at time 1 between attention/working memory performance and cognitive symptoms of depression was also observed at time 2. This time specific association was the most robust and substantial finding of the study.

APPENDIX A.
TABLES & FIGURES

Table 1. Demographics

	Time 1. (T1) Population	Time 2. (T2) Population
Gender		
Male	176 (83%)	32 (80%)
Female	36 (17 %)	8 (20%)
T1 Age		
Mean (SD in months)	10 y. 9 mo. (25.95)	11 y. (27.7)
Range	6 y. 3 mo. – 16 y	6 y. 10 mo. – 16 y.
T2 Age		
Mean (SD in months)	-----	32 y. 5 mo. (33.3)
Range		26 y. 4 mo. – 37 y. 3 mo.
T1 Behavior Subtype		
Depression Only	71 (33.5%)	20 (50%)
Depression & Hyperactivity	53 (25%)	9 (22.5%)
Hyperactivity & “mini” or latent depression	6 (2.8%)	
Hyperactivity Only	32 (15.1%)	3 (7.5%)
No Problem or “mini” Depression	50 (23.6%)	8 (20%)
T2 Mood Diagnosis		
Depression Only		15 (37.5%)
Bipolar Disorder	-----	8 (20%)
No Mood Disorder		17 (42.5%)
T1 Learning Disability (LD)		
Reading Subtype		
LD with reading difficulty	69 (32.5%)	19 (47.5%)
LD without reading difficulty	136 (64.2%)	21 (52.5%)
Missing	7 (3.3%)	-----

Table 2. ABQ & CDS Composite Items & Standardized Reliability Coefficients

	Mood Symptoms	Cognitive Symptoms	Vegetative Symptoms
ABQ Items	<p>Mean of 5 of the following:</p> <ol style="list-style-type: none"> 1. Statements or appearance of sadness, loneliness, unhappiness and/or pessimism 2. Appearance or statements of despair or hopelessness 3. Mood swings, moodiness 4. Hypersensitive, cries easily 5. Less group participation 6. Socially withdrawing 7. Loss of usual social interests 8. Does not enjoy school activities 9. Loss of usual work effort or interest in school subjects 10. Loss of interest in non-academic school activities 11. Loss of usual personal interests or pursuits (other than school) 	<p>Mean of 3 of the following</p> <ol style="list-style-type: none"> 1. Feelings of being worthless, useless, dumb, stupid, ugly, guilty or the cause of someone else's problems 2. Frequent complaints from teachers ("daydreaming," "poor concentration," "poor memory") 3. Many incomplete classroom assignments 4. Much incomplete homework 5. Finds concentrating to complete homework difficult 	<p>Mean of 8 of the following:</p> <ol style="list-style-type: none"> 1. Trouble falling to sleep 2. Restless sleep; awakens during night 3. Awakening too early 4. Difficulty awakening in morning 5. Headaches 6. Stomach aches 7. Muscle aches or pains 8. Other physical concerns or complaints 9. Decreased energy; mental and/or physical fatigue 10. Loss of usual appetite with actual weight loss
ABQ Alpha	Alpha = .817	Alpha = .834	Alpha = .725
CDS Items	<p>Mean of 4 of the following</p> <ol style="list-style-type: none"> 1. Unhappy, sad, depressed 2. Gets angry easily, has a bad temper 3. Temper tantrums 4. Cries or gets upset over little things 5. Not interested in things around him, acts bored 6. Gets his feelings hurt easily 7. Moody, sad one day and happy the next without cause 	<p>Mean of 5 of the following:</p> <ol style="list-style-type: none"> 1. Feels he is not as good as others 2. Can't pay attention for very long at a time 3. Doesn't think he can do things as well as he actually can 4. Poor memory 5. Gets mixed up easily, easily confused 6. Daydreams a lot 7. Can't keep his mind on what he is doing, gets distracted easily 	<p>Mean of 5 of the following</p> <ol style="list-style-type: none"> 1. Trouble in sleeping 2. Trouble around eating 3. Always getting hurt or having an accident 4. Complains of stomach aches or headaches 5. Has pains in muscles and joints 6. Clumsy, awkward, poor muscular coordination 7. Can't relax, seems tense 8. Always seems tired, no "get up and go" 9. Accident prone
CDS Alpha	Alpha = .750	Alpha = .795	Alpha = .719

