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Executive Function Differences In Medicated Depressed, Non-Medicated Depressed, And Non-Medicated Non-Depressed Individuals

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EXECUTIVE FUNCTION DIFFERENCES IN MEDICATED DEPRESSED, NON-MEDICATED DEPRESSED, AND NON-MEDICATED NON-DEPRESSED INDIVIDUALS

by

Michael T. Ransom
Bachelor of Science, Westminster College, 2002
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A Dissertation
Submitted to the Graduate Faculty
of the
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Doctor of Philosophy

Grand Forks, North Dakota
August
2007
This dissertation, submitted by Michael T. Ransom in partial fulfillment of the requirements for the Degree of Doctor of Philosophy from the University of North Dakota, has been read by the Faculty Advisory Committee under whom the work has been done and is hereby approved.

Chairperson

Dean of the Graduate School

Date
PERMISSION

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To my frontal lobes
ABSTRACT

The purpose of the current study was to assess the performance of depressed young adults on tests of executive function, while addressing the variables of age and medication status, which have been inconsistently measured in previous research. It was hypothesized that statistically significant group differences would occur on tests of executive functions in three distinct groups: medicated depressed, non-medicated depressed, and non-medicated non-depressed individuals.

Participants included 53 adults who were medicated depressed (n = 15), non-medicated depressed (n = 16), and non-medicated non-depressed (n = 22) and were between the ages of 19 and 40 years. Participants completed measures assessing depression, psychological well-being (including anxiety), intelligence, and executive functions.

Between group comparisons revealed several statistically significant differences on executive function measures including the Wisconsin Card Sorting Test (WCST) Trials Administered, WCST Failure to Maintain Set, Trail Making Test (TMT) B, Stroop Color, and Stroop Color-Word. The non-medicated non-depressed group performed better overall than those in the medicated depressed and non-medicated depressed groups. Post hoc stepwise regression analyses indicated that anxiety predicted performance on a number of executive function measures to a greater degree than did depression.
Findings suggest that executive functions of young adults are affected by depression, medication status, and anxiety. Results of the present study contradict the assumption that psychotropic medications do not affect cognitive abilities. These findings also suggest that future research investigating the interaction between anxiety and executive functions are necessary.
CHAPTER I

INTRODUCTION

Depression has been documented as far back as the ancient Greeks (Caroll, 2006). Between the 17th and 18th centuries many Europeans used the term melancholia to describe, not only depression, but a vast array of mental illnesses. At that time many physicians believed that mental illnesses, including depression, were disorders of the brain. However, soon after, other theories of mental illness and depression began to flourish. Most of this began with Freud and his psychoanalytic theory.

In the late 19th and early 20th centuries, psychoanalytic explanations gained favor of most in the fields associated with mental illness (see Freud, 1917). Although psychoanalytic and many other purely psychological explanations have been widely accepted and utilized regarding the understanding of depression, discussions of a brain-based illness were never too far off. Geneticists and biologists believed that there were factors to depression that scientists simply had not yet found. Some have argued that complex hormonal feedback loops have gone awry or that the overstimulation of certain hormones results in depression (see Pepper & Kreiger, 1984). Others have argued that there are specific genes that are passed down from one generation to the next often resulting in depression (see Nurnbereger & Gershon, 1984). While all of the aforementioned explanations have gained much respect and credibility within each one’s respective field, the World Health Organization (WHO; 2001) asserted that they all likely share some responsibility in the cause of depression.
More recently, scientists have begun to focus again on the brain’s role in depression. Neuropsychologists have begun to examine the behavioral aspects of depression that are mediated by the brain. The executive functions, one’s skill and ability at performing novel problem-solving tasks from there beginning to there end, have found mixed results regarding the effects of depression on neurocognitive functions (Grant, Thase, & Sweeney, 2001; Landro, Stiles, & Sletvold, 2001; Paradiso, Lamberty, Garvey, & Robinson, 1997; Purcell, Maruff, Kyrios, & Pantelis, 1997). Scientists have argued that the executive function deficits that occur in depressed individuals in some studies and not in others have resulted from numerous confounds. Specifically, age and medication status (medicated or non-medicated) have been implicated as two major inconsistencies. The purpose of the current study was to add to the burgeoning literature regarding executive function deficits in depressed individuals while accounting for age and medication status.

Depression

According to *Webster’s II: New College Dictionary* (Berube et al., 1995), depression is “a neurotic or psychotic condition marked by an inability to concentrate, insomnia, and feelings of dejection and guilt” (p. 304). To many, depression is a source of terrible pain and unrelenting despair about themselves and their future. Of depression, Kay Redfield Jamison (1995) wrote:

Depression is awful beyond words or sounds or images; I would not go through an extended [depression] again. It bleeds relationships through suspicion, lack of confidence and self-respect, the inability to enjoy life, to walk or talk or think normally, the exhaustion, the night terrors, the day terrors. There is nothing good to be said for it except that it gives you the
experience of how it must be to be old, to be old and sick, to be dying; to
be slow of mind; to be lacking in grace, polish, and coordination; to be
ugly; to have no belief in the possibilities of life, the pleasures of sex, the
exquisiteness of music, or the ability to make yourself and others laugh.
(p. 217)

According to WHO (2001), approximately 121 million individuals worldwide
presently experience the deleterious effects of depression. The magnitude of this number
leads some to believe that in the near future it will be the leading cause of disability in the
world. Currently, it is reported to be the leading cause of lost work days for both men and
women (Quinn, 2000). Quinn noted that there have been reports that found depression
costs employers approximately $33 billion a year due to the increased absenteeism and
decreased productivity of employees.

According to the Diagnostic and Statistical Manual of Mental Disorders, Fourth
Edition, Text Revision (DSM-IV-TR; 2000), depression is the combination of several
symptoms simultaneously occurring over the course of a 2-week period that represents a
change from previous levels of functioning. Specifically, an individual must experience
either a depressed mood or a loss of interest or pleasure in daily activities. Furthermore,
an individual must experience other symptoms such as feeling depressed (e.g., feelings of
sadness or emptiness), a noticeable decrease of interest or pleasure in activities, weight
fluctuations, sleep difficulties, observable difficulties with motoric movements, fatigue,
feelings of worthlessness, cognitive processing difficulties, and/or recurrent thoughts of
death and/or suicide.
Comer (2001) has described depression as a decreased mood that is highlighted by pronounced sadness, significant reduction of energy, feelings of low self-worth and guilt. He elaborated by noting, depression “has no redeeming characteristics. It brings severe and long lasting psychological pain that may intensify as time goes by. Those who suffer from it may lose their will to carry out the simplest of life’s activities” (p. 193), while others lose their will to live outright.

WHO (n.d.) noted that fewer than 25% of those experiencing the negative effects of depression actually have access to effective treatment. The DSM-IV-TR (2000) reported that as many as 15% of those with Major Depressive Disorder (MDD) die by suicide. Furthermore, we now know that depression is not usually experienced in one episode (Barlow & Durand, 1999). According to Barlow and Durand (1999), reports have found that as many as 80% of individuals who experience a single episode of depression will have another, more severe, episode at a later time.

As previously noted, numerous explanations of depression have been set forth from the fields of biology, genetics, and psychology. Recently, neuropsychologists have begun to investigate neurocognitive functioning and its relationship with depression, and more specifically, what executive function deficits are present in individuals with depression.

Executive Functions

Zillmer, Speirs, and Culbertson (2001) asserted that executive functions are “higher-order regulatory and supervisory functions that … [are believed to be] subserved, in part, by the frontal lobes. Cognitive operations such as planning, mental flexibility, attentional allocation, working memory, and inhibitory control are considered executive
functions” (p. 552). Johnson-Greene and Adams (1998) elaborate on the previous description by including organization and regulation of affective state to their list of executive functions. Much of what is known about the executive functions of the brain has come from studies of brain damaged individuals.

Sbordone (2000) noted that reports have found associations between damage to the orbitofrontal cortex and certain behavioral manifestations (e.g., decreased social tact, use of crude language, inability to regulate one’s emotions, emotional lability, & insensitivity toward others). Furthermore, neuroimaging studies have shown decreased regional blood flow and metabolism in depressed individuals in the anterior regions of the brain. More specifically, depressed individuals showed decreases in the prefrontal cortex, left dorsolateral prefrontal cortex, Supplementary Motor Area (SMA), anterior cingulate cortex, and premotor areas of the frontal lobes as well as the posterior cingulate cortex and angular gyrus of parietal lobes (Baker, Frith, Dolan, 1997; Bench et al., 1992). Additionally, Positron Emission Tomography (PET) studies have implicated frontal lobe inactivity in depressed individuals (Paradiso et al., 1997).

Age and medication status appear to be common confounds in the literature regarding the executive functions and depression. Operational definitions of young adult, middle age, and late life/elderly individuals have been erratic. For example, while executive function deficits in older individuals have been fairly well documented (Grant et al., 2001), attempts to investigate younger adults have resulted in research participants that range in age from as young as 18 (Merriam et al., 1999) to those as old as 61 years (Porter et al., 2003).
Additionally, medication status has been investigated half-heartedly by most. Although studies such as Porter et al.'s (2003) have focused on depressed medication free individuals, their sample included individuals in their 60's. Furthermore, studies such as Paradiso et al. (1997) clustered their experimental sample together under one variable (depressed) whether they were medicated or not. According to Paradiso and his colleagues, post-hoc analyses showed that there were no differences between their medicated depressed and non-medicated depressed groups. However, comparing only 12 medicated individuals to 8 non-medicated individuals hardly accounts for the confounds inherent to such small sample sizes and loose methodology.

Purpose

Depression has been a recorded part of the human experience since the days of antiquity. To date, various hypotheses and theories have been set forth to explain how depression develops as well as how one can ameliorate the effects of depression in one's life. With a recent emphasis on depression's effects on the brain, researchers have attempted to identify sequelae of depression on a brain-behavior continuum. However, the research to date has overlooked various methodological aspects that have hindered health care providers from utilizing the information to help younger adult's diagnosed with depression. For example, too wide of an age range has been utilized in various studies (e.g. Porter, Gallagher, Thompson, & Young, 2003) and medication status appears to have been studied as an afterthought rather than as the major variable it is. While some studies have attempted to mitigate these confounds, no research has managed both age and medication status adequately. It is well known that the brain continues to develop well into one's early twenties (Zillmer et al., 2001) and that different areas of the
brain are used for the same task at different stages of development (Riccio, 2006). Thus, any comparison of differently aged individuals must address these differences methodologically. Moreover, medication status (e.g., antidepressants) directly affects one’s neurochemistry and is thus of key importance in any discussion of cognitive functioning.

The purpose of the current study was to assess the level of executive functioning in a depressed and non-depressed sample. Furthermore, medication status was managed by separating the depressed group into medicated and non-medicated individuals. Additionally, participants consisted of individuals between the ages of 19 and 40. This age group allows for a purer understanding of the effects of depression on the executive functions in a younger adult population.

It is the hypothesis of the current study that differences in performance will be found between medicated depressed, non-medicated depressed, and non-medicated non-depressed participant groups on measures of executive functioning. More specifically, it is hypothesized that all three of the aforementioned groups will obtain statistically significant differences from one another regarding their performance on five well-established tests of executive functions (i.e., Wisconsin Card Sorting Test [Heaton, Chelune, Talley, Kay, & Curtiss, 1993], Trail Making Test [Reitan, 1993], Tower of London: Drexel Edition [Culbertson & Zillmer, 2005], Stroop Color and Word Test [Golden & Freshwater, 2002], & Controlled Oral Word Association [Spreen & Straus, 1998]).
CHAPTER II

REVIEW OF LITERATURE

In the following sections a discussion occurs regarding the various aspects of depression that include a definition of depression, prevalence rates, and theories. Psychological, genetic, and biological theories of depression are reviewed. Within the psychological domain, the focus is on Psychoanalytic, Interpersonal, and (particularly) Cognitive conceptualizations. Twin studies are emphasized in a discussion focused on genetic implications related to depression, while the serotonin, norepinephrine, and corticotropin-releasing factor hyperactivity models are discussed in terms of biological explanations of depression. These are in no way meant to be exhaustive lists of all conceptualizations of depression in each field. However, these explanations are presented in an effort to provide an overview of some of the most popular psychological, genetic, and biological explanations of depression. Furthermore, descriptions of depression at different developmental points in time (i.e., childhood, adolescence, young adulthood, & later-life) serve to provide context to the varied paths depression takes across the lifespan.

The second half of the literature review overviews the executive functions, with emphasis on the most popular theories of executive functions (including the most common symptoms of executive dysfunction), the development of the executive functions, and different measures that assess aspects of the executive functions. Furthermore, the scientific literature regarding executive functioning and depression are discussed in order to provide context related to what has been accomplished in the
research literature and highlight what further research is necessary in order to further the scientific communities understanding of the complex interaction that results between the executive functions and depression.

Depression

DSM-IV-TR definition of Depression

According to Barlow and Durand (1999), the Major Depressive Episode is the most often diagnosed and most severe type of depression. An extension of this is the DSM-IV-TR (2000) mood disorder, Major Depressive Disorder (MDD), Single Episode. The DSM-IV-TR indicates that individuals diagnosed with a MDD, Single Episode must be in the process of experiencing a Major Depressive Episode that is not better accounted for by a Schizoaffective Disorder nor simply covering over Schizophrenia, Schizophreniform Disorder, Delusional Disorder, or Psychotic Disorder Not Otherwise Specified. Furthermore, the individual must have never experienced a Manic, Mixed, or Hypomanic Episode.

If the full criteria just described are met, the clinician then has several labels at her/his disposal to make the diagnosis more specific. For example, one can further specify the clinical condition by attaching the statement, In Partial Remission, In Full Remission, Chronic, With Catatonic Features, With Melancholic Features, With Atypical Features, or With Postpartum Onset (DSM-IV-TR, 2000). These various labels clearly show how complex and multifaceted depression truly is.

The DSM-IV-TR (2000) criteria for a Major Depressive Episode requires that an individual possess at least five of the following symptoms: depressed mood most of nearly every day, either by self-report or observations of others; strikingly diminished
interest or pleasure in most or all activities every—or nearly every—day, either by self-report or observations of others; significant weight loss, not due to the effects of dieting, or gain; insufficient or too much sleep nearly every day; agitated, delayed, or impeded movement or muscular action due to mental processes nearly every day; fatigue or loss of energy nearly every day; feelings of worthlessness or excessive or inappropriate guilt, which can be delusional, nearly every day; diminished ability to think and/or concentrate or indecisiveness; and/or recurrent thoughts of death or suicidal ideation (DSM-IV-TR).

These symptoms must have been present for at least two weeks and represent a change from one’s previous level of activity. Furthermore, at least one of the symptoms an individual possesses must be a depressed mood or loss of interest or pleasure. Additionally, the symptoms cannot meet criteria for a Mixed Episode, they must cause clinically significant distress or impairment in social, occupational, or other important areas of the individual’s functioning, they must not be symptoms related to the physiological effects of drugs or medication or a medical condition, and the symptoms must not be better explained by Bereavement (DSM-IV-TR). While the DSM-IV-TR has attempted to provide a clear list of descriptive symptoms that manifest themselves during a Major Depressive Episode, others have theorized the underlying causes of this disorder.

Prevalence of Major Depressive Disorder

Depression has been reported to be one of the most common and severe psychological disorders (Weissman, Bruce, Leaf, Florio, & Holzer, 1991) with typical onset occurring in one's mid-twenties (DSM-IV-TR; 2000; Ebmeier, Donaghey, & Steele, 2006; McMahon & DePaulo, 1996). However, Clayton (1983) has asserted that the true prevalence of MDD is unknown as the numerous studies in this area have varied in their
methodology and case criteria. Although an exact prevalence rate will never be known, studies have consistently reported rates of MDD that concur with the DSM-IV-TR (2000) and WHO (2001) rates previously reported (see Weissman & Myers, 1978; Boyd & Weissman, 1981).

Weissman et al. (1991) reported lifetime prevalence rates for individuals living in the United States. They reported that 4.9% of individuals will experience a MDD in her/his lifetime with the subgroups of 30-44 year-olds, females, and Caucasians showing the greatest lifetime prevalence percentages (7.5, 7.0, & 5.1, respectively). The research literature generally agrees that women experience this disorder much more than men (Clayton, 1983; DSM-IV-TR, 2000). Furthermore, while the DSM-IV-TR (2000) reports that the disorder appears to be unrelated to one’s “ethnicity, education, income, or marital status” (p. 372), Weissman et al. (1991) found somewhat contradictory evidence regarding differences related to race. Weissman et al. reported prevalence percentages for European Americans, African Americans, and Hispanic Americans of 5.1, 3.1, and 4.4, respectively.

More recently, of the approximately 121 million individuals who currently deal with depression on a daily basis (WHO, 2001), estimates show that between 5% and 10% of adults in the United States will experience a unipolar (i.e., non bi-polar) depressive episode during any given year. The DSM-IV-TR (2000) reports the prevalence rates of MDD in community samples at between 10% and 25% for women and between 5% and 12% for men.
Theories of Depression

Psychological Theories of Depression. Affective disorder is a commonly used term that refers to a disorder in one’s mood (Thomas, 1993). Taber’s Cyclopedic Medical Dictionary (17th Ed.; Thomas, 1993) defines affective disorders as “a group of disorders characterized by a disturbance of mood accompanied by a full or partial manic or depressive syndrome that is not caused by any other physical or mental disorder” (p. 50). It further describes depression as being characterized by an altered mood (Thomas). Mood is defined as “a pervasive and sustained emotion that may have a major influence on a person’s perception of the world” (Thomas; p. 1236; e.g., elation, joy, anger, depression, & anxiety). Additionally, Taber’s (Thomas) suggests that mood and affect are somewhat interchangeable and describes affect as “the emotional reactions associated with an experience” (p. 50)

Comer (2001) has defined depression as “a low, sad state marked by significant levels of sadness, lack of energy, low self-worth, guilt, or related symptoms” (p. 191). WHO (2001) describes depression as a common mental disorder that is typically “characterized by sadness, loss of interest in activities and by decreased energy” (¶ 2, retrieved March 29, 2005). WHO further states that depression can be distinguished from a normal decrease in one’s mood by the severity, symptoms, and duration of the low mood state and acknowledged that psychosocial, genetic, and biological factors are implicated in causing the onset of a depressive episode.

Past psychological explanations have been put forth by numerous theorists. For example, in 1917 Freud published Mourning and Melancholia which has been heralded as the most influential work on depression to come from psychodynamic theory. Freud’s
seminal work grew from a belief that depressed individuals were much like grief stricken individuals following a loss (Becker, 1977; Comer, 2001). Therefore, as Freud believed that depressed individuals were likely the result of severe, uncontrollable stress associated with a loss, they would regress back to the Oral phase of psychosocial development (Comer, 2001) and appear to others as an individual who’s world and self appeared empty, self-regard severely impaired, and external relations minimal (Becker, 1977). Furthermore, Freud believed that depressed individuals were predisposed to pathological reactions to loss and that regression to earlier psychosexual stages of development (e.g., the Oral phase) were the result (Becker).

Another theory of depression came in the form of Interpersonal theory (Schwartz & Schwartz, 1993; Weissman & Klerman, 1990). The Interpersonal Psychotherapy (IPT) approach to depression was the first of its kind to look at a variety of factors that led to and maintained depression. IPT is based on the theory that although one’s environment, genetics, symptom patterns or severity, childhood experiences, and biological factors all interact and result in depression, these all occur in the context of interpersonal relationships (Schwartz & Schwartz, 1993; Weissman & Klerman, 1990).

Harry Stack Sullivan was another notable theorist whose work influenced the development of IPT. For example, he emphasized that most psychiatric illnesses are related to flawed interpersonal relationships (Schwartz & Schwartz, 1993). Additionally, Meyer viewed psychiatric illnesses “as an expression of the patient’s attempt to adapt to the environment” (Weissman & Klerman, 1990, p. 380). IPT began to focus its work on individual’s interpersonal functioning and the various problems that co-occur with depression (Weissman & Klerman). Thus, relationships between family members,
IPT theorists created a frame of depression from which three main components have been set forth that are seen as being involved in depression. The first of these three components is believed to be symptom formation (Weissman & Klerman, 1990). Symptom formation includes the depressive affect and vegetative signs of depression such as fluctuations in sleep and appetite, loss of interest in pleasurable activities, psychomotor agitation, and fatigue (Weissman & Klerman). The second component is related to social and interpersonal relations. This component reflects social roles, past learning, and current problems in social relationships (Schwartz & Schwartz, 1993; Weissman & Klerman, 1990). The third component is that of personality. This includes one's enduring traits and behaviors, communication deficits, low self-esteem, and inadequate management of anger and guilt (Weissman & Klerman, 1990). Weissman and Klerman asserted that this component is "... the person's unique reactions and patterns of functioning and may contribute to a predisposition to depression..." (p. 381).

According to Schwartz and Schwartz (1993) and Weissman and Klerman (1990), IPT only focuses its energies on intervening at the first two levels due to the lack of research indicating that personality change is fruitful. Klerman, Weissman, Rounsaville, and Chevron (1984) asserted that IPT works to decrease depressive symptoms and help the individual develop healthier ways of dealing with interpersonal relationships that are linked to the depressive episode. Another psychological view of depression has implicated one's own thoughts rather than social interactions. Cognitive theorists, beginning with the work of Aaron Beck in the early 1970's, has attempted to explain
depression through three interacting aspects of one’s cognitions (i.e., the self, one’s environment, and one’s future).

