

Fall 2009

Examining the Association between Depressive Symptoms and Performance on Executive Function Measures in Children

Hillary A. Mangis

Follow this and additional works at: <https://dsc.duq.edu/etd>

Recommended Citation

Mangis, H. (2009). Examining the Association between Depressive Symptoms and Performance on Executive Function Measures in Children (Doctoral dissertation, Duquesne University). Retrieved from <https://dsc.duq.edu/etd/868>

This Immediate Access is brought to you for free and open access by Duquesne Scholarship Collection. It has been accepted for inclusion in Electronic Theses and Dissertations by an authorized administrator of Duquesne Scholarship Collection. For more information, please contact phillips@duq.edu.

EXAMINING THE ASSOCIATION BETWEEN DEPRESSIVE SYMPTOMS AND
PERFORMANCE ON EXECUTIVE FUNCTION MEASURES IN CHILDREN

A Dissertation

Submitted to the School of Education

Duquesne University

In partial fulfillment of the requirements for
the degree of Doctor of Philosophy

By

Hillary A. Mangis

December 2009

Copyright by
Hillary A. Mangis

2009

DUQUESNE UNIVERSITY
SCHOOL OF EDUCATION
Department of Counseling, Psychology and Special Education

Dissertation

Submitted in partial fulfillment of the requirements
for the degree
Doctor of Philosophy (Ph.D.)

School Psychology Doctoral Program

Presented by:

Hillary Anne Mangis
B.A. Liberal Arts, Washington & Jefferson College, 2002
M.S.Ed. School Psychology, Duquesne University, 2003

September 18, 2009

EXAMINING THE ASSOCIATION BETWEEN DEPRESSIVE SYMPTOMS AND
PERFORMANCE ON EXECUTIVE FUNCTION MEASURES IN CHILDREN

Approved by:

_____, Co-Chair
Ara J. Schmitt, Ph.D.
Assistant Professor
Department of Counseling, Psychology, & Special Education
Duquesne University

_____, Co-Chair
Jeffrey A. Miller, Ph.D., ABPP
Professor/Associate Dean
Graduate Studies and Research
Duquesne University

_____, Member
Glen E. Getz, Ph.D.
Assistant Professor, Psychiatry
Drexel College of Medicine
Allegheny General Hospital

ABSTRACT

EXAMINING THE ASSOCIATION BETWEEN DEPRESSIVE SYMPTOMS AND PERFORMANCE ON EXECUTIVE FUNCTION MEASURES IN CHILDREN

By

Hillary A. Mangis

December 2009

Dissertation supervised by Ara J. Schmitt, Ph.D. & Jeffrey A. Miller, Ph.D., ABPP

A variety of cognitive deficits have been linked to depression. In particular, data exists to suggest that persons with depression are subject to poorer executive function compared to normal controls. Establishing the connection between depression and impaired executive function is particularly important in childhood as a child's daily functioning, including social interactions and academic performance, may be impacted. The purpose of this study was to explore if children with significant symptoms of depression displayed deficits on tasks designed to measure the executive functions of attentional control, information processing and cognitive flexibility (Anderson, 2002) compared to a clinical control group. A clinical sample of children referred for outpatient, neuropsychological evaluation was used in this investigation. Results revealed that the sample of children with elevated symptoms of depression did not demonstrate impaired,

or worse executive function performance compared to clinical controls. Further investigations should examine executive function within the context of verified clinical depression, and with an expanded array of executive function measures, including ratings of executive function across settings.

DEDICATION

Eric, Mom, Ryan, and the rest of my family, thank you so much for your love, support, and encouragement over the years. I would like to dedicate this work to my beautiful daughter, Kennedy Aryn Kuntz, to remind you that nothing is beyond your reach. I love you baby girl.

ACKNOWLEDGEMENTS

I would like to thank my committee members for all of the encouragement, support, and expertise that they provided to me throughout this journey. Dr. Miller, thank you for filling my mind with thoughts of executive functioning and providing me with guidance throughout my time at Duquesne. Dr. Getz, thank you for guiding my initial thinking with this project and providing me with the data to realize the end results. Dr. Schmitt, thank you for your support with organizing, polishing, and finishing my dissertation. Lastly, I express my gratitude to Allegheny General Hospital's Department of Psychiatry-Neuropsychiatry for providing the data utilized in this dissertation.

TABLE OF CONTENTS

<i>Heading</i>	<i>Page</i>
CHAPTER I: INTRODUCTION	1
Depression	1
Academic Implications of Depression	4
Neurobiological Etiology of Depression	4
Executive Functions	6
Depression and Executive Functions	8
Critical Analysis of Current Literature	8
Implications of Current Study	10
Research Questions and Hypotheses	11
CHAPTER II: LITERATURE REVIEW	12
Childhood Depression	12
Diagnostic Presentation	12
Developmental Manifestation	16
Clinical Course and Functional Implications	17
Neurobiological Etiology of Childhood Depression	19
Diagnosis	23
Executive Functions	29
Conceptualization	29
Associated Deficits with Executive Dysfunction	31
Measurement of Executive Functions	33

TABLE OF CONTENTS (CONT.)

<i>Heading</i>	<i>Page</i>
Cortical Development	35
Depression and Executive Functions	37
Current Research Linkages	38
Current Study	46
CHAPTER III: METHOD	48
Purpose	48
Participants	48
Measures	49
Research Design	54
Procedures	55
Data Analyses	55
A Priori Statistical Analyses	56
Research Questions and Hypotheses	57
CHAPTER IV: RESULTS	61
Demographic Characteristics	61
Descriptive Statistics	66
Preliminary Analyses	67
Research Question 1	67
Research Question 2	69
Research Question 3	70

TABLE OF CONTENTS (CONT.)

<i>Heading</i>	<i>Page</i>
CHAPTER V: DISCUSSION	73
Summary of Motor Speed Results	74
Summary of Cognitive Fluency Results	76
Summary of Cognitive Flexibility Results	77
Limitations	78
Recommendations for Future Research	81
Implications for School Psychologists	82
Conclusions	82
REFERENCES	84

LIST OF TABLES

<i>Table</i>	<i>Page</i>
Table 1. Primary DSM-IV-TR Diagnosis by Group Status	64
Table 2. Descriptive Statistics Regarding Executive Function Dependent Variables	66
Table 3. Pearson Bivariate Correlations among the Executive Function Scales	67
Table 4. Pearson Bivariate Correlations among CBCL and D-KEFS Variables	68

CHAPTER I

INTRODUCTION

Depression

Childhood depression has increasingly become a public health concern over the past three decades. In fact, the National Institute of Mental Health (2005) reports that up to 2.5 percent of children and 8.3 percent of adolescents suffer from depression. Typical symptoms include decreased concentration and indecisiveness, depressed/irritable mood, failure to make expected weight gains, fatigue, feelings of guilt/worthlessness, insomnia or hypersomnia, morbid thoughts, suicidal ideations/attempts, psychomotor agitation or retardation (Louters, 2004). In turn, these symptoms often adversely affect the daily functioning of children across environments. Widely documented is that impaired daily, social/interpersonal, and academic functioning with an increased likelihood of family problems, substance abuse, and truancy issues are manifestations of childhood depression (Emslie & Mayes, 1999). Furthermore, public health officials are placing an increasing emphasis on early intervention, given that the early onset of depressive symptoms increases the risk for the continued presence of symptoms into adulthood and further negative outcomes (Brent, Ryan, Dahl, & Birmaher, 2005; Kovacs & Devlin, 1998). Of additional concern is the increased risk for suicide attempts and completions (Rudd, Joiner, & Rumzek, 2004). Treatment vigilance is required as research suggests persistent functional impairment even after recovery from a depressive episode (Puig-Antich et al., 1993).

In order to best understand childhood depression and appropriately intervene, four symptom categories should be considered. These include the following: emotional

symptoms, motivational symptoms, behavioral/vegetative symptoms, and cognitive symptoms (Berk, 2003; Bernstein, Clarke-Stewart, Roy, & Wickens, 1997; Birmaher, Brent, & Benson, 1998; Comer, 2001; Dubuque, 1998; Gençoz, Voelz, Gençoz, Pettit, & Joiner Jr., 2001; Global Mental Health Network, n.d.; Kendall, Stark, & Adam, 1990; Lamarine, 1995; Louters, 2004; Mayberg, Keightly, Roderick, & Brannan 2004; Timbermont & Braet, 2004; To, Zepf, & Woods, 2005). Children with depression are emotionally dysregulated, with a presentation commonly marked by feelings of sadness, dejection, frustration, hopelessness, inadequacy, worthlessness, and guilt. These children also exhibit a state of persistent sadness and an irritable mood for extended periods of time, coupled with decreased motivation (Global Mental Health Network, n.d.). Withdraw from friends, family, and activities that once brought them pleasure is often noted. This withdraw is quite pronounced compared to previous levels of energy and participation and is thought to result from anhedonia, listlessness, and an inability to find enjoyment in life (Louters, 2004). Behaviorally, children with depression have difficulty sleeping, appear agitated, demonstrate changes in appetite, and exhibit decreases in concentration and energy (Berk, 2003; To, Zepf, & Woods, 2005).

Moreover, the cognitive distortions associated with depression may adversely affect many aspects of a child's daily functioning. A hallmark characteristic of childhood depressive disorder is the lack of positive, and the presence of negative, cognitive features. For example, children with depression typically have poorly developed self-schemas and present with dysfunctional information processing styles, which tend to focus on the negative aspects of events and situations (Gençoz, Voelz, Gençoz, Pettit, & Joiner Jr., 2001). Children with depression also demonstrate negative self-evaluations,

leading to distorted thought processes that affect numerous areas of functioning, including social interactions and academics (Kendall, Stark, & Adam, 1990; Timbermont & Braet, 2004). Furthermore, soft-signs of cognitive impairment associated with depressive disorders in children include: maladaptive attention, memory, psychomotor speed, motivation, and organizational abilities (Bulbena & Berrios, 1993; Mayberg, Keightly, Roderick & Brannan 2004). Difficulties in language, perception, and spatial abilities have also been identified secondary to the presence of depression in adults (Mayberg, Keightly, Roderick, & Brannan, 2004). A better understanding of the specific cognitive deficits associated with childhood depression is needed in order to establish targets for intervention and treatment planning across functional settings.

Academic Implications of Depression

Children with depression are at increased risk for academic failure. It appears as though academic difficulties commonly associated with depressive disorders may in part be manifestations of cognitive inefficiency (e.g., memory impairment, poor motivation, lack of task initiation, poor organization, decreased concentration and attention, and difficulty monitoring performance) associated with depression, (Louters, 2004; McDonough-Ryan et al., 2002). Often, the first signs of childhood depression are observed in the classroom (Dubuque, 1998), with unexplained deterioration in school performance being one of the more overt signs (House, 1999). Another sign of childhood depression, which puts the student at risk of further negative outcome, is chronic absences from school (Global Mental Health Network, n.d.). This is particularly problematic as the data are clear that lack of academic engagement may directly be related to academic failure (see Shapiro, 2004, for a review). In order to avoid academic

failure, and design effective interventions, a better understanding of cognitive deficits associated with childhood depression is required.

Neurobiological Etiology of Depression

Recent research has focused on the neuropathology of depression. Morphological abnormalities in the left hemisphere of the brain have been observed in participants with depression (Bolla-Wilson, Robinson, Starkstein, Boston, & Price, 1989; Jacobs & Snyder, 1996; Keightley, Winocur, Graham, Mayberg, Hevenor, Grady, 2003). Other studies have indicated bilateral brain activity differences. For example, people with depression have consistently demonstrated decreased activity in the left and increased activity in the right prefrontal cortex (Davidson, 1984; Starkstein & Robinson, 1986). These studies provide compelling evidence that associates depression with impaired functioning of the left hemisphere, as well as both the left and right prefrontal cortex.

Studies using positron emission tomography (PET), single photon emission computered tomograph (SPECT), magnetic resonance spectroscopy (MRS), and structural and functional magnetic resonance imaging (MRI) have also been used to examine and the neurobiological aspects of depression in adults (Kaufmann, Blumberg, & Young, 2004; Mayberg et. al, 2004). In their review, Liotti and Mayberg (2001) state that SPECT and PET studies consistently implicate hypometabolism of the dorsal prefrontal cortices, cingulate cortex, and other paralimbic cortex structures (orbitofrontal, insular, and anterior temporal cortex). Additional research has consistently implicated the components of the limbic system, temporal lobes and frontal lobe areas (Andrewes, 2001; Mayberg, 1997).

Antidepressant treatment of depression has also shed some insight into the neurobiological correlates of depression. The four major categories of antidepressant drugs (tricyclics, monoamine oxidase inhibitors, selective serotonin reuptake inhibitors, and atypical antidepressants) act on various neurotransmitters, particularly norepinephrine and serotonin (Comer, 2001; To, Zeph, & Woods, 2005). In fact, the prevailing hypothesis of depression is that there is a deficiency in the monoamine neurotransmitters, particularly norepinephrine and serotonin combined with the influence of environmental factors (To, Zeph, & Woods, 2005). The physiological implications arising from antidepressant treatment suggest that mood depends on the effects of a combination of neurotransmitters and people with mood disorders have different combinations of neurotransmitter abnormalities (Kalat, 2001).

Research linking depression with the frontal lobes would seem to suggest possible involvement of executive functioning, which is modulated by the structures in the frontal lobes, including the orbitofrontal prefrontal cortex (Kaufmann, Blumberg, & Young, 2004; Mayberg et. al, 2004). Mayberg's (1997) working model of depression implicates failure of the coordinated interactions of a distributed network of limbic-cortical pathways, which would include the connections to systems associated with executive functioning, a very important component in cognitive functioning. Mayberg (2003) discusses findings from blood flow and glucose metabolism studies, which consistently implicate frontal abnormalities in depressed subjects. In particular, there are noted decreases in frontal lobe function and cingulate and limbic-paralimbic abnormalities.

The interaction between the frontal and subcortical circuits in depression is described by Mayberg (2003) as “‘network’ dysfunction” (p. 195). In this model,

depression is theorized to be a disorder involving interconnections between systems that fail to establish homeostasis in emotional control during times of increased stress.

Mayberg et al. (1999) demonstrated this model through two different PET techniques.

They found limbic-paralimbic and neocortical regions to be effected by mood state, with influences of depressed mood on attention.

Using functional-magnetic resource imaging (f-MRI), Keightley et al. (2003) found that frontal lobe functioning operates in a “top-down” fashion to the limbic and temporal areas, whereby cognitive factors such as attentional control have strong implications for depression. Connections between depression and frontal lobe functioning could be particularly important in helping to understand the cognitive deficits associated with depression, with the expectation that this better understanding can lead to research and development of better interventions designed to remediate these deficits in children.

Executive Function

Executive function is thought of as a broad term used to describe higher order cognitive skills such as planning, organizing, and problem solving (Anderson, 2002; Andrewes, 2001; Hughes & Graham, 2002). Research to date has conceptualized executive functions in two different ways: a unidimensional construct, or conversely, a set of multiple, interrelated, and interdependent processes. Based on Stuss and Alexander’s (2000) model, Anderson (2002), elaborated that executive functions can be conceptualized as four distinct domains: attentional control, information processing, cognitive flexibility, and goal setting. Attentional control involves the ability to selectively attend to stimuli and inhibit responses, while focusing attention for a period of time. Information processing involves fluency, efficiency, and speed by which output is

processed. Cognitive flexibility includes the ability “to shift between response sets, learn from mistakes, devise alternative strategies, divide attention, and process multiple sources of information concurrently (Anderson, 2002, p. 74).” Finally, goal setting involves problem solving abilities, particularly the ability to develop and elaborate upon new concepts and determine an efficient course of action. All of the domains are considered discrete functions; however, they operate interactively to execute tasks. Each domain is influenced by and interacts with the others to process stimuli from various sources (Stuss & Alexander, 2000). Related to the current study, it is hypothesized that measures of executive function that tap the domains of attentional control, information processing, and cognitive flexibility will be negatively impacted by the presence of depressive symptoms.

Executive functioning is a cognitive process commonly associated with the frontal lobe and the orbitofrontal prefrontal cortex (Kaufmann, Blumberg, & Young, 2004; Mayberg et al., 2004). Cognitive deficits, like deficits in executive function, are often seen in persons with depression and frontal lobe lesions. As such, recent research into the neurobiological underpinnings of the cognitive deficits associated with depression implicates the prefrontal cortex (Andrewes, 2001; Davidson, 1984; Starkstein & Robinson, 1986; Liotti & Mayberg, 2001; Mayberg, 1997). For example, memory impairment, poor motivation, lack of task initiation, poor organization, decreased concentration and attention, and difficulty monitoring performance are present in both. Likewise, deficits in academic and social functioning are also commonly documented between disorders (Anderson, 2002; Louters, 2004). Establishing which executive

function deficits co-occur with depression would likely lead to the creation of very specific and tailored interventions across settings.

Depression and Executive Functions

There are many overlaps between the cognitive deficits, brain structures, and functional implications associated with depression and executive dysfunction. Similarities in cognitive deficits between depression and executive functioning include memory impairments, poor motivation and initiation, poor organization, decreased concentration and attention, and difficulty monitoring performance (Anderson, 2002; Keightly et al., 2003; Mayberg et al., 2004; Stuss & Alexander, 2000). Additionally, research on the neurobiological origins of both topics has implicated the frontal lobes, prefrontal cortex, temporal lobes, and limbic system. Depression and executive functioning deficits are also both associated with functional impairment in academic and social environments (Anderson, 2002; Louters, 2004; McDonough-Ryan et al., 2002; Roberts & Wallace, 2000; Powell & Kytja, 2004). This would suggest that measures designed to identify deficits in executive function might also aid in the diagnosis and treatment of children with depression.

Critical Analysis of Current Literature

To date, studies examining the relationship between depression and executive functioning primarily have been conducted in geriatric and adult populations. The geriatric literature demonstrates consistent findings that depressed subjects are impaired on tasks of executive functioning (Abas, Sahakian, & Levy, 1990; Alexopoulos et al., 2000; Beats, Sahakian & Levy, 1996; Butters et al., 2000; Butters et al., 2004). However, results from studies examining executive function in non-geriatric populations are less

consistent. Purcell, Maruff, Kyrios, and Pantels (1997), found that adult participants exhibited motor slowing and deficits in attentional set shifting. However, they concluded that no global deficits in executive functioning were identified because performance on tasks assessing spatial span, spatial working memory, planning, and visual memory were not impaired. Limitations of their study include small sample size (n=20) and the medication status of the subjects (where 12 subjects were medicated and 8 were medication free). They concluded that their findings are evidence supporting the variability in the nature and severity of cognitive impairment in depression.

Conversely, Austin et al. (1999) found depressed subjects older than 20 years of age demonstrated impaired performance on several neuropsychological measures suggestive of frontal involvement. Specifically, they reported that the depressed sample was impaired on most mnemonic tasks, simple reaction time and Trail Making Test B, which assesses cognitive flexibility. This study also included sampling limitations, such as, recruitment of depressed subjects from a clinical population and recruitment of controls from a convenience sampling of patient's relatives, staff, and community volunteers at a hospital. Using relatives of depressed patients is particularly problematic given the strong genetic and familial components associated with depression (APA, 2000; Erlenmeyer-Kimling et al., 1997; Kessler et al., 1994; Regier et al., 1993; Rice, Harold, & Thapar, 2002; Weismann et al., 1991; Wender et al., 1986).