Table 3. Descriptive Statistics for Cognitive Composites

Composite	<i>M</i>	<i>SD</i>	<i>N</i>
VERBAL1	10.245	2.515	188
VERBAL2	11.175	2.238	40
VERBALE1	9.942	2.409	188
VERBALE2	10.981	2.083	40
VISUAL1	9.878	2.718	188
VISUAL2	9.900	2.042	40
VISUALE1	10.174	2.295	188
VISUALE2	10.850	2.245	40
ATTN/MEM1	7.508	2.358	181
ATTN/MEM2	8.138	1.676	40
ATTN/MEME1	7.606	2.150	181
ATTN/MEME2	8.125	1.760	40

VERBAL1 = mean of WISC-R Similarities & Comprehension; VERBAL2 = mean of WAIS-III Similarities & Comprehension; VERBALE1 = mean of WISC-R Similarities, Comprehension, Information, & Vocabulary; VERBALE2 = mean of WAIS-III Similarities, Comprehension, Information, & Vocabulary; VISUAL1 = mean of WISC-R Block Design & Object Assembly; VISUAL2 = mean of WAIS-III Block Design & Object Assembly; VISUALE1 = mean of WISC-R Block Design, Object Assembly, Picture Arrangement, & Picture Completion; VISUALE2 = mean of WAIS-III Block Design, Object Assembly, Picture Arrangement, & Picture Completion; ATTN/MEM1 = mean of WISC-R Coding & Digit Span; ATTN/MEM2 = mean of WAIS-III Coding & Digit Span; ATTN/MEME1 = mean of WISC-R Coding, Digit Span, & Arithmetic; ATTN/MEME2 = mean of WAIS-III Coding, Digit Span, & Arithmetic

Table 4. Descriptive Statistics for Mood Composites

Composite	<i>M</i>	<i>SD</i>	<i>N</i>
MD1	1.061	.763	180
CD1	2.196	1.129	177
VD1	.635	.603	178
PAIA	48.375	9.234	40
PAIC	46.400	7.629	40
PAIP	48.375	9.724	40
PAID	47.725	8.750	40

MD1 = mean of mood depression scales of ABQ & CDS; CD1 = mean of cognitive depression scales of ABQ & CDS; VD1 = mean of vegetative depression scales of ABQ & CDS; PAIA = Personality Assessment Inventory affective depression subscale; PAIC = Personality Assessment Inventory cognitive depression subscale; PAIP = Personality Assessment Inventory physical depression subscale; PAID = Personality Assessment Inventory Depression Scale