The cognitive theory of depression hypothesizes that the negative and pessimistic beliefs that are typically associated with depression are not a result of the depression, but are actually a cause of it (Schwartz & Schwartz, 1993). According to Barlow and Durand (1999), these continuous negative and pessimistic beliefs are cognitive errors. A common example of a cognitive error is arbitrary inferences (Barlow & Durand). For instance, an individual may say hello to another person as they pass one another on the street and, for some reason, the person may not say hello back. For the individual who makes inferences arbitrarily, he/she may believe that the person did not say hello because of something he/she said or did. The individual will likely come up with many negative reasons for the person to have not said hello, potentially asking her/himself, “what did I do to make that person upset with me?”

Although many hypotheses are typically made by the individual, it is likely that none of them will include a neutral response, such as the person did not hear the remark. All of the cognitive errors are hypothesized to revolve around three main areas, self, environment, and future; together these are referred to as the cognitive triad of depression (Barlow & Durand; Schwartz & Schwartz, 1993; Young, Weinberger, & Beck, 2001).

The cognitive triad model of depression posits that a depressed individual will typically view her/himself as worthless and/or unable to be loved. A typical view of a depressed person’s environment is that it is unmanageable, overwhelming, and harsh. The future is often believed to be the source of only more pain as many feel what they are
Cognitive theorists hypothesize that childhood and development are integrally related to depression. They have asserted that while growing up people do the best they can to deal with and understand their environment (Schwartz & Schwartz, 1993; Young et al., 2001). In so doing, they unintentionally create negative patterns of dealing with situations resulting in schemas. According to Beck, Shaw, Rush, and Emory (1979), individuals screen, code, and evaluate information, and a schema is the structure one gives to this information. Based on this information and subsequent structure, “the individual is able to orient himself in relation to time, and space and to categorize and interpret experiences in a meaningful way” (Beck et al., p. 283). While schemas can be positive, most schemas in depressed individuals are negative (Schwartz & Schwartz, 1993). Recent work by Jeffrey Young and his colleagues (2001) has extended the concept of schemas.

Young has identified a total of 18 negative schemas that are compiled into five categories (disconnection and rejection, impaired autonomy and performance, impaired limits, other-directedness, and overvigilance and inhibition; Young et al., 2001). Cognitive theorists have not only set forth an explanation of depression, they have also set forth a treatment regimen to ameliorate the negative thinking that is believed to occur in depressed individuals. According to Young et al. the center of attention in cognitive therapy is to change the negative thinking that is so prevalent in depressed individuals. A great deal of research has supported the concepts set forth by cognitive theorists (see Gloaguen, Cottraux, Cucherat, & Black, 1998). Thus, it appears that psychological
theories of depression have made a considerable impact on the diagnosis and treatment of depression and will likely continue to do so well into the future.

The cognitive theory of depression has received a great deal of attention in the scientific literature as it relates to treatment outcomes (see Dobson, 1989; Robins & Hayes, 1993). Furthermore, Hollon, Shelton, and Davis (1993) discussed the debate regarding cognitive therapy's efficacy. Overall, they identified several studies that have shown how cognitive therapy has fared well against other forms of psychotherapy. For example, dynamic (see Covi & Lipman, 1987), non-directive (see Shaw, 1977), and interpersonal therapies (Shaffer, Shapiro, Sank, & Coghlan, 1981; see also Dobson, 1989 for an excellent review of a large number of psychotherapies as compared to cognitive therapy). Robins and Hayes (1993) also noted that several studies have shown that cognitive therapy resulted in better treatment outcomes than pharmacotherapy.

Dobson (1989) reviewed the efficacy of several forms of psychotherapies and pharmacotherapy as compared to cognitive therapy through meta-analysis. Dobson reported, "cognitive therapy is more effective than nothing at all, behavior therapy, or pharmacotherapy in the treatment of clinical depression. It also appears that cognitive therapy is superior to other forms of psychotherapy in the treatment of depression" (p. 417).

*Genetic Theories of Depression.* Genetic models of depression have also been put forth by various researchers. Past research has shed some light on the heritability debate of whether or not genetics play a factor in one's susceptibility to depression. Nurnbereger and Gershon (1984) reported more than 20 years ago that the differences in correlations evidenced in research studies of monozygotic/identical (one fertilized egg splits into two)
and dizygotic/fraternal (two different eggs are fertilized by two different sperm) twins have provided a strong argument for heritability. Research conducted approximately ten years (Kendler et al., 1994) and 20 years (McGue & Christensen, 2003) later emphasized the consistent finding that shared environment (i.e., reared together) accounts for very little, to none, of the variance in depression in twins.

Lyons et al. (1998) asserted that twin studies generally reflect the importance that genetics play in the etiology of depression and thus utilized a proband concordance rate approach in their study. According to Thomas (1993), proband is the initial individual presenting with a condition (generally genetic) “who causes a study of his or her heredity in order to determine if other members of the family have had the same disease or carry it” (p. 1599). Furthermore, concordance “in twins, [is] the equal representation of a genetic trait in each” (Thomas, p. 432).

In their article, Lyons et al. (1998) reported concordance rates from 3,372 pairs of male twins with unipolar depression. Statistical analyses were used to remove the effects of shared and non-shared environments revealing proband concordance rates for monozygotic and dizygotic twins of 22.5% and 14.0%, respectively. Lyons et al. further asserted that their analyses showed strong effects of genetics on major depression while at the same time excluding various environmental factors (e.g., parenting behaviors, peer relationships, and experiences outside of the family) through structural equation modeling.

Additionally, McGue and Christensen (2003) recently completed a longitudinal study of 2100 elderly Danish twins. Over the course of four years the researchers assessed depression in the twins at yearly intervals resulting in pooled (average of the
four trials) concordance rates. They found concordance rates of 33% for male and female monozygotic twins (range = 25% to 39%, 95% confidence interval) while dizygotic twins had a concordance rate of 14% (range = 7% to 21%, 95% confidence interval). McGue and Christensen further noted that their study was consistent with others (e.g., Kendler et al., 1994) that have failed to produce evidence that a shared environment by mono- or dizygotic twins influences the prevalence of depression therefore encouraging further evidence of genetic involvement in depression.

**Biological Theories of Depression.** Biological conceptualizations have been posited by researchers looking at body chemistry imbalances as a primary underlying component of depression. Klein (2000) asserted that Positron Emission Tomography (PET), a device which measures the “metabolic activity of a specific structure of the nervous system in order to determine neuronal functioning” (p. 492), has confirmed that there is decreased cortical brain activity during depressive episodes and increased activity during manic episodes. More specifically, Davidson (1992) has reported decreased activity in the left frontal cortex during depressive episodes. Klein (2000) asserted that the correlation between metabolic activity in the brain and one’s mood is key evidence that invites a biological explanation of depression.

Nemeroff (1997) reported that of the numerous biological theories of depression that have been set forth in recent years, three major theories have progressed to the forefront: (a) the serotonin theory (the hypothesis that there is a deficiency of serotonin at specific synaptic sites, (b) the norepinephrine theory of depression (the hypothesis that there is decreased norepinephrine at specific synaptic sites within the central nervous system), and (c) the corticotropin-releasing factor hyperactivity model (the hypothesis
that there is a hypersecretion of corticotropin-releasing factor in the central nervous system). Furthermore, Nemeroff asserted that the serotonergic and corticotropin-releasing factor hyperactivity models have received the most empirical attention.

In 1984, von Praag asserted that the link between serotonin and depression had been studied for over 20 years. With serotonin affecting one’s mood, behavior, and thought processes (Barlow & Durand, 1999), it seems quite appropriate to put efforts into studying the relationship between serotonin and depression.

Siever, Guttmacher, and Murphy (1984) presented a discussion and review of the literature implicating serotonin’s role in mood disorders. They asserted that “the serotonergic system has known contributions to a variety of affect-related behaviors” (p. 587). For example, they noted that the link between aggression, psychomotor activity, and irritability and the serotonergic system have led many researchers to investigate their relationship.

Studies have investigated serotonin levels and found lower levels of serotonin in blood, blood cells, and cerebrospinal fluid of depressed individuals (Siever et al., 1984). von Praag (1984) acknowledged that various studies of serotonin arose from the fact that certain antidepressant medications decreased the amount of serotonin in the presynaptic neuron by blocking its reuptake into the synapse (Vasko & Gutierrez, 2003), which decreased depressive symptoms. Furthermore, assertions maintaining that medications that increase the uptake of serotonin can induce a depressive state (e.g., reserpine, methyldopa, & propanolol; Vasko & Gutierrez; van Praag, 1984) have also provided a great deal of insight into the serotonergic aspects of depression.
More recently, Nelson, Mazure, Jatlow, Bowers, and Price (2004) showed that a sample of 38 hospital psychiatric inpatients that met criteria for MDD had lower depressive rating scale scores after taking a combination of serotonin and norepinephrine reuptake inhibiting medications than when taking one medication without the other. While the differences in scores were not statistically significant, the authors asserted that the combination of drugs still produced a greater change than either drug acting alone. This may be true, however, the fact that the difference is only semantically different and not statistically different casts definite doubt on the necessity to prescribe or ingest two medications when one will apparently do. Although this study failed to gather statistical backing for its hypothesis, the results appear to back the serotonergic hypothesis of depression in that there were noteworthy decreases in the depressive rating scales of the participants.

The corticotropin-releasing factor hyperactivity model is another popular biological theory of depression (Nemeroff, 1997). According to this theory of depression an oversecretion of Corticotropin-releasing factor is secreted into the central nervous system which triggers a cascade effect within various other parts of the body that, in turn, ultimately result in depression. The hypothesis is that a hypothalamic-pituitary-adrenal axis is activated when the brain is stimulated by stress, or other glucocorticoid hormones (Pepper & Kreiger, 1984). In this type of situation, the brain is stimulated, which then activates the hypothalamus to release corticotropin-releasing factor; this stimulates the anterior pituitary gland which then releases adrenocorticotropic hormone (ACTH). The ACTH is carried via the blood stream to the adrenal cortex where it activates the release of glucocorticoids, especially cortisol (Huether & Zekauskus, 1996).
According to Huether and Zekauskus (1996), one of the major roles of the adrenal system is to maintain an optimal internal body environment throughout one's life. Clearly, if there is a hypersecretion along the way, it offsets the homeostatic balance that is achieved through the hormonal regulation system resulting in untoward effects. As ACTH stimulates secretion in the adrenal gland, cortisol, the main glucosteroid, is produced and can cause major problems in the feedback loop. For example, if there is a problem of oversecretion at the point of corticotropin-releasing factor, there will ultimately be an oversecretion of ACTH, thus resulting in an overproduction of cortisol. Therefore it would logically make sense that stress and worry associated with depression would trigger such adverse biological hypersecretions.

Müller and Wurst (2004) suggest that brief situations of stress, either experienced with excitement or fear, result in the adaptive hypothalamic-pituitary-adrenal axis response. However, an uncontrolled and unrelenting activation of this feedback loop can leave one in a chronic state of distress that results in a surplus of stress hormones which is believed to enhance one's vulnerability to problems such as affective disorders (Müller & Wurst). The corticotropin-releasing factor hyperactivity model has been supported by investigations that found increased secretory impulses of ACTH and cortisol as well as elevated urinary levels of cortisol (Rubin et al., 1987), elevated levels of corticotropin-releasing hormone in cerebrospinal fluid (Nemeroff et al., 1984), increased secretion of corticotropin-releasing hormone from neurons in the limbic area of the brain (Raadsheer et al., 1994), and elevated levels of corticotropin-releasing hormone as compared to the number of available binding sites in the prefrontal cortex of the research sample (Nemeroff et al., 1988).
Of the three views of depression that have been discussed (psychological, genetic, & biological), it is the biological view that addresses the issue of medication. A common sense approach related to the biological view is that if there is a chemical imbalance, then a medication could help to reestablish equilibrium. This assertion is key to the present study in that the body chemistry being discussed in these biological models is actually brain chemistry. Furthermore, medication that is designed to affect brain chemistry may result in untoward effects on brain functioning.

*Depression Across the Lifespan*

*School Aged Children.* School aged children (6-12 years) are a group that is not often discussed in MDD literature, likely due in part to the typical onset occurring in one’s mid-twenties (DSM-IV-TR, 2000). While MDD is much less common in school aged children, prevalence rates have been reported as being between 0.04% and 2.5% (Birmaher & Rozel, 2003). A noteworthy difference between adolescent and adult onset MDD and school aged child MDD is that the disorder occurs at approximately the same rate in boys as it does in girls (Birmaher & Rozel). Another noteworthy detail regarding childhood MDD is that the prevalence has been increasing in this population (Birmaher & Rozel). Although there is an age of onset difference between children and adults, the underlying etiological factors are similar (i.e., psychological, genetic, & biological factors; Watts & Markham, 1997)

The clinical characteristics of school aged MDD are also fairly similar to that of the DSM-IV-TR (2000) MDD previously described. However, Birmaher and Rozel (2003) reported that children with MDD often are irritable or report feeling bored rather than sad, and they typically experience “more symptoms of separation anxiety, phobias,
somatic complaints, and behavioral problems” (p. 48) than do adolescents with MDD. Another stark difference in this population is that there is rarely melancholic school aged MDD children, however, many (40%-70%) have at least one comorbid psychiatric disorder, while between 20% and 50% have two or more (Costello et al., 1999). With such high comorbidity rates, Birmaher and Rozel (2003) asserted that “…the presence of comorbid psychiatric disorders is more the rule than the exception” (p. 48).

As noted earlier, psychological, biological, and genetic factors are believed to play vital roles in the etiology of MDD in school aged children (Birmaher & Rozel, 2003). The course of MDD in children has been reported to last between 8 and 13 months with high relapse rates ranging between 30% and 70% within 2-7 years (Goodyer et al., 1997). Outcomes in school aged MDD children appear to be hampered by the psychosocial dysfunction experienced during the disorder (Puig-Antich et al., 1985). Puig-Antich et al. asserted that most children’s interpersonal relations improve as the MDD remits although some impairment has been found to remain. While there appears to be a base of research attempting to elucidate the connection between MDD and school aged child, more understanding is needed in order to make more declarative statements regarding how to treat this group of individuals.

Adolescents. Adolescents experiencing depression may have had difficulties in the past receiving appropriate treatment due to a commonly held professional belief that adolescent turmoil is normal (Harrington, 2003). However, there has been research indicating that adolescent depressive symptoms resemble that of the adult criteria for depression (see Pearce, 1978; Puig-Antich, 1982; Weinberg et al., 1973). While depressive symptoms appear to manifest themselves in adolescents, the effects are
somewhat different than those in adult populations. For example, Harrington and Clark (1998) discussed a study in which no participants were found to have the full five or more depressive symptoms necessary to receive the diagnosis of Major Depressive Episode. However, parental reports suggested that students with as little as one or two symptoms were experiencing significant problems in school as a result of the symptoms. Harrington and Clark asserted that such information indicates that “it is better for adolescents to have no depression at all than to be averagely depressed” (p. 34) by having only one or two symptoms.

As with school aged children, the etiological factors of depression are similar in adolescents (i.e., psychological, genetic, & biological factors), as are the increased comorbid difficulties that commonly occur. Costello, Federman, Erkanli, and Angold (1999) presented meta-analytic research indicating that depression is highly comorbid with other psychiatric disorders; specifically with anxiety, conduct disorder, and attention deficit hyperactivity disorder (ADHD; with median odds ratios of 8.2, 6.6, & 5.5, respectively at 95% confidence intervals). These comorbidity estimates match the results reported by van Dulmen et al. (2002) in which they found the comorbidity of depression and conduct disorder/oppositional defiant disorder to be at 5.9%, which they report is consistent with those of the DSM-IV. Furthermore, van Dulman et al. reported adolescent prevalence rates for MDD at 1%. However, Lewinsohn, Rohde, and Seeley (1998) forecasted that 28% of all adolescents will experience a MDD by their 19th birthday.

An interesting detail emerges in regard to gender and adolescent depression. Harrington (2003) reported that there are greater numbers of girls who experience MDD than boys of similar age. Furthermore, Angold, Costello, and Worthman (1998) reported
that in the adolescent population the question of onset may not be so much of how old the adolescent is, but more of whether or not he/she has started puberty. This is apparently due to rates of depression increasing with advancing cycles of puberty (Angold et al.).

While there appears to be more adolescents who experience MDD than school aged children, research suggests that adolescents have good prognoses of remittance. For example, Harrington suggested that “… most young people with major depression will recover to a significant extent …” (p. 72). However, not all is positive as Kovacs et al. (1984) reported that approximately 40% of their sample of adolescents with MDD had a MDD relapse. It appears that many gains have been made in regards to adolescent MDD; however, Harrington (2003) warned that more research must be done in order to validate the concept of adolescent depression.

*Young Adulthood.* Depressed younger adults (approximately 19-40 years for the current study) have also been somewhat overlooked in the research literature regarding MDD. However, while onset of MDD can occur at any age, the most common age at onset is in one’s mid-twenties (Ebmeier et al., 2006; McMahon & DePaulo, 1996). McMahon and DePaulo (1996) asserted that there is, however, some debate as to what exactly constitutes onset. Some argue that it is age at first treatment while others define onset as the age at identification of first depressive symptoms. However, McMahon and DePaulo acknowledged that most investigators typically use age at first hospitalization, age at first diagnosis of MDD, or age at first identified impairment. Regardless of the definition used, as noted earlier, young adulthood is the most common onset period for depression.
Numerous prevalence rates have been published regarding MDD. The Epidemiologic Catchment Area (ECA) and the National Comorbid Survey (NCS) data have suggested that depression occurs in considerably higher numbers than does bipolar disorder (Zarate & Tohen, 1996). Weissman et al. (1988) reported the lifetime prevalence rate of at least one episode of MDD at 4.4%. However, their range was between 2.9% at a site in Baltimore, MD and 5.8% at a site in New Haven, CT. More specifically, Zarate and Tohen (1996) reported the NCS prevalence rates by age and sex. For 15 to 24 year-old males and females, rates were 11% and 20.8% respectively (total of 15.7%), 13.1% and 19.4% for 25 to 34 year-old males and females respectively (total of 16.5%), and 14.7% and 23.8% for 35 to 44 year-old males and females respectively (total of 19.2%).

Although etiology remains similar to that as has been discussed in the previous age groups (i.e., psychological, genetic, & biological), there are different factors that affect younger adult individuals. For example, it appears that a staple of young adult depression is that it is often invoked as a result of stressful life events (Friis, Wittchen, Pfister & Lieb, 2002).

Friis, et al. (2002) reported that while stressful life events appear to be an antecedent to depression, stressors do not necessarily need to be negative. In their research they found that negative life stressors showed a significantly elevated odds ratio of resulting in a depressive condition (e.g., school, family, work, & living situations). However, positive life stressors (e.g., graduating college, marriage, beginning a family, purchasing a home) also showed a significantly elevated odds ratio as an antecedent of depression (Friis et al.; Horwitz & White, 1991). Lewinsohn and Seeley (2003) have recently been investigating what effects previous episodes of MDD in adolescence may
have on MDD episodes of younger adults. Having found that the symptoms of MDD are somewhat similar between the closely aged groups, researchers are now beginning to look at other areas where the two may be similar regarding their depressive disorder in an attempt to better understand the younger adult depressive phenomena.

One area that appears to be more isolated to the younger adult depressed population is the fact that depression in young adulthood appears to lead to role impairment in more than 50% of depressed individuals (Ebmeier et al., 2006). Furthermore, use of professional health care services in order to assuage depressive symptomatology is low in the younger adult population. Ebmeier et al. reported that approximately half of those needing treatment for depression in the United States were not receiving such treatment. Druss, Hoff, and Rosenheck (2000) reported that less than 1 in 13 young adults were receiving antidepressant therapy. They suggested that patient, clinician, and systemic issues combined to contribute to overall poor treatment situations. Moreover, only 1 in 9 individuals experiencing symptoms consistent with MDD and with suicidal ideation were receiving antidepressant therapy (Druss et al., 2000).

Ebmeier et al. (2006) have speculated that there has been an increased worry among the general population regarding the untoward side effects of antidepressant medication (e.g., increased suicide among antidepressant users). Organizations such as the National Institute for Clinical Excellence have recommended that antidepressant therapy for mild cases of depression in adults be contraindicated due to the poor risk-benefit ratio (Ebmeier et al.). However, this is in sharp contrast to the recommended guidelines made by the American Psychiatric Association (2000) regarding pharmacotherapy for depression, which asserts that "if preferred by the patient,
antidepressant medications may be provided as an initial primary treatment modality for mild major depressive disorder” (p. 2). While some things are similar as far as depression in younger adults as compared to children and adolescents, this review suggests that there are age specific factors that play into younger adult depression. This is important in the context of the current study in order to obtain a better understanding of this population in order to facilitate better overall treatment.

_Later Life_. Depression has historically been a common condition in later life individuals and continues to remain so (O’Brien & Thomas, 2003). Lobo, Saz, Marcos, Dia, and De la Camara (1995) reported prevalence rates of 4.8% for depressive disorders in their sample. Similarly, Steffens et al. (2000) reported prevalence rates of 4.4% in women and 2.7% in men. Overall, however, prevalence rates from various other research studies have ranged from as low as 1% to as high as 20% (see Ernst & Angst, 1995; Katona, 1994; Palsson & Skoog, 1997). Additionally, a noteworthy gender difference is found in later life depression. While women with depression still outnumber men, the difference becomes much less as age increases (O’Brien & Thomas, 2003). Bebbington (1998) observed that this decline in female cases of depression coincided with female menopause. However, he elaborated by stating that the decrease in depression is likely a result of psychological and social change rather than a hormonal change from menopause.