Limited studies have been conducted with children, though similar research found that boys, ages 9-11, with symptoms of anxiety and depression demonstrate impaired frontal functioning including slower processing speed, number of perseverative errors, set shifting, hypothesis testing, and categorical problem solving (Emerson, Mollet, &

Harrison, 2005). However, this sample consisted of only male subjects and failed to study the separate effects of depression and anxiety, making generalizability tenuous at best. This previous research demonstrates a need to further understand the relationship between depression and executive functioning at all age levels, and specifically in childhood. However, there continues to be a paucity of research regarding the effect of depression on executive functions in children.

Implications of Current Study

Understanding the cognitive deficits associated with childhood depression will help explain problems that occur in the academic setting and result in academic failure, as well as aid in the design of effective interventions. Cognitive deficits in attention, memory, psychomotor speed, motivation and organizational abilities have been reported in depressed adults (Bulbena & Berios, 1993, Mayberg, Keightly, Kendrics, & Brannan, 2004). However, a link between psychometric assessment of executive function and depression in children has not been firmly established. Given the cognitive symptom overlap between depression and those with executive function deficits (e.g., deficits in information processing, attention, psychomotor speed, motivation, and organizational abilities), this study represents an attempt to add to the literature base by exploring the presence of executive dysfunction in depressed youth (Mayberg et al., 2004). The current study adds to the literature by examining if the mere presence of elevated symptoms of depression is related to impaired executive function. The current study utilized a battery of neuropsychological instruments to investigate the nature and degree of executive dysfunction in children with elevated depressive symptoms.

Research Questions and Hypotheses

This study explored whether children with clinically significant symptoms of depression displayed deficits on tasks designed to measure aspects of executive functioning. Specifically, three research questions were investigated:

1. Does the motor speed of children with elevated symptoms of depression differ from a clinical population of children without elevated symptoms of depression?

Hypothesis 1: Children with elevated depressive symptoms will perform poorer on tasks assessing motor speed compared to children without symptoms of depression.

2. Does cognitive fluency in children with elevated depressive symptoms differ from that of a clinical population of children without elevated depressive symptoms?

Hypothesis 2: Children with elevated depressive symptoms will perform poorer on tasks assessing cognitive fluency than children without depressive symptoms.

3. Does cognitive flexibility differ in children with elevated symptoms of depression differ from that of a clinical population of children without elevated depressive symptoms?

Hypothesis 3: Children with elevated depressive symptoms will perform poorer and within an impaired range on tasks assessing cognitive flexibility compared to children without clinically elevated depressive symptoms.

CHAPTER II

LITERATURE REVIEW

For the purpose of understanding the proposed relationship between symptoms of depression in children and executive function deficits, the following literature review examines proposed models of depression and executive function and derives conclusions as to the most empirically and theoretically sound model to be utilized in the current study. Extant research involving depression and executive functions is then discussed. This information is used to hypothesize relationships between depressive symptoms and executive functioning.

CHILDHOOD DEPRESSION

Diagnostic Presentation

Depression is a form of mood disorder consisting of depressive episodes that can range from occasional and short-lived to severe and long-lasting episodes (Bernstein, Clarke-Stewart, Roy, & Wickens, 1997). It consists of feeling low, sad, dark, and overwhelmed by life. Another type of mood disorder associated with depression is mania: a euphoric, breathless state marked by frenzied energy and exaggerated belief in one's abilities (Comer, 2001).

Many individuals diagnosed with a mood disorder suffer from unipolar depression, marked by feelings of depression only. Unipolar depression can take on different forms based on the length of the episode. Two different forms of unipolar depression include major depressive disorder and dysthymic disorder. Major depressive disorder is characterized by one or more major depressive episodes. Dysthymic disorder is characterized by at least 2 years of depressed mood for more days than not, but does

not meet the criteria for a major depressive episode (American Psychiatric Association, 2000). Another form of mood disorder is bipolar disorder, which is marked by alternating periods of depression and mania (Comer, 2001).

Childhood onset of affective illness has been recognized as a significant health problem over the past three decades (Brent, Ryan, Dahl, & Birmaher, 2005). The 2005 National Institute of Mental Health report indicates that up to 2.5 percent of children and 8.3 percent of adolescents suffer from depression. At elementary school age, affective disorders are equally common in males and females, a trend that is replaced with a higher occurrence of depression in females following the onset of puberty (Brent et al., 2005). Depression can affect children at any age and is the most common psychological problem of adolescence, with both severity and number of symptoms typically increasing sharply during adolescence (Berk, 2003).

Through examining the research, the symptoms of depression can be broken out into four categories: emotional symptoms, motivational symptoms, behavioral/vegetative symptoms, and cognitive symptoms (Berk, 2003; Bernstein, Clarke-Stewart, Roy, & Wickens, 1999; Birmaher, Brent, & Benson, 1998; Comer, 2001; Dubuque, 1998; Gençoz, Voelz, Gençoz, Pettit, & Joiner, 2001; Global Mental Health Network, n.d.; Kendall, Stark, & Adam, 1990; Lamarine, 1995; Louters, 2004; Mayberg, Keightly, Kendrics, & Brannan 2004; Timbermont & Braet, 2004; To, Zepf, & Woods, 2005).

Emotional Features

Emotional symptoms are the defining feature of mood disorders. Depression is marked by the child feeling sad and dejected (Comer, 2001). Additionally, they feel frustrated and hopeless, often thinking that things will never get better (Berk, 2003).

Children will typically exhibit persistent sadness and an irritable mood for an extended period of time (Global Mental Health Network, n.d.). Additionally, they may feel a pronounced sense of inadequacy, worthlessness, hopeless, or guilt (Bernstein, Clarke-Stewart, Roy, & Wickens, 1999). Crying spells are also common symptoms, sometimes for no apparent reason (Comer, 2001). The emotional features such as crying and irritability are the most overt indicators of impaired functioning in children with depression.

Motivational Features

The motivational features of depression include anhedonia, or the ability to derive feelings of pleasure from anything, including activities once enjoyed (Comer, 2001; Berk 2003). Children with depression may outright refuse to participate or they may comply with task demands but will exhibit little interest and exert little effort (Dubuque, 1998). Childhood depression is marked by listlessness, withdrawal, and an inability to find enjoyment in life (Louters, 2004). These children may pull away from friends and family members, feeling overwhelmed and exhausted by daily forms of social interaction (Comer, 2001). In summary, children with depression may withdraw from friends, family, and activities that once brought them pleasure. Often, this withdraw is quite pronounced from previous levels of energy and participation.

Behavioral/Vegetative Features

Behavior disturbances are also common in children with depression (Birmaher, Brent, & Benson, 1998). Behavioral manifestations in children with depression can often be mistaken for oppositionality and laziness (Dubuque, 1998). They are less active and less involved compared to non-depressed peers (Comer, 2001). Additionally, they have

difficulty sleeping, appear agitated, demonstrate changes in appetite, as well as decreased concentration and energy (Berk, 2003; To, Zepf, & Woods, 2005). They may also have difficulty getting along with others because they are negative, restless, grouchy, and full of complaints (Dubuque, 1998).

Cognitive Features

The associated cognitive deficits of depression have adverse effects on many aspects of a child's daily functioning and may be the cornerstone in helping to differentiate childhood depression from other psychological problems. A major characteristic of childhood depressive disorders is a shortage of positive cognitive features. For example, children with depression typically have poorly developed self-schemas and present with dysfunctional information processing styles, which tend to focus on the negative aspects of events and situations (Gençoz, Voelz, Gençoz, Pettit, & Joiner, 2001). They demonstrate negative self-evaluations, leading to distorted thought processes that affect numerous areas of functioning including social interactions and academics (Kendall, Stark, & Adam, 1990; Timbermont & Braet, 2004). Further, in adult populations, cognitive deficits have been observed in attention, memory, psychomotor speed, motivation, and organizational abilities (Bulbena & Berrios, 1993; Mayberg, Keightly, Mahurin, & Brannan, 2004). These problems can produce secondary difficulties in language, perception, and spatial abilities (Mayberg et al, 2004). It is likely that a better understanding of the cognitive deficits evidenced in children with depression could help to increase understanding with regards to the academic difficulties associated with childhood depressive disorders. This is especially important given that when depression is left untreated, there is opportunity for serious lifelong difficulties to arise

(Lamarine, 1995). Examining the cognitive functioning of children suspected of suffering from depression may be the essential factor in helping to differentiate depression from other diagnoses.

Developmental Manifestation

As childhood depression has become more appreciated as a serious childhood psychological disorder, developing a better understanding of the symptoms of and diagnosing mood disorders in children has become a focus of researchers. Understanding the role of development on the expression and manifestation of depressive symptoms has become a central idea of several researchers (Kovacs, Devlin, & House, 1999; Louters, 2004), whose work has demonstrated that children with depression demonstrate different symptom patterns based on age. Broadly speaking, typically observed symptoms in children include decreased concentration and indecisiveness, depressed/irritable mood, failure to make expected weight gains, fatigue, feelings of guilt/worthlessness, insomnia or hypersomnia, morbid thoughts, suicidal ideations/attempts, psychomotor agitation or retardation (Louters, 2004).

However, recent data suggests that developmental features associated with age impact the expression of symptoms of childhood depressive disorders (Birmaher, Brent, & Benson, 1998; Kashani, Rosenberg, & Reid, 1989; Kovacs & Devlin, 1998; House, 1999). This can complicate the recognition and treatment of childhood depression. The rapid changes that occur in normal childhood development can cause symptoms of depression to go unnoticed and/or be attributed to normal development. For example, normally developing children and adolescents may experience many rapid changes in physiological states, which can contribute to changes in mood and emotional states that

vary across developmental trajectories (Larson, Csikszentmihalyi, & Graef, 1980). Additionally, young children are likely to experience more externalized symptoms, somatic complaints, auditory hallucinations, temper tantrums, crying, stomachaches, failure to stand up for self, nightmares, anhedonia and/or behavior problems (Birmaher, Brent, & Benson, 1998).

The symptoms distinguishing depression in early childhood tend to diminish in middle to late childhood. At this age, symptoms are more likely to include more of the internalizing components associated with depression which typically include dysphoric mood, low self-esteem, reports of fatigue, not caring if hurting oneself, agitation when sad, frequent irritability, not liking to go out, feeling bored, and feelings of guilt and hopelessness (Birmaher, Brent, & Benson, 1998). On the other hand, adolescents manifest more sleep and appetite disturbances, delusions, suicidal ideation and attempts, and irritability (Birmaher et al., 1998; Kashani, Rosenberg, & Reid, 1989). These feelings and symptoms cause significant distress and impairment in daily functioning (Bernstein, Clarke-Stewart, Roy, & Wickens, 1997).

Clinical Course and Functional Implications

It is important to understand the symptoms of childhood depression because they have many adverse affects on the day to day functioning of childhood sufferers. If left untreated, depression can be long lasting and recurring. There is general agreement that major depression in childhood and adolescence is a chronic and recurrent condition, lasting on average between 7-9 months, and with over half of those diagnosed relapsing at some point in the future (Birmaher et al., 1998; Lamarine, 1995). An early onset diagnosis is associated with a pattern of depressive symptoms that continues into

adulthood (Brent et al., 2005; Kovacs & Devlin, 1998; Kessler, Avenevoli, & Merikangas, 2001), with research suggesting persistent functional impairment after recovery from a depressive episode (Puig-Antich et al., 1993).

Another significant risk is that childhood affective disorders are prevalent in completed suicide attempts and multiple attempters are frequently diagnosed with major depressive disorder (Rudd, Joiner, & Rumzek, 2004), indicating significant morbidity and mortality (Birmaher, Brent, & Benson, 1998). The significant functional impairment found in children with depression suggests that researchers and practitioners must strive for early and accurate identification of depression to help alleviate the impact the illness has on children's functioning. Moreover, early and accurate diagnosis is crucial in attempts to prevent the social/interpersonal and academic difficulties that are associated with depression.

Interpersonal/Psychosocial Outcomes

Depressive disorders are associated with poor interpersonal and psychosocial outcomes for children suffering with the symptoms of depression (Louters, 2004; Birmaher et al., 1998). Typically, children with depressive disorders are poor communicators who are likely to socially isolate themselves from others, a problem that causes significant distress to children (Global Mental Health Network, n.d.). Depressed children can also be aggressive and angry (Dubuque, 1998), which can make them difficult to get along with and ultimately strain relationships. Children diagnosed with depression are likely to have strained or poor relationships with others including their peers (Birmaher, Brent, & Benson, 1998). They have a difficult time interpreting social cues that enable them to communicate and interact effectively. These psychosocial

difficulties produce great strain and turmoil for the child suffering from depression, significantly impacting their day to day interactions.

Academic Difficulties

Academic difficulties are a common associated difficulty of depressive disorders as well (Louters, 2004; McDonough-Ryan et al., 2002). In fact, signs of childhood depression will often be noticed in the classroom (Dubuque, 1998), with unexplained deterioration in school performance being one of the more overt indicators of a depressive disorder (House, 1999). Not only are these children more likely to perform poorly (Rapport, Denney, Chung, & Hustace, 2001), but they also demonstrate more frequent absences from school (Global Mental Health Network, n.d.). They show decreased concentration and increased indecisiveness, which appears to significantly influence academic outcomes (Louters, 2004). They are also more sensitive to rejection or failure and are often labeled as lazy (Global Mental Health Network, n.d.).

Neurobiological Etiology of Childhood Depression

Understanding the neurobiological etiology of depression may aid in early identification and diagnosis of early onset depression. Recent research in both adult and childhood depression has focused on genetic connections in an attempt to aid in understanding the causes and identification of childhood depression. Overall, it appears that childhood onset depression shows a strong genetic component, both in twin and family studies (APA, 2000; Erlenmeyer-Kimling et al., 1997; Kessler et al., 1994; Regier et al., 1993; Rice, Harold, & Thapar, 2002; Weismann et al., 1991; Wender et al., 1986). In fact, adopted children tend to resemble their biological parents more closely than their

adoptive parents (Wender et al., 1986). That is, early-onset depression is associated with increased risk among immediate family members.

Also, family history of depression increases risk of onset more strongly in females than males (Bierut et al., 1999). However, the cause of this finding has yet to be explained, namely because the probability of depression does not correlate strongly with hormone levels (Roca, Schmidt, & Rubinow, 1999). Moreover, no specific gene or constellation of genes has been located that is strongly linked to depression. It is likely that a combination of several genes as well as environmental stressors increase the risk of symptoms of depression (McQuillin, Lawrence, Kalsi, Chen, & Gurling, 1999).

Additionally, research has focused on the neurobiological origins of depression, finding strong relationships between depression and neurobiological abnormalities. Some studies have demonstrated that morphological abnormalities in the left hemisphere of the brain have been observed in depressed subjects (Bolla-Wilson, Robinson, Starkstein, Boston, & Price, 1989; Jacobs & Snyder, 1996; Keightley, Winocur, Graham, Mayberg, Hevenor, Grady, 2003). Other studies have indicated bilateral differences in activity patterns in different brain structures. For example, depressed people have consistently demonstrated decreased activity in the left and increased activity in the right prefrontal cortex (Davidson, 1984; Starkstein & Robinson, 1986). These studies provide strong evidence implicating the involvement of the left hemisphere and the right prefrontal cortex in depressed patients.

Studies using positron emission tomography (PET), single photon emission computered tomograph (SPECT), magnetic resonance spectroscopy (MRS), and structural and functional magnetic resonance imaging (MRI) have also examined and

shed light on the neurobiological aspects of depression (Kaufmann, Blumberg, & Young, 2004; Mayberg et al., 2004). In their review, Liotti and Mayberg (2001) state that SPECT and PET studies consistently implicate hypometabolism of the dorsal prefrontal cortex, cingulate cortex, and other paralimbic cortex (orbitofrontal, insular, and anterior temporal cortex). Mayberg, Brannan, Mahurin, Jerabek, Brickman, Tekell, Silva, McGinnis, Glass, Martin, & Fox (1997) found an important role of the cingulate as a bridge linking the dorsal and ventral pathways necessary for processing mood and cognitive behaviors in a normal, non-negative manner. Additional research has consistently implicated the components of the limbic system, temporal lobes and frontal lobe areas. The frontal lobes have been demonstrated to have established connections with the limbic system, the area in the brain associated with emotion (Andrewes, 2001; Mayberg, 1997). Further, Mayberg (2002) reported that the dorsal and ventral prefrontal cortex, the inferior parietal region, the anterior cingulate gyrus, the anterior insula, and the posterior cingulate display hypometabolism in depressed patients. She adds that this supports the associated altered cognitive performances in depressed patients.

Antidepressant treatment of depression has also shed some insight into the neurobiological etiology of depression. The four major categories of antidepressant drugs (tricyclics, monoamine oxidase inhibitors, selective serotonin reuptake inhibitors, and atypical antidepressants) act on various neurotransmitters, particularly norepinephrine and serotonin (Comer, 2001; To, Zeph, & Woods, 2005). In fact, the prevailing hypothesis of depression is that there is a deficiency in the monoamine neurotransmitters, particularly norepinephrine and serotonin combined with the influence of environmental factors (To, Zeph, & Woods, 2005). The physiological implications from antidepressant

treatment demonstrate that mood depends on the effects of a combination of neurotransmitters and different people with mood disorders have different combinations of neurotransmitter abnormalities (Kalat, 2001).

Research linking depression with the frontal lobes would seem to suggest possible involvement of executive functioning, which is modulated by the frontal lobes, particularly the orbitofrontal prefrontal cortex (Kaufmann, Blumberg, & Young, 2004; Mayberg et al., 2004). Mayberg's (1997) working model of depression implicates failure of the coordinated interactions of a distributed network of limbic-cortical pathways, which would include the connections to systems associated with executive functioning, a very important component in cognitive functioning. Mayberg (2003) discusses findings from blood flow and glucose metabolism studies, which consistently implicate frontal abnormalities in depressed subjects. In particular, there are noted decreased frontal lobe function and cingulate and limbic-paralimbic abnormalities. Similar findings were reported by Castillo, Kwock, Courvoisie, & Hooper (2000), who reported increased glutamate/glutamine in the frontal lobes of children with bipolar disorder, which related to impaired performance on the executive function, attention, sensorimotor, and memory subscales on the NEPSY.

The interaction between the frontal and subcortical circuits in depression is described by Mayberg (2003) as "'network' dysfunction" (p. 195). In this model, depression is theorized to be a disorder involving interconnections between systems that fail to establish homeostasis in emotional control during times of increased stress. Mayberg, Liotti, Brannan, et al. (1999) demonstrated this model through two different PET techniques. They found limbic-paralimbic and neocortical regions to be effected by

mood state, with influences of depressed mood on attention. Further research regarding neurobiological origins of depression points to the limbic-hypothalamic-pituitary-adrenal (LHPA) system. Lopez (2005) reports that hyperactivity of the LHPA system is found in depression, which is observable through the overproduction of cortisol, the stress hormone, in both the hippocampus and the prefrontal cortex.