Table 5. Pearson Correlations for Model 1. Composite Variables

	Sex	VER1	VIS1	AM1	MD1	CD1	VD1	VER2	VIS2	AM2	PAIC	PAIA	PAIP
sex	-----												
VER1	-.173* p=.017 n=188	-----											
VIS1	-.131 p=.072 n=188	.453** p=.000 n=188	-----										
AM1	.052 p=.486 n=181	.360** p=.000 n=181	.377** p=.000 n=181	-----									
MD1	.009 p=.908 n=180	-.016 p=.838 n=163	-.075 p=.345 n=163	-.079 p=.324 n=158	-----								
CD1	-.038 p=.618 n=177	.052 p=.509 n=161	-.007 p=.928 n=161	-.076 p=.346 n=156	.634** p=.000 n=177	-----							
VD1	-.096 p=.204 n=178	.108 p=.173 n=161	-.005 p=.950 n=161	-.134 p=.095 n=156	.617** p=.000 n=177	.490** p=.000 n=175	-----						
VER2	-.181 p=.264 n=40	.577** p=.000 n=40	.299 p=.093 n=40	.194 p=.243 n=38	-.198 p=.278 n=32	-.229 p=.206 n=32	.081 p=.666 n=31	-----					
VIS2	-.130 p=.423 n=40	.309 p=.052 n=40	.627** p=.000 n=40	.125 p=.453 n=38	-.147 p=.423 n=32	-.218 p=.231 n=32	.122 p=.513 n=31	.565** p=.000 n=40	-----				
AM2	-.249 p=.121 n=40	.231 p=.152 n=40	.171 p=.291 n=40	.439** p=.006 n=38	-.291 p=.106 n=32	-.092 p=.617 n=32	-.100 p=.593 n=31	.400* p=.010 n=40	.270 p=.092 n=40	-----			
PAIC	.480** p=.002 n=40	-.296 p=.064 n=40	.067 p=.680 n=40	-.199 p=.232 n=38	.395* p=.025 n=32	.474** p=.006 n=32	.147 p=.431 n=31	-.220 p=.173 n=40	.022 p=.891 n=40	-.057 p=.729 n=40	-----		
PAIA	.459** p=.003 n=40	-.280 p=.080 n=40	.112 p=.491 n=40	-.295 p=.073 n=38	.202 p=.267 n=32	.312 p=.082 n=32	.165 p=.376 n=31	-.175 p=.280 n=40	.050 p=.758 n=40	-.081 p=.618 n=40	.659** p=.000 n=40	-----	
PAIP	.241 p=.134 n=40	-.115 p=.481 n=40	.081 p=.621 n=40	-.134 p=.421 n=38	.419* p=.017 n=32	.402* p=.022 n=32	.310 p=.089 n=31	-.240 p=.136 n=40	-.131 p=.420 n=40	-.235 p=.144 n=40	.469** p=.002 n=40	.517 p=.001 n=40	-----
PAID	.443** p=.004 n=40	-.257 p=.110 n=40	.119 p=.463 n=40	-.244 p=.140 n=38	.416* p=.018 n=32	.465** p=.007 n=32	.281 p=.125 n=31	-.255 p=.112 n=40	-.020 p=.905 n=40	-.167 p=.303 n=40	.792** p=.000 n=40	.860** p=.000 n=40	.838** p=.000 n=40

Table 6. Pearson Correlations for Model 2. Composite Variables

	Sex	VERE1	WISE1	AME1	MD1	CD1	VD1	VERE2	WISE2	AME2	PAIC	PAIA	PAIP
sex	-----												
VERE1	-.216** p=.003 n=188	-----											
WISE1	-.162* p=.026 n=188	.567** p=.000 n=188	-----										
AME1	.020 p=.794 n=181	.457** p=.000 n=181	.400** p=.000 n=181	-----									
MD1	.009 p=.908 n=180	-.005 p=.946 n=163	-.061 p=.441 n=163	-.097 p=.224 n=158	-----								
CD1	-.038 p=.618 n=177	.069 p=.381 n=161	-.026 p=.743 n=161	-.127 p=.114 n=156	.634** p=.000 n=177	-----							
VD1	-.096 p=.204 n=178	.130 p=.101 n=161	.005 p=.950 n=161	-.112 p=.165 n=156	.617** p=.000 n=177	.490** p=.000 n=175	-----						
VERE2	-.223 p=.166 n=40	.646** p=.000 n=40	.495** p=.001 n=40	.342* p=.036 n=38	-.122 p=.507 n=32	-.136 p=.457 n=32	.148 p=.427 n=31	-----					
WISE2	-.178 p=.273 n=40	.460** p=.003 n=40	.760** p=.000 n=40	.264 p=.109 n=38	-.059 p=.749 n=32	-.122 p=.505 n=32	.181 p=.330 n=31	.572** p=.000 n=40	-----				
AME2	-.252 p=.117 n=40	.476** p=.002 n=40	.467** p=.002 n=40	.547** p=.000 n=38	-.221 p=.223 n=32	-.194 p=.288 n=32	-.038 p=.837 n=31	.634** p=.000 n=40	.485** p=.002 n=40	-----			
PAIC	.480** p=.002 n=40	-.156 p=.338 n=40	.064 p=.694 n=40	-.134 p=.422 n=38	.395* p=.025 n=32	.474** p=.006 n=32	.147 p=.431 n=31	-.154 p=.344 n=40	-.048 p=.770 n=40	-.009 p=.956 n=40	-----		
PAIA	.459** p=.003 n=40	-.239 p=.138 n=40	.012 p=.943 n=40	-.218 p=.189 n=38	.202 p=.267 n=32	.312 p=.082 n=32	.165 p=.376 n=31	-.121 p=.456 n=40	-.003 p=.986 n=40	-.141 p=.386 n=40	.659** p=.000 n=40	-----	
PAIP	.241 p=.134 n=40	-.117 p=.471 n=40	.096 p=.558 n=40	-.137 p=.413 n=38	.419* p=.017 n=32	.402* p=.022 n=32	.310 p=.089 n=31	-.145 p=.373 n=40	-.132 p=.415 n=40	-.271 p=.091 n=40	.469** p=.002 n=40	.517 p=.001 n=40	-----
PAID	.443** p=.004 n=40	-.200 p=.216 n=40	-.044 p=.788 n=40	-.197 p=.237 n=38	.416* p=.018 n=32	.465** p=.007 n=32	.281 p=.125 n=31	-.172 p=.288 n=40	-.069 p=.674 n=40	-.190 p=.241 n=40	.792** p=.000 n=40	.860** p=.000 n=40	.838** p=.000 n=40