The clinical presentation of depression in later life individuals has been reported to be quite similar to younger adults (O’Brien & Thomas, 2003). Along with this is the evidence suggesting that cognitive impairments, while common in all adults with depression, (Austin, Mitchell, & Goodwin, 2001) are likely more severe in later life
persons with depression (O’Brien & Thomas, 2003). Much like that of younger adults, the etiologic factors believed to be at work in later life depression are genetics, biology, and life events. However, these take on quite different presentations in the later life individuals. For example, in later life individuals life stressors revolve more around bereavement, illness, and financial issues (O’Brien & Thomas, 2003), whereas in younger adults they are school, work, or living situation related (Friis et al., 2002).

Biological factors appear to affect depression in later life individuals more than any other grouping of people. Coronary artery disease (CAD), myocardial infarction (MI), and cerebrovascular disease (e.g., stroke and hypertension) have all been implicated in increased cases of depression as well as poorer outcomes (O’Brien & Thomas, 2003).

While the underlying factors related to the etiology of depression (e.g., psychology, genetics, & biology) are fairly similar throughout the lifespan, it appears that specific factors are more relevant or less relevant depending on the developmental group an individual is in. Therefore, just as it is important to study depression in the context of the overall human experience, it is just as important to study each group separately in order to identify group specific issues that are related to depression in order to adjust treatment to best help individuals.

As was noted earlier, more research is looking into how the brain is affected by depression. Regardless of age, recent research has begun to use neuroimaging techniques to investigate brain structure and function in an attempt to gain an increased understanding of how these two aspects may affect or be affected by depression. Neuroimaging studies have suggested various links between frontal lobe structure and/or function and depression (O’Brien & Thomas, 2003).
Parashos, Tupler, Blitchington, and Krishnan (1998) used a type of magnetic resonance imaging (MRI) that measured brain volume of numerous anatomical areas. They found various subcortical areas (e.g., caudate and putamen) that were significantly decreased in size in a group of 72 depressed inpatients with a mean age of 55.4 years (SD = 16.8). Furthermore, the frontal lobes of their sample were found to be smaller with increased age (Parashos et al.). Additionally, Kumar, Bilker, Lavretsky, and Gottlieb (2000) found normal asymmetry of the frontal lobes in their control group using MRI technology. However, the minor and major depressed groups had less asymmetry with the highest degree of symmetry being in the major depressed group.

The frontal lobes have been described as the executive of the brain, helping one to self regulate, plan, and execute behaviors (Malloy, Cohen, & Jenkins, 1998). O’Brien and Thomas (2003) asserted that research has highlighted the importance of the frontal and subcortical areas of the brain in regards to later life depression and conclude that structural differences in the brain may account for the cognitive deficits that remain after the clinical symptoms of depression have been assuaged. Executive functioning (regardless of life stage) is considered more fully in the following section.

Executive Functions

Executive function is a term used in neuropsychology that refers to a varied group of skills that are goal directed and future oriented (Bennetto & Pennington, 2003). Burgess (2003) asserted that the study of the executive functions is likely the newest area of investigation within the field of neuropsychology and are “probably the most technically and theoretically complex aspect of neuropsychological assessment” (p. 313).
Yet, a generally accepted definition of the executive functions has eluded scientists to date.

Alexander and Stuss (2006) have gone so far as to state that the “frontal functions, or impairment, have achieved the status of pornography: everyone knows them when they see them, but there is little agreement on their exact defining properties” (p. 192). Riccio (2006) stressed that there has been difficulty in operationally defining the executive functions as a frontal lobe problem due to the possibility that there may be other brain areas involved (e.g., one cannot do a number of tasks that utilize the executive functions without using a part of the brain that is considered non-executive). Burgess (2003) asserted that test "performance can be affected by dysfunction in other cognitive systems (e.g., memory, etc.)" (p. 313). Therefore, one is not necessarily purely investigating frontal lobe dysfunction. Rather, researchers have had to theorize what cognitive abilities are affected by damage to specific brain areas.

Most of the previous literature regarding the executive functions has come from correlational work (i.e., frontal lobe damage resulting in poorer performance on a given measure). However, a large majority of the measures used to assess the executive functions were not created to be used as such (Burgess, 2003). Alexander and Stuss (2006) have noted that planning, monitoring, sequencing, and inhibiting responses are typical impairments in individuals with frontal lobe lesions. They further describe frontal lobe lesioned individuals as “simultaneously unaware and distractible, irritable and apathetic, violent and passive, impulsive and perseverative” (Alexander & Stuss, p. 192). These individuals tend to have difficulties related to feelings of empathy, lack self-awareness, and are unable to emotionally regulate themselves (Alexander & Stuss).
Riccio (2006) asserted that planning, attention, abstract reasoning, self-regulation, sequencing, temporal orientation, organization, and even motor control are considered executive functions, depending on who is identifying them. Different definitions of executive functions result from each particular researcher. However, most acknowledge that these separate entities can be compartmentalized into approximately four main categories.

Lezak, Howieson, and Loring (2004) described the executive functions as consisting of volition, planning, purposive action, and effective performance. Malloy et al. (1998) added that self-monitoring the adequacy and correctness of one’s behavior, correcting and modifying one’s behaviors when situations change, and persisting in the face of distraction are also key components of the executive functions. Alexander and Stuss (2000) identified cognitive flexibility, goal setting, information processing, and attentional control as four inter-related, yet distinct, components of executive functions.

Lezak et al.’s (2004) and Alexander and Stuss’ (2000) conceptualizations overlap considerably in their subcomponent parts (e.g., both discuss planning of tasks, initiation of tasks, etc.). The following section describes the components set forth by Lezak and her colleagues (2004). Additionally, when overlap is present, Alexander and Stuss’ (2000) categorical system is discussed within the context of Lezak et al. (2004). For the purpose of the current study, executive functions will follow an integrated view of executive functions that utilizes aspects of both Lezak et al.’s and Alexander and Stuss’ (2000) conceptions of executive functions. Additionally, as not all identified areas of executive functioning are directly measured in isolation by tests of executive functioning, symptom identification (Burgess, 2003), by way of established measures, will be utilized.
Lezak et al. (2004) identified volition as one component of the executive functions. This “refers to the complex process of determining what one needs or wants and conceptualizing some kind of future realization of that need or want” (Lezak et al.). Additionally, an ability to form intentions, create goals, and initiate activities are also elements that fall into the volition component of executive functions (Lezak et al.).

Alexander and Stuss (2000) describe several of these activities in their goal setting component (i.e., the ability to initiate, plan, and carry out a task). According to Lezak and her colleagues (2004), “persons who lack volitional capacity simply do not think of anything to do” (p. 612). Alexander and Stuss (2000) report that there is specific frontal lobe brain structures relevant to self-awareness, for example, the right frontal lobe consisting of the hypothalamic limbic, posterior cortical, and cingulate associations. Problems associated with this area include feelings of apathy or indifference, deterioration of personal hygiene, loss of curiosity and/or self-awareness, decreased awareness of cognitive problems, loss if interest in previously enjoyed activities, and decreased social awareness (Sbordone, 2000).

It should be noted that there are no formal tests of volitional capacity and Lezak et al. (2004) suggest that assessment of volition be done by observation and self-report of day-to-day living by collateral interview. However, one can assess volition by way of neuropsychological measures via qualitative data such as whether or not an individual makes spontaneous speech, engages in conversation, asks questions, actively participates in testing by turning cards, moving beads, or exerts effort that is noticeable to the examiner (Lezak et al., 2004); claims of validity are common practice in
neuropsychological evaluations based on examinee effort. With this information, it could be argued that all neuropsychological instruments are tests of volition.

Planning is the second of Lezak et al.'s (2004) components of executive functioning. She and her colleagues note that while one must identify and organize steps and materials needed to carry out a task, one must also think of alternatives, make choices, and entertain different ways in which a plan might be carried out. Alexander and Stuss (2000) also describe many subcomponents of this area in their goal setting component. They stress the ability to initiate, plan, and carry out a task. Sbordone (2000) notes that those who exhibit "planning deficits are unable to formulate a set of plans to achieve a desired goal, evaluate the effectiveness of such plans, or select a particular plan to be executed" (p. 445).

Deficits in the area of planning exhibit themselves as a loss of abstract or conceptual thinking, disorganized behaviors and thoughts, inflexible thinking, poor planning and organization, decrease in goals or planning for the future, and social inappropriateness (Sbordone, 2000). Several instruments have been recommended as tests of planning. For example, mazes, visual search tests, and tower tests (Lezak et al., 2004; Sbordone, 2000).

Purposive action is the third component of Lezak et al.'s (2004) main executive function components. According to Sbordone (2000), "purposive action involves the patient's ability to initiate a particular plan while simultaneously ignoring irrelevant or competing needs, wants, or other plans" (p. 446). Lezak et al. (2004) elaborate by noting "the translation of an intention or plan into productive, self-serving activity requires the
actor to initiate, maintain, switch, and stop sequences of complex behavior in an orderly and integrated manner” (p. 621).

Alexander and Stuss (2000) discuss such concepts in their information processing area. This area includes the subcomponents of efficiency, fluency, and speed of processing. Efficiency and speed of processing can be thought of in terms of how fast one is able to maximally achieve a task (mental or physical) while expending the least amount of effort possible. Those who have difficulties in this area will exhibit distractibility, loss of initiative, problems processing external activities simultaneously, an inability to maintain ongoing cognitive and/or motor response sets, disorganization in thinking, emotional lability and impatience, discrepancies between expressed intentions and actions, difficulties in maintaining train of thought, circumstantial or tangential thinking, decreased ability to perform novel activities, and/or poor work habits/frequent terminations (Sbordone, 2000).

Several instruments (e.g., TOL\textsuperscript{DX} [Culbertson & Zillmer, 2005]; Tinkertoy Test [Lezak, 1982]) that assess purposive action and its subcomponents have been identified (Lezak et al., 2004). However, all require the “ability to initiate, maintain, switch, and stop sequences of complex behavior in an orderly and integrated manner” (Sbordone, 2000, p. 447).

The last area conceived by Lezak and her colleagues (2004) was that of effective performance. Effective performance refers to one’s ability to “monitor, self-correct, and regulate goal-directed behavior” (Sbordone, 2000, p. 447). Individuals with impairment in this area often exhibit erratic performances (a hallmark of executive dysfunction) as they are unable to self-correct mistakes or self-monitor their actions.
Impairments related to poor effective performance include perseveration (persistence of a same response even though its inappropriateness has been established), cognitive rigidity, decreased ability to complete or follow through on tasks, inability to recognize or rectify errors, poor work habits or employment history, clear problem solving difficulties, and an inability to utilize plans or strategies that were previously effective (Sbordone, 2000).

Any instrument that requires self-monitoring of behavior can be considered a test of effective performance. For example, fluency tests do not allow repetition and thus require an individual to self-monitor for success while an inhibition task would require one to refrain from some action in order to accomplish another. Lezak (1982) asserted that “constructional or drawing tasks in which more of the solution process is overt … can be particularly instructive in bringing to light how the patient errs, self-corrects or not, and monitors his performance” (p. 294) and are thus helpful in assessing effectiveness of performance.

Lezak et al. (2004) left out certain areas of executive functioning that have been incorporated by several scholars. For example, Alexander and Stuss’ (2000) area of attentional control has also been utilized by several others in their definitions of executive functioning (see Dagenbach & Carr, 1994; Denckla, 1996). According to Loring (1999), attention is a “process that enables an individual to engage in certain cognitive operations while ignoring others. Thus, attention involves a selective awareness or responsiveness. Attention also refers to the ability to focus and maintain interest for a given task or activity” (p. 24). Thus, attentional control refers to one’s ability to monitor and regulate one’s attention and is an important aspect of the executive functions.
There are various areas of attention that are utilized, for example, selective attention ("the capacity to highlight the one or two important stimuli or ideas being dealt with while suppressing awareness of competing distractions" Lezak et al., 2004, p. 34, also known as concentration) and sustained attention ("the capacity to maintain an attentional activity over a period of time," Lezak et al., 2004, p. 34). Alexander and Stuss (2000) also include self-monitoring and self-regulation under the attentional control heading in their conceptualization whereas Lezak et al. (2004) holds it as its own peripheral component of executive functioning. Self-regulation is characterized by decreased productivity and dissociation between an intention and an action (Lezak et al.).

Lezak et al. (2004) also fully described self-regulation as a "peripheral but equally important executive capacity[y]" (p. 612). Lezak et al. divided self-regulation into two main areas that coincide with Alexander and Stuss' (2000) main component of cognitive flexibility, productivity and flexibility and the capacity to shift. Riccio (2006) described several aspects of cognitive flexibility. She asserted that components such as perseveration (i.e., "persistence of the same response, even when it is shown to be inappropriate" [Loring, 1999, p. 125]), the ability to shift between sets (i.e., ability to adapt to a change in rules such as identifying items by color and then changing to shape), learn from mistakes, and working memory function (i.e., the ability to take in information, hold it briefly, process it, and then formulate a response [Groth-Marnat, 2003]) are all subcomponents of cognitive flexibility. With an emphasis on aspects such as set shifting and cognitive flexibility, the WCST (Heaton et al., 1993) is a common and often used measure to assess this area of executive functioning.
When these components are disturbed, one is inflexible in his/her thought patterns, often times attempting to solve a problem in the same manner repeatedly (i.e., perseveration). Such difficulties may be seen as an inability to shift one’s train of thought or a certain behavior in order to meet the demands of a given situation (Lezak et al., 2004). Problem solving is also impaired as one will not be able to organize adequately. This is problematic as one will not be able to inhibit one action in order to carry out another. It is interesting to note that Lezak et al. conceptualize the former as a self-regulation difficulty and consider the area a peripheral part of the executive functions. Instruments that assess productivity and flexibility and the capacity to shift have been reported as any test that reveals slowing in completion time and any “tests of abstraction that emphasize shifts in concept formation touch upon mental flexibility” (Lezak et al., p. 628).

Throughout Lezak et al.’s (2004) four components of executive functioning, the various subcomponents can, and do, overlap significantly. This highlights the difficulty in defining what pure executive functions are and what they are not. Riccio (2006) emphasized this difficulty by noting that although there are several overlapping subcomponents, they are all unique in some ways and related in others; and sometimes dependent on one another.

Therefore, overall, it is generally accepted that the executive functions serve to “facilitate adaptation to novel situations” (Burgess, 1997, p. 83) and maintain self-regulation (locus of control is transferred from external to internal control). They also consist of the abilities one needs in order to solve novel problems from beginning to end by way of identifying a problem, evaluating the problem, formulating a plan or goals in
order to solve the problem (e.g., establishing new behaviors and patterns of thinking and critically analyzing the appropriateness of each) and modifying them as necessary, terminating all actions when plan is complete, and finally storing information in order to access it later when the same or similar situations arise (Burgess, 2003; Sbordone, 2000).

Due to several difficulties in theoretically defining the executive functions, Burgess (2003) highlighted the most commonly reported symptoms of executive dysfunction. The top five symptoms reported by caregivers were planning (48%), Distractibility (42%), Lack of Insight (39%), Poor Decision-Making (38%), and Unconcern for Social Rules (38%). Following closely behind these were Apathy (27%), Perseveration (26%), Lack of Concern (26%), Poor Temporal Sequencing (25%), Aggression (25%), Impulsivity (22%), Know-Do Dissociations (21%), Poor Abstract Thinking (21%), and Inability to Inhibit Responses (21%).

Instruments used to assess executive functions and/or component parts are discussed in greater detail later in this chapter. However, it should be noted that there are not one-to-one relationships with any current conceptualization of executive functions and any neuropsychological measures. As noted previously, the major instruments used to assess executive functioning were not developed to specifically assess particular executive function symptoms. Rather, they came from research investigating individuals with frontal lobe damage who performed poorly on certain neuropsychological measures. While the executive functions have been described (i.e., theoretically defined), they have not been operationally defined.

Burgess (1997) points out that this is a major obstacle for researchers in this area. He adds that this is commonly due to the well-established link between executive
dysfunction and frontal lobe lesions. Heppner, Kivlighan, & Wampold (1999) noted that
to operationally define a construct, one must specify “the activities or operations
necessary to measure it in this particular experiment” (p. 39). However, without a one-to-
one relationship between an instrument and a component of the executive functions, an
operational definition is impossible.

Burgess (2003) stated that the ideal assessment of executive functions would
address all symptoms. However, he goes onto note that such an effort is impractical and
that formal measures do not yet exist for many executive dysfunction symptoms.
Moreover, "little is known about what many of the tests shown to be sensitive to frontal
lobe lesions are actually measuring... (... a theoretical problem that is far more complex
than it seems at first)” (Burgess, p. 306). Burgess (1997) warns that until executive
functions are clearly operationally and theoretically defined, allowing for identification
by way of psychological grounds alone, the topic will not receive equivalence with other
well established areas of neuropsychology.

Although not all measures associated with decreased executive functioning fit
nicely into Lezak et al.’s (2004) or Alexander and Stuss’ (2000) component systems,
several instruments have been identified as primary measures associated with frontal lobe
damage, and thus, executive dysfunction. These instruments will be discussed later in this
chapter.

_Brain and Executive Function Development_

At birth the human brain weighs approximately 368.5 grams (Dawson & Guare,
2004), which equates to just about one-fourth its adult weight of 1,300 to 1,500 grams
(Majovski, 1997). This difference in weight from birth to adulthood shows that as a child
grows and develops so does his/her brain. Various processes occur during brain development in the human being. Approximately 18 days after conception the neural tube is created. This event gives rise to the process of brain development that does not reach its culmination until many years later in late adolescence to early adulthood (Zillmer et al., 2001). Key points within brain development include cell migration/arborization, synaptogenesis, and pruning.

Cell migration is the act of neuronal travel along glial fiber tracks throughout the brain that function to aid the neurons travel to various destinations within the brain. Thus, at approximately the 18th week of gestation, most, if not all, cortical neurons have reached the location to which they will ultimately serve (Zillmer et al., 2001). At this point, dendrites begin to sprout, creating various spines throughout the brain enabling ultimate communication throughout the brain system. Although this action begins prenatally, a majority of the arborization and spine growth occurs postnatally from birth up to 18 months of age (Zillmer et al.).

As axons and dendrites develop, synaptogenesis also takes place. This is the act of synaptic development, where connections between the various axons and dendrites begin to appear. Zillmer et al. (2001) reported that increases in synaptic connections are responsible for the behaviors displayed in children. For example, increases in synaptic density within the frontal lobes during the first 12 months of life parallels the emergence of basic executive functions (e.g., delayed response performance; Zillmer et al.). It is interesting that the brain actually overproduces the number of neurons people need to function in our world. Through the pruning process, our own natural development actually eliminates unneeded neurons.
In the pruning process, large numbers of neurons are eliminated in order to facilitate structural and functional economy within the brain. Estimates have reported that as much as 40% of the brain's total cortical neurons can be pruned (Zillmer et al.). An interesting fact of pruning is that it is not random. Zillmer et al. (2001) asserted that it is a calculated shaping of the neural network that functions to eliminate weak and/or redundant neural connections and promote neural efficiency. Pfefferbaum et al. (1994) noted that prefrontal cortex pruning takes place from approximately age 5 through 16 years.

As these various processes take place, so too does the development of functional areas of the brain. For example, speech, language, vision, and executive functions just to name a few. Developmentally, it is generally believed that the executive functions develop early in life (i.e., infancy) and follow a protracted course through late adolescence (Dawson & Guare, 2004; Gioia et al., 2000; Zillmer et al., 2001). Gioia et al. (2000) further expanded upon this concept by noting that infants and toddlers display intentional self-control capacities that aid them in goal-directed problem solving. They warn, however, that while these skills become apparent at an early point in the developmental scheme, self-control is variable, fragile, and highly dependent upon the external environment in which the child lives before becoming more stable around the 18th through 30th month of life (Gioia et al.).

In a study of Finnish children aged 3-12 years, Klenberg, Korkman, and Lahti-Nuuttila (2001) used various tests of executive functioning to sequence the developmental course of executive function skills. According to their findings, they
began seeing developmental changes in executive functioning in their lowest age group (3-5 years).

In an inhibition task (e.g., ability to stop or modulate an ongoing task, particularly when there is a strong association involved), children as young as 6 and 7 years-old were showing relative proficiency. In a test of planning (ability to identify and organize steps and materials in order to carry out a task), monitoring (i.e., assessing progress), self-regulation (ability to maintain productivity), and problem solving (cognitive process requiring modulation and control of routine skills in order to complete a task) children were showing relative mastery of the task by 8 years of age. The last area of executive functioning assessed was that of fluency (aspects of verbalized speech related to ease of articulation, production of words, and use of sentence structure; Loring, 1999). Children in the sample were able to reach a relative mastery of Semantic Fluency (generating words from specific semantic categories [e.g., animal names or foods], which likely involves strategy and self-regulation skills) by age 10 and Phonemic Fluency (similar to Semantic, although here the child must generate words that begin with a specific letter) and Design Fluency (this is a visualmotor task that requires an individual to generate novel designs and likely assesses systematic production and use of strategy) by age 11, respectively (Klenberg et al., 2001).