Using fMRI, Keightley et al. (2003) found that frontal lobe functioning operates in a “top-down” fashion to the limbic and temporal areas, whereby cognitive factors such as attentional control have strong implications for depression. Connections between depression and frontal lobe functioning could be particularly important in helping to understand the cognitive deficits associated with depression, with the expectation that this better understanding can lead to research and development of better interventions designed at remediating these deficits in children.

Diagnosis

Since childhood depression is recognized as a serious health problem in children, marked by significantly impaired functioning (Ryan, 2001), it is important that the diagnostic criteria for depression be accurate and clear to ensure accurate and proper diagnosis of childhood depressive disorders. Currently, depression in children is diagnosed according to the criteria established by the American Psychiatric Association’s *Diagnostic and Statistical Manual of Mental Disorder, Fourth Edition, Text Revision* (DSM-IV-TR, 2000). The criteria for a diagnosis of major depressive episode include five or more of the following symptoms present during the same 2-week period:

“(1) depressed mood most of the day nearly every day, which is replaced by irritable mood in children, (2) markedly diminished interest or pleasure in all

or most all of activities, (3) significant weight loss or weight gain, (4) insomnia or hypersomnia, (5) psychomotor agitation or retardation, (6) fatigue or loss of energy, (7) feelings of worthlessness or excessive/inappropriate guilt, (8) diminished ability to think or concentrate or indecisiveness, (9) recurrent thoughts of death” (American Psychiatric Association, 2000, p. 356).

The most commonly diagnosed forms of depression in children include Major Depressive Disorder, Dysthymic Disorder, and Depressive Disorder, NOS (Emslie & Mayes, 1999). Diagnosis of mood disorders typically includes interviews of the parent and child (Kowatch, DelBello, Mayes, Kennard, & Emslie, 2006).

Measurement tools. Evaluation of childhood depressive symptoms is often aided by the use of unstructured clinical/developmental interviews, structured-interviews, semi-structured interviews, and self- and observer-rating scales (Carlson, 2000). These measurement instruments are developed to serve different functions, either diagnostic identification in the case of interviews or symptom evaluation in the case of rating scales. For a complete and comprehensive review of available tools for the diagnosis and measurement of adolescent depression, please refer to Brooks & Kutcher (2001).

The following list includes some of the interview tools available to assess the presence of depression in children: Schedule for Affective Disorders and Schizophrenia in School-Age Children (K-SADS; Puig-Antich & Chambers, 1978), Child and Adolescent Psychiatric Assessment (CAPA; Angold et al., 1995), Children’s Interview for Psychiatric Symptoms (ChIPS; Fristad et al., 1998a, Fristad et al., 1998b), Diagnostic Interview for Children and Adolescents (DICA; Herjanic & Reich, 1982), and the Diagnostic Interview Schedule for Children (DISC; Shaffer, Fisher, & Lucas, 1999).

Limitations of structured and semi-structured interviews are that administration is time-intensive, expensive, and often requires administration by a skilled clinician (Costello & Angold, 1988; Klein, Dougherty, & Olino, 2005). Also, these interviews are designed to assess overall psychosocial functioning, and are not specific to depressive symptoms. Further, interviews are typically administered to multiple informants and there remains uncertainty regarding how to combine the data to yield a diagnosis (Kessler, Avenevoli, & Merikangas, 2001). However, structured interviews for children have been shown to demonstrate comparable reliability and validity in assessing child and adolescent mental health concerns as adult instruments (Ryan, 2001).

On the other hand, rating scales are widely administered as screening tools to assess current symptoms and behaviors associated with pathology (Klein, Dougherty, & Olino, 2005). Rating scales can include self-report and observer-report (parent or teacher report) measures and can assess broad-band or narrow band pathology. Rating scale checklists serve as a cost-effective and efficient way to screen for psychopathology in children and adolescents (Doyle, Ostrander, Skare, Crosby, & August, 1997). Additionally, rating scales are attractive in that they can be completed by clients themselves and do not require extensive training to score (Costello & Angold, 1988). However, rating scales are not sufficient to warrant a diagnosis (Klein, Dougherty, & Olino, 2005). Some of the available self-rating scales include: the Child Depression Inventory (CDI; Kovacs, 1992) and The Mood and Feelings Questionnaire (MFQ; Angold, Costello, Messer, & Pickles, 1995).

An example of a narrow-band observer-rating scale for depressive symptoms includes the Child Depression Rating Scale (CDRS; Poznanski, Cook, & Carroll, 1979;

CDRS-R; Poznanski, E.O., & Morkros, H.B, 1999). Additionally, several broad-band observer rating scales exist including the Behavior Assessment Scale for Children (BASC; Reynolds & Kamphaus, 1992) and the Child Behavior Checklist (CBCL; Achenbach & Rescorla, 2001). One problem with observer-rating scales is that they often rely solely on behavioral manifestations rather than internal psychological processes, which can result in under-diagnosis (Puura et al., 1998).

Difficulty with Diagnosis

Despite the presence of established and universal diagnostic criteria, there are many problems associated with diagnosing children with affective disorders. One problem is that the symptoms are often hard to recognize in children because they may present differently from the typical symptoms seen in depressed adult populations (Louters, 2004). It is recognized in clinical practice that the expression of the symptoms of childhood onset depression tends to differ slightly from adult onset depression. For example, the melancholy features typically observed in adults is replaced with observed agitation and irritability in children (American Psychiatric Association, 2000; Birmaher, Brent, & Benson, 1998). In younger children, comorbid separation anxiety, phobias, somatic complaints and behavioral problems are more frequently observed than in adolescent and adult populations (Ryan, 2001). Additionally, children have greater difficulty verbally expressing their emotions, so much of the overt symptoms of depression are expressed through somatic complaints, anxiety, and irritability (Son & Kirchner, 2000). Ryan (2001) describes children as having limited abilities to identify and label their internal affective state. The developmental differences are more pronounced the younger the age of the child at onset (Clarizo, 1989; Lamarine, 1995),

with younger age of onset associated with more severe symptoms and recurrence rates (Clarizio, 1989; Kovacs, 1998), as well as lifelong patterns of impaired psychosocial functioning (Lewinshohn, Rohde, Seely, Klein, & Gotlib, 2003). Furthermore, children with depression are more likely than adults to have comorbid diagnoses (Carlson, 2000). The above-mentioned associations of childhood depression make it difficult to recognize the expression of common diagnostic symptoms in children with depression.

Another problem in diagnosing depression in children relates to the available diagnostic tools. Traditionally, measures used to aid in diagnosing depressive illness, including the DSM-IV-TR, have typically been developed for use with adult populations. Only recently have attempts been made to adapt measures for children (Kaufman, Birmaher, Brent, Ryan, 1996; Komar, 1999; Kovacs, 1982) with limited diagnostic utility being demonstrated by these self-report measures because children may often be unable to “identify and appropriately express their emotional experiences” (Louters, 2004, p.20). Subsequently, there is a lack consensus within the field of psychology on the applicability and utility of the DSM-IV-TR criteria in diagnosing childhood depression (Louters, 2004). This debate is driven by the understanding that the clinical manifestation of depression varies across developmental stages and the difficulty children experience with expressing emotional states, both of which lead to poor accuracy in diagnostic identification (Brent et al., 2005; Birmaher et al., 1998; Louters, 2004; Son & Kirchner, 2000). Further, there is a lack of consensus amongst researchers regarding which is the most reliable and valid diagnostic tool (Brooks & Kutcher, 2001). Therefore, it is imperative that the field continues to expand upon the current research regarding the development of diagnostic tools and accuracy for childhood depression. Through

continued research of diagnostic tools, researchers and clinicians can continue to develop a more complete understanding of the clinical manifestation and presentation of childhood depression.

Comorbid Disorders and Symptoms

Depressive disorders in children are associated with increased chances of comorbid symptoms with other psychological disorders, which can further complicate timely and accurate diagnosis (Alessi & Magen, 1988; Birmaher et al., 1998; Gerhardt, Compas, Connor, & Achenbach, 1999; Kovacs & Devlin, 1998; Shoaf, Graham, & Mayes, 2001). Specifically symptoms in children with depression may be misattributed to comorbid disorders where similar symptoms overlap with disorders such as Attention Deficit Hyperactivity Disorder (ADHD), Oppositional Defiant Disorder, and/or Anxiety Disorder (Dubuque, 1998). Diler, Daviss, Lopez, Axelson, Iyengar, and Birmaher (2007) report that the cognitive symptoms of depression, including social withdrawal, anhedonia, depressive cognitions, suicidal thoughts, and psychomotor retardation are important in differentiating Major Depressive Disorder from ADHD. Further, children with depression are also more likely to have comorbid learning disabilities (Lamarine, 1995). These comorbid conditions can often mask the actual symptoms of the depressive disorder leading to missed opportunities for early identification and treatment, which in turn can lead to the development of further cognitive impairments. However, understanding the overlapping symptoms and comorbid conditions associated with depression may help guide researchers in developing more precise diagnostic criteria to distinguish depression from these disorders, as well as contribute to the understanding of childhood depression.

EXECUTIVE FUNCTIONS

Conceptualization

There have been many attempts to define executive functioning; however, no common agreed upon definition is established to date. Currently it is commonly used as a broad term used to describe higher order cognitive skills such as planning, organizing, and problem solving (Anderson, 2002; Andrewes, 2001; Hughes & Graham, 2002). A simplified way of explaining executive functions is thinking of them as the “cognitive abilities responsible for controlling and coordinating performance in complex cognitive tasks” (Klenberg, Korkman, & Lahti-Nuutila, 2001).

Andrewes (2001) describes the executive system as “a series of systems, with each system vulnerable to interference or disruption at various levels” (p.85). These systems include control, organization/synthesis/judgment, attention, planning/sequencing/monitoring, and personality. According to Anderson (2002), executive functioning incorporates several interdependent processes that mediate goal directed behaviors and have often been conceptualized in two different ways. In the first conceptualization, executive functioning is a single or unidimensional construct, typically referred to as the central executive. The central executive is responsible for multimodal processing and higher level cognitive skills (Baddeley & Hitch, 1974; Baddeley, 1996; Della Sala, et al., 1998; Shallice, 1990). According to Baddeley (1996), the central executive is housed in the frontal lobes and is the least developed and researched component of his working memory model in terms of its impact on cognition.

Another conceptualization is that executive functions are multiple processes that are interrelated and interdependent. This system is referred to as the supervisory control

system, which provides the ability to selectively attend to specific stimuli and inhibit proponent responses, as well as focus attention for prolonged periods of time (Stuss & Alexander, 2000; Anderson, 2002). All of the domains are considered discrete functions; however, they operate interactively to execute tasks. Each domain is influenced by and interacts with the others to process stimuli from various sources. The conceptualization of executive functions as a complex system was supported by an exploratory factor analysis conducted by Pineda & Merchan (2003), who reported a five-factor structure. Miller (2005) discusses executive functions as tasks of self-regulation and tasks of metacognition. Self-regulation tasks are described as guiding current mental and behavioral activities and encompass inhibition, flexibility (shifting), and emotional control. Metacognitive tasks involve the coordination of complex activities and include working memory, problem solving, and monitoring. Based on Stuss and Alexander's (2000) model Anderson (2002) elaborated that executive functions can be conceptualized as four distinct domains: attentional control, information processing, cognitive flexibility, and goal setting. Attentional control involves the ability to selectively attend to stimuli and inhibit responses, while focusing attention for a period of time. Information processing involves fluency, efficiency, and speed by which output is processed. Cognitive flexibility includes the ability "to shift between response sets, learn from mistakes, devise alternative strategies, divide attention, and process multiple sources of information concurrently (p. 74)." Finally, goal setting involves problem solving abilities, particularly the ability to develop and elaborate upon new concepts and determine an efficient course of action.

Associated Deficits with Executive Dysfunction

Deficits in any one of the four domains can result in profound implications for functional living (Anderson et al., 2001; Andrewes, 2001). Anderson (2002) notes that executive dysfunction is not one disorder, but rather any number of combinations of deficits in the four domains of executive functioning. Research supports that there are numerous functional deficits associated with executive dysfunction including emotional, behavioral, and cognitive impairments. Lesions in the frontal lobe are associated with deficits in inhibitory control and difficulties with affective processing (Roberts & Wallace, 2000). Behavior regulation difficulties include problems with initiation of movements or behaviors, inhibition of automatic responses, sustaining motor performance over time, shifting motor responses when appropriate, ability to delay gratification, and the anticipation of future consequences of present actions. Impairments in attentional control are likely to produce children who are “impulsive, lack self-control, fail to complete tasks, commit procedural mistakes which they fail to correct, and respond inappropriately” (Anderson, 2002 p. 74). Specific emotion regulation difficulties include modulation of emotional arousal, modulation of mood, and use of self-soothing strategies (Powell & Kytja, 2004).

It is likely that there will be impairments in cognitive regulation of various tasks including those involving working memory and regulation of attention. Additionally, it is likely that planning, goal setting, time estimation, time management, organizational strategies, mental flexibility, fluency, abstract reasoning/concept formation, problem solving, judgment, and maintaining self-awareness will all be impacted (Dickstein et al., 2004; Meyer et al., 2004; Powell & Kytja, 2004). Verbal and visual learning and memory

impairments have also been reported (Duff, Schoenberg, Scott, & Adams, 2005). Also, differences in information processing approaches have been demonstrated between clinically depressed subjects compared to normal controls and other psychological disorders (Channon & Green, 1999; Dalgleish et al., 2003).

Although clinical lore suggests that executive functions may covary as a function of intelligence, there is a growing body of research that suggests that deficits in executive function are independent of the construct of intelligence (Welsh, Pennington, & Grossier, 1991; Ardila, Pineda, & Rosselli, 2000; Bogood, Mateer, & MacDonald, 2003).

Traditionally, deficits in executive functioning in children have been strongly associated with Attention Deficit Hyperactivity Disorder (ADHD) (Berlin, Bohlin, & Rydell, 2003; Bayliss & Roodenrys, 2000; Barkley, Murphy, Dupaul, & Bush, 2002; Clark, Prior, & Kinsella, 2000; Houghton et al., 1999; Shallice et al., 2002; Piek et al., 2004). Berlin, Bohlin, & Rydell (2003) observed that inhibition deficits were strongly related to inattentive ADHD symptoms in boys. Douglas' (2005) working model of ADHD emphasizes the importance of self-regulation and effortful attention, implicating a role for the prefrontal areas in the cognitive and motor deficits of ADHD. Planning deficits have also been found to clearly differentiate between children with attention deficits and controls (Papadopoulos, Panayiotou, Spanoudis, & Natsopoulos, 2005). However, recent research indicates that executive dysfunction is not specific to ADHD, but rather has been associated with several other disorders as well (Sergeant, Geurts, & Oosterlann, 2002). In fact, Jonsdottir, Bouma, Sergeant, & Scherder (2006) reported findings which indicated no significant relationship between parent/teacher ratings of ADHD and performance on executive function tasks. Rather, they explained that correlational analyses revealed that

executive function deficits in ADHD may be better explained for by comorbid depressive and autistic symptoms. Wilding (2003) explains that children with attentional difficulties may perform poorly on executive function tasks related to difficulty modulating arousal and motivation when faced with difficult tasks above and beyond their attentional difficulties. Additionally, executive dysfunction has been observed in children with moderate to severe Traumatic Brain Injury (TBI) (Vriezen & Pigott, 2002; Ylvisaker & DeBonis, 2000). Also, executive dysfunction has been linked to high-functioning Autism (Goldberg et al., 2005; Miriam et al., 2001; Sergeant, Geurts, & Oosterlann, 2002), Oppositional Defiant Disorder (ODD), Conduct Disorder (CD), and Tourette Syndrome (TS) (Sergeant, Geurts, & Oosterlann, 2002). Dawson and Guare (2004) implicate depression, anxiety, fatigue, situational stress, and attentional deficits as adversely impacting executive skills. Mattison, Hooper, and Carlson (2006) found impaired performance on the Language and Attention subscales of the NEPSY in children with serious emotional/behavioral disorders. As deficits in executive functioning become associated with other disorders and is associated with numerous processes, it seems likely that research will demonstrate its involvement in many more disorders, perhaps even a connection with the cognitive deficits associated with depression.

Measurement of Executive Functions

There are several neuropsychological tests available that measure the various aspects of executive functioning. Some of the most utilized tests include Stroop Color and Word Test (STROOP)(Golden, 1978), Wisconsin Card Sorting Test-64 Card Version (WCST-64)(Kongs, Thompson, Iverson, & Heaton, 2000), Trail Making Test A & B (TMT; Halstead, 1947, Reitan, 1958, Reitan & Davison, 1974, Reitan, 1971), Multi-

lingual Aphasia Examination (COWAT; Benton & Hamsher, 1989, and versions of the original Continuous Performance Task (CPT; Rosvold, Mirsky, Sarason, Bransome, & Beck, 1956). A more recent addition to the repertoire of tests available is the Delis Kaplan Executive Function System (D-KEFS; Delis, Kaplan, and Kramer 2001). Manchester, Priestley, and Jackson (2004) argue that measures of executive function are of most clinical utility when they can be used to hypothesize about difficulties that have been observed in natural settings and can make predictions about functional behavior.

However, there are many problems associated with measuring executive dysfunction in children (Manchester, Priestley, & Jackson, 2004). For example, some argue that an inherent problem with measurement of executive functioning is that EF is still a theoretical construct, not an operationalized definition (Hughes & Graham, 2002; Jurado & Roselli, 2007). Additionally, most measures of executive functions are “complex and involve a wide range of skills, thus complicating efforts to identify specific processes (Jurado & Roselli, 2007, p. 227). Delis, Kaplan, and Kramer (2001), developers of a test designed to measure executive functions, The Delis Kaplan Executive Function System (D-KEFS), state that existing models and theories of frontal lobe functioning are “at best, preliminary conceptualizations in need of extensive empirical testing and refinement” (p. 14). Another difficulty is that detecting deficiencies is difficult in a clinical setting because the structure of the setting may mask problems (Anderson, Anderson, Northam, Jacobs, & Catroppa, 2001). Further, traditional executive function measures often fail to demonstrate correlation amongst each other, thus placing limits on the construct and ecological validity of the tests (Jurado & Roselli, 2007).

Additionally, measures of EF currently do not take developmental differences into consideration, specifically related to limited language abilities in children (Hughes & Graham, 2002). A number of these tests have been used in studies of depressed adults and geriatric populations, but very few of these tests have been used in studies of depressed children. Given the occurrence of cognitive deficits observed in depressed children, it seems imperative to examine performance on measures of the aspects of executive functioning.