Table 7. Model Fit Results

Model	df	χ^2	NFI	NNFI	CFI	MFI	RMSEA	SRMR
<u>Model 1. WISC-DP</u>	34	42.468	.935	.972	.986	.973	.041	.054
<u>Model 2. WISCE-DP</u>	99	121.156	.886	.967	.976	.930	.039	.062

Note. NFI = Bentler-Bonett Normed Fit Index; NNFI = Bentler-Bonett Nonnormed Fit Index; CFI = Comparative Fit Index; MFI = McDonald Fit Index; RMSEA = Root Mean Square Error of Application; SRMR = Standardized Root Mean Squared Residual

Table 8. Standardized parameter latent variable and error loadings: Model 1. WISC-DP (N=154)

	Model WISC-DP - Total sample								Error
	1	2	3	4	5	6	7	8	
1. WISC Similarities	.78								.63
1. WISC Comprehension	.62								.79
2. WISC Block Design		.80							.59
2. WISC Object Assembly		.61							.79
3. WISC Digit Span			.60						.80
3. WISC Coding			.58						.82
4. ABQ Mood				.77					.64
4. CDS Mood				.74					.67
5. ABQ Cognitive					.53				.80
5. CDS Cognitive					.99				.14
6. ABQ Vegetative						.96			.29
6. CDS Vegetative						.73			.68

Table 9. Standardized parameter latent variable and error loadings: Model 2. WISCE-DP (N=154)

	Model 2. WISCE-DP - Total sample								Error
	1	2	3	4	5	6	7	8	
1. WISC Information	.77								.64
1. WISC Vocabulary	.80								.60
1. WISC Similarities	.75								.67
1. WISC Comprehension	.64								.77
2. WISC Block Design		.66							.75
2. WISC Object Assembly		.62							.78
2. WISC Picture Arrangement		.59							.81
2. WISC Picture Completion		.62							.79
3. WISC Digit Span			.63						.78
3. WISC Coding			.56						.83
3. WISC Arithmetic			.76						.65
4. ABQ Mood				.79					.62
4. CDS Mood				.72					.70
5. ABQ Cognitive					.52				.86
5. CDS Cognitive					.99				.14
6. ABQ Vegetative						.99			.15
6. CDS Vegetative						.71			.71

Table 10. Model 1. Confirmatory Factor Analysis (CFA) Latent Variable Intercorrelations (N=154)

Latent Variables	Total Sample					
	1	2	3	4	5	6
1. Verbal.....	.57	.68	-.13	-.18*	.06	
2. Visual65	-.11	-.15^	.01	
3. Attention/ Memory.....			-.15^	-.27**	-.20^	
4. Mood Depression82	.75	
5. Cognitive Depression.....					.56	
6. Vegetative Depression						

Table 11. Model 2. Confirmatory Factor Analysis (CFA) Latent Variable Intercorrelations (N=154)