There are caveats to the interpretation of findings such as this. While studies, such as Klenberg et al. (2001) have found rapid development in some areas at certain times, stages of executive function development are likely not clearly differentiated and various skills probably emerge before, during, and after peak developmental periods (Gioia et al., 2000; Klenberg et al., 2001).
Various studies have attempted to chart the development of the executive functions in childhood and have, to varying degrees, been successful. Although clear and more distinguishable changes in children have been reported, less research has tracked the development into and throughout adolescence (Anderson, Anderson, Northam, Jacobs, and Catroppa, 2001; Dawson & Guare, 2004; Gioia et al., 2000; Zillmer et al., 2001). Anderson et al. (2001) completed a study looking at children aged 11 years to 17 years and 11 months and primarily found flat trajectories of the executive functions throughout adolescence. They concluded that while development of executive functions is quite rapid through early and middle childhood they slow considerably during late childhood and adolescence (Anderson et al.).

Symptomatic Measures of Executive Function

As noted in the previous section, there can be great difficulty when choosing which neuropsychological test to use for a given situation. Many have been found to be highly correlated with brain impairment and serve to distinguish between brain impaired and non-impaired individuals. In regards to tests of executive functioning, the majority of measures were not specifically created to assess executive dysfunction (Burgess, 2003). Moreover, when choosing an approach to assessment, one must also consider many issues. For example, does one pick tests according to time, instrument psychometrics, or theory (Burgess)? None of the previous examples are without merit and none are devoid of fault.

Theoretically driven instrument identification will assess the domains that have been identified as being within a certain area of dysfunction. However, as is the case with the executive functions, there are myriad theories with no real consensus. Time is
questionable as to its justifiability. One can simply give as many tests as possible given a certain amount of time, but a shotgun approach is haphazard and may result in false-positive data (Burgess, 2003).

Psychometrics allows us to understand a measures merits and selection can be made upon such information. However, Burgess asserts that this is more difficult when assessing executive functions as measurement of something like strategy formation can be difficult. He elaborated by stating that this "can subvert [a] theory behind traditional psychometrics, and render the values a poor guide to a test's actual clinical utility" (Burgess, p. 307). Moreover, as one's level of performance alters, so does the construct validity of a test (Burgess), therefore making it difficult to simply use a test with the best psychometrics regardless of other factors. Instead, Burgess encourages one to use a test with sound psychometrics with all things being equal, "however, all things are unlikely to often be equal" (p. 307).

There are many instruments available for an individual to use to assess the various facets of the executive functions. Following is a description of some of the most popular tests of executive functioning. This list is in no way meant to be exhaustive; however, it is comprised of those most often utilized in research studies investigating the executive functions.

Controlled Oral Word Association Test (COWA; Spreen & Strauss, 1998) – The purpose of this measure is to assess an individuals spontaneous production of words that begin with a certain letter, most commonly F, A, and S (Golden, Espe-Pfeifer, & Wachtsler-Felder, 2000; Johnstone, Holland, & Larimore, 2000). This measure is known
differentiate individuals with left frontal or bilateral frontal lesions from non-lesioned individuals (Anderson, 1994; Lezak et al., 2004).

To administer the COWA, the examiner instructs the examinee to generate as many words as possible in 1 minute. The rules of the test are that the examinee cannot state proper nouns (e.g., peoples names) or create variations of the same word (e.g., fog and foggy; Johnstone et al., 2000). Other variations of the measure have been used, for example one can use different letters, such as C, F, L or P, R, W. Children’s versions have been put forth where examinees are asked to generate words that belong in a certain category (e.g., fruits, vegetables, or animals). However, Johnstone et al. warn to be cautious when using varied letters in the COWA as letters have differing frequencies in how they are used in words. Interpretation of this measure is based on normative data. The COWA is believed to assess areas of the dorsolateral prefrontal cortex, inhibition, perseveration, and may be more left frontal lobe sensitive (Malloy et al., 1998).

The Tower of London – Drexel University (TOL\textsuperscript{DX}): 2\textsuperscript{nd} Edition (Culbertson & Zillmer, 2005) – The TOL\textsuperscript{DX} is based on the original Tower of London test described by Shallice (1982) that was designed to assess adult patients with frontal lobe injuries and is widely recognized as a test of planning ability. The actual test is comprised of two boards (one for the examiner and one for the examinee) with three pegs on each board arranged from shortest to longest. Three colored beads (red, blue, & green) are arranged on the pegs in a specific manner.

The object of the test is for the examiner to set up his/her board with the beads in a certain configuration and then ask the examinee to “[n]ow make one on your board in as few moves as you can” (Culbertson & Zillmer, p. 11). The examiner is responsible for
keeping time of how long it takes the examinee to complete the task (i.e., making the examinee board look just like the examiner’s board), counting number of moves, and ensuring that the examinee does not violate either of two rules. The first rule is that the examinee may not place more beads on a peg than the peg will hold (the longest peg can hold three beads, the middle peg can hold two, and the shortest bead can hold one bead; Culbertson & Zillmer). The second rule is that the examinee is forbidden to move more than one bead off a peg at a time (Culbertson & Zillmer).

Culbertson and Zillmer (2005) noted that they made several modifications in the administration and scoring in order to improve “clinical utility, applicability, and standardization” (p. 1) of the instrument. In order to maintain novelty of the task, the authors removed the previous administrative technique of repeating trials of failed attempts. Alterations were also made in order to make the test useful across the lifespan. The authors note that the TOLD $^{DX}$ is meant to assess the frontal lobes’ executive ability of planning. Culbertson and Zillmer elaborated by stating that “systematic interaction with other cortical and subcortical regions are centrally involved in executive planning…” (p. 1). Insult to these areas of the brain often result in decreased executive planning abilities. Culbertson and Zillmer asserted “the value of the TOLD $^{DX}$ assessment as a measure of executive planning relates to its novel and anticipatory problem-solving demands” (p. 2). Successful completion of the tasks is reportedly indicative of expected abilities to plan sequentially, execution of repetitive moves, and monitoring (Culbertson & Zillmer).

Wisconsin Card Sorting Test (WCST; Heaton, Chelune, Talley, Kay, & Curtiss, 1993) – “The WCST is often referred to as a measure of ‘frontal’ or ‘prefrontal’ functioning” (Heaton, Chelune et al., p. 1) as it is believed to assess areas of the brain
related to the dorsolateral prefrontal cortex (Malloy et al., 1998). The main function of this test is to evaluate one's ability to form abstract concepts, shift and maintain a set (i.e. a particular sequence), and make use of feedback (Lezak et al., 2004; Spreen & Straus, 1998). Malloy et al. (1998) assert that the WCST “has been considered the premier test of executive functions for many years. It taps a variety of executive abilities, including maintenance of task set, flexibility in response to feedback or changing circumstances, and perseverative tendencies” (Spreen & Straus, 1998, p. 584).

The WCST requires an individual to sort cards that consist of varying forms (crosses, circles, triangles, or stars), colors, (red, yellow, blue, or green), and numbers of the figures (one, two, three, or four; Heaton et al., 1993) onto one of four stimulus cards. The stimulus cards are set in front of the examinee who then must match her/his cards to the stimulus cards according to color, form, or number. The examinee is never told how exactly to complete the test, only whether their choice was correct or incorrect according to the sorting principle in effect.

The first sorting principle is color (Spreen & Straus, 1998). Thus, after the examinee has placed 10 cards down matching the color to the stimulus card (10 correct responses), the examiner then changes the rule, without warning, to form (i.e., shape; Spreen & Straus). The examinee must again utilize the feedback in order to figure out the new sorting principle (Heaton et al., 1993). According to Spreen and Straus (1998), this procedure continues on until the examinee has completed the principles of color, form, number, color, form, number, or until all 128 cards have been used. Scoring dimensions of the WCST are: Trials Administered, Correct-Incorrect, Perseverative-Nonperseverative, Number of Categories Completed, Trials to Complete First Category,
Percent Perseverative Errors, Failure to Maintain Set, Percent Conceptual Level Responses, Learning to Learn, and Percent Errors (Heaton et al., 1993).

Trail Making Test (TMT; Reitan & Wolfson, 1993) – This measure has proven to be quite useful in assessing “visual conceptual abilities, cognitive flexibility, set shifting [changing concept of a task], sequencing ability, visual-motor tracking, and visual-spatial functioning” (Golden et al., 2000) in the dorsolateral prefrontal cortex and the frontal eye fields (Malloy et al., 1998). It consists of two parts, A and B, with the former measuring cognitive processing speed and the latter assessing complex cognitive processing speed and mental flexibility (Malloy et al.). Malloy et al. also noted that the Trail Making Test (Reitan & Wolfson, 1993) is a test of abstraction, set maintenance, and cognitive flexibility.

Part A consists of numbers from 1 to 25 with circles around the numbers. The examinee is instructed to draw a line from 1 to 2, 2 to 3, and so on to the end as fast as possible. If the examinee makes a mistake, the examiner is to point out the error and redirect the examinee to begin at the number/circle previous to the error. Time is recorded in seconds. Similar to Part A, there are circled numbers on Part B. However, there are also circled letters as well. Thus, the examinee is instructed to draw a continuous line from circle to circle, but this time the sequence must be number to letter (e.g., 1-A, A-2, B-3, C, etc.).

Errors are noted on the TMT; however, no penalty is assessed, as increased errors will result in increased time to complete the task, thus affecting one’s score. Broshek and Barth (2000) point out that slower times on the trails tests can indicate cerebral dysfunction, potential problems related to the anterior frontal lobes, slow cognitive
processing speed, visual scanning deficits, decreased motivation/effort, and depression with psychomotor deficits (especially on Part B), and memory (Golden et al., 2000).

Stroop Color and Word Test (Stroop; Golden & Freshwater, 2002) – The Stroop is a fairly quick and easily administered measure that assesses one’s ability to shift perceptual set (a mental readiness to see one thing based on expectations, experiences, emotions, or assumptions) to conform to changing stimuli and resist habitual responding in favor of more unconventional responding (inhibition; Spreen & Straus, 1998) in the orbital prefrontal cortex (Malloy et al., 1998).

One part of the Stroop requires an examinee to read words (i.e., the words RED, BLUE, & GREEN) from a list. Another has the words printed in different colors (e.g., the word RED is printed in blue colored ink). In all, there are three pages of required activities, a word page, color page, and a color-word page. On the word page, the examinee is informed that he/she must read the words as fast as possible. On the color page, four X’s are set in columns in one of the three colors previously noted. The examinee is required to name the color of the four X’s as fast as possible. For the color-word page, examinees are told to “... name the color of the ink the words are printed in, ignoring the word that is printed for each item” (Golden & Freshwater, 2002 p. 4). One needs to work as fast as possible while still attempting to make correct responses.

The use of these and other assessments to measure executive function deficits has grown tremendously in recent years. The next section explores that line of scholarly inquiry, especially as it relates to the association between the executive functions and depression. The primary focus is on research that has both shown executive deficits in those with depression as well as studies that have provided conflicting information.
Research Literature Regarding Executive Functioning and Depression

One group of researchers that have investigated the relationship between executive functions and depression is Paradiso, Lamberty, Garvey, and Robinson (1997). They noted that although much research has been done investigating neurocognitive impairment in depressed individuals, it has been confounded by inconsistent applications of the term depressed. Paradiso et al. asserted that much of the previous research has combined both unipolar and bipolar type depressed individuals into one experimental group and simply labeled them all as depressed. Furthermore, according to Paradiso et al., the previous literature has looked at individuals while they were experiencing a depressive episode.

In an attempt to distinguish any potential differences between unipolar and bipolar type individuals, as well as explore whether or not remission of depressed state affects neurocognitive impairment, Paradiso et al. examined the neurocognitive impairments in euthymic phase unipolar (age $M = 55.9$, $SD = 11.3$), bipolar (age $M = 57.0$, $SD = 11.3$), and healthy comparison groups (age $M = 49.8$, $SD = 11.5$). The number of years of education was comparable across groups (unipolar $M = 12.2$, $SD = 3.0$; bipolar $M = 13.5$, $SD = 2.7$; and healthy comparison $M = 13.5$, $SD = 1.9$). Furthermore, citing previous literature that there are gender differences in “brain organization of cognition and emotion” (Paradiso et al., p.748), only males were investigated. A total of 65 individuals were enrolled in the study, of which 20 participants had a history of unipolar depression and 11 participants had a history of bipolar disorder. All participants with a history of neurologic and/or active substance abuse issues were excluded from their study (Paradiso et al.).
All participants completed the Trail Making Test (TMT) Parts A and B, CERAD word list memory test, Stroop Color and Word Test, and the Digit Symbol subtest of the Wechsler Adult Intelligence Scale-Revised (Paradiso et al., 1997). Analyses of the data indicated that the neuropsychological measures revealed group effects where unipolar depressed individuals performed more slowly than the healthy comparison group on the TMT Part A, Stroop Color and Word Test, Digit Symbol subtest, and the CERAD word list memory test at a statistically significant level \((p < .05)\). However, the only measure in which the unipolar depressed individuals performed worse than the bipolar group at a statistically significant level \((p < .05)\) was on the TMT Part B. Paradiso et al. acknowledged that the healthy comparison group typically performed better on all measures in their study, however, no differences reached statistical significance between the healthy comparison group and the bipolar disorder group.

Various conclusions reached by Paradiso et al. (1997) are of importance. For example, they concluded that the cognitive deficits that have been associated with depression continue on after symptom remittance. Furthermore, it also appears that bipolar and unipolar types of depression are not unitary disorders and should be investigated separately. However, a major caveat should be noted with regard to Paradiso et al.'s work. All but three individuals in the experimental sample were taking medications (Lithium; \(n = 12\), Antiepileptics; \(n = 8\), Tricyclic Antidepressants; \(n = 4\), Selective Serotonin Reuptake Inhibitors; \(n = 7\), Bupropion or trazodone; \(n = 11\), or benzodiazepines or buspirone; \(n = 6\)) at the time of testing.

According to DiMicco and Gutierrez (2003) and Vasko and Gutierrez (2003), several of these medications have sedative/hypnotic or confusional effects upon cognitive
functioning. Paradiso et al. (1997) attempted to analyze and explain the effects of these medications with post-hoc analyses and found that there were no statistically significant differences between the medicated and non-medicated groups. However, this goes against conventional wisdom (e.g., DiMicco & Gutierrez, 2003; Vasko & Gutierrez, 2003) and research (Elliot, 1998) that says otherwise. Additionally, such results would need to be replicated before investigators could accept that tricyclic antidepressants, benzodiazepines, and trazodone have no neurocognitive effects.

Another example of work that has investigated the connection between depression and neuropsychological functioning is that by Landrø, Stiles, and Sletvold (2001). Landrø et al. recently examined a sample of depressed individuals who were identified via a structured clinical interview and a depression inventory.

All individuals were screened and participants were excluded if they were taking an antidepressant, lithium, or neuroleptic medications, or were actively abusing alcohol or drugs (Landrø et al., 2001). A total experimental sample of 22 individuals (82% women) and thirty healthy comparisons (73% women) were enrolled based on their report of no psychiatric illnesses and a score of at least 9 or below on the Beck Depression Inventory (Landrø et al.). All participants completed a test of motor function (two finger tapping task), selective attention (choice reaction time test), mental flexibility (Trail Making Test [TMT], Parts A and B), visuomotor tracking (Kimura Recurring Recognition Figures Test), verbal fluency (Controlled Oral Word Association test), visuospatial function (Block Design subtest from the original WAIS), and the Similarities subtest from the original WAIS.
Statistical analyses, including MANCOVA’s, ANOVA’s, t-tests, and Chi squares, produced a variety of interesting results. Most notable was the significant overall group differences between the depressed and non-depressed groups in neuropsychological test performance (Landro et al., 2001). More specifically, there were statistically significant group differences in selective attention, working memory, verbal long-term memory, and verbal fluency (Landro et al.). Quite interesting was the lack of findings regarding mental flexibility (TMT). Inconsistent with much of the young adult literature regarding executive functioning, Landro et al. showed no differences between the depressed and non-depressed groups on the TMT. While some studies have shown robust differences (Paradiso et al., 1997), others (Grant et al., 2001)—including Landro et al., (2001)—have shown no differences, thus adding to the debate of whether or not the executive skill of mental flexibility is affected by depression.

While Landro et al., (2001) matched participants well in regards to age, education, and estimated intelligence, there are important pieces missing from their report. For instance, although the mean age of 40.6 ($SD = 10.7$) was reported for the depressed group, this suggests that over 95% of the sample was approximately between the ages of 30 and 50 years-old. Some have argued that this age group is considered middle-aged (Paykel & Kennedy, 2003) and may possess different neurocognitive profiles than young adults. According to Landro et al., (2001), the results of their study match the literature investigating neurocognitive differences in depressed and non-depressed individuals. Although there were limitations and inconsistencies in the Landro et al. study, the overall results were consistent with the literature and provide support for the hypothesis that there are neurocognitive deficits associated with depression.
In another study looking at the neurobehavioral effects of depression, Merriam, Thase, Haas, Keshaven, and Sweeney (1999) used previous neuroimaging research that showed decreased prefrontal cortical blood flow and metabolism in depressed individuals to hypothesize that such an event would lead to neurocognitive decreases. The participants ranged in age from 18 to 50, were not medicated, and were excluded if they had a history of electroconvulsive therapy, neurological disorder, head injury, or substance dependence (Merriam et al.). Unipolar major depressed individuals (five with psychotic features), individuals with schizophrenia, and a healthy comparison group made up the participant pool.

An ANCOVA (with age and IQ serving as covariates) showed that age did not impact WCST performance (the depressed group was appreciably older than both the schizophrenia and healthy comparison groups), while IQ impacted all groups (Merriam et al.). It is noteworthy that the depressed group performed significantly poorer than both the healthy comparison and schizophrenia groups on all aspects of the WCST \((p < .001)\) except the failure to maintain set task (i.e., participants were able to maintain a task once it was figured out; Merriam et al.). Golden et al. (2000) noted that deficits in this area are typical of frontal dysfunction (i.e., memory difficulties or increased distractibility).

Merriam et al. (1999) warn that their findings should be viewed with some caution as their depressed group consisted mainly of ambulatory outpatients and the schizophrenia group was primarily acutely psychotic inpatients. However, the significant deficits found on the WCST in the depressed group definitely warrants further investigation as the depressed group’s performance was so poor across the WCST subtests which are a typical mainstay of any test of executive functions.
In another study utilizing the WCST, Channon (1996) tested Dehaene and Changeux's (1991) theory of the WCST. Dehaene and Changeux's hypothesized that evaluating too many sources of failure on the WCST and neglecting the reward signal (being informed one is correct), or failing to alter behavior in response to feedback (not changing a strategy when informed a response was incorrect) are the reasons for errors on the WCST.

A total of 56 college students participated by completing the Beck Depression Inventory, and based on the score, were asked to participate in a study looking at reasoning (Channon, 1996). This resulted in 28 individuals assigned to two groups, a dysphoric group (male = 6, female = 22, age; $M = 22.3$, $SD = 4.2$) and a healthy control group (male = 7, female = 21, age; $M = 21.0$, $SD = 2.8$). Both groups were administered the BDI twice; once during a classroom lecture, which resulted in an invitation to participate in the study, and another just before testing (dysphoric group; $M = 20.0$ & 17.8; control group; $M = 2.3$ & 1.6; Channon). No mention was made regarding antidepressant medication use by the authors. Other inclusion criteria included average or above vocabulary scores on the Wechsler Adult Intelligence Scale-Revised (WAIS-R) and no significant physical or psychiatric illness (other than depression or dyslexia). Statistical analyses ($t$-tests) indicated that the groups did not differ significantly in vocabulary or age (Channon).

The results from Channon's (1996) study revealed that dysphoric participants took more trials to carry out a task and made significantly more perseverative and non-perseverative errors than the comparison group. Previous data has typically looked at MDD instead of a subthreshold depression (i.e., dysphoria).
Overall, Channon’s (1996) work contributed to the body of literature suggesting that depressed individuals suffer deficits on the WCST. More importantly, the finding that dysphoric individuals performed worse than healthy comparisons suggests that even lower levels of depression may impact frontal lobe function and the executive functions. Additionally, Channon (1996) has also provided evidence supporting Dehaene and Changeux’s (1991) theory of the WCST that failing to alter behavior in response to feedback are the reasons for error on the WCST. Adding to the executive function belief that individuals with decreased executive functions cannot self-regulate. Channon (1996) further proposed that while fatigue has been suggested to affect depressed individuals performance on tests, his study found no significant differences between the two groups over the duration of the testing (Channon).

While Channon (1996) asserted that many aspects of his study support and further the connection between depression and executive dysfunction, there are some limitations that went unmentioned by the author. For example, the gender discrepancy should have been addressed by Channon. As noted earlier in this paper, there are many more women who are diagnosed with depression than men. However, without limiting the results of his study to women he may have erred in regards to the generalizability of the results. Additionally, Channon chose to use a sample of dysphoric individuals instead of a clinically depressed sample. It was noted that previous research has found similar deficits in those with clinical depression as in those with dysphoria (see Hartlage, Alloy, Vazquez, & Dykman, 1993; Vredenburg, Flett, & Krames, 1993), although the clinical samples with more severe levels of depression tended to have more severe executive deficits (Channon, 1996). Instead of viewing this as a limitation, Channon asserted that
his results support the hypothesis that depression, even mild depression, impairs executive functions.

Based on literature that there are executive function deficits in depressed individuals (Paradiso et al., 1997), Channon and Green (1999) recently investigated whether or not providing individuals with a strategy aid would affect their performance on tasks related to executive functioning. It was hypothesized that strategy aids (i.e., hints) would help depressed individuals make up for the reported deficits on many traditional neuropsychological tests (Channon & Green).