Cortical Development

The prefrontal cortex is the frontal lobe area typically associated with executive functions (Anderson et al., 2001). Developmentally, this is one of the last areas of the brain to develop and it continues to develop until young adulthood (Andrewes, 2001). The prefrontal cortex is described as overseeing cognitive processes to ensure appropriate movements are selected at the appropriate time (Kolb & Whishaw, 2003). Hale and Fiorello (2004) describe the prefrontal cortex as consisting of the dorsolateral prefrontal cortex, the orbital frontal cortex, and the medial section (includes anterior cingulate). Further, they state that these regions have rich interconnections with subcortical areas (including the limbic system which is implicated in depression). Within the prefrontal cortex, the anterior cingulate cortex has consistently been demonstrated to be associated with tasks of executive functions during functional-magnetic resource imaging (f-MRI) studies. The anterior cingulate cortex has bidirectional connections with many areas of the brain including the frontal lobe and the amygdala. The anterior cingulate cortex serves an evaluative role and a signaling role for activating strategic processes (Tamminga & Carter, 2000) and modulating the interactions between the various

posterior and prefrontal areas by responding to novelty, self-monitoring performance, inhibiting automatic responses, shifting cognitive set, and complex decision making (Hale & Fiorello, 2004). Additionally, the dorsolateral prefrontal cortex has been shown to be responsible for motor planning, organization, and regulation. Further it involved with planning, organizing, strategizing, initiating, monitoring, evaluating, modifying, changing and shifting behaviors. The orbital frontal region is responsible for behavioral and emotional regulation (Hale & Fiorello, 2004). Jurado & Rosselli (2007) provide a comprehensive review of research that additionally implicates other brain areas as serving critical roles in executive function processes, including subcortical regions and the posterior cortex.

Similar to the development of the frontal lobes, executive function skills develop rapidly and nonlinearly throughout childhood. This rapid and nonlinear development makes it difficult to measure and explain executive functioning strengths and deficits in children. However, more research has been conducted in an attempt to better understand the development of executive skills along a developmental trajectory. These studies indicate that executive skills begin developing in infancy, but do not become functional until later in the developmental sequence (Anderson, 2002), with continued myelination and maturation of frontal lobe structures (Anderson et al., 2001).

Anderson (2002) discussed approximate ages of development for the 4 areas of executive functioning: attentional control, information processing, cognitive flexibility, and goal setting. Development of attentional control begins at 9 months of age and approaches complete development by 11-years-old. Gains in information processing, via verbal fluency, are observed in children beginning at age 3-5 years of age, but leveling

off after age 15. The perseverative behaviors common in early and middle childhood are replaced by cognitive flexibility in adolescence. Goal setting behaviors begin with simple task planning at 4 years of age, become more organized between ages 7-11, and continue to refine throughout adolescence (Anderson, 2002). Of the four areas discussed, goal setting has been found to develop the most during adolescence (Anderson et al., 2001).

This developmental trajectory was replicated in an empirical study of four hundred 3- through 12-year old Finnish children conducted by Klenberg, Korkman, & Lahti-Nuutila (2001). They concluded that development of executive functions proceeds sequentially from motor inhibition and impulse control, to selective and sustained attention and finally to fluency.

DEPRESSION AND EXECUTIVE FUNCTIONS: A POSSIBLE RELATIONSHIP?

There are many overlaps between the cognitive deficits, brain structures, and functional implications associated with depression and executive dysfunction suggesting a relationship between the two. For example, similarities in cognitive deficits between depression and executive functioning include memory impairments, poor motivation and initiation, poor organization, decreased concentration and attention, and difficulty monitoring performance. Additionally, research on the neurobiological origins of both topics has implicated the frontal lobes, prefrontal cortex, temporal lobes, and limbic systems. Furthermore, recent research refutes the association between ADHD and executive deficits (Jonsdottir, Bouma, Sergeant, & Scherder, 2006; Wilding, 2003). Depression and executive functioning deficits are also both associated with functional impairment in academic and social environments. This would suggest that measures

designed to identify deficits in executive function might also aid in the diagnosis and treatment of children with depression.

Current Research Linkages

Adult studies

To date, the majority of current neuropsychological studies of depression predominantly examine performance in geriatric patients. These geriatric studies consistently demonstrate the presence of neuropsychological impairments in depressed subjects, particularly on executive function tasks (Abas, Sahakian, & Levy, 1990; Alexopoulos et al, 2000; Beats, Sahakian & Levy, 1996; Butters et al., 2000; Butters et al., 2004). The findings in non-geriatric samples have been mixed. For example, similar results were not replicated in a study (n = 20) of depressed younger patients (mean age = 37.5, range = 18-52) using the computerized CANTAB battery (Purcell, Maruff, Kyrios, & Pantels, 1997). While they demonstrated that subjects exhibited deficits in motor slowing and attentional set shifting, they failed to demonstrate that there were global deficits in executive functioning when examining performance on constructs assessing short-term memory capacity, spatial working memory, planning ability, cognitive speed, and recognition memory. They suggest that this is evidence of variability in the nature and severity of cognitive impairment in depression, specifically because subjects with observed cognitive impairments were more likely to have been hospitalized for their depression. However, it is possible that their results may have been due to limitations in their study, including a small sample size.

Veiel (1997) conducted a meta-analysis of 13 studies investigating young-middle aged neurologically unimpaired individuals with a diagnosis of major depressive disorder

to derive a profile of cognitive deficits. Neuropsychological measures were divided into nine categories: Attention/Concentration, Verbal Fluency, Scanning & Visuo-motor Tracking, Verbal Learning-Acquisition, Verbal Learning-Retention/Retrieval, Nonverbal Learning-Acquisition, Nonverbal Learning-Retention/Retrieval, Visual Spatial Functions, and Mental Flexibility/Control. When compared to normal controls, depressed subjects showed clear deficits on tasks assessing verbal fluency (COWAT), scanning & visuo-motor tracking (TMT-B; WAIS-R Digit Symbol), visual spatial functions (Rey Complex Figure Test, WAIS-R Block Design and Object Assembly), and mental flexibility and control (TMT-B and Stroop Color-Word). He noted a higher variability in scores on all measures and concluded that the verbal and nonverbal acquisition and retention tasks may have been mediated by other factors including motivation. He concluded that the cognitive deficits observed in depressed subjects were similar to organic brain damage associated with the frontal lobes.

Using the CANTAB battery of neuropsychological tests, Elliot, Sahakian, McKay, Herrod, Robbins, & Paykel (1996), investigated differences in performance of 28 middle-aged patients with unipolar depression to 22 age and IQ matched controls. They reported neuropsychological deficits across cognitive domains including recognition memory, matching to sample, working memory, and planning tasks. They argued that motivational deficits related to perceived failures contributed to the observed differences between subjects with depression and controls and concluded that there is evidence to support frontostriatal component of dysfunction in depression. This study provides support to the presence of executive functioning deficits in people with

depression. Limitations of the study include a small sample size, unequal group sizes, and the use of outpatient clinical patients in the depressed group.

Austin et al. (1999) conducted an experimental study of cognitive functioning in depressed subjects age 20 and older in an attempt to find patterns of deficits on frontal lobe tasks. Using an extensive battery of neuropsychological tests including Digit Span Forwards and Backwards, Reaction Time, Trails A & B, Stroop Task, Verbal Fluency, Abbreviated Wisconsin Card Sorting Test, Similarities, Rey Auditory Verbal Learning Test, Visual Reproduction, and Digit Symbol Substitution, they found that depressed subjects demonstrated impaired performance on several neuropsychological measures suggestive of frontal impairment. Impairments were reported on verbal and visual recall tasks, reaction time, and set shifting on the Wisconsin Card Sort task and Trails B. They suggest that their findings are supportive of frontal-subcortical and temporal impairment, which is consistent with current. Though this study included numerous neuropsychological tests, adequately covering areas associated with executive functions, there were major limitations to the participant sample that warrants caution in interpretation of results. First, all of the subjects recruited for the study were inpatients from a clinical hospital population. Control subjects were a convenience sample of patient's relatives, staff, and community volunteers at the hospital. Given these limitations in sampling, it is difficult to generalize the results to the entire population of people with depression. Additional research in this area could help to distinguish whether the difficulties on measures implicating frontal lobe impairment in depressed subjects are a result of some permanent impairment occurring as a result of a developmental brain anomaly or a temporary impairment associated with the symptoms of depression.

Landro, Stiles, & Sletvold (2001) examined neuropsychological deficits in 22 unmedicated patients with nonpsychotic unipolar major depressive disorder compared to 30 controls. They examined performance on neuropsychological tests assessing motor function, selective attention, mental flexibility, visuomotor tracking, working memory, short-term memory, verbal long-term memory, nonverbal long-term memory, verbal fluency, and visuospatial function. They reported significant deficits in the depressed group on tasks assessing selective attention, working memory, verbal long-term memory, and verbal fluency. They suggested that their results were suggestive of diffuse impairment of brain function, particularly associated with frontal lobe involvement. Further, they concluded that their results were supportive of a prefrontal cortical dysfunction in major depression.

In their 2001 study comparing 20 adults with Obsessive Compulsive Disorder (OCD) to control subjects, Basso, Bornstein, Carona, and Morton reported that abnormalities involving frontal lobe functioning were related to co-morbid depression. Specifically, the subjects with OCD performed more poorly on Verbal Concept Attainment test, WCST percent perseverative errors, and TMT A&B. However, these deficits were diminished and no longer statistically significant when depressive severity was accounted for. They concluded that their findings were due to the tasks assessed being associated with dorsolateral arousal, which is implicated in depression and not OCD. However, caution is warranted when interpreting the results of this study as they failed to correct for Type 1 error, used a narrow band of neuropsychological performance, and use of a convenience sample.

A study by Biringer, Lundervold, Stordal, Mykletun, Egeland, Bottlender, and Lund (2005) investigated the state vs. trait hypothesis of the cognitive deficits associated with depression by examining whether improvement in performance of subjects with recurrent unipolar depression on executive function measures were observed. They reported a “medium sized improvement of depressive symptoms and improvement on a composite score of change of executive function. However, no improvements were observed on several of the measures that comprised the composite score for executive function. Further, they suggested that speeded attention-demanding tasks (e.g. COWAT SEM, Stroop C/W, and the PASAT) were impaired. They concluded that there may be both state and trait related deficits associated with depression because some of the measures failed to improve completely to the level of controls (Semantic Fluency and Stroop C/W Inhibition). A relative strength of the study was the relatively homogeneous sample, which was comprised of younger subjects with recurrent unipolar depression. Also, the study used a time between the depressive episode and remittance of approximately 2 years. This study provides additional support for the presence of executive function deficits in depression. Similarly, Merriam, Thase, Haas, Keshavan, & Sweeney (1999) reported significant deficits on the Wisconsin Card Sorting Test number of categories, learning to learn, perseverative and non-perseverative errors, perseverative responses, percent of conceptual level responses, and items to first category in a sample of adult patients with major depression compared to controls. They suggested a possible relationship between cognitive deficits and the state-related physiological abnormalities in the prefrontal cortex during episodes of depression.

Further support for executive function deficits in depression was reported in a literature review of cognitive deficits in depression, which suggested mnemonic deficits and set-shifting executive deficits, noting that some of the deficits persisted upon recovery of depression (Austin, Mitchell, & Goodwin, 2001). Channon & Green (1999) reported that depressed participants displayed impaired cognitive flexibility and shifting abilities, perseverative difficulty, and poor judgment on three executive function tasks (memory for categorized words, response suppression task, and multiple scheduling task) compared to controls. Further, the depressed participants made less spontaneous use of strategies for task performance, made more errors, and were significantly slower than controls. They suggest that the observed deficits are related to reduced or diverted attentional resources associated with depression. Additionally, they hypothesize that subsets of depressed groups may show impairment on specific aspects of executive functioning, particularly memory, set-shifting, and planning.

A growing body of research has been investigating the presence of executive deficits associated with bipolar disorder (Malini, Ivanhosuski, Szekeres, & Olley, 2004; Daban et al., 2006; Pavuluri et al., 2006). Malini et al. (2004) conducted a meta-analytic review of 27 studies and found no significant difference in the severity and pattern of neuropsychological impairment between bipolar and unipolar subjects. Additionally, they reported impairments in working memory, executive functioning, and verbal learning in patients with euthymia, which suggests that euthymic states should not be considered or associated with clinical recovery. Daban et al. (2006) compared cognitive deficits in bipolar disorder versus schizophrenia. They concluded that although subjects with bipolar disorder were impaired on tasks assessing attention, memory, and executive function, the

impairments were more significant in schizophrenia. However, they cautioned that the presence of impairment in bipolar disorder has implications for quality of life and social adaptation. Similarly, Pavuluri et al. (2006) reported neurocognitive deficits in the domains of sustained attention, working memory, verbal memory, and executive functions in pediatric bipolar disorder. They described the implication of their findings as suggestive of dorsolateral prefrontal cortex dysfunction. However, this study did not compare euthymic, manic, and depressed states of pediatric bipolar disorder. Taken together, the research examining executive functioning in bipolar disorder is suggestive of impairments associated with frontal lobe functioning.

Child studies

Emerson, Mollet, & Harrison (2005), examined the effects of anxiety and depression on frontal lobe functioning by comparing performance on the Trail Making Test (Forms A and B) and the Concept Formation subtest of the Woodcock Johnson. Their study consisted of 38 male subjects (age range = 9-11) selected because each subject demonstrated elevated scores on the Child Depression Inventory (CDI). They found that boys with symptoms of anxiety and depression demonstrate impaired frontal functioning including slower processing speed, number of perseverative errors, set shifting, hypothesis testing, and categorical problem solving. The results of their study begin to establish the presence of executive functioning deficits in children with depression.

However, several methodological issues should be considered when interpreting the results of this study. For example, the sample size was limited (n=38) and only included males, making generalizability to the child population tentative at best.

Additionally, no demographic information about the subjects was provided, again warranting caution in generalizing the findings of this study. Also, it is difficult to determine the impact of either depression or anxiety on executive deficits. Further, the measures used in this study are not exhaustive of the associated areas of executive function, making it difficult to support their finding that children with anxiety and depression demonstrate impaired executive functioning. Despite these limitations, their study indicates that research needs to further examine executive functioning deficits in children with depression.

Based on extensive evidence suggesting neuropsychological impairments in adults with major depression and research supporting the importance of the prefrontal cortex in systems involved in modulating and inhibiting emotional behavior, executive functioning in adolescents with major depression was investigated by Kyte, Goodyer, and Sahakian (2005). They utilized tasks assessing attentional flexibility, behavioral inhibition, and decision making from the CANTAB battery to compare performance of 30 subjects with a recent history of major depression to 49 controls. They reported no significant differences between subjects with major depression and controls on attentional flexibility and behavioral inhibition. However, they noted that subjects with depression exhibited a significant bias towards processing negative emotional stimuli. Additionally, the subjects with depression responded significantly faster and less conservatively on the decision making task, which is suggestive of elevated impulsivity and risk taking behaviors. There were several limitations to the study including a high percentage of comorbid diagnoses in the sample. Further, the absence of depressive symptoms was

determined by a self-report measure alone. Also, some of the patients were being treated with antidepressants; however, others had never been treated with antidepressants.

The results of studies examining executive function in depression are mixed. However, limited studies have been done in age groups other than geriatric populations. This previous research demonstrates a need to further understand the relationship between depression and executive functioning at all age levels. To date, there is no clear understanding whether similar deficits of executive function occur in depressed children, and if so, what the nature and extent of these deficits may be. The current study utilized a battery of neuropsychological tests measuring aspects of executive functioning to investigate a profile of executive functioning in children with depression.

Childhood Depression and Executive Functions: Current Study

As depression is becoming more recognized as a childhood diagnosis, it is imperative that symptoms are recognized early and effective treatments and interventions are implemented. It is possible that through improved identification, the importance of early identification can be established as critical in preventing complicating factors of childhood depression such as poor peer relations and academic failure, particularly in the academic setting where children spend a great deal of their day. Understanding the cognitive deficits associated with childhood depression could aid in understanding the problems that are occurring in the academic setting, as well as assist in the development of specific interventions targeting cognitive impairments. The current study adds to the literature by examining if the mere presence of elevated symptoms of depression is related to impaired executive function.

Symptom recognition and understanding is necessary for successful early identification. Influenced by the strong presence of cognitive dysfunction, particularly deficits in information processing, attention, psychomotor speed, motivation, and organizational abilities, in children with depression, this study represents an initial effort to establish the presence of executive dysfunction in depressed youth (Mayberg et al., 2004). The outcome of this study has the potential to contribute to the understanding of childhood depression and help parents and teachers implement more effective intervention strategies. Finally, this study has strong implications for the refinement and improvement of neuropsychological measures of executive function. Thus, in an attempt to determine the existence of executive function deficits in children with depression, measures of motor speed, cognitive fluency, and cognitive flexibility, which are encompassed in the diagnostic criteria as possible impairments in depression (American Psychological Association, 2000), were examined.

CHAPTER III

METHOD

Purpose

This paper seeks to better understand the relationship of children with depressive symptoms and executive functioning. Specifically, this study aims to discover if children with elevated significant symptoms of depression exhibit executive dysfunction related to attentional control, information processing, and cognitive flexibility on lab-based measures. To that end, a select subset of data was utilized from a pre-existing data set compiled within the Neuropsychology section at Allegheny General Hospital's Department of Psychiatry.

Participants

The participant information was collected through use of a pre-existing data set. The data set was created through a retrospective chart review of children assessed in an outpatient neuropsychology clinic between the years 2003 to 2008 in order to examine whether children with symptoms of depression display impaired performance on measures of executive functioning, irrespective of the presence of symptoms of other psychiatric diagnoses. The data are comprised of consecutive clinically referred subjects from Pittsburgh, Pennsylvania and surrounding areas, and includes all genders and ethnic backgrounds. Participants were referred to the clinic for behavioral, psychological, and academic difficulties. All tests were administered to the children by a postgraduate Ph.D. with experience in and training in neuropsychological assessment. All participants were referred for evaluation for clinical rather than research purposes. The database and all associated procedures for data entry have been approved by the Institutional Review

Board of Allegheny General Hospital. Parents completed informed consent for assessment and treatment prior to the initiation of evaluation. Identifying information is removed from all data prior to entry into the database. Subjects with an IQ score less than or equal to 70 were excluded from this study, as were subjects with an Autism Spectrum Diagnosis.

Measures

Depression

Child Behavior Checklist (CBCL/6-18)

The CBCL (Achenbach, 2001) is a standardized behavior rating scale used to address children's social competencies as well as behavioral/emotional problems. The child's parent or immediate caregiver fills out the form which consists of 20 competence items, and 118 items that describe specific behavioral and emotional problems. The items are rated for how true each item is now or within the past six months using a 0-2 point Likert scale. *t*-scores ranging from 60-65 are considered at-risk and scores greater than 65 are considered clinically significant.

Test-Retest reliabilities for the competence scales are moderately high, ranging from .63 to .79. Reliability coefficients for the empirical based problem scales ranged from .78 to .97. Alpha coefficients for the DSM-oriented scales ranged from .72 to .91 (Achenbach, 1991). Similar reliability coefficients were reported by Durta, Cambell, and Westen (2004) when investigating a clinician-report version of the CBCL.

The CBCL has been validated in broad population-based studies and in studies involving children and adolescents with general psychiatric disorders (Edelbrock & Costello, 1998; Dole, 2001; Flanagan, & Steuart, 2005). Content validity for the CBCL

has been established through four decades of research, consultation, feedback, and revisions, as well as by findings that the all of the items significantly discriminate between referred and non-referred children ($p < .01$). Criterion related validity has also been shown to significantly discriminate between preferred and non-referred children ($p < .01$) using multiple regressions, odds ratios, and discriminant analysis (Achenbach, 1991). Again, validity estimates of the CBCL as a clinician report tool were reported to be impressive (Durta, Campbell, & Westen, 2004).