Latent Variables	Total Sample					
	1	2	3	4	5	6
1. Verbal Expanded.....	.66	.61	-.10	-.16*	.10	
2. Visual Expanded53	-.14	-.15^	-.02	
3. Attention/ Mem Expanded.....			-.18^	-.34**	-.15^	
4. Mood Depression81	.74	
5. Cognitive Depression.....					.55	
6. Vegetative Depression						

*p<.05
** p<.01

Table 12. Stepwise Regression Analysis for Model 1.(N= 28)

Adult Criterion	Predictor	Beta	Adjusted R2	F	p
VERBAL2	VERBAL1	.575	.306	13.339	<.002
VISUAL2	VISUAL1	.687	.452	24.083	<.001
ATTN/MEM2	ATTN/MEM1	.465	.187	7.459	<.02
PAIC	CD1	.449			
	Sex	.401	.372	9.309	<.02
PAIA	Sex	.506			
	VISUAL1	.358	.281	6.461	<.006
PAIP	MD1	.468	.190	7.572	<.02
PAID	CD1	.479	.201	8.042	<.01

Sex = 1 =male, 2=female; VERBAL1 = mean of WISC-R Similarities & Comprehension; VERBAL2 = mean of WAIS-III Similarities & Comprehension; VISUAL1 = mean of WISC-R Block Design & Object Assembly; VISUAL2 = mean of WAIS-III Block Design & Object Assembly; ATTN/MEM1 = mean of WISC-R Coding & Digit Span; ATTN/MEM2 = mean of WAIS-III Coding & Digit Span; MD1 = mean of mood depression scales of ABQ & CDS; CD1 = mean of cognitive depression scales of ABQ & CDS; VD1 = mean of vegetative depression scales of ABQ & CDS; PAIA = Personality Assessment Inventory affective depression subscale; PAIC = Personality Assessment Inventory cognitive depression subscale; PAIP = Personality Assessment Inventory physical depression subscale; PAID = Personality Assessment Inventory Depression Scale

Table 13. Stepwise Regression Analysis for Model 2.(N=28)

Adult Criterion	Predictor	Beta	Adjusted R2	F	p
VERBALE2	VERBALE1	.644	.393	19.138	<.001
VISUALE2	VISUALE1	.777	.589	41.208	<.001
ATTN/MEME2	ATTN/MEME1	.604	.341	15.472	<.002
PAIC	CD1	.483			
	Sex	.515			
	ATTN/MEME2	.348	.465	9.126	<.001
PAIA	Sex	.556			
	VISUALE2	.357	.272	6.218	<.007
PAIP	MD1	.468	.190	7.572	<.02
PAID	CD1	.479	.201	8.042	<.01

Sex = 1 =male, 2=female; VERBALE1= mean of WISC-R Similarities, Comprehension, Information, & Vocabulary; VERBALE2 = mean of WAIS-III Similarities, Comprehension, Information, & Vocabulary; VISUALE1 = mean of WISC-R Block Design, Object Assembly, Picture Arrangement, & Picture Completion; VISUALE2 = mean of WAIS-III Block Design, Object Assembly, Picture Arrangement, & Picture Completion; ATTN/MEME1 = mean of WISC-R Coding, Digit Span, & Arithmetic; ATTN/MEME2 = mean of WAIS-III Coding, Digit Span, & Arithmetic; MD1 = mean of mood depression scales of ABQ & CDS; CD1 = mean of cognitive depression scales of ABQ & CDS; VD1 = mean of vegetative depression scales of ABQ & CDS; PAIA = Personality Assessment Inventory affective depression subscale; PAIC = Personality Assessment Inventory cognitive depression subscale; PAIP = Personality Assessment Inventory physical depression subscale; PAID = Personality Assessment Inventory Depression Scale