Two groups of participants (depressed and healthy comparisons) were assigned to strategy aid and no strategy aid groups and administered three measures related to executive functioning (memory for categorized words task, response suppression task, & multiple scheduling task; Channon & Green, 1999). The depressed participants were identified via a structured clinical interview and two measures of depression (one clinician rated form and the other self-report). Participants who scored more than one standard deviation below normal on the spot the word test, had a relevant history of physical or neurological illness, mania, hypomania, other psychiatric illness, alcohol/substance abuse dependence issues, or had electroconvulsive therapy within the previous 12 months were excluded (Channon & Green).

Regarding the testing procedures, all participants received the same introductory instructions for all three of the executive function measures. However, the strategy aid group was given further instructions that served as hints as to how to manage the test in a more efficient and productive manner (Channon & Green, 1999). For example, on the response suppression task, individuals were required to finish a sentence stem with a one-
word non-contextual response as quickly as possible. Additionally, the word had to be unrelated to the sentence as much as possible. The strategy aid group was further “... told to imagine that they were on a [vacation] and to think of words relating to this to complete the sentence” (Channon & Green, p. 164).

Channon and Green’s (1999) analyses of their data revealed no statistically significant differences in regards to participant age, years of education, or spot the word test performance. Using analyses of variance, statistically significant differences were found regarding group performance on all three of the measures utilized in this study, with non-depressed individuals performing better than depressed individuals (both strategy aid and non-strategy aid, $p = .006$; Channon & Green). Furthermore, Channon and Green asserted that a qualitative (i.e., subjective) examination of participant performance suggested that depressed individuals failed to spontaneously utilize the performance strategies and when they did, it did not necessarily equate to improved performance.

Although support was not found for strategy aids assisting depressed individuals erase the performance deficits on neurocognitive tasks, Channon and Green (1999) asserted that strategy aids in the form of hints might still prove beneficial in the everyday treatment of depression. However, their study suggested that information alone does not appear to eradicate deficits. Instead, the authors suggested that hints, along with practice and training, may be helpful (Channon & Green). Furthermore research into this hypothesis will have to be completed in order to gain a fuller understanding of what may and may not be beneficial to depressed individuals performance on measures of neurocognitive functioning.
Remaining consistent with the previously described research, Grant, Thase, and Sweeney (2001) investigated a large group of individuals throughout the young adult to midlife age range. According to Grant et al., their goal was to comprehensively assess a younger adult group of outpatient non-medicated individuals with major depression utilizing various neuropsychological measures of attention, memory, abstract problem solving, and motor skills as well as with a computerized battery of cognitive tests. Without a direct hypothesis stated, these authors ultimately provide an exploratory look at the relationship between the previously mentioned neuropsychological constructs and participants with major depression.

A total of 123 outpatient individuals (age; $M = 39.0$, $SD = 10.4$) who met DSM-IV criteria for MDD without psychotic features were compared to a group of healthy comparison participants ($n = 36$; age; $M = 40.2$, $SD = 9.7$). Interestingly, only 36 participants were involved in the comparison group, equating to almost 3.5 times less individuals than were used in the experimental group. However, there was no explanation provided by the authors for this.

The overall sample was established by use of a structured clinical interview, a clinician rated and a self-report depression scale, and each participant was assigned a Global Assessment of Functioning (GAF) score (Grant et al., 2001). Of the outpatient depressed participant group, two were also diagnosed with social phobia, one was concurrently diagnosed with anorexia, and one with substance abuse issues (Grant et al.). While the number of persons with these disorders were small (2, 1, & 1, respectively), the fact that the comparison group was so small to begin with presents these individuals as potential confounds to the Grant et al. study as any one of these diagnoses can
deleteriously affect the ultimate results and any potential identification of depressions affects upon the executive functions. Additionally, depressed participants were required to be medication free for at least 28 days prior to the testing while the healthy comparison participants were expected to be completely medication free.

Results of $t$ and chi square tests indicated group differences between the depressed group and the non-depressed group in the domain of executive functions but not on tests of attention, motor, or memory (Grant et al., 2001). In the executive function domain, the depressed group showed particular impairment on the WCST. Specifically, deficits were found in areas of number of categories completed, perseverative responses, perseverative errors, and failures to maintain set (Grant et al.). It is important to note that all other tests of executive function (Trail Making Test B, Halstead-Reitan Categories Test, & the COWA [FAS version]) revealed no group differences (Grant et al.). Grant et al. further reported that the computerized assessments, the Cambridge Neuropsychological Test Automated Battery (CANTAB), also revealed no group differences on tests of executive function or memory (Grant et al.).

Overall, the Grant et al. (2001) study contrasts with some previous research studies that have reported deficits in depressed younger individuals. It is important to note that the mean age of individuals participating was 39 years. However, Grant et al. defined their sample as young adults, thus attempting to show deficits in executive functioning in depressed individuals other than mid-life and elderly individuals. Some scholars have operationally defined young adulthood as those between the ages of 19-23, not 39 (Lewinsohn & Seeley, 2003). Additionally, Grant et al.'s (2001) sample appears to be looking at milder cases of depression, which may ultimately account for some of the
discrepant (non-significant) findings regarding executive functioning in their sample. Moreover, the results of this study have to be generalized with caution. The presence of other mental health diagnoses among the depressed group is problematic and should have been addressed theoretically by the authors. However, as such information was not addressed, further research may need to be done in these other areas of mental health functioning in order to fully understand their impact on the executive functions.

Purcell, Maruff, Kyrios, and Pantelis (1997) also investigated the neuropsychological deficits in depressed young adults. In their study, 20 individuals (19 outpatients and 1 inpatient at time of testing) who met Anxiety Disorders Interview Schedule for DSM-IV criteria for unipolar depression were assessed. Twenty depressed individuals (male = 8; female = 12; age; $M = 37.5$, range = 18-52 [standard deviations not reported by authors]; Purcell et al.) and all participants completed the National Adult Reading Test (NART; a vocabulary test that correlates with IQ scores) in order to estimate intelligence level. Seven of the 20 participants reported previous hospitalizations due to their depression and 12 were taking antidepressant medication (medications not named by authors) at the time of testing. It is noteworthy that the participants’ scores assessing depression revealed a moderate level of depressive symptoms. Participants were excused from the study if they endorsed a history of hypomania, neurological and/or medical illness, or major alcohol/drug abuse (Purcell et al.). A healthy comparison group ($n = 20$) was matched to the depressed group on sex, age, years of education, and NART score (Purcell et al.).

All participants completed tests of executive functioning (spatial span, spatial working memory, Tower of London planning task, & intradimensional/extradimensional
[ID/ED] set shift task; shifting from a reinforced condition to a previously irrelevant condition) and visual memory tasks (delayed matching to sample, spatial recognition, pattern recognition; Purcell et al., 1997). Statistical analyses, that included ANOVA’s, t-tests, chi square tests, Pearson’s product moment correlation coefficients, and Spearman’s correlation coefficients, revealed results that are discrepant with previous literature.

Specifically, no statistically significant differences were found on most of the measures of neuropsychological functioning (Purcell et al., 1997). Tests that produced statistically significant differences between the two groups were TOL time of single moves and the ID/ED set shifting task with the healthy comparison participants outperforming the depressed group. Purcell et al. noted that they found no statistically significant differences between the medicated and non-medicated participants; however with an uncontrolled 12:8 ratio, of medicated to non-medicated, their assertion should be interpreted cautiously.

Purcell et al. (1997) reported findings that support motor speed and attentional set shifting deficits in depressed individuals. However, they were unable to replicate past findings of executive function deficits in their population. It should be noted that Purcell et al.'s depressed participants were all outpatients, except one, and scored in the moderately depressed range on a measure of depression. Based on this, Purcell et al. hypothesized that non-hospitalized depressed individuals may have more intact cognitive abilities than those needing hospitalization for treatment of their depression. Thus, the authors argued that hospitalized and non-hospitalized depressed individuals may possess different neurocognitive profiles.
Another aspect of Purcell et al.’s (1997) work that limits its generalizability is their definition of “young patients.” They argue that they have provided evidence that young adults may not necessarily possess the same executive function deficits that middle aged and elderly individuals do. However, the mean age of the Purcell et al. group was 37.5 years with ages ranging from 18 to 52, an age range that only places some of the participants into the “young adult” category according to some authors (see Paykel & Kennedy, 2003). Purcell et al. (1997) note that future research should examine the depressed young adult in order to determine and understand the relationship between neuropsychological functioning and depression.

Porter, Gallagher, Thompson, and Young (2003) also investigated young adults and the potential neurocognitive deficits associated with depression. They asserted that there have been inconsistent findings in neurocognitive impairment research with depressed individuals. They attributed these inconsistencies to the wide variety of participants in various studies in regards to “age, hospitalisation (sic), severity and subtype of depression, and most importantly, the effect of psychotropic medication” (Porter et al., p. 214). In order to address such issues, they hypothesized that non-medicated depressed individuals would perform more poorly than a well-matched healthy comparison group.

The Montgomery-Asberg Depression Rating Scale (\(M=28.9\)) and the Beck Depression Inventory (\(M=27.9\)) were used to identify participants for the depressed group. Individuals for the non-depressed comparison group were required to obtain scores of less than seven on the Beck Depression Inventory. A total of 44 individuals in each group (age; \(M=32.9, SD=10.6\) for the out-patient depressed group and \(M=32.3\), \(SD=10.6\) for the comparison group).
SD = 11.4 for the control group) participated in the Porter et al. (2003) study. Participants were matched for age, gender, premorbid IQ (via the National Adult Reading Test), years of formal education, and season of testing; females were also matched for phase of menstrual cycle.

All participants completed the Digit Symbol Solution Task, Rey Auditory Verbal Learning Test, Controlled Oral Word Association Test, ‘exclude letter’ fluency test, and vigil continuous performance test. Furthermore, the Paired Associates learning, pattern recognition, spatial recognition, simultaneous/delayed matching to sample, spatial working memory, and the Tower of London were all completed from the Cambridge Neuropsychological Test Automated Battery (CANTAB; Porter et al., 2003).

The results of Porter et al.’s (2003) data revealed that most of the depressed individuals (68%) were experiencing their first episode of depression, had never taken antidepressant medication (58%; others were medication free for at least 6 weeks prior to testing), and had never received electroconvulsive therapy (100%). On tests of neurocognitive functioning, ANOVA’s showed depressed participants performed poorer than a group of healthy comparison individuals on aspects of verbal and visualspatial learning and memory as well as sustained attention and executive functioning. (Porter et al.). By executive functioning, Porter et al. are referring to verbal fluency, spatial working memory, and planning. Overall, Porter et al. suggested that their work indicates that there are significant neurocognitive deficits in young adult unipolar depressed out-patient individuals. It is noteworthy that the most robust findings were in the area of attention and executive functioning. Porter et al. asserted that while large effect sizes for executive functions were present, participant performance correlated with level of depression.
Porter et al. suggested that the executive functions may be quite sensitive to depressive symptoms and that even the slightest amount of depressive symptomatology may substantially affect one's executive functioning.

Since Porter et al.'s (2003) results cannot be attributed to the effects of psychotropic or central nervous system active medications their work lends solid support to the theory of prefrontal cortex dysfunction in depressed individuals. However, while the mean age of the depressed sample was 32.9 years, the participants' ages ranged from 19 to 61 years (Porter et al.). This age range has been defined by others as midlife (ages 40 to 60; Paykel & Kennedy, 2003) and later life/elderly (O'Brien & Thomas, 2003). Therefore, the results of Porter et al.'s (2003) research should be interpreted cautiously as executive deficits have been reported in middle aged and elderly individuals.

The research studies just discussed have all displayed various neurocognitive deficits in depressed individuals. However, in the process of doing so, these studies have not been able to operationally define the executive functions. Although this would appear an easy task, it is not (Burgess, 2003). The executive functions are the newest, and likely the least well understood, area of brain functioning research. Due to the infancy of executive functioning research, there is no shortage of explanations for what they are. Unfortunately, this has hindered research activities and understanding of the related phenomena that are the executive functions. As a result most research has been completed by assessing symptoms of executive dysfunction (e.g., difficulties related to planning, cognitive flexibility, inhibition, aspects of attention, etc.; Burgess). However, this is not a complete detriment to this area of study. A great deal of clinical utility has resulted from such work and has served several of the quotidian needs of clinicians (e.g.,
a better understanding of how frontal lobe impairment will affect functional ability).

Although work is not complete in this area, great utility can come from this system of symptom assessment. Symptom identification and assessment may lead to further theoretical clarity, and—ultimately—an operational definition.

Summary

Information has been presented discussing depression, executive functions, and research that has investigated the interaction of the two. The previous sections have established that depression has long been an issue for many people. Estimates suggest that some 121 million people worldwide suffer from the effects of depression (WHO, 2001). Not only is this of great concern for health care providers, it has also begun to affect the economy where estimates suggest that depression costs United States businesses some $33 billion per year due to decreased work productivity and absenteeism (Quinn, 2000). What is more, less than 25% of these individuals have access to effective treatment (WHO, n.d.) and over 80% of all depressed individuals will have a second, more severe, depressive episode later in their life (Barlow & Durand, 1999).

The executive functions have proven to be a difficult set of brain functions to operationally define (Burgess, 2003; Riccio, 2006). Too often, the definition simply depends upon who is carrying out the research (Riccio, 2006). Thus, the field has been left with a series of complex and often incomplete conceptualizations of the executive functions. Furthermore, the instruments used to assess the executive functions are not theoretically driven. Most were developed in order to assess functions that are not part of the executive functions at all. However, it has been found that these “hallmark” measures of executive functioning are able to detect deficiencies in tasks that are associated with
certain brain areas (e.g., the frontal lobes; Burgess, 2003). Symptoms have ultimately been identified by Burgess that have allowed scientists to infer executive dysfunction by correlation with brain damaged individuals. Thus, there has been a disconnect between those needing clinical utility from an instrument (i.e., clinicians) and those needing theoretical backing (i.e., academicians/scholars).

Age and medication status have been common confounds in the literature regarding the executive functions and depression. Definitions of young adult, middle age, and late life/elderly individuals have been erratic (e.g., see Channon, 1996; Grant et al., 2001; Purcell et al., 1997). This is problematic as one cannot generalize to young adults when one does not know what a young adult is. Furthermore, medication status has not been addressed as the major variable it is in previous research studies. Several investigations have analyzed medication status as a post-hoc variable (e.g., see Paradiso et al., 1997). This is insufficient as antidepressant medications affect brain chemistry and thus have the potential to affect neurocognitive functioning.

Purpose of Current Study

The purpose of the current study was to assess and compare the level of executive functioning in a medicated depressed, non-medicated depressed, and non-medicated non-depressed sample. In so doing, the present study assesses executive functions based on typical executive dysfunction symptoms (e.g., problems with planning, inhibition, cognitive flexibility, etc.; Alexander and Stuss, 2000; Burgess, 2003, Lezak et al., 2004). Furthermore, medication status is addressed by separating the depressed participants into medicated and non-medicated groups. Additionally, participants consisted of individuals
between the ages of 19 and 40. This age group allows for a purer understanding of the effects of depression on the executive functions in a younger adult population.

Main Hypotheses

Given the information presented throughout this review, the main hypothesis of the current study is that differences in performance will be found between the medicated depressed, non-medicated depressed, and non-medicated non-depressed groups on measures of executive functioning. More specifically:

1a. Differences will be found between the non-medicated non-depressed group and the depressed groups on The Wisconsin Card Sorting Test.
1b. Differences will be found between the medicated depressed group and the non-medicated depressed group on The Wisconsin Card Sorting Test.
2a. Differences will be found between the non-medicated non-depressed group and the depressed groups on the Trail Making Test (Parts A & B).
2b. Differences will be found between the medicated depressed group and the non-medicated depressed group on the Trail Making Test (Parts A & B).
3a. Differences will be found between the non-medicated non-depressed group and the depressed groups on the Tower of London, Drexel University (2nd Ed.).
3b. Differences will be found between the medicated depressed group and the non-medicated depressed group on the Tower of London, Drexel University (2nd Ed.).
4a. Differences will be found between the non-medicated non-depressed group and the depressed groups on the Stroop Color and Word Test.
4b. Differences will be found between the medicated depressed group and the non-medicated depressed group on the Stroop Color and Word Test.

5a. Differences will be found between the non-medicated non-depressed group and the depressed groups on the Controlled Oral Word Association test.

5b. Differences will be found between the medicated depressed group and the non-medicated depressed group on the Controlled Oral Word Association test.
CHAPTER III

METHOD

Participants

The participants in the current study were divided into three different groups. One group was a collection of individuals experiencing depression and taking psychotropic medication \((n = 15)\) while another group included individuals experiencing depression who were not taking any psychotropic medication \((n = 16)\). Individuals in these two groups were referred to the principal investigator from various clinical practices located in Northeastern North Dakota and Northwestern Minnesota. The third group comprised non-medicated non-depressed individuals \((n = 22)\) enrolled in undergraduate or graduate courses at a mid-sized university located in the upper Midwestern United States.

Participants were required to meet certain criteria in order to participate in the current study. All participants were required to have abstained from taking any central nervous system active medications for at least 28 days and have no history of psychosis or Schizophrenia. In addition to these basic requirements, there were also group specific requirements. The medicated depressed participants were required to obtain a score of 17 or higher on the Beck Depression Inventory-II (BDI-II; Beck, Steer, & Brown, 1996), meet criteria for Major Depressive Disorder based on the Mood Disorders module of the Structured Clinical Interview for DSM-IV-I (clinical version; SCID-I; First et al., 1997), and be taking either a Selective Serotonin Reuptake Inhibitor (SSRI) or a Monoamine
Oxidase Inhibitor (MAOI), but no Tricyclic medication due to known cognitive impairments associated with such medications (Vasko & Guiterrez, 2003). The non-medicated depressed group was also required to obtain a score of 17 or higher on the BDI-II, meet criteria for Major Depressive Disorder (MDD) based on the Mood Disorders module of the SCID-I (First et al.). Additionally, they must also be free from ingesting any psychotropic medication for at least 28 days prior to testing. The non-medicated non-depressed group participants were required to have scored a six or lower on the BDI-II, not meet criteria for MDD based on the Mood Disorders module of the SCID-I (First et al.), have no history of diagnosed depression, and be free of any antidepressant medications.

Overall, a total of 53 participants completed the BDI-II (Beck et al. 1996), the clinical interview assessing depressive symptoms based on modules A (Mood Episodes, Dysthymic Disorder, Mood Disorder Due to a General Medical Condition, & Substance Induced Mood Disorder) and D (Mood Disorders) of the SCID-I (clinical version; First et al, 1997), a measure of ADHD symptoms (Current Symptoms Scale–Self-Report Form; Barkley & Murphy, 1998), a demographic questionnaire, measures of group equivalence (Vocabulary subtest of WAIS-III [Wechsler, 1997] & Symptom Checklist-90-Revised [SCL-90-R; Derogatis, 1994]), and executive functions (Wisconsin Card Sorting Test [WCST; Heaton, Chelune, Talley, Kay, & Curtiss, 1993], Trail Making Test [TMT A & B; Reitan, 1996], Tower of London: Drexel Edition [TOL\textsuperscript{DX}; Culbertson & Zillmer, 2005], Stroop Color and Word Test [Stroop; Golden & Freshwater, 2002], & Controlled Oral Word Association test [COWA; Spreen & Straus, 1998]).
The participants in the current study (N = 53) resided in the upper Midwestern United States. Table 1 presents the frequencies and percentages of the participant demographics found in the present study. The participants in the current study consisted

Table 1. Participant Demographic Information.

<table>
<thead>
<tr>
<th></th>
<th>Medicated Depressed</th>
<th>Non-Medicated Depressed</th>
<th>Non-Medicated Non-Depressed</th>
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<tr>
<td></td>
<td>n(%)</td>
<td>n(%)</td>
<td>n(%)</td>
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<tr>
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<td>13(24.5)</td>
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<td>1(1.9)</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Single (Never Married)</td>
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<td>8(15.1)</td>
<td>12(22.6)</td>
<td>26(49.1)</td>
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Table 1 (cont.). Participant Demographic Information.

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<tr>
<td>of Depression, age at</td>
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Table 1 (cont.). Participant Demographic Information.

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<th>Non-Medicated Non-Depressed</th>
<th>All Groups Together</th>
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<td>n(%)</td>
<td>n(%)</td>
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of 44 women (83%) and 9 men (17%) who ranged in age from 19 years to 40 years with a mean age of 27.19 years and a standard deviation of 6.70 years. Of the 53 participants, 90.6% (48) self-identified as Caucasian/White, 3.8% (2) as African American, 1.9% (1) as First Nations Aboriginal, 1.9% (1) as Asian American, and 1.9% (1) as Asian.

Nearly half of the participants (49.1%, 26) reported that they were ‘Single (never married),’ while 32.1% (17) reported that they were ‘Married,’ 1.9% (1) ‘Partnered,’ 1.9% (1) ‘Separated,’ 7.5% (4) ‘Divorced,’ 1.9% (1) ‘Widowed,’ and 5.7% (3) ‘Engaged.’
Twenty-nine participants (54.7%) reported that their yearly household income was between $0 and $25,000, 20.8% (11) between $25,000 and $50,000, 13.2% (7) between $50,000 and $75,000, 7.5% (4) between $75,000 and $100,000, 1.9% (1) between $100,000 and $150,000, 1.9% (1) greater than $200,000.