The CBCL has been utilized in several studies to screen for childhood psychopathology. The CBCL has demonstrated clinical utility in screening for externalizing disorders, particularly ADHD (Hudziak, Copeland, Stranger, & Wadsworth, 2004). The CBCL has been found to be an effective screening instrument for comorbid diagnoses in children with ADHD (Biederman, Monuteaux, Kendrick, Klein, & Faraone, 2005; Bird, Canino, Gould, Ribera, Rubio-Stipec, Woodbury, Huertas-Goldman, & Sesman, 1986; Bird, Gould, Rubio-Stipec, Staghezza, & Canino, 1991).

The CBCL has also been found to distinguish between depressed and non-depressed subjects. For example, Kazdin & Heidish (1984) found that items on the CBCL that reflected inner-directed or emotional problems successfully distinguished between children with and without a diagnosis of depression. Additionally, Biederman, Faraone, Mick, Moore, and LeLon (1996) reported that the CBCL discriminated between children with and without major depression irrespective of comorbid ADHD. Further, the CBCL has demonstrated utility in discriminating children with mania from those with ADHD (Biederman, Wozniak, Kiely, Ablon, Faraone, Mick, Mundy, & Kraus, 1995).

The CBCL was selected to serve as a method for deriving observer based estimations of depressive symptoms. For the purposes of this study, subjects were considered to have symptoms of depression if they demonstrated at-risk T-scores ($T \geq 65$) on the Anxious/Depressed subscale of the Child Behavior Checklist 6-18, Parent Response Form (CBCL). Support for use of this scale is reported in the conclusions of Rey & Morris-Yates (1992), who reported that the Anxious/Depressed subscale taps mainly a depression construct because it discriminated between major depression and separation anxiety as accurately as it discriminated between major depression and other disorders. Based upon these findings observer based estimations of depression will be generated through an examination of the CBCL's Anxious/Depressed Scale.

Executive Functions

Delis Kaplan Executive Function System (D-KEFS)

The Delis-Kaplan Executive Function System (D-KEFS; Delis, Kaplan, & Kramer, 2001) is a set of nine, individually administered standardized tests designed to assess executive functions including attention, language, perception, as well as levels of creative and abstract thought. The D-KEFS is normed for ages 8-89 and is the first nationally-normed set of tests designed to measure executive functions. The D-KEFS Trail Making Test and Verbal Fluency Test were considered for this study. Because these subtests are relatively new or modifications of long-standing clinical or experimental tests, information regarding their validity is based on the original forms of the tests, as opposed to current research.

In their review of the D-KEFS, Homack, Lee, and Ricco (2005) report several advantages of the test including that it is the first comprehensive set of executive tests co-

normed on a large and nationally representative sample. Further it is based on a cognitive-process approach, which they note is appropriate as researchers of executive functions have yet to agree on a theoretical construct. Additionally, they describe that primary measures on the D-KEFS demonstrate adequate reliability and reasonable validity for differentiating clinical groups. They cite limitations of the test as possible oversimplification of instructions and lower reliability for optional measures.

The D-KEFS Trail Making Test consists of five conditions. The conditions are administered as follows: 1. Visual Scanning, 2. Number Sequencing, 3. Letter Sequencing, 4. Number-Letter Switching, and 5. Motor Speed. Number-Letter Switch condition measures flexibility of thinking and is considered the primary executive functioning task. For this condition, the participant searches for and crosses out all occurrences of the number 3 on the page. For Number Sequencing and Letter Sequencing, the participant connects the numbers and letters in numerical or alphabetical order respectively. For the Motor Speed condition, the participant traces a dotted-line around the page. The primary executive function condition is Number-Letter Switching. In this condition, the participant alternates connecting numbers in numerical order and letters in alphabetical order. Standard scores for the Trail-Making Test are based on task completion time.

Baron (2004) notes that the five condition administration allows the examiner to consider the effects of slowed information processing, slow psychomotor speed, fine motor impairment, or impaired ability to sequence numbers and/or letters of the cognitive flexibility task. Additionally, the D-KEFS Trail Making Test provides published

normative data for error types, which provides additional clinical information (Delis, Kaplan et al, 2001).

Lezak and colleagues (2004) reported that the shifting condition of trails demonstrates a high correlation with traditional cognitive flexibility such as the Wisconsin Card Sorting Test. The other four conditions allow the examiner to rule out difficulties on the key component processes needed to perform the switching task (visual scanning, number sequencing, letter sequencing, and motor speed). Internal consistency for the Trail Making Test ranges from .59 to .81 across ages 8-89. Test-Retest Reliability Coefficients range from .20 to .82 across the five conditions for all ages. The validity of the Trail Making Test has been demonstrated in various neuropsychological studies conducted over the past 50 years (Delis, Kaplan, & Kramer, 2001). The findings by Lezak et al. (2004) provide further support for the validity of the D-KEFS Trail Making Test.

Verbal Fluency is based on the Controlled Oral Word Association Test (COWAT; Benton & Hamsher, 1989), a task designed to measure difficulty with initiation and verbal fluency when provided a specific response cue (Duff, Schoenberg, Scott, & Adams, 2005). The D-KEFS Verbal Fluency Test consists of three conditions: Letter Fluency, Category Fluency, and Category Switching. For the Letter Fluency condition, participants are asked to list as many words as they can, beginning with the given letter (e.g. F, A, S). For the Category Fluency condition, the participant is asked to name as many words as they can within the specified category (e.g. animals and boys names). The Category Switching task incorporates the cognitive flexibility component. For this task, participants are asked to alternate between words between two categories (e.g. fruit and

pieces of furniture). For each condition, the time limit is 60 seconds. Additional guidelines are that the words cannot be a proper noun and the participant cannot simply change the ending of a word (i.e. the participant cannot say both runs and running). These rules apply for all conditions of the test. The score for each condition is the total number of correct words the participant is able to produce within the 60 second time limit. Additional scores include repetition errors and set-loss errors. Scores for each condition involve the total number of correct words produced within the 60 second time limit. Additional scores are calculated for repetition errors and set-loss errors (Delis, Kaplan, & Kramer, 2001).

Internal consistency values range from .37 to .90 across the three conditions for all age groups. Test-retest reliability coefficients range from .24 to .88 across all conditions and ages. The validity of the Verbal Fluency task has been demonstrated in various neuropsychological studies conducted over the past 50 years (Delis, Kaplan, & Kramer, 2001).

Research Design

This study utilized a quasi-experimental research design. The independent variable is clinically significant symptoms of depression as operationalized by the CBCL Anxious/Depressed subscale. The dependant variable in this study was performance of subjects on executive function tasks including motor speed, cognitive fluency, and cognitive flexibility. Motor Speed was operationalized by D-KEFS Trail Making Test Condition 5 (Motor Speed). Cognitive fluency was operationalized by the Letter and Category Fluency conditions of the D-KEFS Verbal Fluency subtest. Cognitive flexibility

was operationalized by the D-KEFS Trail Making Test Condition 4 (Number-Letter Switching) and the Verbal Fluency Category Switch.

Procedures

This study analyzed a select dataset from the Neuropsychology section of Allegheny General Hospital's Department of Psychiatry database to better understand the association between depressive symptoms on performance on measures of executive functioning. Allegheny General Hospital's Department of Psychiatry provided a Microsoft Excel database that did not contain any identifying subject information. SPSS was used for all statistical analyses. Extracted data was analyzed to address the specific research questions described below.

Data Analysis

The purpose of this study is to determine if children with symptoms of depression demonstrate impairment on measures of executive functioning irrespective of attentional difficulties. Statistical analysis utilized in this study included a priori analysis, descriptive statistics, and a correlation matrix of all study variables. Additionally, a series of Analyses of Variance (ANOVA), Multivariate Analyses of Variances (MANOVA), (Stevens, 1999) were conducted. Statistical analyses were performed using SPSS 12.0 for Windows (SPSS, Inc, 2003). A significance level of $\alpha=.05$ was used to determine statistical significance.

A Priori Statistical Analysis

An a priori power analysis was conducted to establish the minimum number of participants needed to achieve adequate power for deriving a moderate effect size (.40) using two (CBCL and the covariate GAI) predictors at a .5 alpha level. Power analysis

was calculated with G*Power version 3.0.10, a general power analysis program (Faul, Erdfelder, Lang, & Buchner, in press). Results of the a priori power analysis suggest that a minimum n of 42 per group ($F=3.2381$) would generate sufficient power to generate a moderate effect size.

Means and standard deviations for each variable will also be reported. Symptoms of depression will be examined for outliers using the Mahalanobis Distance compared to chi-square critical values, as Mahalanobis measures multivariate outliers (Stevens, 1999). To determine if the dependent variables are normally distributed, residuals were examined. Residuals greater than positive or negative 3 were considered outliers (Stevens, 1999). Additionally, an a priori *t*-test was conducted to determine if there was a significant difference in mean IQ scores between depressed and non-depressed subjects.

Assumptions for ANOVA

There are three assumptions to consider when conducting an ANOVA: normality, homogeneity of variances, and independence of observations (Tabachnik & Fidel, 2007). Normality assumes the observations are normally distributed on the dependent variable in each group. Violation of this assumption affects the Type 1 error rate. This assumption can be impacted by the number of observations, with greater numbers of observations leading to approximate normality. Lack of normality also has a slight impact on power when skewness is involved.

Homogeneity of the population variances assumes that the variances of dependent variable are the same for all populations. This assumption is influenced by group sizes. If group sizes are equal or approximately equal, then the test is considered robust for unequal variances (the actual α is approximately equal to the nominal α). Violation of this

assumption is likely when group sizes are largely unequal (largest/smallest > 1.5) and the test shows the population variances are unequal. This results in an F statistic that is too liberal, where false rejections are occurring too often. This is important because smaller α values cause a decrease in Type 1 error. This assumption will be observed using Levene's Test for Equality of Variances as it is less sensitive to non-normality. The independence assumption that asserts cases represent random samples from the populations and the scores on the test variable are independent of one another is critical to satisfy. If this assumption is violated, inaccurate p values are yielded from the ANOVA, impacting both the power and significance of the statistic.

Research Questions and Hypotheses

Research Question #1:

Does the motor speed of children with elevated symptoms of depression differ from a clinical population of children without elevated symptoms of depression?

Hypothesis #1:

Children with elevated depressive symptoms will perform poorer on tasks assessing motor speed compared to children without elevated symptoms of depression related to the depression diagnostic criteria which includes psychomotor slowing.

Statistical Analysis

To examine if children with elevated depressive symptoms significantly differ from non-depressed clinical controls in regard to motor speed a one-way analysis of variance was conducted.

Independent Variable: Depression as measured by standard scores on the Anxious/Depressed subscale of the CBCL: (A) depressed and (B) non-depressed

Dependent Variables: Motor Speed as measured by standard score performance on the D-KEFS Trail Making Test Motor Speed Condition.

Research Question #2:

Does cognitive fluency in children with elevated depressive symptoms differ from that of a clinical population of children without elevated depressive symptoms?

Hypothesis #2:

Children with elevated depressive symptoms will perform poorer on tasks assessing cognitive fluency than children without elevated depressive symptoms.

Statistical Analysis

To examine if children with elevated depressive symptoms differ from clinical controls without depressive symptoms in regard to cognitive fluency, a multivariate analysis of variance will be conducted. If the F statistic indicates that there is a significant overall difference, the Tukey correction will be employed as the post-hoc statistic to detect where the differences occur.

Independent Variable: Depression as measured by standard scores on the Anxious/Depressed subscale of the CBCL: depressed (A) and non-depressed (B)

Dependent Variable: Cognitive fluency as measured by a standard score of performances on the Letter Fluency and Category Fluency subtests of the D-KEFS Verbal Fluency Task.

Assumptions for MANOVA

Four assumptions were considered when conducting this MANOVA: independence of observations, multivariate normality, linearity, and homogeneity of variance-covariance matrices (Stevens, 1999; Tabachnick & Fidell, 2007). Independence of

observations refers to finding a correlation among observations of a greater magnitude than would be expected by chance. Violation of this assumption occurs when within group variance is underestimated, in turn leading to the underestimation of standard error which ultimately results in an increased risk of Type-1 error (Stevens, 1999). An intraclass correlation will be conducted to determine if the independence of observations has been violated.

Multivariate normality is the assumption that all variables and all combinations of the variables conform to a normal distribution. To check the assumption of normality a review of histograms for the residuals and an examination of normal probability plots for skewedness and/or kurtosis were conducted. Nonsymmetrical distributions are skewed either positively or negatively. Kurtosis references the distribution's degree of peakedness. Normal distributions' skewedness and Kurtosis values are 0, values greater than +1.5 or less than -1.5 are considered extreme when divided by the standard error of measurement (Tabachnick & Fidell, 2007). If the major variables of interest do not conform to a normal distribution then the assumption is likely violated.

Linear relationships among pairs of dependent variables are another assumption of MANOVA. The assumption of linearity is that there is a straight-line relationship between variables. This can be problematic because the linear combination of the dependent variables would not maximize separation of groups for the independent variables if the relationship is non-linear. This assumption is assessed by inspection of bivariate scatterplots. A normally distributed and linearly related scatterplot will be oval-shaped (Tabachnick & Fidell, 2007).

Multivariate outliers are cases where combinations of scores on two or more variables are so variable that they distort statistics. Outliers are important to consider because they have much more impact on both Type-I and Type-II error and can lead to results which do not generalize, except to samples which have a similar outlier. The presence of outliers will be considered

Homogeneity of variance-covariance involves the assumption that the covariance matrices for each group are equal. When this assumption is violated, Type 1 error rates can be affected. Violation of this assumption typically occurs when group sizes are unequal, or if the ratio of larger group n is no larger than 1.5 times the smaller group n . The Box M test for equality of covariance matrices will be examined to determine if this assumption has been violated because it is known to be a sensitive test of homogeneity of covariance (Tabachnick & Fidell, 2007).

Research Question #3:

Does cognitive flexibility differ in children with elevated symptoms of depression differ from that of a clinical population of children without elevated depressive symptoms above and beyond deficits associated with motor speed?

Hypothesis #3:

Children with depressive symptoms will perform poorer on tasks assessing cognitive flexibility versus children without elevated depressive symptoms.

Statistical Analysis

To examine if children with clinically significant depressive symptoms differ from clinical controls without depressive symptoms in regard to cognitive flexibility, a multivariate analysis of variance was conducted.

Independent Variable: Depression as measured by standard scores on the Anxious/Depressed subscale of the CBCL: depressed (A) and non-depressed (B)

Dependent Variable: Cognitive flexibility as measured by standard score performances on the Number-Letter Switch subtest of the D-KEFS Trail Making Test and Category Switch Total Correct Responses on the D-KEFS Verbal Fluency Test

CHAPTER IV

RESULTS

The results section is organized as follows: demographic characteristics, descriptive statistics, preliminary analyses, and then an examination of statistical assumptions for each research question, followed by the statistical analyses for each research question. Demographic statistics are considered to provide context for the descriptive and inferential statistics. The descriptive statistics present information concerning the independent and dependent variables. Pre-analyses investigate correlations and significant differences among the variables in this study. Statistical assumptions for each research question are then examined in order to assure the appropriateness of running the proposed analyses for each research question. Lastly, statistical results for each research question are provided.

Demographic Characteristics

The original sample consisted of 147 participants, of which approximately 70% were male ($n = 95$), while the rest were female ($n = 42$). Within the data-set, sex was not identified in 10 of the subjects. Mean age of the population was 10.79 years with a standard deviation of 3.1 years. After eliminating participants who were not administered the measures utilized in this study, as well as those with full scale IQ scores below 70 and those with diagnoses on the Autism Spectrum, 79 participants were included in the analyses. Of the 79 participants included in the analysis, 56 were male and 23 were female. The mean age of the final sample was 11.94 with a standard deviation of 2.56. The non-depressed group consisted of 51 participants and the depressed group consisted of 28 participants. Approximately 83% of the participants were Caucasian, 10% Blacks,

3% Bi-racial, 1% Asian, and 1% Other. IQ assessment was part of the standard protocol for the neuropsychological clinic and Table 1 presents the means and standard deviations of full scale intelligence (FSIQ) by group status. The mean FSIQ for the Depressed group was 94 ($SD = 2.88$), while the mean FSIQ for the Non-Depressed group was 91 ($SD = 1.883$). An independent sample t -test was conducted to examine the difference of FSIQ between depressed and non-depressed participants. Results of the t -test suggest that the means of FSIQ were similar for depressed and non-depressed children $t(112) = -1.10, p = .27$.

Because the sample consisted of clinic referred children, it is important to consider participant's primary diagnosis. Table 1 displays the primary diagnoses by group.

Table 1

Primary DSM-IV-TR Diagnosis by Group Status

	Non-Depressed	Depressed
293.83 Mood Disorder (Medical)	1	0
249.9 Cognitive Disorder, NOS	1	0
295.90 Schizophrenia	1	0
296.21 Major Depressive Disorder (Single Episode, Mild)	0	1
296.23 Major Depressive Disorder (Single Episode, Severe)	0	1
296.32 Major Depressive Disorder (Recurrent Moderate)	1	1
297.07 Bipolar Disorder	0	1
300.00 Anxiety Disorder, NOS	2	3
300.02 Generalized Anxiety Disorder	1	5
300.3 Obsessive Compulsive Disorder	1	1
309 Adjustment Disorder with Depressed Mood	2	0
309.21 Separation Anxiety	1	0
309.24 Adjustment Disorder with Anxiety	5	2
309.28 Adjustment Disorder with Mixed Anxiety/Depressed Mood	3	4

Table 1 Continued

309.4	Adjustment Disorder with Mixed Disturbance of Emotions/Conduct	2	2
309.9	Adjustment Disorder, Unspecified	1	0
310.1	Personality Disorder due to General Medical Condition	2	0
311	Depressive Disorder, NOS	0	3
312.81	Conduct Disorder	2	1
312.9	Disruptive Behavior Disorder, NOS	2	0
313.81	Oppositional Defiant Disorder	3	2
314.00	Attention Deficit Hyperactivity Disorder, Primarily Inattentive Type	20	8
314.01	Attention Deficit Hyperactivity Disorder, Combined Type	24	11
314.9	Attention Deficit Hyperactivity Disorder, NOS	2	0
315.00	Reading Disorder	2	0
315.32	Mixed Receptive-Expressive Language Disorder	0	1
315.39	Phonological Disorder	1	0
315.9	Learning Disorder, NOS	5	1
	No Diagnosis Given	12	0

Within the sample, 42 of the non-depressed participants carried multiple diagnoses, as did 35 of the depressed participants.

Descriptive Statistics

The independent variable in this study was group status. The decision rule for group membership was a *t*-score of greater than or equal to 65 on the Anxious/Depressed subscale of the CBCL. The mean score on the Anxious/Depressed subscale for the Depressed group was 70.37 (*SD* = 6.09) and the mean score for the Non-Depressed group was 53.92 (*SD* = 4.59). Descriptive statistics were also computed for the dependent variables. Table 2 includes the mean and standard deviations of the executive function measures by group status.