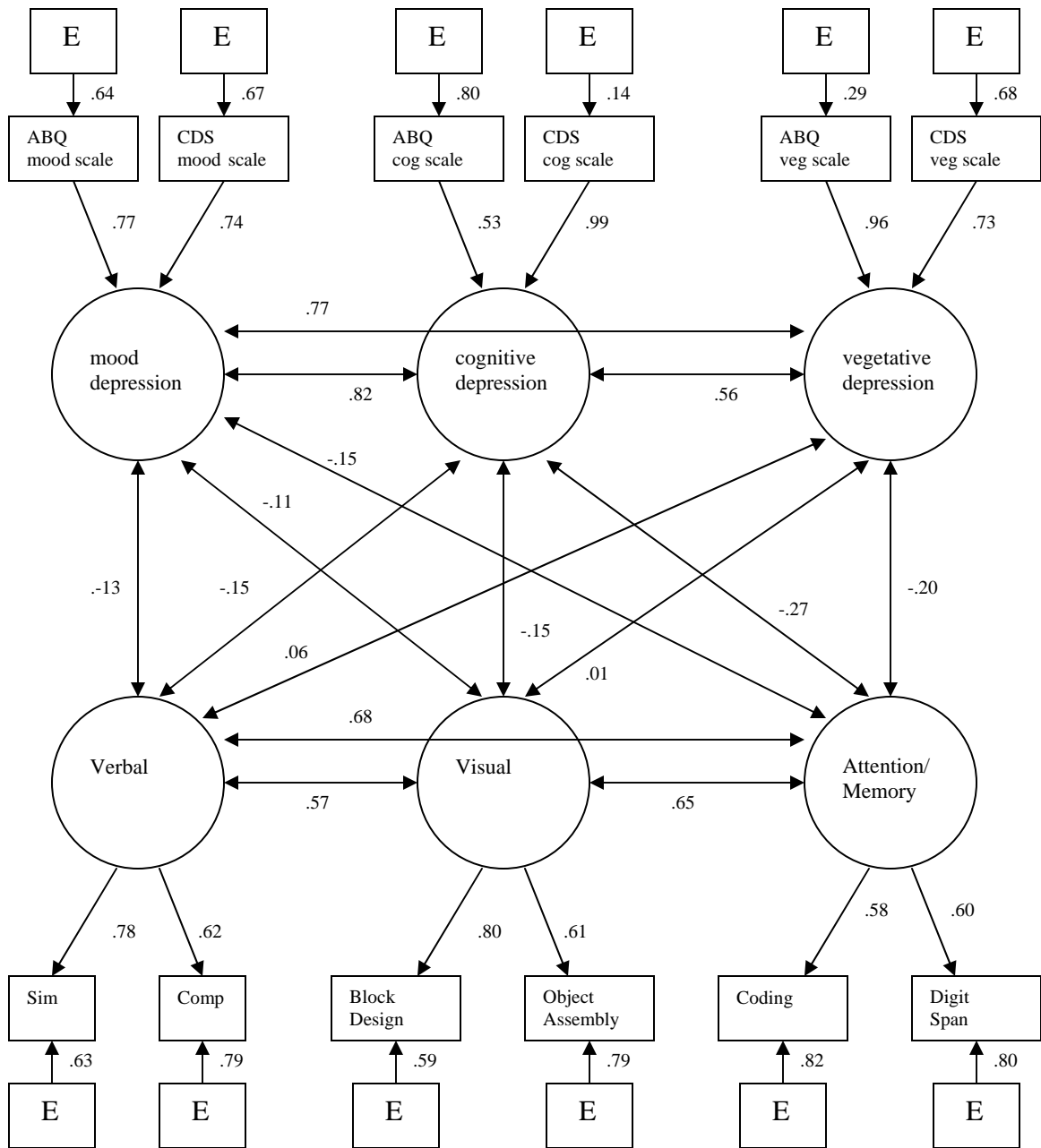


Figure 1. Model 1.