A majority of the participants reported that they were right handed (92.5%, n = 49), while 3.8% (2) endorsed being left handed and 3.8% (2) endorsed being ambidextrous. In regards to years of education completed, 1.9% (1) of the individuals reported completing the 10th grade, 24.5% (13) graduated high school (12 years of education), 11.3% (6) completed 13 years of education, 24.5% (13) completed 14 years of education, 18.9% (10) completed 15 years of education, 15.1% (8) completed 16 years of education, 1.9% (1) completed 17 years of education, and 1.9% (1) completed 18 years of education.

While 88.7% (47) of the participants reported that they had never previously been diagnosed with depression, 11.3% (6) endorsed that they had. Of these six individuals who reported a previous diagnosis of depression, 1.9% (1) reported the age of first onset at 9 years of age, 1.9% (1) reported the age of first onset at age 17, 1.9% (1) reported the age of first onset at age 22, 1.9% (1) reported the age of first onset at age 23, 1.9% (1) reported the age of first onset at age 24, and 1.9% (1) reported the age of first onset at age 25. Furthermore, of these six previously diagnosed individuals, 5.7% (3) reported experiencing at least one previous depressive episode while 1.9% (1) reported experiencing two, 1.9% (1) reported experiencing twelve, and 1.9% (1) reported experiencing 20.
Of the 15 participants in the medicated depressed group, 5 (9.4% of total) reported taking Prozac, 3 (5.7% of total) Zoloft, 3 (5.7% of total) Paxil, 3 (5.7% of total) Wellbutrin, 2 (3.8% of total) Effexor, 1 (1.9% of total) Lorazepam, 1 (1.9% of total) Celexa, and 1 (1.9% of total) Xanax. The sum of these numbers is more than the number of medicated depressed participants as some participants were taking more than one medication.

Of the 53 total participants, 18.9% (10) were engaged in psychotherapy at the time of assessment (medicated depressed = 6, non-medicated depressed = 4). Of these ten individuals, 3.8% (2) had engaged in three psychotherapy sessions, 1.9% (1) had engaged in four, 1.9% (1) had engaged in five, 1.9% (1) had engaged in six, 1.9% (1) had engaged in ten, 1.9% (1) had engaged in 12, 1.9% (1) had engaged in 13, 1.9% (1) had engaged in 16, and 1.9% (1) had engaged in 27 psychotherapy sessions.

Two participants (3.8%) reported previously receiving a mental health diagnosis. One participant reported receiving a diagnosis of Obsessive-Compulsive Disorder (OCD) and the other reported a prior diagnosis of depression. Both participants were in one of the two depressed groups and the participant whom reported a previous history of OCD reported being symptom free and not in treatment at time of testing.

It should be noted that all participants (100%, N = 53) reported that they had never received electroconvulsive therapy (ECT), never been diagnosed with ADHD, never been hospitalized for a psychiatric condition, and never lost consciousness due to a blow to the head.
Materials

All participants were asked to complete a demographic questionnaire as well as various inventories and measures regarding concomitant psychological functioning, intelligence, depressive symptoms, and executive functions.

Measures of Group Equivalence

Psychological Functioning. Concomitant psychological functioning was assessed using the Symptom Checklist 90, Revised (SCL-90-R, Derogatis, 1994). The SCL-90-R is a 90 item measure that was developed to be a brief self-report that screens for a wide range of psychological problems and symptoms of psychopathology of psychiatric inpatients, medical patients, and non-patient individuals in a community for those aged 13 years and older (Derogatis). Derogatis asserted that the SCL-90-R is helpful in identifying "... current, point-in-time, psychological symptom status" (p. 5).

The SCL-90-R consists of nine primary symptom scales and three global indices. The primary symptom dimensions are: 1) Somatization, 2) Obsessive-Compulsive, 3) Interpersonal Sensitivity, 4) Depression, 5) Anxiety, 6) Hostility, 7) Phobic Anxiety, 8) Paranoid Ideation, and 9) Psychoticism. The three global indices include: (a) The Global Severity Index (GSI), (b) The Positive Symptom Distress Index (PSDI), and (c) The Positive Symptom Total (PST). The SCL-90-R utilizes a T-score distribution that ranges from 0-100 ($M = 50$, $SD = 10$; Derogatis, 1994).

Four groups were used to establish norms that consisted of adult psychiatric outpatients ($n = 1,002$; Males = 425, Females = 577, 32.9% non-White), adult nonpatients ($n = 974$; Males = 494, Females = 480, 14.5% non-White), adult psychiatric
inpatients (n = 423; Males = 158, Females = 265, 44.2% non-White), and adolescent nonpatients (n = 806; Males = 327, Females = 479, 0.3% non-White; Derogatis, 1994).

Derogatis (1994) reported that the internal consistency coefficients for the nine primary dimensions were obtained from two separate studies, first of which was research conducted by Derogatis, Rickels, and Rock (1976). The group reported internal consistencies ranging from .77 to .90 and test-retest coefficients that ranged from .78 to .90. Horowitz, Rosenberg, Baer, Ureno, and Villasenor (1988) showed alpha coefficients that ranged from .79 to .90 with test-retest reliabilities ranging from .68 to .83. However, in a study by Hafkenscheid (1993), Cronbach alphas ranged from .14 to 1.0, leaving him to conclude that this instrument would be better described as “... a measure of global distress, i.e. a unidimensional measure” (p.755).

In regards to validity, Schmitz et al. (2000) compared the SCL-90-R to the Inventory of Interpersonal Problems (IIP-C) and the General Health Questionnaire (GHQ-12). Their findings showed concurrent validity with Pearson correlations between the SCL-90-R and the IIP-C at .63. Criterion validity was shown using receiver operating characteristic analysis (ROC). The area under the curve for the SCL-90-R GSI was 0.917 (SD = 0.049).

Due to the similarity between Attention-Deficit Hyperactivity-Disorder (ADHD) symptoms and those of executive function deficits, the Current Symptoms Scale—Self-Report Form (Barkley & Murphy, 1998) was used to rule out the presence of ADHD. This scale is an 18-item questionnaire that asks examinees to answer questions related to ADHD symptoms on a Likert-type scale where 0 equals “Never or Rarely” and 3 equals “Very Often.” The scoring method employed in this study, as described by
Barkley and Murphy, was to identify odd items (those pertaining to inattention) or even items (those pertaining to hyperactivity-impulsivity) endorsed with a 2 or 3 by the participant. If six or more items on either set (those pertaining to inattention or hyperactivity-impulsivity) were endorsed, the participant would have been considered to possess clinically significant ADHD symptoms and excluded from the current study. No individual in the current study obtained a score of six or higher on this measure.

Intelligence. General intelligence was assessed through the Wechsler Adult Intelligence Scale-Third Edition (WAIS-III) Vocabulary subtest. Channon (1996) noted that this subtest possesses the best correlation with the Verbal IQ (VIQ). Additionally, Sattler (1992) noted that this subtest is often administered as an estimate of general intelligence. Hartlage, Alloy, Vasquez, and Dykman (1993) asserted that one's performance on the vocabulary subtest remains fairly unimpaired by depression. Thus, the Vocabulary subtest was chosen in order to estimate level of intelligence in the overall participant sample, which allowed for comparisons to be made between groups.

The Vocabulary subtest of the WAIS-III requires individuals to tell an examiner what a stimulus word means. It consists of “a series of orally and visually presented words that the examinee orally defines” (Wechsler, 1997, p. 2). There are 2, 1, and 0 point answers that must be assessed for quality by the examiner with the help of an administration manual.

The Vocabulary subtest of the WAIS-III has shown reliability coefficient averages of .93 across a broad range of ages (approximately 16 to 89; No author, 1997). Measures of test-retest stability on the Vocabulary subtest revealed strong coefficients that ranged from .89 to .94 (No author). Lastly, inter-rater reliability coefficients were
also calculated for the Vocabulary subtest which revealed a very high coefficient of .95 (No author). Validity has been shown for the WAIS-III in general and, more specifically, for the Vocabulary subtest.

Criterion related validity has been found by correlating the WAIS-III with the WAIS-R (its previous version) and the WISC-III (the children’s version of this test). The Vocabulary subtest revealed a .90 correlation with the WAIS-R and .83 with the WISC-III indicating strong validity in this area (No author, 1997). Construct validity has been shown by correlating the subtests of the VIQ with one another as well as the rest of the subtests from the WAIS-III. It was believed that the VIQ subtests would correlate highest with one another (No author). Results of the validation effort ultimately revealed that the Vocabulary subtest correlated the highest with the overall VIQ (.89; No author).

As other measures of intelligence may not have a specific Vocabulary subtest, validation efforts were made to correlate the overall VIQ Index of the WAIS-III, which the Vocabulary subtest is part of, with other valid and standard measures in the field. For example, the VIQ was found to correlate well with The Stanford-Binet Intelligence Scale-Fourth Edition Verbal Reasoning score (.72; No author).

Measures of Independent Variable

Depression. Objective levels of depression were measured by the Beck Depression Inventory-Second Edition (BDI-II; Beck et al., 1996). The BDI-II is a 21-item self-report measure created to assess the severity of depression (based on the DSM-IV) in those aged 13 years and older. Each item of the BDI-II is rated on a 4-point scale that ranges from 0 to 3. Beck et al. reported that normative data was collected from four different psychiatric outpatient clinics as well as one college-student group. The
outpatient psychiatric group was comprised of approximately 63% women and 37% men and was almost entirely European American (91%). The college-student sample was comprised of 56% women and 44% men and was also predominately European American (Beck et al.).

Measures of internal consistency revealed coefficient alphas of .92 and .91 for the outpatients and students respectively (Beck et al., 1996). Beck et al. also reported that all 21 of the corrected item-total correlations for the BDI-II were significant at the .05 level. To establish the BDI-II's stability over time, test-retest correlations were completed and revealed a correlation of .93 (Beck et al.).

Content, construct, and factorial validity were also assessed by Beck et al. The content validity of the BDI-II (Beck et al.), as noted earlier, is based on the DSM-IV criteria for depression. Convergent validity was assessed by comparing the BDI-II to its predecessor, the BDI-IA. In so doing, Beck et al. reported correlation coefficients of .93 and .84. It is noteworthy that the BDI-II was correlated with the Hamilton Psychiatric Rating Scale for Depression (HRSD; a popular and often used measure of depression in research), which revealed a correlation coefficient of .71 (Beck et al.). Discriminant validity has also been established for the BDI-II by way of correlating it with the Hamilton Rating Scale for Anxiety (Hamilton, 1959) which revealed a correlation coefficient of only .47 (Beck et al., 1996). Beck et al. asserted that due to a .51 correlation coefficient between the HRSD and the Hamilton Rating Scale for Anxiety, robust discriminant validity was shown. Factorial validity reported by Beck et al. cited evidence for two factors based on factor analyses, however there was a difference of almost 7 between the two highest eigenvalues.
In addition to the BDI-II, the Structured Clinical Interview for DSM-IV Axis I Disorders (Clinical Version; First et al., 1997), modules A (Mood Episodes, Dysthymic Disorder, Mood Disorder Due to a General Medical Condition, & Substance Induced Mood Disorder) and D (Mood Disorders; hereafter referred to as SCID-I) were also used to categorize individuals into the depressed and non-depressed groups. The SCID-I is a “semi-structured interview for making the major DSM-IV Axis I diagnoses” (First et al., p. 1).

This measure was used to ensure standardization in clinical judgment regarding participant depression. The SCID-I interview protocol was chosen due to its intent to be an efficient and user-friendly instrument. The SCID-I has extensive instructions on how to administer and score responses and further allows for administration of individual modules for research purposes (First et al.). The SCID-I is a popular and widely used semi-structured interview in clinical and research settings.

First et al. described the reliability of the SCID-I as being comparable to other diagnostic instruments. For example, they noted recent studies that have reported kappa statistics that ranged from .70 to 1.00. Furthermore, First et al. asserted that procedural validity is desired for a semi-structured interview, yet noted that this can be difficult to achieve since there is no “gold standard” way that has been established to diagnose psychiatric disorders. However, initial work is being completed and has begun to build the procedural validity needed in order to have a sound measure (see Kranzler et al., 1995; Skodol, Rosnick, Kellman, Oldham, & Hyler, 1988).
Measures of Dependent Variable

Executive functioning was assessed by way of the Wisconsin Card Sorting Test (WCST; Heaton et al., 1993), Trail Making Test, Parts A and B (TMT; Reitan, 1993), Tower of London, Drexel Edition (TOL\textsuperscript{DX}; Culbertson & Zillmer, 2005), Stroop Color and Word Test (Golden & Freshwater, 2002), and Controlled Oral Word Association test (COWA; Spreen & Straus, 1998).

*Wisconsin Card Sorting Test.* The Wisconsin Card Sorting Test (WCST) "... has been considered the premier test of executive functions for many years" (Malloy, Cohen, & Jenkins, 1998, p. 584) and is considered to be the most common tool to assess executive functions (Golden et al., 2000). Various researchers have set numerous versions of the WCST forth. However, the presently accepted method of administration and scoring are based on the work of Heaton et al. (1993). In this revised and updated method, one can complete the WCST either by hand (with a deck of cards) or on a computer. Both methods require an examinee to match cards to a set of four stimulus cards (one card with a red triangle, one with two green stars, one with three yellow crosses, and one with four blue circles) on three parameters, color, form, or number (Heaton et al.).

Success on the WCST necessitates an individual to determine the correct sorting principle based on examiner feedback (correct or incorrect) and then maintain said principle or *set* while the stimuli changes (Heaton et al.). Two main areas that the WCST assesses are failure to maintain set and perseveration. A failure to maintain a set occurs when 5 or more consecutive correct answers are made and an error occurs before the category (completion of 10 consecutive correct answers) has been established.
Furthermore, the WCST assesses one’s level of perseveration. This is said to exist when one is unable to inhibit one’s self from persisting with an older rule and must use the examiner’s feedback to identify the correct sorting principle (Heaton et al.).

Scores on the WCST are compared to a normative stratified group of nearly 900 individuals from six different samples. Heaton et al. (1993) reported that the normative group closely approximated the U.S. census data for age for 1995. Inter-rater and intra-rater reliabilities have been investigated for the WCST. Axelrod, Goldman, and Woodard (1992) reported inter-rater reliability coefficients of .93, .92, and .88 while the samples consistency was reported as .96, .94, and .91 for Perseverative Responses, Perseverative Errors, and Nonperseverative errors.

Evidence of concurrent validity for the WCST has been shown. Perrine (1993) used a Veterans Administration sample to establish discriminate validity between the WCST and the Halstead Categories Test (HCT). He found that these two measures shared only 30% of the variance leading him to suggest that they measure separate things. For example, the WCST was associated with attribute identification whereas the HCT was associated more with rule learning.

*Trail Making Test*. The Trail Making Test (TMT) consists of two parts (A & B) that assesses “visual conceptual abilities, cognitive flexibility, set shifting, sequencing ability, visual-motor tracking, and visual-spatial functioning” (Golden et al, 2000, p. 171). Part A has 25 encircled numbers in various positions and requires one to connect the circles by pencil line in correct order as fast as one is able (Spreen & Strauss, 1998). While Part B employs the same requirements, it differs in that it also utilizes letters as well as numbers in alternating order. Scoring consists of calculating time in seconds.
taken to complete the measure from beginning to end and comparing the time to a normative group (Spreen & Strauss).

Research has shown that the two parts of the TMT only correlate at a .49 level, suggesting that the two assess somewhat different functions (Heilbrunner et al., 1991). Ehrenstein, Heister, and Cohen (as cited in Spreen & Strauss, 1998) showed construct validity for the TMT by establishing positive correlation coefficients that ranged between .36 and .93 with an object finding and hidden pattern test.

Other research has shown that the TMT can discriminate between psychiatric patients and healthy controls. In a study by Warner et al. (1987), it was found that times for all of the psychiatric patients in the study exceeded the lowest percentile norms of the healthy controls. Furthermore, Part B is believed to be the more sensitive of the two measures to general cortical dysfunction and has been utilized a great deal in research investigating executive functioning (Spreen & Strauss, 1998). This has been a consistently reliable finding since it was initially believed to be a measure that could distinguish between brain impaired and non-impaired individuals (Reitan, 1955; Reitan & Wolfson, 1993; Spreen & Strauss, 1998).

Tower of London Drexel University: 2nd Edition (TOL\textsuperscript{DX}). The Tower of London is believed to be an instrument that can “get to the heart of planning disorders” (Lezak et al., 2004). This test has been heralded as not only an excellent test of frontal lobe damage, but also of attention and executive functions (Multi-Health Systems, Inc. 2005). According to Culbertson and Zillmer (2005), the TOL\textsuperscript{DX} was “designed to assess higher-order problem solving—specifically, executive planning abilities—in children and adults” (p. 1). They further noted that executive planning impairments are often implicated in
both acquired and developmental disorders and, thus, may be of great importance to understanding its etiology and advancing treatment.

The TOL\textsuperscript{DX} consists of two rectangular boards with three pegs on each board pointing upward from shortest to longest. Examinees are required to move around three different colored beads that fit onto the previously mentioned pegs. In this task, the examinee is told to match his/her board to the evaluator's board. The test begins with the evaluator asking the examinee to match the bead formation on his/her board to the evaluator's in as few moves as possible. After a demonstration problem is completed, the evaluator informs the examinee of two rules that must be followed. The first rule is that one can only place as many beads on a peg as the peg was intended to hold (e.g., one peg is designed to hold one bead, while another can hold two, and the other can hold three). The second rule is that only one bead can be off of a peg at one time. The evaluator provides demonstrations of each rule and how it may be violated. Following this, two practice problems are completed and then the ten test problems of ascending difficulty. All bead formations are produced on the evaluator's board and then the examinee is asked to "now make one like this" on his/her board in as few moves as possible.

The TOL\textsuperscript{DX} provides eight standard scores based on executive planning performance: Total Moves, Total Correct, Total Rule Violation, Total Time Violation, Total Initiation Time, Total Execution Time, Total Problem-Solving Time, and Total Stimulus Bound (only used in populations age 60 years and over).

Normative data were compiled by Culbertson and Zillmer (2005) and represent a moderate racially diverse sample. Culbertson et al. (2004) reported test-retest reliability coefficients regarding the temporal stability of the TOL\textsuperscript{DX} obtained from Parkinson's
disease patients. The Bivariate correlations revealed that scores were adequately stable over time for Move Score \( (r = .80, p < .001) \); Correct Score \( (r = .48, p = .007) \), Time Violation Score \( (r = .77, p < .001) \), Rule Violation Score \( (r = .37, p < .047) \), Stimulus-Bound Score \( (r = .63, p < .001) \), Initiation Time Score \( (r = .74, p < .001) \), Execution Time Score \( (r = .83, p < .001) \), and Total Problem Solving Time Score \( (r = .87, p < .001) \).

The validity of the TOL\textsuperscript{DX} has been assessed through criterion related and construct related methods and is considered to be a valid measure of executive planning (Culbertson & Zillmer, 2005).

**The Stroop Color and Word Test.** The Stroop Color and Word Test (Stroop; Golden & Freshwater, 2002) was first discussed in professional literature in the late 1800’s (Golden & Freshwater). However, it was not until 1935 that the Stroop began to take the form of its present state. Its present form has been established through the work of Charles Golden and is currently felt to test one’s ability to “… sort information from his or her environment and to selectively react to this information” (2002, p. 2). Golden et al. (2000) asserted that the Stroop is most frequently used to screen for brain damage, more specifically frontal lobe damage. The most current form of the Stroop consists of a three-page form consisting of 5 columns with 20 items in each column. The *Word* page consists of the words RED, GREEN, and BLUE printed in black ink. The *Color* page consists of XXXX symbols printed in red, green, or blue ink. The *Color-Word* page consists of words from the Word page printed in red, green, or blue ink; however, no words are printed in the color of the word itself (e.g., the word BLUE is never printed in blue ink; Golden & Freshwater, 2002).
The current accepted scoring method requires the examinee to complete as many items as he/she can in 45 seconds (Golden & Freshwater, 2002). All scores are ultimately converted into standardized $t$-scores. Original normative data was gathered by Golden between 1974 and 1977 (Golden & Freshwater). Golden and Freshwater reported that since the early collection of normative data, updated and expanded norming information has been collected and implemented in the Stroop manual, which provides adjusted scores based on one’s age and actual years of education. Reliability of the Stroop has also been reported. Jensen (1965) reported reliabilities of .88, .79, and .71 for the three subtests of the Stroop. Furthermore, Golden (1975) found reliabilities of .86, .82, and .73 for the three subtests. It is noteworthy that Raskin, Friedman, and DiMascio (1982) investigated the Stroop in a sample of depressed individuals and found that there were significant differences between depressed individuals and healthy controls with the depressed individuals demonstrating lower scores on the Stroop.

**Controlled Oral Word Association.** The Controlled Oral Word Association test (COWA; Spreen &Straus, 1998) evaluates one’s ability to spontaneously produce as many words as one is able that begin with the letters F, A, and S. Golden et al. (2000) asserted that one must have adequate mental flexibility and inhibition of verbal output skills in order to perform successfully on the COWA. If one does not possess mental flexibility, he/she will likely perseverate and repeat words or produce quite similar words (Golden et al.). Additionally, if one is unable to inhibit certain responses one may either produce socially inappropriate words or words that do not fit the category.

While no specific materials are needed, the COWA can be found in some current neuropsychological batteries (Spreen & Strauss, 1998). Scoring of the COWA consists of
summing all admissible words from the three letters. Spreen and Strauss asserted that slang and foreign words that are part of Standard English are acceptable (e.g., faux pas, lasagna; p. 448). While errors do not necessarily count against an individual in terms of points, errors waste time and can also be reviewed for insight into potential disorders (e.g., repetitions and intrusions can indicate potential perseveration and distraction problems; Spreen & Strauss). Scores are then compared to normative data, which yields percentile scores. Tombaugh, Kozak, and Rees (as cited in Spreen & Strauss) have developed acceptable normative data for adults ranging between the ages of 16 and 95 as well as for specific years of education based on age grouping from a sample of nearly 900 individuals from Ottawa, Ontario, Canada.