Table 2

Descriptive Statistics Regarding Executive Function Dependent Variables

Measure	Depressed		Non-Depressed	
	Mean	SD	Mean	SD
Motor Speed	11.46	2.20	11.02	2.57
Letter Fluency	8.96	3.81	8.98	2.85
Category Fluency	10.04	3.14	9.60	2.50
Number Letter Switch	8.67	3.86	8.74	3.41
Category Switch	9.15	3.85	9.26	2.53

Preliminary Analyses

Pearson bivariate correlation coefficients were computed among the dependent variables. Correlations are presented in Table 3. Significant positive correlations were observed between the D-KEFS Motor Speed and Letter Fluency, Motor Speed and Category Fluency, Motor Speed and Category Switch, Number Letter Switch and Letter Fluency, Number Letter Switch and Category Switch, Letter Fluency and Category Fluency, Letter Fluency and Category Switch, and Category Fluency and Category Switch subtests.

Table 3

Pearson Bivariate Correlations among Executive Function Scales

	Motor Speed	Number Letter Switch	Letter Fluency	Category Fluency
Number Letter Switch	.20			
Letter Fluency	.35*	.41*		
Category Fluency	.33*	.18	.53*	
Category Switch	.32*	.39*	.39*	.52*

Note. * $p < .01$

In addition, Pearson bivariate correlation coefficients were computed among the dependent variables. Correlations are presented in Table 4. No significant correlations were found between ratings on the CBCL Anxious/Depressed scale and scores on executive function measures.

Table 4

Pearson Bivariate Correlations among CBCL and Executive Function Measures

	CBCL Anxious/Depressed
Motor Speed	.049
Letter Fluency	-.08
Category Fluency	.22
Number/Letter Switch	-.01
Category Switch Total Correct Responses	-.05

Research Question 1

The first research question examined the impact of depressive symptoms on a motor speed task. A one-way analysis of variance was conducted to evaluate the relationship between motor speed and presence of depressive symptoms in children. The independent variable, presence of depression, included two levels: depressed and not-depressed. The dependent variable was performance on the D-KEFS Trail Making Test Motor Speed.

Before calculating the ANOVA, the independence assumption was considered. The independence assumption requires that the responses of participants are not related. As each participant included in the data set was tested individually and did not have contact with any other participant, this assumption is satisfied for the current analyses. Second, the assumption of normality was considered through graphical and statistical

methods. Graphical methods involved examination of histograms and stem and leaf plots. For the non-depressed group, histograms and plots suggested negatively skewed data with a leptokurtic shape. The extent of skewness and kurtosis were also examined by dividing the skewness and kurtosis values by its standard error to obtain a z-score. If the z-score fell outside the +/- 2 range, it was determined that the skewness or kurtosis was violated. For example, the motor speed variable in the non-depressed group the skewness value was -1.488 with a standard error of 3.073. When the skewness value was divided by the standard error (-1.488/3.073), the value was within the range of +/- 2 (-.484). Additionally, the calculation for kurtosis was also within the range ($2.357/2.565 = .919$). As such, the assumption of normality is not considered violated. Similarly, the skewness value of the depressed group was .104 with a standard error of 2.202. When the skewness value is divided by the standard error ($.104/2.202$), the z-score is within the range of +/- 2 (.047), and is therefore robust to violation of the assumption of normality. The z-score calculation for the kurtosis value in the depressed group was also within the range of +/- 2, and therefore not violated ($.109/2.202 = .049$). Finally, the assumption of homogeneity of variances was satisfied through examination of Levene's Test of Equality of Error Variances ($p = .59$). The ANOVA was not significant, $F(1, 77) = .598, p = .442$.

Research Question 2

To answer the question does cognitive fluency in children with clinically significant depressive symptoms differ from that of clinic referred children without depressive symptoms, a one-way multivariate analysis of variance (MANOVA) was conducted. Group status was considered the independent variable and the two levels included depressed and non-depressed participants. D-KEFS Letter Fluency and Category

Fluency subsets were considered together as dependent variables in the analyses to control for experiment-wide error (i.e., reduce the likelihood of committing Type 1 error).

Before computing the MANOVA, assumption tests related to MANOVA were conducted. The independence assumption requires that the responses of participants are not related. As each participant included in the data set was tested individually and did not have contact with any other participant, this assumption is satisfied for the current analyses. Second, the assumption of normality was considered through graphical and statistical methods. Graphical methods involved examination of histograms and stem and leaf plots. Examination of the graphs and plots suggested that both groups followed a normal distribution for both variables, Letter Fluency and Category Fluency. The extent of skewness and kurtosis were also examined by dividing the skewness and kurtosis values by its standard error to obtain a z-score. If the z-score fell outside the ± 2 range, it was determined that the skewness or kurtosis was violated. For example, for the letter fluency variable in the non-depressed group the skewness value was .039 with a standard error of 2.847. When the skewness value was divided by the standard error ($.039/2.847$), the value was within the range of ± 2 (.014). Additionally, the calculation for kurtosis was also within the range ($-.566/2.847 = -.199$). As such, the assumption of normality is not considered violated. Similarly, the skewness value of the depressed group was .403 with a standard error of 3.805. When the skewness value is divided by the standard error ($.403/3.805$), the z-score is within the range of ± 2 (.106), and is therefore robust to violation of the assumption of normality. The z-score calculation for the kurtosis value in the depressed group was also within the range of ± 2 , and therefore not violated ($-.455/3.805 = -.120$). When performing similar calculations for the category fluency

variable, all values for skewness and kurtosis are considered robust to violation of the normality assumption. Finally, the assumption of homogeneity of variances is considered and determined to be satisfied through examination of Box's M Test ($p = .150$).

Regarding the MANOVA, no significant differences were found between the depressed and non-depressed subjects on the measures of cognitive fluency considered together, $Wilk's \Lambda = .992, F(2, 73) = .306, p > .05$.

Research Question 3

To determine whether there was a difference between groups on measures of cognitive flexibility, a one-way MANOVA was conducted to determine the effect of group status (depressed, non-depressed), on the two dependent variables, Number-Letter Switch (D-KEFS TMT) and Category Switch Total Correct Responses (D-KEFS VF).

Before calculating the MANOVA, MANOVA assumptions were again examined. As each participant included in the data set was tested individually and did not have contact with any other participant, the independence assumption was satisfied. Second, the assumption of normality was considered through graphical and statistical methods. Graphical methods involved examination of histograms and stem and leaf plots. Examination of the graphs and plots suggested that the distribution for the depressed group for the Number-Letter Switch variable was negatively skewed and leptokurtic. The non-depressed group appeared normally distributed for the Number-Letter Switch variable. For the Category Switch variable, examination of the histograms and plots suggested that both the depressed and non-depressed groups were normally distributed. The extent of skewness and kurtosis were also examined by dividing the skewness and kurtosis values by its standard error to obtain a z-score. If the z-score fell outside the ± 2

range, it was determined that the skewness or kurtosis was violated. For example, for the number letter switch variable in the non-depressed group the skewness value was $-.754$ with a standard error of 3.410 . When the skewness value was divided by the standard error ($-.754/3.410$), the value was within the range of ± 2 ($-.221$). Additionally, the calculation for kurtosis was also within the acceptable range ($-.139/3.410 = -.041$). As such, the assumption of normality is not considered violated. Similarly, the skewness value of the depressed group was $-.370$ with a standard error of 3.863 . When the skewness value is divided by the standard error ($-.370/3.863$), the z-score is within the range of ± 2 ($-.096$), and is therefore robust to violation of the assumption of normality. The z-score calculation for the kurtosis value in the depressed group was also within the range of ± 2 , and therefore not violated ($-1.007/3.863 = -.261$). When performing similar calculations for the category switch total correct response variable, all values for skewness and kurtosis are considered robust to violation of the normality assumption. Finally, the assumption of homogeneity of variances was considered and determined to not be violated through examination of Box's M Test ($p = .081$). No significant differences were found between the depressed and non-depressed subjects on the two measures of cognitive fluency considered together, *Wilk's Λ* = 1.000, $F(2, 71) = .012$, $p > .05$.

CHAPTER V

DISCUSSION

A growing literature base is present that describes the relationship between depression and executive functioning in both adult and geriatric populations. The geriatric literature consistently links depression to impaired performance on tasks of global executive functioning (Abas, Sahakian, & Levy, 1990; Beas, Sahakian & Levy, 1996). In fact, Alexopoulos' (2003) work with geriatric subjects with depression resulted in a theory of depression called the depression-executive dysfunction syndrome that implicates frontal lobe dysfunction. The theory states that geriatric persons with depression may evidence reduced fluency, impaired visual naming, decreased interest in activities, poor/delayed anti-depressant response, impaired selective and sustained attention, abnormal initiation, perseveration, deficits in inhibitory control and sustained effort, impaired problem solving, deficits in set-shifting, decreased psychomotor speed, and poor disability understanding.

Memory, set-shifting, and planning have also been implicated in adults with depression (Channon & Green, 1999). Likewise, Purcell, Maruff, Kyrios, and Pantels (1997) discovered that adult participants with depression exhibited motor slowing and deficits in attentional set shifting. However, no global deficits in executive functioning were identified as no differences were observed on tasks assessing spatial span, spatial working memory, planning, and visual memory. On the other hand, Austin et al. (1999) did find evidence of global executive impairment in adult participants with depression evidenced by impaired memory, set-shifting, selective attention, mnemonic fluency, and reaction time. At least one meta-analysis has been conducted that examined the link

between depression and executive function deficits in adults (Veiel, 1997). This study found evidence of diffuse impairments with deficits noted on tasks assessing verbal fluency, scanning and visuo-motor tracking, visual spatial functions, and mental flexibility and control. Some of these same executive impairments may be present in persons with bipolar depression (Daban et al., 2006; Dickstein et al., 2004; Malini et al., 2004; Meyer et al., 2004; & Pavuluri et al., 2006).

With respect to childhood depression and executive dysfunction, there currently is a paucity of research. Emerson, Mollett, and Harrison (2005), found that boys with anxiety and depression demonstrated impairments in processing speed, number of perseverative errors, set shifting, hypothesis testing, and categorical problem solving. Kyte, Goodyer, & Sahakian (2005) reported elevated impulsivity and risk taking behaviors, as well as significant bias towards processing negative emotional stimuli. Differences between adolescents with depression and controls on attentional flexibility and behavioral inhibition were not observed, suggesting lack of global executive function deficits. Inconsistencies and gaps in previous research demonstrate a need to further understand the relationship between depression and executive functioning at all age levels, and specifically during childhood. The present study explored if children with clinically elevated symptoms of depression display deficits on tasks designed to measure aspects of executive functioning, namely on tasks assessing motor speed, cognitive fluency, and cognitive flexibility.

Summary of Motor Speed Results

The first research question explored whether the motor speed of children with elevated symptoms of depression differed from children without elevated symptoms of

depression. Although it was hypothesized that children with elevated symptoms of depression would perform poorer on motor speed tasks, this was not supported by the findings of the current study. No significant difference was found between these two groups regarding tasks that measure motor speed. This result was unexpected given that in geriatric populations, decreased psychomotor speed has been consistently reported (Abas et al., 1990, Alexopoulos, 2003, & Beats et al., 1996). Similarly, psychomotor slowing has been observed in studies of adults with depression (Biringier et al., 2005 & Purcell et al., 1997). Germaine to the present study, there is data present that links psychomotor slowing to depression in childhood (Emerson, Mollet, and Harrison, 2005). It is difficult to determine the exact reason for the differences in findings between the current study and that of Emerson, Mollet, & Harrison (2005) as little demographic information regarding their sampling was presented. Their study utilized participants with symptoms of both anxiety and depression. As such, one should consider the impact that the comorbidity of anxiety and depression contributed to the significance of their findings. Still in the current study, elevated symptoms of depression were defined by the Anxious/Depressed subscale of the CBCL, and as such, symptoms of anxiety were also present in participants. Another consideration is that their sample consisted of only adolescent males, leading one to consider the possibility that such deficits exist only in adolescent males with depression and anxiety. The findings of the current study are also seemingly inconsistent with the diagnostic criteria established in the *DSM-IV-TR* (American Psychological Association, 2000) which lists psychomotor agitation or retardation as a symptom linked to depression. Therefore, it is possible that psychomotor

agitation or retardation may not be linked to elevated symptoms of depression as measured by broad-band behavior rating scales.

Summary of Cognitive Fluency Results

The second hypothesis that children with elevated symptoms of depression would perform poorer on tasks assessing cognitive fluency was not supported. Additionally, no significant results were found when separate one way ANOVAs comparing the performance of children with elevated symptoms of depression to clinical controls were computed for each of the dependent variables (Letter Fluency and Category Fluency subtests of the D-KEFS). These findings are not consistent with existing research that suggests the presence of impaired fluency in persons with depression. For example, in studies examining cognitive functioning in geriatric subjects with depression, deficits on cognitive fluency tasks assessing psychomotor speed, visual naming, inhibitory control, problem-solving, set shifting, and initiation, are reported (Alexopoulos, 2003). This finding has been replicated in adult populations as well (Biringier et al., 2005; Elliot et al., 1996). Lardo, Stiles, & Stevold (2001) found significant deficits in depressed groups on verbal fluency tasks. Verbal fluency deficits have also been reported in studies involving participants with bipolar disorder (Daban et al, 2006; Malini et al, 2004). A meta-analysis conducted by Veiel (1997) suggests controlled oral fluency is an executive function consistently impacted in adults with depression. To date, no studies exist which document the presence of fluency deficits in children with depression, which may attribute for differences in findings of the current study compared to the above mentioned studies. One must consider the possibility that cognitive fluency is a later developing executive

function skill and, as such, deficits will not be observable until adulthood when performance of such skills are expected, as proposed by Barkley (1997).

Summary of Cognitive Flexibility Results

The final hypothesis, which suggested that children with elevated symptoms of depression would evidence poorer performance on tasks assessing cognitive flexibility, was also not supported by the findings of this study. Similarly, no significant differences were observed when separate one-way ANOVAs were conducted for each of the dependent variables (D-KEFS TMT Number-Letter Switch and D-KEFS Verbal Fluency Category Switch Total Correct Responses). However, set-shifting deficits are consistently reported in persons with depression (Austin, Mitchell, & Goodwin, 2001; Channon & Green, 1999). In the adult literature, attentional set-shifting and working memory have been shown to be impaired in participants with depression (Elliot, Sahakian, McKay, Herrod, Robbins, & Paykel 1996; Lardo, Stiles, & Stevlod, 2001; Purcell, Maruff, Kyrios, & Pantels, 1997). Depressed participants have also shown deficits on Wisconsin Card Sorting Task, TMT B and Stroop Color Word (Austin et al, 1999; Veiel, 1997). Similarly, Basso, Bornstein, Carona, and Morton (2001) reported subjects with OCD performed poorer than controls on Verbal Concept Attainment test, WCST percent perseverative errors, and TMT A&B. However, these deficits were diminished and no longer statistically significant when depressive severity was controlled, which implicates the role of the depressive symptoms on performance. Unipolar participants with depression have also been found to demonstrate deficits in verbal fluency, information processing speed, flexibility, and calculation ability (Biringier et al., 2005). Similarly, Emerson, Mollet, & Harrison (2005) and have reported the presence of set-shifting

deficits in depressed youth, though Kyte, Goodyer, & Sahakian (2005) did not report similar findings. Given the inconsistent findings in the extant literature involving youth with depression, one must again consider that flexibility is an executive skill that is not fully developed until adulthood, and therefore, cognitive deficits may not be observable until then. Conversely, the lack of participant information provided in the Emerson, Mollet, & Harrison (2005), may provide insight into the differences in findings between their study and that of the current study, as well as the findings reported by Kyte, Goodyer, & Sahakian (2005).

Limitations of Study

This study represents an attempt to contribute to the extant literature examining the association between executive function deficits and childhood depression. Data exists supporting the reliability and validity of the measures used in the current study. However, several study limitations are present and may account for the differences between the findings of the current study and comparison studies. First, in examining the preliminary analyses, no correlation was found between the CBCL Anxious/Depressed subscale and the D-KEFS executive function measures utilized in the current study. Given theory and data linking depression to poor executive function in adults, it was expected that significant inverse correlations would result when considering the two constructs in children. One explanation for the lack of inverse correlation is that the two constructs are simply unrelated in children. Conversely, the possibility exists that the lack of significant findings is an artifact of the low reliability of some of the executive function measures. Uncovering systematic differences between independent and dependent variables becomes more difficult as the reliability of used measures decreases (Stevens, 1999).

Additionally, the present study utilized an operational definition of depression that was different from other comparison studies. In the current study, “depression” was defined by *t*-score elevations of 65 or greater on the Anxious/Depressed scale of the CBCL, not DSM-IV-TR diagnostic criteria. In comparison studies, participants were included in the depression clinical group based on a formal diagnosis of depression consistent with DSM-IV-TR. Many of the participants in the current sample were not diagnosed with clinical depression. It is known that severity of depression is linked to degree of executive function impairment. For example, Purcell, Maruff, Kyrios, & Pantels (1997) reported that participants with diagnoses of depression who were hospitalized were more likely to exhibit deficits in executive functioning; implicating the severity of depression is linked to the presence of executive deficits. Future studies that confirm the presence of clinical depression may uncover executive function deficits.

Also, in this study, quantitative means via cut scores were utilized to establish group status to verify the presence of elevated symptoms, though these categorizations were not consistent with reported diagnoses. In particular, the primary diagnosis observed in the sample was ADHD, and few of the participants utilized in the current study met DSM-IV-TR diagnostic criteria for a depressive disorder. Use of this mixed sample likely introduced more error variance into the analyses. Comorbid diagnoses often share many of the symptoms that are associated with depression (Alessi & Magen, 1988; Birmaher et al., 1998; Gerhardt, Compas, Connor, & Achenbach, 1999; Kovacs & Devlin, 1998; Shoaf, Graham, & Mayes, 2001) and could also contribute to executive skill deficits. By tightening the definition of depression and eliminating comorbid disorders to the greatest extent, it is possible that different results may have been observed.

Furthermore, this study used lab-based, or performance-based, measures of executive functions. Extant literature suggests that children with ADHD, a clinical group theorized to experience executive dysfunction (Berlin, Bohlin, & Rydell, 2003; Bayliss & Roodenrys, 2000; Barkley, Murphy, Dupaul, & Bush, 2002; Clark, Prior, & Kinsella, 2000; Houghton et al., 1999; Shallice et al., 2002; Piek et al., 2004), may not perform poorer than normal controls on many lab-based measures of executive function (Jonsdottir, Bouma, Sergeant, & Scherder, 2006 & Sergeant, Geurts, & Oosterlann, 2002). At the same time, this group is consistently rated as impaired on rating scales measuring executive dysfunction (Barkley, 1991; Sergeant, Geurts, & Oosterlann, 2002; Jonsdottir, Bouma, Sergeant, & Scherder, 2006; Riccio, Homack, Jarratt, & Wolfe, 2006). A more recent line of research has examined the utility in rating scales, like the BRIEF (*Behavior Rating Inventory of Executive Function*, Gioia, Isquith, Guy, & Kentworthy, 2000), as they provide assessment by significant others who observe children in their everyday environments. Research using the BRIEF has suggested that it is effective in establishing the presence of executive dysfunction in ADHD (Isquith & Gioia, 2000; Mahone et al., 2002; Riccio, Homack, Jarratt, & Wolfe, 2006). Applying this line of research to another clinical population with suspected executive function deficits, similar findings may be found using ratings of executive function in children with depression.

Finally, the sample size was small and there was a substantial difference in the size of groups (>1:1.5). Utilizing smaller sample sizes makes it more difficult to detect clinically significant differences.