Figure 1. Legend

E = Measurement Error

ABQ = Affective Behavior Questionnaire

CDS = Child Development Study Questionnaire

Sim = WISC-R Subtest Similarities

Comp = WISC-R Subtest Comprehension

Block Des = WISC-R Subtest Block Design

Obj Assly = WISC-R Subtest Object Assembly

Coding = WISC-R Subtest Digit Symbol Coding

Digit Span = WISC-R Subtest Digit Span

PO = Perceptual Organization

Uni-directional arrows = latent variable loadings

Bi-directional arrows = Latent variable correlations

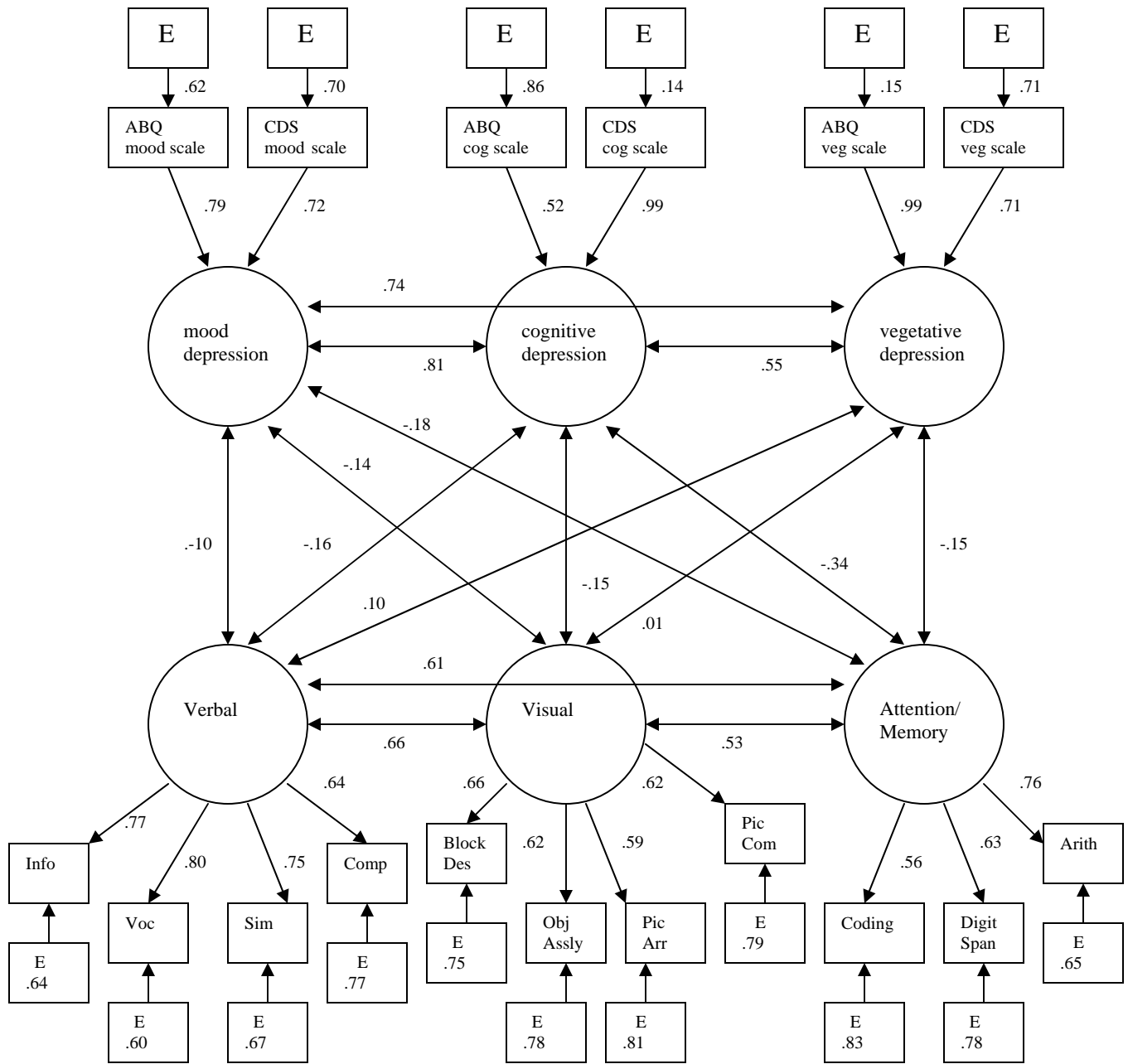


Figure 2. Model 2.