Inter-rater reliability estimates have been reported at .70 overall and .70, .60, and .71 for F, A, and S, respectively (as cited in Spreen & Strauss, 1998). Spreen and Strauss reported that concurrent validity has been widely established for the COWA using the letters F, A, and S. Crockett (1977) found that the COWA related to many abilities that are implicated in executive functions. For example, problem solving, sequencing, resisting distraction, perseveration, and memory were each related to the COWA.

Procedure

A number of medical and psychological/psychiatric clinics located in the upper Midwestern United States were asked to participate in the current study by offering patients/clients whom reported depressive symptoms a free depression evaluation through participation in the current research study. Clinicians then referred patients/clients whom expressed interest in the free depression evaluation to the principal investigator. The
principle investigator then contacted these individuals and scheduled evaluation appointments that ranged in time from 60 minutes (primarily the non-medicated non-depressed participants) to 120 minutes. During the scheduling telephone phone calls, potential participants were reminded that they were being asked to participate in a research study and that the free depression evaluation would be completed regardless of their decision to participate or not participate in the current research study. They were also informed that while both the participant and the referring source would receive a letter reporting the findings of the depression evaluation as well as treatment recommendations, they would not receive any information regarding their performance on the tests of group equivalence or executive functioning.

Individuals in the non-medicated non-depressed comparison group were recruited by going into various undergraduate and graduate classes at a mid-sized university in the upper Midwestern United States and asking for volunteers. This group’s participants were offered a $5 gift certificate to a local sandwich or coffee shop as compensation for their participation. It was decided that this group could be given a gift certificate as incentive to participate as the two depressed groups were receiving a free depression evaluation.

All participants were evaluated in a well lit, climate controlled room with appropriate testing supplies (e.g., adequately sized table, chairs, etc.). At the beginning of the testing sessions, all participants were provided with an informed consent statement to read and sign. All participants were administered the BDI-II (Beck et al., 1996) and the SCID-I (First et al., 1997) to establish the presence of depressive symptoms in the depressed groups and the absence of depressive symptoms in the non-depressed group. Beck et al. (1996) recommend that certain score cutoffs be used to differentiate between
depressed and non-depressed individuals. They noted that their research has shown that a cut score of 17 yielded a 93% true-positive rate while only experiencing an 18% false-positive rate for major depression (Beck et al.). Thus, based on Beck et al.'s assertions, this study employed a cut score of 17 on the BDI-II for inclusion into both of the depressed groups (medicated & non-medicated). Using the BDI, Channon (1996) secured a sample of depressed individuals for his research of executive functions and depression. In his study, a cut score of 5 or below was used as inclusion criteria for the healthy control group. Based on Beck et al.'s (1996) raw score conversion chart, a score of 5 on the BDI-A equates to a score of 6 on the BDI-II. Therefore, a cut score of 6 or below was employed in the current study as inclusion criteria for the non-depressed group.

Following this, each participant completed the demographic questionnaire and the Current Symptoms Scale—Self-Report Form to rule out ADHD symptoms. To establish group equivalence, participants then completed the SCL-90-R and the Vocabulary subtest of the WAIS-III. Participants were then administered the tests of executive function in the following order, WCST, TMT Part A and B, TOL$^{DX}$, Stroop, and the COWA. There was no particular reason for the order of tests.

After the completion of all measures, participants from the depressed groups were briefed regarding the information obtained from the depression evaluation. Information was explained and participants were allowed to ask questions regarding their depression evaluation. After all questions were answered, the participants were thanked for their time and asked to scribe their name on an envelope in order to send the short report regarding their depression evaluation. This served to maintain the highest level of confidentiality as they could address the envelope to wherever they desired and have it be
in their writing. Non-depressed participants were provided their gift certificate at the end of testing and also thanked for their participation.

Data Analyses

All analyses were completed using SPSS Version 13.0. During the initial stages of data analyses, two-tailed bivariate correlations were completed in order to analyze the numerous variables to ascertain any correlations that would help decipher which later analyses would be completed. Two noteworthy trends were revealed from this action. First, the Vocabulary subtest of the WAIS-III (Wechsler, 1997) significantly correlated with a large proportion of the other variables. Such correlation suggested that Vocabulary scores be used as a covariate in the later analyses. The second noteworthy trend was that the sub-scores of each of the executive function measures were intercorrelated (e.g. the sub-scores of the WCST correlated with each other at a statistically significant level).

Preliminary analyses employed an analysis of variance (ANOVA) on participant age, the Vocabulary subtest of the WAIS-III (Wechsler), yearly household income, and years of education completed as well as a multivariate analysis of covariance (MANCOVA), with age and vocabulary score of WAIS-III serving as covariates, on the SCL-90-R. Based on this information, it was decided that multivariate analyses of covariance would be completed for the main analyses.

The main analyses consisted of completing MANCOVAs on the various scores obtained on the tests of executive functioning with participant age and the Vocabulary subtest of the WAIS-III as covariates. This served to remove the influence of estimated level of intelligence and age from the comparison of groups on the factor of performance on tests of executive functioning. The main area of interest in the current study lies at the
Post-Hoc Pairwise Comparison level as this allows for identification of specific differences between the medicated depressed, non-medicated depressed, and non-medicated non-depressed groups.

A multiple linear regression was also completed as a post-hoc analysis in order to explore any potential differences that depression and anxiety may have upon executive function test performance. Using the BDI-II (Beck et al., 1996) and the Anxiety subscale of the SCL-90-R (Derogatis, 1994), regression analyses were completed in order to ascertain whether or not anxiety added any predictive ability to how well or poorly one would perform on tests of executive function over depression (as measured by the BDI-II [Beck et al., 1996]) alone.
CHAPTER IV

RESULTS

The results of the current study are presented in this chapter in three sections. The first section reports the results of preliminary analyses. The second section reports the results of the main analyses regarding the main hypotheses of the study and the final section reports the results of post hoc analyses.

Preliminary Analyses

A correlation matrix was constructed in order to visually analyze any trends in the data that necessitated certain statistical procedures in the main analyses. High Pearson correlations (e.g., .65 or higher and -.65 or lower) between variables were identified and evaluated to determine the necessity of using Multivariate Analyses of Variance (MANOVA) in the main analyses. Furthermore, the three groups (medicated depressed, non-medicated depressed, & non-medicated non-depressed) were analyzed in terms of their group equivalence on the Vocabulary section of the WAIS-III (Wechsler, 1997; estimated level of intelligence) and the Symptom Checklist-90-Revised (SCL-90-R; estimated level of psychological well-being).

According to Pagano (2001), “Pearson r is a measure of the extent to which paired scores occupy the same or opposite positions within their own distributions” (p. 109). A visual search of correlations revealed several significant relationships, both positive and negative. Several of the 48 variables looked at throughout the following analyses reached statistical significance and were highly correlated ($r \geq .65$ or $r \leq -.65$).
See Table 2 for a full list of correlations between the measures of executive functioning and Table 3 for correlations between the measures of executive functioning and measures of affective group equivalence.

Several of the variables were expected to covary to some degree. For example, the SCL-90-R is a measure of psychological well-being and covers psychological distress on a variety of continuums. Therefore, Depression, Anxiety, Phobic Anxiety, Interpersonal Sensitivity, etc., would be expected to covary as one who scores higher on Depression would presumably score higher on Interpersonal Sensitivity as they share similar characteristics. Furthermore, the BDI-II (Beck et al., 1996) was expected to covary with subscales of the SCL-90-R for the same reasons, especially the depression subscale. Others, however, were expected to have low or no correlations, as this would suggest that the test was measuring separate constructs as evidenced by a lower correlation coefficient.

The subscales of the five measures of the executive functions were not expected to covary with the subscales of the other tests. However, some subscales were expected to covary with subscales within the same test. For example, analyses revealed several correlations between the subscales within the Wisconsin Card Sorting Test (WCST; Heaton et al., 1993).

Higher correlations would be expected between subscales that were affected by the same event. For example, the Trials Administered subscale correlated highly with the Total Errors ($r = .90$), Perseverative Responses ($r = .87$), Perseverative Errors ($r = .87$), Nonperseverative Errors ($r = .86$), and Failure to Maintain Set ($r = .69$) subscales. This
Table 2. Pearson Correlations ($r$) of Executive Function Measures.

|   | 1  | 2  | 3  | 4  | 5  | 6  | 7  | 8  | 9  | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | 19 | 20 | 21 | 22 | 23 | 24 | 25 |
| 1 |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |
| 2 | .51|   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |
| 3 | .86| .07|   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |
| 4 | .87| .14| .94|   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |
| 5 | .87| .12| .95| .99|   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |
| 6 | .86| .03| .97| .84| .85|   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |
| 7 | .06| .81| -.49| -.39| -.41| -.51|   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |
| 8 | -.71| -.13| -.89| -.84| -.85| -.87| .63|   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |
| 9 | .47| -.33| .72| .59| .64| .73| -.68| -.79|   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |
| 10| .69| .70| .44| .44| .44| .41| .35| .42| .14|   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |
| 11| -.20| .05| -.27| -.36| -.33| -.20| .23| .21| .10| .05|   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |
| 12| -.27| .30| .15| .21| .20| .11| .17| -.02| -.01| .24| -.05|   |   |   |   |   |   |   |   |   |   |   |   |   |   |   |
| 13| .50| .05| .57| .46| .48| .57| -.27| -.59| .58| .36| .04| .31|   |   |   |   |   |   |   |   |   |   |   |   |   |   |
| 14| -.04| -.09| -.00| -.06| -.05| -.03| -.02| -.04| -.13| -.05| -.03| .16| -.16|   |   |   |   |   |   |   |   |   |   |   |   |   |
| 15| -.35| -.43| -.19| -.24| -.22| -.15| -.22| .11| .06| -.39| .18| -.28| -.33| .38|   |   |   |   |   |   |   |   |   |   |   |   |   |
| 16| -.29| -.13| -.27| -.29| -.29| -.25| .06| .32| -.22| -.35| .05| -.29| -.43| .17| .67|   |   |   |   |   |   |   |   |   |   |   |   |   |
| 17| -.45| -.18| -.43| -.44| -.43| -.40| .08| -.38| -.29| -.36| -.05| -.20| -.49| .33| .49| .48|   |   |   |   |   |   |   |   |   |   |   |   |
| 18| -.28| -.09| -.27| -.30| -.29| -.24| .04| .21| -.21| -.17| -.12| -.07| -.21| .24| .09| .01| .84|   |   |   |   |   |   |   |   |   |   |   |
| 19| -.34| -.17| -.30| -.25| -.27| -.30| -.04| -.27| -.28| -.43| -.16| -.14| -.32| -.03| .04| .33| .05| -.13|   |   |   |   |   |   |   |   |   |   |
| 20| .34| .18| .30| .22| .24| .33| .03| -.26| .23| .45| .15| .11| .30| .05| .08| -.30| -.11| .08| .88|   |   |   |   |   |   |   |
| 21| .06| -.05| .10| .10| .09| .10| -.15| -.05| -.03| -.06| -.32| .26| .18| -.11| -.33| -.09| -.16| -.05| .37| .23|   |   |   |   |   |   |
| 22| .30| -.02| .36| .27| .27| .40| -.22| -.32| .35| .24| .14| .13| .50| -.29| -.15| -.15| -.22| -.06| -.56| .62| .03|   |   |   |   |
| 23| .30| -.04| .37| .29| .28| .41| -.27| -.32| .31| .18| -.03| .23| .53| -.28| -.28| -.20| -.26| -.04| -.36| .47| .48| .88|   |   |   |
| 24| .21| .09| .19| .14| .13| .23| -.10| -.17| .03| .19| -.11| .19| .30| -.36| -.28| -.12| -.10| -.08| -.15| .27| .48| .69| .83|   |
| 25| .06| -.05| .10| -.00| -.00| .17| -.06| -.06| .07| .14| .11| .10| .16| .11| .04| -.09| -.09| .04| -.30| .40| -.13| .25| .21| .05|   |

WCST 3 Total Errors 6 Nonpersev. Er. 9 Trials to 1st 12 TMT Part A 15 Stroop Word 18 Stroop Inter. 20 Tot. Move 23 Tot. Time
1 Trials Ad. 4 Persev. Resp. 7 Concept. Level Res. 10 Fail Maintain Set 13 TMT Part B 16 Stroop Color TOL DAX 21 Tot. Ini. 24 Time Violat.
Table 3. Pearson Correlations ($r$) between Measures of Executive Functioning and Measures of Affective Group Equivalence.

<table>
<thead>
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<th>SCL-90-R Anx</th>
<th>SCL-90-R GSI</th>
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<td>.49</td>
<td>.49</td>
<td>.55</td>
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<td>.42</td>
<td>.41</td>
<td>.52</td>
</tr>
<tr>
<td>WCST Learn to Learn</td>
<td>.09</td>
<td>-.01</td>
<td>-.11</td>
<td>-.01</td>
</tr>
<tr>
<td>TMT Part A</td>
<td>.42</td>
<td>.44</td>
<td>.42</td>
<td>.42</td>
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<tr>
<td>TMT Part B</td>
<td>.45</td>
<td>.36</td>
<td>.48</td>
<td>.48</td>
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<tr>
<td>COWA</td>
<td>-.22</td>
<td>-.24</td>
<td>-.26</td>
<td>-.25</td>
</tr>
<tr>
<td>Stroop Word</td>
<td>-.41</td>
<td>-.44</td>
<td>-.53</td>
<td>-.50</td>
</tr>
<tr>
<td>Stroop Color</td>
<td>-.55</td>
<td>-.51</td>
<td>-.64</td>
<td>-.65</td>
</tr>
<tr>
<td>Stroop Color-Word</td>
<td>-.42</td>
<td>-.36</td>
<td>-.34</td>
<td>-.37</td>
</tr>
<tr>
<td>Stroop Interference</td>
<td>-.17</td>
<td>-.14</td>
<td>-.05</td>
<td>-.05</td>
</tr>
<tr>
<td>$TOL^{DX}$ Total Correct</td>
<td>-.33</td>
<td>-.28</td>
<td>-.30</td>
<td>-.35</td>
</tr>
<tr>
<td>$TOL^{DX}$ Total Move</td>
<td>.25</td>
<td>.16</td>
<td>.21</td>
<td>.28</td>
</tr>
<tr>
<td>$TOL^{DX}$ Total Initiation</td>
<td>.06</td>
<td>.09</td>
<td>.22</td>
<td>.12</td>
</tr>
<tr>
<td>$TOL^{DX}$ Total Execution</td>
<td>.25</td>
<td>.25</td>
<td>.29</td>
<td>.31</td>
</tr>
<tr>
<td>$TOL^{DX}$ Total Time</td>
<td>.27</td>
<td>.27</td>
<td>.36</td>
<td>.34</td>
</tr>
<tr>
<td>$TOL^{DX}$ Time Violation</td>
<td>.13</td>
<td>.16</td>
<td>.29</td>
<td>.26</td>
</tr>
<tr>
<td>$TOL^{DX}$ Rule Violation</td>
<td>.11</td>
<td>.09</td>
<td>-.10</td>
<td>.01</td>
</tr>
</tbody>
</table>

Dep = Depression; Anx = Anxiety; GSI = Global Severity Index

makes conceptual sense, as one would have the opportunity to attempt more trials as one makes more errors. Additionally, the more errors and trials one attempts, the less likely one is to maintain a steady action (i.e., maintain a set). Moreover, as this is happening, one is not going to be completing as many categories due to the fact that the individual does not comprehend the task. Thus, there was a high inverse correlation between the Trials Administered subscale and the Categories Completed subscale ($r = -.71$).

Similarly, there were high correlations between the Total Errors subscale and the Perseverative Responses ($r = .94$), Perseverative Errors ($r = .95$), Nonperseverative
Errors \((r = .97)\), and Trial to Complete 1\textsuperscript{st} Category \((r = .72)\) subscales. Just the same, an inverse correlation was revealed between the Total Errors subscale and the Categories Completed \((r = -.89)\) subscale. In this same vein, high correlations were revealed between the Perseverative Responses subscale and the Perseverative Errors \((r = .99)\) and Nonperseverative Errors \((r = .84)\) subscales; as was an inverse correlation between the Perseverative Responses subscale and Categories Completed \((r = -.84)\) subscale. Analyses also revealed high correlations between the Perseverative Errors and Nonperseverative Errors \((r = .85)\) subscales and an inverse relationship between the Perseverative Errors and Categories Completed \((r = -.85)\) subscales. The Nonperseverative Errors subscale was inversely related to the Categories Completed \((r = -.87)\) and Trials to Complete 1\textsuperscript{st} Category \((r = -.68)\) subscales, respectively. Lastly, the Categories Completed and Trials to Complete First Category were inversely related \((r = -.79)\). As one would expect, the more trials it takes to complete the first trial the less likely it becomes that the individual will complete a higher number of categories.

The Stroop Color and Word test (Golden & Freshwater, 2002) revealed one expected correlation between the Color-Word and Interference subscales \((r = .84)\). This is an expected correlation due to the fact that the Interference score is primarily based upon the Color-Word subscale. The requirement of the Color-Word task is to state the color of ink the word is printed in, ignoring the word that is spelled out. This is difficult to do, as it requires one to inhibit reading the word. Thus, the more difficulty one has with the Color-Word task, the higher one’s Interference score will be.

The TOL\textsuperscript{DX} (Culbertson & Zillmer, 2005) revealed similar correlations. High positive correlations were found between the Total Execution Time and Total Time
(r = .88) and Time Violation (r = .69) subscales. This makes conceptual sense in that the total time of the task from start to finish is impacted a great deal by the time it takes one to execute the task. Furthermore, individuals who violate rules are told of their violation and required to undo the violation and begin from the point at which they violated the rule. Thus, this takes a good deal of time resulting in increased total times and increased execution times for anyone who violates a rule.

A relationship was revealed between the TOL\textsuperscript{DX} Total Time and Time Violation (r = .83) subscales. Again, this is conceptually appropriate, as an individual would have an increased total time if he/she were not completing the tasks in less than 60 seconds (the amount of time one has to complete the task before one obtains a Time Violation). Also, the Total Correct and Total Move scores were inversely related (r = -.88) as the two represent opposite ends of a continuum of success. One would not be able to do well in terms of Total Correct score if he/she were also obtaining high scores on the Total Moves subscale as the more moves one makes the less likely it is that the individual will complete the task in the least amount of moves possible. Therefore, while several variables correlated highly with others, none appeared to impact the current study in a way that would render any of the measures obsolete or indicate that any measure was measuring similar entities.

Four one-way analyses of variance (ANOVAs) were completed in order to evaluate the relationship between participant age, estimated level of intelligence (i.e., the raw Vocabulary subtest scores of the WAIS-III), yearly household income, and years of education completed and group membership. The independent variable in each analysis was group membership (i.e., medicated depressed, non-medicated depressed, and non-
medicated non-depressed) while the dependent variable was participant age, Vocabulary subtest raw scores of the WAIS-III, yearly household income, and years of education completed. ANOVAs were statistically significant for participant age, \( F(2, 50) = 5.5, p = .007 \), partial \( \eta^2 = .18 \) and Vocabulary subtest raw scores of the WAIS-III, \( F(2, 50) = 6.1, p = .004 \), partial \( \eta^2 = .20 \) indicating that there are statistically significant differences between the groups regarding participant age and estimated level of intelligence. However, these ANOVAs also revealed nonsignificant results for yearly household income, \( F(2, 50) = 0.3, p = .761 \), partial \( \eta^2 = .01 \) and years of education completed, \( F(2, 50) = 1.9, p = .157 \), partial \( \eta^2 = .07 \).

Follow-up tests were conducted to evaluate pairwise differences among the means using the Tukey Honestly Significant Differences test (Tukey HSD). In regards to participant age, statistically significant differences were found between the medicated depressed (\( M = 31.6, SD = 6.9 \)) group and both the non-medicated depressed (\( M = 24.8, SD = 5.1; p = .01 \)) and the non-medicated non-depressed (\( M = 25.9, SD = 6.4; p = .02 \)) groups. The difference between the non-medicated depressed and non-medicated non-depressed groups failed to reach statistical significance (\( p = .85 \)).

Post-hoc analyses of the Vocabulary subtest of the WAIS-III revealed statistically significant differences between the medicated depressed (\( M = 39.60, SD = 11.10 \)) and both the non-medicated depressed (\( M = 40.56, SD = 9.07, p = .02 \)) and non-medicated non-depressed groups (\( M = 48.55, SD = 6.31, p = .01 \)), while there was not a statistically significant difference between the two depressed groups (\( p = .95 \)). Overall, the non-medicated non-depressed group showed higher levels of intelligence than the other two groups at a statistically significant level. Based on the information provided by the
analyses of the participant’s age and the Vocabulary subtest of the WAIS-III, participant age and the Vocabulary variables were used as covariates in the remaining analyses.

In the second hypothesis of the preliminary analyses, it was hypothesized that the two depressed groups (medicated depressed & non-medicated depressed) would differ significantly from the non-medicated non-depressed group on a measure of psychological well-being as measured by the SCL-90-R. Specifically, differences were expected between the two depressed groups and the non-medicated non-depressed group on the depression subscale of the SCL-90-R. Based on the obtained correlations (described previously) and the observation by Derogatis (1994) that the Global Severity Index (GSI) “represents the best single indicator of the current level or depth of the disorder, and should be utilized in most instances where a single summary measure is required” (p. 11), the GSI was used to assess overall group differences related to overall psychological well-being.