Recommendations for Future Research

It is specifically recommended that in future studies, replication of the current study occur between a control samples and a sample with *DSM-IV-TR* diagnoses of depressive disorders, to determine if executive function deficits occur in children with depression. It is also recommended that larger, more equal sample sizes be utilized for future research to ensure adequate power. Future studies should aim to limit the existence of comorbid disorders and obtain a normal control group for comparison. Further, restrictions in the age range could result in more significant findings as development of executive functions are reported to develop across the life span (Anderson, 2002). Still, it is possible that executive dysfunction given depression does not appear until adulthood, namely because executive functions are amongst the last cognitive skills to fully develop and therefore may not be at risk until later in life. Therefore, a cross-sectional study may be useful in determining if executive functioning deficits are more likely observable in adulthood.

Another consideration for future studies relates to how executive functions are measured. Given the previous discussion regarding executive impairments in children with ADHD, intuitively, a rating scale measure of daily executive functioning within the child's environment may also be used to study executive dysfunction of children with depression. Behavior ratings allow for multiple raters to evaluate a child's behavior across settings and under the day to day conditions where executive skills are required.

Another avenue for future research could involve measuring the effectiveness of executive function interventions in a population of children with depression. Such studies could compare performance on a particular task pre and post intervention. Dawson and

Guare (2004) provide a comprehensive review of strategies for development of interventions to promote executive skills: intervening at the environmental level, intervening at the individual level, classroom level interventions, and interventions for specific executive deficits. Given that cognitive deficits including impaired attention, memory, psychomotor speed, motivation, and organizational abilities, as well as increased focus on negative thoughts (Bulbena & Berrios, 1993; Mayberg, Keightly, Mahurin, & Brannan, 2004), have been reported in people with depression, interventions targeting working memory, self regulation of affect, sustained attention, task initiation, planning, organization, goal directed persistence, and/or flexibility should be considered (Dawson & Guare, 2004).

Implications for School Psychologists

As the presence of depression, as well as other mental health concerns, increase in the classroom setting, the school psychologist plays an integral role in identifying the strengths and needs of students and assisting in development and implementation of interventions to remediate student needs. School psychologist can utilize their training in assessment, observation, intervention design, and progress monitoring to examine the impact of executive functioning in the classroom. Given the growing literature implicating executive function deficits across various diagnoses (Goldberg et al., 2005; Jonsdottir, Bouma, Sergeant, & Scherder, 2006; Miriam et al., 2001; & Sergeant, Geurts, & Oosterlann, 2002), school psychologists should consider screening for executive deficits when assessing students if specific referral concerns implicate deficits in executive skills. Another potential implication for the field of school psychology is that elevated ratings on depression subscales may not be sufficient to detect impaired

performance on lab-based measures of executive function, despite reports from parents and/or teachers implicating executive skills. Thus, it would be imperative for school psychologists to further investigate executive functions through interviews, rating scales and/or observations across settings.

Conclusion

Though no significant findings were reported in the current study, attempts to establish the presence of executive dysfunction in children with depression should not be abandoned, especially in light of the extant literature with adult populations, which suggest implications of depression on executive functioning. By developing a better understanding of the specific cognitive deficits related to childhood depression, interventions that address any cognitive deficits and functional impairments associated with depression may be developed.

References

- Abas, M., Sahakian, B.J., & Levy, R. (1990). Neuropsychological deficits and CT scan changes in elderly depressives. *Psychological Medicine*, 20, 507-520.
- Achenbach, T.M., & Rescorla, L.A. (2001). *Manual for the ASEBA School-Age forms and profiles*. Burlington: University of Vermont.
- Alessi, N.E. & Magen, J. (1988). Comorbidity of other pediatric disturbances in depressed, psychiatrically hospitalized children. *The American Journal of Psychiatry*, 145, 12, 1582-1584.
- Alexopoulos, G.S. (2003). Role of executive function in late-life depression. *Journal of Clinical Psychiatry*, 64, 14, 18-23.
- Alexopoulos, G.S., Meyers, B.S., Young, R.C., Kalayam, B., Kakuma, T., Gabrielle, M., Sirey, J.A., & Hull, J., (2000). Executive dysfunction and long-term outcomes of geriatric depression. *Archives of General Psychiatry*, 57, 285-290.
- American Psychiatric Association (2000). *Diagnostic and statistical manual of mental disorders* (4th Ed. Text Revision). American Psychiatric Association, Washington, D.C.
- Anderson, P. (2002). Assessment and development of executive functioning (EF) during childhood. *Child Neuropsychology*, 8, 2, 71-82.
- Anderson, V., Anderson, P., Northam, E., Jacobs, R., & Catroppa, C. (2001). Development of executive functions through childhood and late adolescence in an Australian sample. *Developmental Neuropsychology*, 20, 1, 385-406.
- Andrewes, D. (2001). *Neuropsychology from theory to practice*. Psychology Press: New York.

- Angold, A., & Costello, E.J. (2000). The Child and Adolescent Psychiatric Instrument (CAPA). *Journal of the American Academy of Child and Adolescent Psychiatry*, 39, 39-48.
- Angold, A., Costello, E.J., Messer, S.C., & Pickles, A. (1995). Development of a short questionnaire for use in epidemiological studies of depression in children and adolescents. *International Journal of Methods in Psychiatric Research*, 5, 237-249.
- Ardila, A., Pineda, D., & Rosselli, M. (2000). Correlation between intelligence test scores and executive function measures. *Archives of Clinical Neuropsychology*, 15, 1, 31-36.
- Austin, M.P, Mitchell, P., & Goodwin, G.M. (2001). Cognitive deficits in depression: possible implications for functional neuropathology. *British Journal of Psychiatry*, 178, 200-206.
- Austin, M.P., Mitchell, P., Wilhelm, K., Parker, G., Hickie, I., Brodaty, H., Chan, J., Eysers, K., Milic, M., & Hadzi-Pavlovic, D. (1999). Cognitive function in depression: a distinct pattern of frontal impairment in melancholic?. *Psychological Medicine*, 29, 1, 73-85.
- Azuma, T. (2004). Working memory and perseveration in verbal fluency. *Neuropsychology*, 18, 69-77.
- Baddeley, A. (1996). Exploring the central executive. *The Quarterly Journal of Experimental Psychology*, 49A, 1, 5-28.
- Baddeley, A.D., & Hitch, G.J. (1974). Working memory. In G.H. Bower (Ed.), *The psychology of learning and motivation*, 8, 47-89. New York: Academic Press.

- Barkley, R. A. (1997). *ADHD and the nature of self-control*. New York: Guilford Press.
- Barkley, R.A., Murphy, K.R., Dupaul, G.J., & Bush, T. (2002). Driving in adults with attention deficit hyperactivity disorder: knowledge, performance, adverse outcomes, and the role of executive functioning. *Journal of the International Neuropsychological Society*, 8, 655-672.
- Baron, I.S. (2004) *Neuropsychological Evaluation of the Child*. New York: Oxford University Press.
- Basso, M.R., Bornstein, R.A., Carona, F., & Morton, R. (2001). Depression accounts for executive function deficits in obsessive-compulsive disorder. *Neuropsychiatry, Neuropsychology, and Behavioral Neurology*, 14, 4, 241-245.
- Bayliss, D.M., & Roodenrys, S. (2000). Executive processing and attention deficit hyperactivity disorder: an application of the supervisory attentional system. *Developmental Neuropsychology*. 17,2, 161-180.
- Beats, B.C., Sahakian, B.J., & Levy, R. (1996). Cognitive performance in tests sensitive to frontal lobe dysfunction in the elderly depressed. *Psychological Medicine*, 26, 591-603.
- Benton, A.L., & Hamsher, K. Eds. (1989). *Multilingual aphasia examination*. Iowa City: AJA Associates.
- Berk, L.E. (2003). *Child Development* (6th ed.). Boston: Allyn and Bacon.
- Berlin, L., Bohlin, G., & Rydell, A.M. (2003). Relations between inhibition, executive functioning, and ADHD symptoms: a longitudinal study from 5 to 8½ years. *Child Neuropsychology*, 9, 4, 255-256.

- Bernstein, D.A., Clarke-Stewart, A., Roy, E.J., & Wickens, C.D. (1997). *Psychology* (4th ed.). Boston: Houghton Mifflin Company.
- Biederman, J., Faraone, S., Mick, E., Moore, P., & Lelon, E. (1996). Child behavior checklist findings further support comorbidity between ADHD and major depression in a referred sample. *Journal of the American Academy of Child and Adolescent Psychiatry*, 35, 6, 734-742.
- Biederman, J., Monuteaux, M.C., Kendrick, E., Klein, K.L., & Faraone, S.V. (2005). The CBCL as a screen for psychiatric comorbidity in paediatric patients with ADHD. *Archives of Disease in Childhood*, 90, 1010-1015.
- Biederman, J., Wozniak, J., Kiely, K., Ablon, S., Faraone, S., Mick, E., Mundy, E., & Kraus, I. (1995). CBCL clinical scales discriminate pre-pubertal children with structured interview-derived diagnosis of mania from those with ADHD. *Journal of the American Academy of Child and Adolescent Psychiatry*, 34, 4, 464-471.
- Bierut, L.J., Heath, A.C. Bucholz, K.K., Dinwiddie, S.H., Madden, P.A.F., Statham, D.J., Dunne, M.P., & Martin, N.G. (1999). Major depressive disorder in a community-based twin sample. *Archives of General Psychiatry*, 56, 557-563.
- Bird, H.R., Canino, G., Gould, M.S., Ribera, J., Rubio-Stipec, M., Woodbury, M., Huertas-Goldman, S., & Sesman, M. (1986). Use of the child behavior checklist as a screening instrument for epidemiological research in child psychiatry: results of a pilot study. *American Academy of Child and Adolescent Psychiatry*, 26, 2, 207-213.
- Bird, H.R., Gould, M.S., Rubio-Stipec, M., Staghezza, B.M., & Canino, G. (1991). Screening for childhood psychopathology in the community using the child

- behavior checklist. *Journal of the American Academy of Child and Adolescent Psychiatry*, 30, 1, 116-123.
- Biringer, E., Lundervold, A., Stordal, K., Mykletun, A., Egeland, J., Bottlender, R., & Lund, A. (2005). Executive function improvement upon remission of recurrent unipolar depression. *Archives of Psychiatry and Clinical Neurosciences*, 255, 373-380.
- Birmaher, B., Brent, D., & Benson, R.S. (1998). *Summary of the practice parameters for the assessment and treatment of children and adolescents with depressive disorders*. Retrieved January 31, 2005, from <http://www.healthyplace.com>.
- Bogood, N.M., Mateer, C.A., & MacDonald, S.W.S. (2003). Self-awareness after traumatic brain injury: a comparison of measures and their relationship to executive functions. *Journal of the International Neuropsychological Society*, 9, 450-458.
- Bolla-Wilson, K., Robinson, R.G., Starkstein, S.E., Boston, J., & Price, T.R. (1989). Lateralization of dementia of depression in stroke patients. *American Journal of Psychiatry*, 146, 627-634.
- Brent, D., Ryan, N., Dahl, R., & Birmaher, B. (2005, January). *Early-onset mood disorder*. Psychopharmacology – Fourth Generation of Progress, The American College of Neuropsychopharmacology. Retrieved January 31, 2005, from www.acnp.org/G4/GN401000158/CH154.html.
- Brooks, S.J., & Kutcher, S. (2001). Diagnosis and measurement of adolescent depression: a review of commonly utilized instruments. *Journal of Child and Adolescent Psychopharmacology*, 11, 4, 341-376.

- Bulbena, A., & Berrios, G.E. (1993). Cognitive functions in the affective disorders: a prospective study. *Psychopathology*, 26, 6-12.
- Butters, M.A., Becker, J.T., Nebes, R.D., Zmuda, M.D., Mulsant, M.H., Pollock, B.G., & Reynolds, C.G., (2000). Changes in cognitive functioning following treatment of late-life depression. *American Journal of Psychiatry*, 157, 1494-1954.
- Butters, M.A., White, E.M., Nebes, R.D., Beagley, A.E., Dew, M.A., Mulsant, B.H., Zmuda, M.D., Bhalla, R., Meltzer, C.C., Pollock, B.G., Reynolds, C.F., & Becker, J.T., (2004). The nature and determinants of neuropsychological functioning in late-life depression. *Archives of General Psychiatry*, 61, 587-595.
- Carlson, G.A. (2000). The challenge of diagnosing depression in childhood and adolescence. *Journal of Affective Disorders*, 61, 3-8.
- Castillo, M., Kwock, L., Courvoisie, H., & Hooper, S.R. (2000). Proton MR spectroscopy in children with bipolar disorder: preliminary observations. *American Journal of Neuroradiology*, 21, 832-838.
- Channon, S., & Green, P.S.S. (1999). Executive function in depression: the role of performance strategies in aiding depressed and non-depressed participants. *Journal of Neurological Neurosurgery and Psychiatry*, 66, 162-171.
- Clarizio, H.F. (1989). Continuity in childhood depression. *Adolescence*, 24, 94, 253-267.
- Comer, R.J. (2001). *Abnormal Psychology* (4th ed.). New York: Worth Publishers.
- Costello, E.J., & Angold, A. (1988). Scales to assess child and adolescent depression: checklists, screens, and nets. *Journal of the American Academy of Child and Adolescent Psychiatry*, 27, 6, 726-737.

- Daban, C., Martinez-Aran, A., Torrent, C., Tabares-Seisdedos, R., Balanza-Martinez, V., Salazar-Fraile, J., Sela-Vera, G., & Vieta, E. (2006). Specificity of cognitive deficits in bipolar disorder versus schizophrenia. *Psychotherapy and Psychosomatics*, 75, 72-84.
- Dagleish, T., Taghavi, R., Neshat-Doost, H., Moradi, A., Canterbury, R., & Yule, W. (2003). Patterns of processing bias for emotional information across clinical disorders: a comparison of attention, memory, and prospective cognition in children and adolescents with depression, generalized anxiety, and post-traumatic stress disorder. *Journal of Clinical Child and Adolescent Psychiatry*, 32, 1, 10-21.
- Davidson, R.J. (1984). Affect, cognition, and hemispheric specialization. In C.E. Izard, J. Kagan, & R.B. Zajonc (eds.). *Emotions, cognition, and behavior*, 320-365. Cambridge, England: Cambridge University Press.
- Dawson, P., & Guare, R. (2004). *Executive skills in children and adolescents: a practical guide to assessment and intervention*. New York: The Guilford Press.
- Delis, D.C., Kaplan, E., & Kramer, J.H. (2001). D-KEFS Executive Function System.
- Della Sala, S., Gray, C., Spinnler, H. & Trivelli, C. (1998). Frontal lobe functioning in man: the riddle revisited. *Archives of Clinical Neuropsychology*, 13, 663-682.
- Dickstein, D.P., Treland, J.E., Snow, J., McClure, E.B., Mehta, M.S., Towbin, K.E., Pine, D.S., & Leibenluft, E. (2004). Neuropsychological performance in pediatric bipolar disorder. *Biological Psychiatry*. 55, 32-39.
- Diler, R.S., Daviss, W.B., Lopez, A., Axelson, D., Iyengar, S., & Birmaher, B. (2007). Differentiating major depressive disorder in youths with attention deficit hyperactivity disorder. *Journal of Affective Disorders*. 102, 125-130.

- Douglas, V. (2005). Cognitive deficits in children with attention deficit hyperactivity disorder: a long-term follow-up. *Canadian Psychology*, 46, 1, 23-31.
- Doyle, A., Ostrander, R., Skare, S., Crosby, R.D., & August, G.J. (1997). Convergent and criterion-related validity of the Behavior Assessment System for Children-Parent Rating Scale. *Journal of Clinical Child Psychology*, 26, 3, 276-284.
- Dubuque, S.E. (1998). Fighting childhood depression. *The Education Digest*, 63,6, 65-69.
- Duff, K., Schoenberg, M.R., Scott, J.G., & Adams, R.L. (2005). The relationship between executive functioning and verbal and visual learning and memory. *Archives of Clinical Neuropsychology*, 20, 111-122.
- Durta, L., Campbell, L., & Westen, D. (2004). Quantifying clinical judgment in the assessment of adolescent psychopathology: reliability, validity, and factor structure of the Child Behavior Checklist for clinician report. *Journal of Clinical Psychology*, 60, 1, 65-85.
- Edelbrock, C., & Costello, A.J. (1988). Convergence between statistically derived behavior problem syndromes and child psychiatric diagnoses. *Journal of Abnormal Child Psychology*, 16, 2, 219-231.
- Elliot, R., Sahakian, B.J., McKay, A.P., Herrod, J.J., Robbins, T.W., & Paykel, E.S. (1996). Neuropsychological impairments in unipolar depression: the influence of perceived failure on subsequent performance. *Psychological Medicine*, 26, 5, 975-990.
- Emerson, C.S., Mollet, G.A., & Harrison, D.W. (2005). Anxious-depression in boys: an evaluation of executive functioning. *Archives of Clinical Neuropsychology*, 20, 539-546.