Figure 2. Legend

E = Measurement Error
 ABQ = Affective Behavior Questionnaire
 CDS = Child Development Study Questionnaire
 Info = WISC-R Subtest Information
 Voc = WISC-R Subtest Vocabulary
 Comp = WISC-R Subtest Comprehension
 PO = Perceptual Organization
 Block Des = WISC-R Subtest Block Design

Obj Assly = WISC-R Subtest Object Assembly
 Pic Arr = WISC-R Subtest Picture Arrangement
 Pic Com = WISC-R Subtest Picture Completion
 Coding = WISC-R Subtest Digit Symbol Coding
 Digit Span = WISC-R Subtest Digit Span
 Arith = WISC-R Subtest Arithmetic
 Sim = WISC-R Subtest Similarities
 Uni-directional arrows = latent variable loadings
 Bi-directional arrows = Latent variable correlations

APPENDIX B
CONSENT FORM

The University of Texas Southwestern Medical Center at Dallas
Children's Medical Center of Dallas
Then Winston School, Dallas

CONSENT TO PARTICIPATE IN RESEARCH

Title of Research: Children with Learning Disabilities Grown Up: A Longitudinal Study
Sponsor: Association for Citizens with Learning Disabilities Foundation (ACLD)

Investigators:

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INVITATION: You were diagnosed as a child as having a learning disability. In addition, you attended the Winston School during the years 1975-1979. We are asking you to participate in this follow-up study of the initial 227 students that entered the Winston School between 1975-1979. We are asking 10% of you to participate in our initial follow-up study. It is anticipated that 23 of your colleagues from the Winston School will participate in this study.

RIGHTS OF PARTICIPATION IN RESEARCH: You have certain rights. These rights include, but are not limited to, the right to have information about the purpose and nature of the research, the procedures, any risks, and benefits. You have the right to ask any questions about the research and to receive answers to all of your questions.

VOLUNTARY PARTICIPATION: You have the right to agree or refuse to participate in this research. Refusal to participate will involve no penalty or loss of benefits to which you are otherwise entitled. If you decide to participate and later change your mind, you are free to discontinue participation in the research without affecting your legal rights or the quality of health care that you will receive at this medical center. Any significant new information which becomes available during your participation in the research that may affect your willingness to continue in the study will be given to you.

CONFIDENTIALITY: You have the right to privacy. All information obtained from this research that can be identified with you will remain confidential within the limits of the law. Such private information will be released only to the investigators. The results of this research may appear in scientific publications without identifying you by name.

An Institutional Review Board (IRB) is a group of people who are responsible for assuring the community that the rights of participants in research are respected. The records of your participation in this research may be reviewed by members and staff of the IRB at this medical center. You may be contacted by a representative of the Board for information about your experience with this research. If you wish, you may refuse to answer any questions the representative of the Board may ask.

COMPLICATIONS: We do not anticipate any complications resulting from participation in this study.

YOUR QUESTIONS: Julie Baudoin is available to answer your questions about this research during regular office hours. Further information can also be obtained from Stephanie Jones, M.A. or Warren A. Weinberg, M.D. The Chairman of the IRB is available to answer any questions about your rights as a participant in research or to answer your questions. You may telephone the Chairman of the IRB during regular office hours at (214) 648-2258.

YOU WILL HAVE A COPY OF THIS SIGNED AND DATED CONSENT FORM TO KEEP.

PURPOSE: The purpose of this research is to follow-up on past Winston School students and determine their current status.

PROCEDURES: Intellectual functioning, academic skill development, social-emotional characteristics, and life adjustment will be measured. Standardized tests and clinical interviews will be used to gather information. Testing and interviews will be performed by a trained clinician. Tests will be administered once and will be completed in one-two days (8-10 hours of contact).

POSSIBLE RISKS: There are no anticipated risks with participation in this study.

POSSIBLE BENEFITS: You may benefit from this study by receiving the opportunity to discuss individual test results with a trained professional. In the future, other children and adults with learning disabilities may benefit from the results of this research. New information may lead to improved treatment for children and adults with learning disabilities. Very important will be information gleaned from the study toward the understanding of what happens to children and adolescents with learning disabilities over a long period of time.

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