- Emslie, G.J., & Mayes, T.L. (1999). Depression in children and adolescents: a guide to diagnosis and treatment. *Disease Management*, 11,3, 181-189.
- Erlenmeyer-Kimling, L., Adamo, U.H., Rock, D., Roberts, S.A., Bassett, A.S., Squires-Wheeler, E., Cornblatt, B.A., Endicott, J., Pape, S., & Gottesman, I.I. (1997). The New York high-risk project. *Archives of General Psychiatry*, 54, 1096-1102.
- Faul, F., Erdfelder, E., Lang, A.-G., & Buchner, A. (2007). G*Power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behavior Research Methods*.
- Fristad, M.A., Cummins, J., Verducci, J.S., Teare, M., Weller, E.B., & Weller, R.A. (1998a). Study IV: concurrent validity of the DSM-IV revised Children's Interview for Psychiatric Syndromes (ChIPS). *Journal of Child and Adolescent Psychopharmacology*, 8, 227-236.
- Fristad, M.A., Glickman, A.R., Verducci, J.S., Teare, M., Weller, E.B., & Weller, R.A. (1998b). Study V: Children's Interview for Psychiatric Syndromes (ChIPS): psychometrics in two community samples. *Journal of Child and Adolescent Psychopharmacology*, 8, 237-245.
- Gençoz, T., Voelz, Z.R., Gençoz, F., Petit, J.W., & Joiner Jr., T.E. (2001). Specificity of information processing styles to depressive symptoms in youth psychiatric inpatients. *Journal of Abnormal Child Psychology*, 29, 3, 355-262.
- Global Mental Health Network (n.d.). *Child and adolescent bipolar disorder*. Retrieved January 27, 2005, from: <http://www.gmhnet.com>

- Gerhardt, C.A., Compas, B.E., Connor, J.K., & Achenbach, T.M. (1999). Association of mixed anxiety-depression and symptoms of major depressive disorder in adolescence. *Journal of Youth and Adolescence*, 28, 3, 305-323.
- Goldberg, M.C., Mostofsky, S.H., Cutting, L.E., Mahone, E.M., Astor, B.C., Denckla, M.B., & Landa, R.J. (2005). Subtle executive impairment in children with Autism and children with ADHD. *Journal of Autism and Developmental Disorders*, 35, 3, 279-293.
- Golden, C.J. (1978). Stroop Color and Word Test. PAR, Inc.
- Gravetter, F.J., & Wallnau, L.B. (2000). *Statistics for the Behavioral Sciences* (5th ed.). California: Wadsworth.
- Hale, J.B., & Fiorello, C.A. (2004). *School Neuropsychology A Practitioner's Handbook*. New York: The Guilford Press.
- Halstead, W.C. (1947). *Brain and Intelligence*. Chicago, IL: University of Chicago Press.
- Herjanic, B., & Reich, W. (1982). Development of a structured psychiatric interview for children: Agreement between child and parent on individual symptoms. *Journal of Abnormal Child Psychology*, 10, 307-324.
- Homack, S., Lee, D., & Riccio, C.A. (2005). Test review: Delis-Kaplan Executive Function System. *Journal of Clinical and Experimental Neuropsychology*, 27, 599-609.
- House, A.E. (1999). *DSM-IV diagnosis in the schools*. New York: The Guilford Press.
- Hudziak, J., Copeland, W., Stranger, C., & Wadsworth, M. (2004). Screening for DSM-IV externalizing disorders with the child behavior checklist: a receiver operating

- characteristic analysis. *Journal of Child Psychology and Psychiatry*, 45, 7, 1299-1307.
- Hughes, C., & Graham, A. (2002). Measuring executive functions in childhood: problems and solutions? *Child and Adolescent Mental Health*, 7,3, 131-142.
- Jacobs, G.D., & Snyder, D. (1996). Frontal brain asymmetry predicts affective style in men. *Behavioral Neuroscience*, 110, 3-6.
- Jonsdottir, S., Bouma, A., Sergeant, J.A., & Scherder, E.J.A. (2006). Relationships between neuropsychological measures of executive function and behavioral measures of ADHD symptoms and comorbid behavior. *Archives of Clinical Neuropsychology*, 21, 383-394.
- Jurado, M.B., & Roselli, M. (2007). The elusive nature of executive functions: a review of our current understanding. *Neuropsychological Review*, 17, 213-233.
- Kalat, J.W. (2001). *Biological Psychology* (7th ed.). Belmont, CA: Wadsworth, a division of Thompson Learning, Inc.
- Kashani, J.H., Rosenberg, T.K., & Reid, J.C. (1989). Developmental perspectives in child and adolescent depressive symptoms in a community sample. *The American Journal of Psychiatry*, 146, 7, 871-875.
- Kaufman, J., Birmaher, B., Brent, D., & Ryan, N. (1996). *Kiddie-SADS, present and future version*. 10, 1.0.
- Kaufman, J., Blumberg, H., & Young, C.M. (2004). Neurobiology of early-onset mood disorders. In D.S. Charney & E.J. Nestler (Eds.), *Neurobiology of mental illness* (2nd ed., 1007-1021). Oxford University Press.

- Kazdin, A.E., & Heidish, I.E. (1984). Convergence of clinically derived diagnoses and parent checklists among inpatient children. *Journal of Abnormal Child Psychology*, 12, 3, 421-435.
- Keightley, M.L., Winocur, G., Graham, S.J., Mayberg, H.S., Hevenor, S.J., & Grady, C.L. (2003). An fMRI study investigating cognitive modulation of brain regions associated with emotional processing of visual stimuli. *Neuropsychologica*, 41, 585-596.
- Kendall, P.C., Stark, K.D., & Adam, T. (1990). Cognitive deficit or cognitive distortion in childhood depression. *Journal of Abnormal Child Psychology*, 18, 3, 255-270.
- Kessler, R.C., Avenevoli, S., & Merikangas, K.R. (2001). Mood disorders in children and adolescents: an epidemiologic perspective. *Biological Psychiatry*, 49, 1002-1014.
- Kessler, R.C., McGonagle, K.A., Zhao, S., Nelson, C.B., Hughes, M., Eshleman, S., Wittchen, H.-U., & Kendler, K.S. (1994). Lifetime and 12-month prevalence of *DSM-III-R* psychiatric disorders in the United States. *Archives of General Psychiatry*, 51, 8-19.
- Klein, D.N., Dougherty, L.R., & Olino, T.M. (2005). Toward guidelines for evidence-based assessment of depression in children and adolescents. *Journal of Clinical Child and Adolescent Psychology*, 34, 3, 412-432.
- Klenberg, L., Korkman, M., & Lahti-Nuutila, P. (2001). Differential development of attention and executive functions in 3-to 12-year old Finnish children. *Developmental Neuropsychology*, 20, 1, 407-428.
- Kolb, B., & Wishaw, I.Q. (2003). *Fundamentals of Human Neuropsychology* (5th ed.). New York: Worth Publishers.

- Komar, C.R. (1999). *Depression self-rating scale*.
- Kongs, S.K., Thompson, L.L., Iverson, G.L., & Heaton, R.K. (2000). Wisconsin Card Sorting Test-64 Card Version.
- Kovacs, M. (1992). *Children's depression inventory*. Pearson Assessments.
- Kovacs, M., & Devlin, B. (1998). Internalizing disorders in childhood. *Journal of Child Psychology and Psychiatry*, 39,1, 47-63.
- Kowatch, R.A., DelBello, M.P., Mayes, T., Kennard, B.D., & Emslie, G.J. (2006). Pediatric Mood Disorders. In C. E. Coffey & R.A. Brumback (Eds.), *Pediatric Neuropsychiatry*. Philadelphia: Lippincott, Williams, & Wilkins.
- Kyte, Z.A., Goodyer, I.M., & Sahakian, B.J. (2005). Selected executive skills in adolescents with recent first episode major depression. *Journal of Child Psychology and Psychiatry*, 46, 9, 995-1005.
- Lamarine, R.J. (1995). Child and adolescent depression. *The Journal of School Health*, 65,9, 390-393.
- Landro, N.I., Stiles, T.C., & Sletvold, H. (2001). Neuropsychological function in nonpsychotic unipolar major depression. *Neuropsychiatry, Neuropsychology and Behavioral Neurology*, 14, 4, 233-240.
- Larson, R., Csikszentmihalyi, M., & Graef, R. (1980). Mood variability and the psychosocial adjustment of adolescents. *Journal of Youth and Adolescence*, 9, 6, 469-490.
- Lewinsohn, P.M., Rohde, P., Seeley, J.R., Klein, D.N., & Gotlib, I.H. (2003). Psychosocial functioning of young adults who have experienced and recovered

- from major depressive disorder during adolescence. *Journal of Abnormal Psychology*, 112,3, 353-363.
- Liotti, M., & Mayberg, H.S. (2001). The role of functional neuroimaging in the neuropsychology of depression. *Journal of Clinical and Experimental Neuropsychology*, 23, 121-136.
- Lopez, J. (2005). The neurobiology of depression. From:
http://www.thedoctorwillseeyounow.com/articles/behavior/depression_5.
(01/27/2005).
- Louters, L.L. (2004). Don't overlook childhood depression. *Journal of the American Academy of Physicians Assistants*, 19, 9, 18-24.
- Malini, G.S., Ivanhouski, B., Szekeres, V., & Olley, A. (2004). Bipolar disorder: it's all in your mind? The neuropsychological profile of bipolar disorder. *Canadian Journal of Psychiatry*, 49, 12, 813-819.
- Manchester, D., Priestley, N., & Jackson, H. (2004). The assessment of executive functions: coming out of the office. *Brain Injury*, 18, 11, 1067-1081.
- Mattison, R.E., Hooper, S.R., & Carlson, G.A. (2006). Neuropsychological characteristics of special education students with serious emotional/behavioral disorders. *Behavioral Disorders*, 31, 2, 176-188.
- Mayberg, H.S. (1997). Limbic-cortical dysregulation: a proposed model of depression. *Journal of Neuropsychiatry*, 9, 471-478.
- Mayberg, H.S. (2002). Depression, II: localization of pathophysiology. *The American Journal of Psychiatry*, 159, 12, 1979.

- Mayberg, H.S. (2003). Modulating dysfunctional limbic-cortical circuits in depression: towards development of brain-based algorithms for diagnosis and optimized treatment. *British Medical Bulletin*, 65, 193-207.
- Mayberg, H.S., Brannan, S.K., Mahurin, R.K., Jerabek, P.A., Brickman, J.S., Tekell, J.L., Silva, A., McGinnis, S., Glass, T.G., Martin, C.C., & Fox, P.T. (1997). Cingulate function in depression: a potential predictor of treatment response. *NeuroReport*, 8, 1057-1061.
- Mayberg, H.S., Keightley, M., Roderick, K.M., & Brannan, S.K. (2004). Neuropsychiatric aspects of mood and affective disorders. In S.C. Yudofsky & R.E. Hales (Eds.), *Essentials of neuropsychiatry and clinical neurosciences* (pp.489-517). Washington, D.C.: American Psychiatric Publishing, Inc.
- Mayberg, H.S., Liotti, M., Brannan, S.K., McGinnis, S., Mahurin, R.K., Jerabek, P.A., Silva, J.A., Tekell, J.L., Martin, C.C., Lancaster, J.L., & Fox, P.T. (1999). Reciprocal limbic-cortical function and negative mood: converging PET findings in depression and normal sadness. *The American Journal of Psychiatry*, 156, 675-682.
- McDonough-Ryan, P., DelBello, M., Shear, P.K., Douglas-Ris, M., Soutullo, C., & Strakowski, S.M. (2002). Academic and cognitive abilities in children of parents with bipolar disorder: a test of the nonverbal learning disability model. *Journal of Clinical and Experimental Neuropsychology*, 24, 3, 280-285.
- McQuillin, A., Lawrence, J., Kalsi, G., Chen, A., & Gurling, H. (1999). No allelic association between bipolar affective disorder and the tryptophan hydroxylase gene. *Archives of General Psychiatry*, 56, 99-100.

- Merriam, E.P., Thase, M.E., Haas, G.L., Keshavan, M.S., & Sweeney, J.A. (1999). Prefrontal cortical dysfunction in depression as determined by Wisconsin Card Sorting Test performance. *American Journal of Psychiatry*, 156, 5, 780-782.
- Meyer, S.E., Carlson, G.A., Wiggs, E.A., Martinez, P.E., Ronsaville, D.S., Klimes-Dugan, B., Gold, P.W., & Radke-Yarrow, M. (2004). A prospective study of the association among impaired executive functioning, childhood attentional problems, and the development of bipolar disorder. *Development and Psychopathology*, 16, 461-476.
- Miller, K.J. (2005). Executive functions. *Pediatric Annals*, 34, 4, 310-317.
- Miriam, L., Fein, D., Allen, D., Dunn, M., Feinstein, C., Morris, R., Waterhouse, L., & Rapin, I. (2001). Executive functioning in high-functioning children with autism. *Journal of Child Psychology and Psychiatry*, 42,2, 261-270.
- National Institute of Mental Health (2005, March). *Child and Adolescent Depression*.
<http://www.nimh.org>
- Paapadopoulos, T.C., Panayioto, G., Spanoudis, G., & Natsopoulos, D. (2005). Evidence of poor planning in children with attention deficits. *Journal of Abnormal Child Psychology*, 33, 5, 611-623.
- Piek, J.P., Dyck, M.J., Nieman, A., Anderson, M., Hay, D., Smith, L.M., et al., (2004). The relationship between motor coordination, executive functioning and attention in school aged children. *Archives of Clinical Neuropsychology*, 19, 1063-1076.
- Pineda, D.A., & Merchan, V. (2003). Executive function in young Colombian adults. *International journal of neuroscience*, 113, 397-410.

- Powell, K.B., & Kytja, K.S. (2004). Prefrontal executive function syndromes in children. *Journal of Child Neurology*, 19, 10, 785-797.
- Poznanski, E.O., Cook, S.C., & Carroll, B.J. (1979). A depression rating scale for children. *Pediatrics*, 157, 1195-1203.
- Poznanski, E.O., & Morkos, H.B. (1999). *Child Depression Rating Scale-Revised (CDRS-R)*. Los Angeles: Western Psychological Services.
- Puig-Antich, J., & Chambers, W. (1978). *The Schedule for Affective Disorders and Schizophrenia for School-Aged Children (KIDDIE-SADS)*. New York: New York State Psychiatric Institute.
- Puig-Antich, J., Kaufmann, J., Ryan, N.D., Williamson, D.E., Dahl, R.E., Lukens, E., et al., (1993). The psychosocial functioning and family environment of depressed adolescents. *Journal of the American Academy of Child and Adolescent Psychiatry*, 32, 244-253.
- Purcell, R., Maruff, P., Kyrios, & M., Pantelis, C. (1997). Neuropsychological function in young patients with unipolar major depression. *Psychological Medicine*, 27, 6, 1277-1285.
- Puura, K., Almqvist, F., Tamminen, T., Piha, J., Kumpulainen, K., Rasanen, E., Moilanen, I., & Koivisto, A-M. (1998). Children with symptoms of depression – what do the adults see? *Journal of Child Psychology and Psychiatry*, 39, 4, 577-585.
- Rappoport, M.D., Denney, C.B., Chung, K-M., & Hustace, K. (2001). Internalizing behavior problems and scholastic achievement in children: cognitive and

- behavioral pathways as mediators of outcome. *Journal of Clinical Child Psychology*, 30,4, 536-551.
- Reitan, R. (1958). The validity of the Trail Making Test as an indication of organic brain damage. *Perceptual and Motor Skills*, 8, 271-276.
- Reitan, R. (1971). Trail Making Test results for normal and brain-damaged children. *Perceptual and Motor Skills*, 33, 575-581.
- Reitan, R., & Davison, L. (1974). *Clinical neuropsychology: current status and applications*. New York: Hemisphere.
- Regier, D.A., Narrow, W.E., Rae, D.S., Manderscheid, R.W., Locke, B.Z., & Goodwin, F.K. (1993). The de facto US mental and addictive disorders service system: epidemiologic catchment area prospective 1-year prevalence rates of disorders in services. *Archives of General Psychiatry*, 50, 85-94.
- Rey, J.M., & Morris-Yates, A. (1992). Diagnostic accuracy in adolescents of several depression rating scales extracted from a general purpose behavior checklist. *Journal of Affective Disorders*, 26, 7-16.
- Reynolds, C.R., & Kamphaus, R.W. (1992). *Behavior Assessment System for Children: Manual*. Circle Pines, MN: American Guidance Service, Inc.
- Rice, F., Harold, J., & Thapar, A. (2002). The genetic aetiology of childhood depression: a review. *Journal of Child Psychology and Psychiatry*, 43, 1, 65-79.
- Roberts, A.C., & Wallis, J.D. (2000). Inhibitory control and affective processing in the prefrontal cortex: neuropsychological studies in the common marmoset. *Cerebral Cortex*, 10, 3, 252-262.

- Roca, C.A., Schmidt, P.J., & Rubinow, D.R. (1999). Gnadal steroids and affective illness. *Neuroscientist*, 5, 227-237.
- Rosvold, H., Mirsky, A., Sarason, I., Bransome, E., & Beck, L. (1956). A continuous performance task for brain damage. *Journal of Clinical and Consulting Psychology*, 20, 343-350.
- Rudd, M.D., Joiner, T.J., & Rumzek, H. (2004). Childhood diagnoses and later risk for multiple suicide attempts. *Suicide & Life-Threatening Behavior*, 34, 2, 113-125.
- Ryan, N.D. (2001). Diagnosing pediatric depression. *Biological Psychiatry*, 49, 12, 1050-1054.
- Sergeant, J.A., Geurts, H., & Oosterlaan, J. (2002). How specific is a deficit of executive functioning for Attention-Deficit/Hyperactivity Disorder?. *Behavioral Brain Research*, 130, 3-28.
- Shaffer, D., Fisher, P.W., & Lucas, C.P. (1999). Respondent-based interviews. In D. Shaffer, C.P. Lucas, & J.E. Richters (Eds.), *Diagnostic assessment in child and adolescent psychopathology*, 3-33. New York: Guilford.
- Shallice, T., Marzocchi, G.M., Coser, S., DelSavio, M., Meuter, R.F., & Rumiati, R.I. (2002). Executive function profile of children with attention hyperactivity disorder. *Developmental Neuropsychology*. 21, 1, 43-71.
- Shapiro, E.S. (2004). *Academic Skills Problems: Direct Assessment and Intervention (3rd ed.)*. New York: The Guilford Press.
- Shoaf, T.L., Graham, E.J., & Mayes, T.L. (2001). Childhood depression and treatment strategies in general pediatrics. *Pediatric Annuals*, 30,3, 130-137.

- Son, S.E., & Kirchner, J.T. (2000). Depression in children and adolescents. *American Family Physician*, 1-14.
- Starkstein, S.E., & Robinson, R.G. (1986). Cerebral lateralization in depression. *American Journal of Psychiatry*, 143, 1631.
- Stevens, J. (1999). *Intermediate statistics, second edition*. New Jersey: Lawrence Erlbaum Associates, Publishers.
- Stuss, D., & Alexander, M. (2000). Executive functions and the frontal lobes. *Psychological Research*, 63, 289-298.
- Tamminga, C.A., & Carter, C. (2000). Images in neuroscience: executive function. *American Journal of Psychiatry*, 157, 1, 3.
- Thapar, A., Harold, G., & McGuffin, P. (1998). Life events and depressive symptoms in childhood – shared genes or shared adversity? – a research note. *Journal of Child Psychology and Psychiatry*, 39, 8, 1153-1158.
- Timbermont, B., & Braet, C. (2004). Cognitive vulnerability in remitted depressed children and adolescents. *Behavioral Research and Therapy*, 42, 423-437.
- To, S.E., Zepf, R.A., & Woods, A.G. (2005). The symptoms, neurobiology, and current pharmacological treatment of depression. *Journal of Neuroscience and Neurosurgery*, 37, 2, 102-107.
- Veiel, H.O.F. (1997). A preliminary profile of neuropsychological deficits associated with major depression. *Journal of Clinical and Experimental Neuropsychology*, 19, 4, 587-603.

- Vriezen, E.R., & Pigott, S.E. (2002). The relationship between parental report on the BRIEF and performance-based measures of executive function in children with moderate to severe traumatic brain injury. *Child Neuropsychology*, 8,4, 296-303.
- Weissman, M.M., Livingston, B.M, Leaf, P.J, Florio, L.P., & Holzer, C., III. (1991). Affective disorders. In L.N. Robbins & D.A. Regeir (eds.) *Psychiatric disorders in America: the epidemiologic catchment area study*. New York: Free Press.
- Welsh, M.C., Pennington, B.F., & Groisser, D.B. (1991). A normative-developmental study of executive function: a window on prefrontal function in children. *Developmental Neuropsychology*, 7, 2, 131-149.
- Wender, P.H., Kety, S.S., Rosenthal, D., Schulsinger, F., Ortmann, J., & Lunde, I. (1986). Psychiatric disorders in the biological and adoptive families of adoptive individuals with affective disorders. *Archives of General Psychiatry*, 43, 923-929.
- Wilding, J.M. (2003). Attentional difficulties in children: weaknesses in executive function or problems coping with difficult tasks. *British Journal of Psychology*, 94, 427-436.
- Ylvisaker, M., & DeBonis, D. (2000). Executive function impairment in adolescence: TBI and ADHD. *Topics in Language Disorders*, 20, 2, 29-57.