A one-way univariate analyses of covariance (ANCOVA), with participant age and WAIS-III Vocabulary subtest scores serving as covariates, was carried out to determine if significant differences were present on the GSI based on group membership (depressed or not). Additionally, this analysis was completed in order to lend support to the hypothesis that the two depressed groups would not differ from one another in terms of overall psychological well-being as measured by the GSI of the SCL-90-R.

A preliminary analysis evaluating the homogeneity-of-slopes assumption indicated that the relationship between the covariates (participant age and Vocabulary score) and the dependent variable (GSI) did not differ significantly as a function of the independent variable (group membership), $F(3, 45), \text{MSE} = 0.18, p = .05$, partial $\eta^2 = .16$. 
The ANCOVA was significant, $F(2, 48), MSE = 0.20, p < .001$. The strength of the relationship between the group membership factor and GSI was very strong as assessed by a partial $\eta^2$ (traditionally a partial $\eta^2$ [effect size] of .01 is small, .06 is medium, and .14 is large; Green, Salkind, & Akey, 1999), with the group membership factor accounting for 43% of the variance on the dependent variable, holding constant the GSI score.

The mean GSI score, adjusting for initial differences, were ordered as expected across the three groups. Adjusted means resulted in small changes in the three groups (medicated depressed; $M = 1.24, SE = 0.13$; non-medicated depressed; $M = 0.91, SE = 0.12$; & non-medicated non-depressed; $M = 0.26, SE = 0.10$). Follow-up tests were conducted to evaluate pairwise differences among these adjusted means. The Holm's sequential Bonferroni procedure was used to control for Type I errors across the three pairwise comparisons. There were significant differences in the adjusted means between the non-medicated non-depressed group and both depressed groups (medicated depressed, $p < .001$; non-medicated depressed, $p < .001$). However, there was not a statistically significant difference found between the two depressed groups on the GSI ($p = .208$).

Based on the correlations previously described, a one-way Multivariate Analysis of Covariance (MANCOVA), with participant age and WAIS-III Vocabulary subtest score serving as covariates, was carried out to determine the effect of group membership on the SCL-90-R subscales. Significant differences were found among the three groups on the dependent variables of the SCL-90-R, Wilkes' $\Lambda = 0.149, F(24, 74) = 4.898, p < .001$, partial $\eta^2 = .61$. The strength of the relationship between group membership and scores on the SCL-90-R was quite strong (i.e., roughly 60% of the effect is attributable to...
group membership) as assessed by the partial $\eta^2$. Table 4 contains raw score means, standard deviations, estimated marginal means, and standard errors for the dependant variables and ANCOVA results for the three groups.

Analyses of covariance (ANCOVAs) were conducted on each dependent variable as follow-up tests to the MANCOVA. A more conservative alpha (.01) was incorporated in order to control for experimentwise error, thus reducing the chance of Type I error. All but two (Phobic Anxiety and Hostility) of the dependent variable $F$ statistics were significant. Table 4 contains the ANCOVA results ($F$ statistics & partial $\eta^2$s) as well as means, standard deviations, estimated marginal means, and standard errors for all SCL-90-R subscales across the three groups (medicated depressed, non-medicated depressed, non-medicated non-depressed).

Post hoc pairwise analyses to the ANCOVAs were completed for the SCL-90-R subscales at the .01 level. While the majority of the subscales revealed statistically significant differences between the two depressed groups and the non-medicated non-depressed group, the Interpersonal Sensitivity (non-medicated depressed, $p = .05$), Anxiety (non-medicated depressed, $p = .16$), Phobic Anxiety (medicated depressed, $p = .02$; non-medicated depressed, $p = 1.0$), Hostility (medicated depressed, $p = .02$; non-medicated depressed, $p = .11$), and Paranoid (non-medicated depressed, $p = .13$) subscales failed to reach statistical significance. As hypothesized, there were no statistically significant differences on the subscales between the medicated depressed group and the non-medicated depressed group.
Table 4. Raw Score Means, Standard Deviations, Estimated Marginal Means (EMM), Standard Errors (SE), and ANCOVA results for SCL-90-R.

|                       | Medicated Depressed | Non-Medicated Depressed | Non-Medicated Non-Depressed | All Groups
group ANCOVA |
<table>
<thead>
<tr>
<th></th>
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<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n = 15</td>
<td>n = 16</td>
<td>n = 22</td>
<td>N = 53</td>
</tr>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
<td>(EMM)</td>
<td>M</td>
</tr>
<tr>
<td>SCL-90-R</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depression</td>
<td>1.97</td>
<td>0.80</td>
<td>(1.82)</td>
<td>1.78</td>
</tr>
<tr>
<td>Somatization</td>
<td>1.05</td>
<td>0.70</td>
<td>(0.90)</td>
<td>1.00</td>
</tr>
<tr>
<td>Psychoticism</td>
<td>0.91</td>
<td>0.60</td>
<td>(0.89)</td>
<td>0.63</td>
</tr>
<tr>
<td>Interpersonal Sensitivity</td>
<td>1.64</td>
<td>0.84</td>
<td>(1.60)</td>
<td>0.92</td>
</tr>
<tr>
<td>Anxiety</td>
<td>1.15</td>
<td>0.81</td>
<td>(1.03)</td>
<td>0.73</td>
</tr>
<tr>
<td>Obsessive-Compulsive</td>
<td>1.75</td>
<td>0.89</td>
<td>(1.63)</td>
<td>1.16</td>
</tr>
<tr>
<td>Phobic Anxiety</td>
<td>0.64</td>
<td>0.54</td>
<td>(0.54)</td>
<td>0.23</td>
</tr>
<tr>
<td>Hostility</td>
<td>0.82</td>
<td>0.64</td>
<td>(0.77)</td>
<td>0.63</td>
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</tbody>
</table>
Table 4 (cont.). Raw Score Means, Standard Deviations, Estimated Marginal Means (EMM), Standard Errors (SE), and ANCOVA results for SCL-90-R.

<table>
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<tr>
<th></th>
<th>Medicated Depressed n = 15</th>
<th>Non-Medicated Depressed n = 16</th>
<th>Non-Medicated Non-Depressed n = 22</th>
<th>All Groups* ANCOVA N = 53</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M (EMM)</td>
<td>SD (SE)</td>
<td>M (EMM)</td>
<td>SD (SE)</td>
</tr>
<tr>
<td>SCL-90-R</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Paranoid Ideation</td>
<td>1.05</td>
<td>0.8</td>
<td>0.70</td>
<td>0.67</td>
</tr>
<tr>
<td></td>
<td>(1.08)</td>
<td>(0.16)</td>
<td>(0.61)</td>
<td>(0.15)</td>
</tr>
<tr>
<td>Global Severity Index</td>
<td>1.32</td>
<td>0.62</td>
<td>0.95</td>
<td>0.55</td>
</tr>
<tr>
<td></td>
<td>(1.24)</td>
<td>(0.13)</td>
<td>(0.91)</td>
<td>(0.12)</td>
</tr>
<tr>
<td>Positive Symptom Distress Index</td>
<td>2.13</td>
<td>0.58</td>
<td>2.06</td>
<td>0.64</td>
</tr>
<tr>
<td></td>
<td>(2.03)</td>
<td>(0.15)</td>
<td>(2.08)</td>
<td>(0.14)</td>
</tr>
<tr>
<td>Positive Symptom Total</td>
<td>54.07</td>
<td>14.96</td>
<td>39.81</td>
<td>16.29</td>
</tr>
<tr>
<td></td>
<td>(52.56)</td>
<td>(3.87)</td>
<td>(38.13)</td>
<td>(3.63)</td>
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</table>

*All degrees of freedom = 2, 48, *\( p \leq .01, **p < .001. \)
Main Analyses

The main hypotheses of this study were that differences in performance would be found between the medicated depressed, non-medicated depressed, and non-medicated non-depressed groups (independent variables) on five measures of executive functioning (depaendant variables; WCST, TMT A & B, TOL, Stroop, & COWA), and primarily at the group Pairwise Comparison level. The following text reviews the results of those analyses.

Hypothesis I

To test the first hypothesis that differences would be found between the medicated depressed, non-medicated depressed, and non-medicated non-depressed groups on the WCST, a MANCOVA was performed using the eleven subscales of Wisconsin Card Sorting Test (WCST) as the dependent variables, participant age and WAIS-III Vocabulary scores as the covariates, and group membership as the independent variables. The overall MANCOVA was not significant, Wilkes' $\Lambda = .54, F(18, 80) = 1.61, p = .078,$ partial $\eta^2 = .27$. However, the strength of the relationship between group membership and scores on the WCST was quite strong as assessed by the partial $\eta^2$.

Despite the non-significant omnibus test, specific analyses (ANCOVAs) were performed on each subscale of the WCST (Trials Administered, Total Correct, Total Errors, Perseverative Responses, Perseverative Errors, Nonperseverative Errors, Conceptual Level Response, Categories Completed, Trials to Complete 1st Category, Failure to Maintain Set, & Learning to Learn) as follow-up tests to the MANCOVA. Each ANCOVA was tested at the .05 level and of the eleven subscales only two revealed statistically significant differences, the Trials Administered, $F(2, 48) = 3.74, p = .031,$
partial $\eta^2 = .135$ and the Failure to Maintain Set, $F(2, 48) = 3.30, p = .045$, partial $\eta^2 = .121$.

Post hoc pairwise analyses to the ANCOVAs (with participant age and WAIS-III Vocabulary scores serving as the covariates) for the Trials Administered and Failure to Maintain Set subscales of the WCST were performed using a Bonferroni correction with a .005 significance level in order to control for Type I error. No group comparisons reached statistical significance. Full ANCOVA results are presented in Table 5 comparing the three groups on the various subscales of the WCST as well as the raw score means and standard deviations.

**Hypothesis II**

To test the second hypothesis that differences would be found between the medicated depressed, non-medicated depressed, and non-medicated non-depressed groups on the Trail Making Test (TMT) parts A and B (Reitan, 1993), a MANCOVA was performed using the TMT parts A and B as the dependent variables, participant age and WAIS-III Vocabulary scores as the covariates, and group membership as the independent variables. The overall MANCOVA was not significant, Wilkes' $\Lambda = .826, F(4, 94) = 2.3514, p = .060$, partial $\eta^2 = .091$. The strength of the relationship between group membership and scores on the TMT was fairly strong as assessed by the partial $\eta^2$.

ANCOVAs were also performed as follow-up tests to the MANCOVA. Each ANCOVA was tested at the .05 level. The ANCOVA for TMT Part B was statistically significant, $F(2, 48) = 4.45, p = .017$, partial $\eta^2 = .156$, while the ANCOVA on TMT Part A was not, $F(2, 48) = 0.75, p = .479$, partial $\eta^2 = .030$. 

<table>
<thead>
<tr>
<th>WCST</th>
<th>Medicated Depressed n = 15</th>
<th>Non-Medicated Depressed n = 16</th>
<th>Non-Medicated Non-Depressed n = 22</th>
<th>All Groups(^a) ANCOVA N = 53</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trials Administered</td>
<td>M = 102.67 (EMM) 100.13 (SE) 5.75</td>
<td>M = 103.06 (EMM) 103.10 (SE) 5.39</td>
<td>M = 82.82 (EMM) 84.52 (SE) 4.67</td>
<td>F = 3.740* (\text{Partial } \eta^2 = 0.135)</td>
</tr>
<tr>
<td>Total Correct</td>
<td>75.33 (EMM) 75.06 (SE) 2.75</td>
<td>71.19 (EMM) 70.71 (SE) 2.76</td>
<td>67.73 (EMM) 68.27 (SE) 2.23</td>
<td>F = 1.680 (\text{Partial } \eta^2 = 0.065)</td>
</tr>
<tr>
<td>Total Errors</td>
<td>27.33 (EMM) 25.08 (SE) 5.12</td>
<td>31.88 (EMM) 32.39 (SE) 4.79</td>
<td>15.09 (EMM) 16.25 (SE) 4.16</td>
<td>F = 3.104 (\text{Partial } \eta^2 = 0.115)</td>
</tr>
<tr>
<td>Perseverative Responses</td>
<td>14.27 (EMM) 13.10 (SE) 2.64</td>
<td>16.19 (EMM) 16.65 (SE) 2.47</td>
<td>8.05 (EMM) 8.51 (SE) 2.14</td>
<td>F = 2.986 (\text{Partial } \eta^2 = 0.111)</td>
</tr>
<tr>
<td>Perseverative Errors</td>
<td>12.87 (EMM) 12.01 (SE) 2.25</td>
<td>14.44 (EMM) 14.78 (SE) 2.11</td>
<td>7.68 (EMM) 8.01 (SE) 1.83</td>
<td>F = 2.854 (\text{Partial } \eta^2 = 0.106)</td>
</tr>
<tr>
<td>Nonperseverative Errors</td>
<td>14.47 (EMM) 13.07 (SE) 3.10</td>
<td>17.44 (EMM) 17.61 (SE) 2.90</td>
<td>7.41 (EMM) 8.24 (SE) 2.52</td>
<td>F = 2.838 (\text{Partial } \eta^2 = 0.106)</td>
</tr>
<tr>
<td>Conceptual Level Response</td>
<td>67.27 (EMM) 67.62 (SE) 3.26</td>
<td>63.81 (EMM) 63.17 (SE) 3.06</td>
<td>64.86 (EMM) 65.10 (SE) 2.65</td>
<td>F = 0.476 (\text{Partial } \eta^2 = 0.019)</td>
</tr>
</tbody>
</table>
Table 5 (cont.). Raw Score Means, Standard Deviations, Estimated Marginal Means, Standard Errors, and ANCOVA results for Wisconsin Card Sorting Test (WCST).

<table>
<thead>
<tr>
<th></th>
<th>Medicated Depressed n = 15</th>
<th>Non-Medicated Depressed n = 16</th>
<th>Non-Medicated Non-Depressed n = 22</th>
<th>All Groups ANCOVA N = 53</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M (EMM) SD (SE)</td>
<td>M (EMM) SD (SE)</td>
<td>M (EMM) SD (SE)</td>
<td>F          Partial $\eta^2$</td>
</tr>
<tr>
<td>WCST</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Categories Completed</td>
<td>4.93 (5.09) 1.94 (0.44)</td>
<td>4.69 (4.69) 1.99 (0.41)</td>
<td>5.86 (5.76) 0.64 (0.36)</td>
<td>1.849      0.072</td>
</tr>
<tr>
<td>Trials to Complete 1st Category</td>
<td>25.40 (26.01) 21.72 (6.65)</td>
<td>24.19 (24.01) 29.16 (6.23)</td>
<td>11.86 (11.53) 2.25 (5.40)</td>
<td>1.678      0.065</td>
</tr>
<tr>
<td>Failure to Maintain Set</td>
<td>1.40 (1.35) 1.50 (0.32)</td>
<td>1.19 (1.18) 1.28 (0.30)</td>
<td>0.32 (0.36) 0.48 (0.26)</td>
<td>3.303*     0.121</td>
</tr>
<tr>
<td>Learning to Learn</td>
<td>0.166 (0.86) 1.78 (1.18)</td>
<td>-0.27 (-0.56) 3.11 (1.11)</td>
<td>-1.35 (-1.60) 5.78 (0.96)</td>
<td>1.193      0.047</td>
</tr>
</tbody>
</table>

*All degrees of freedom = 2, 48, *p < .05.
Post hoc pairwise analyses to the ANCOVAs (with participant age and the WAIS-III Vocabulary scores serving as covariates) for the TMT parts A and B were performed using the Bonferroni correction with a .025 significance level in order to control for Type I errors. While significant differences were found between the medicated depressed (Seconds to completion; \( M = 71.6; \ SD = 24.2 \)) and non-medicated non-depressed (Seconds to completion; \( M = 44.3; \ SD = 10.0 \)) groups on TMT part B \( (p = .017) \), no other comparisons reached statistical significance. Full ANCOVA results are presented in Table 6 comparing the three groups on the various subscales of the TMT parts A and B as well as the raw score means, standard deviations, estimated marginal means (EMM), standard error of estimates (SEE), and ANCOVA results.

Table 6. Raw Score Means, Standard Deviations, Estimated Marginal Means (EMM), Standard Error of Estimates (SEE), and ANCOVA results for Trail Making Test, Part A & B.

<table>
<thead>
<tr>
<th>Group</th>
<th>Statistic</th>
<th>( M )</th>
<th>( SD )</th>
<th>EMM</th>
<th>SEE</th>
<th>( F )</th>
<th>( df )</th>
<th>( \text{Partial } \eta^2 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Medicated</td>
<td></td>
<td>27.80</td>
<td>7.21</td>
<td>25.57</td>
<td>2.04</td>
<td>0.748</td>
<td>2.48</td>
<td>0.030</td>
</tr>
<tr>
<td>Depressed (n = 15)</td>
<td></td>
<td>71.60</td>
<td>24.20</td>
<td>66.40</td>
<td>5.04</td>
<td>4.445*</td>
<td>2.48</td>
<td>0.156</td>
</tr>
<tr>
<td>Non-Medicated</td>
<td></td>
<td>25.06</td>
<td>9.26</td>
<td>25.74</td>
<td>1.92</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depressed (n = 16)</td>
<td></td>
<td>56.88</td>
<td>22.29</td>
<td>58.59</td>
<td>4.72</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-Medicated</td>
<td></td>
<td>21.86</td>
<td>6.69</td>
<td>22.90</td>
<td>1.66</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-Depressed (n = 22)</td>
<td></td>
<td>44.323</td>
<td>9.97</td>
<td>46.62</td>
<td>4.10</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All Groups(^a)</td>
<td></td>
<td>0.748</td>
<td>4.445*</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

\(^a\)All degrees of freedom = 2, 48 , \(^*\)p < .05.
Hypothesis III

To test the third hypothesis that differences would be found between the medicated depressed, non-medicated depressed, and non-medicated non-depressed groups on the Tower of London: Drexel Edition (TOL\textsuperscript{DX}; Culbertson & Zillmer, 2005), a MANCOVA was performed using the five subscales of TOL\textsuperscript{DX} as the dependent variables, participant age and WAIS-III Vocabulary scores as the covariates, and group membership as the independent variables. The overall MANCOVA was not significant, Wilkes' $\Lambda = .813$, $F(10, 88) = .960$, $p = .483$, partial $\eta^2 = .098$. The effect of group membership on scores of the TOL\textsuperscript{DX} was fairly strong as assessed by the partial $\eta^2$.

ANCOVAs, with participant age and the WAIS-III Vocabulary scores serving as covariates, were performed on each subscale of the TOL\textsuperscript{DX} (Total Correct, Total Move Score, Total Time, Time Violation, & Rule Violation) as follow-up tests to the MANCOVA. Each ANCOVA was tested at the .05 level. Of the five subscales tested, none revealed statistically significant differences. Full ANCOVA results are presented in Table 7 comparing the three groups on the five subscales of the TOL\textsuperscript{DX} as well as the raw score means and standard deviations.

Table 7. Raw Score Means, Standard Deviations, Estimated Marginal Means (EMM), Standard Error of Estimates (SEE), and ANCOVA results for Tower of London-Drexel University Test (TOL\textsuperscript{DX}).

<table>
<thead>
<tr>
<th>Group</th>
<th>Statistic</th>
<th>Total Correct</th>
<th>Total Move Score</th>
<th>Total Time (sec.)</th>
<th>Total Time Violation</th>
<th>Rule Violation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Medicated</td>
<td>$M$</td>
<td>3.87</td>
<td>34.00</td>
<td>315.27</td>
<td>1.07</td>
<td>0.27</td>
</tr>
<tr>
<td>Depressed</td>
<td>$SD$</td>
<td>2.95</td>
<td>20.92</td>
<td>139.78</td>
<td>1.44</td>
<td>0.60</td>
</tr>
<tr>
<td>(n = 15)</td>
<td>EMM</td>
<td>3.96</td>
<td>33.64</td>
<td>301.68</td>
<td>0.93</td>
<td>0.20</td>
</tr>
<tr>
<td></td>
<td>SEE</td>
<td>0.67</td>
<td>4.90</td>
<td>29.43</td>
<td>0.30</td>
<td>0.15</td>
</tr>
</tbody>
</table>
Table 7 cont. Raw Score Means, Standard Deviations, Estimated Marginal Means (EMM), Standard Error of Estimates (SEE), and ANCOVA results for Tower of London-Drexel University Test (TOLDX).

<table>
<thead>
<tr>
<th>Group</th>
<th>Statistic</th>
<th>Total Correct</th>
<th>Total Move Score</th>
<th>Total Time (sec.)</th>
<th>Total Time Violation</th>
<th>Rule Violation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-Medicated</td>
<td>$M$</td>
<td>5.06</td>
<td>26.38</td>
<td>266.69</td>
<td>0.50</td>
<td>0.38</td>
</tr>
<tr>
<td>Depressed</td>
<td>$SD$</td>
<td>2.62</td>
<td>19.52</td>
<td>106.54</td>
<td>0.82</td>
<td>0.62</td>
</tr>
<tr>
<td>(n = 16)</td>
<td>EMM</td>
<td>5.55</td>
<td>23.68</td>
<td>268.14</td>
<td>0.50</td>
<td>0.42</td>
</tr>
<tr>
<td></td>
<td>SEE</td>
<td>0.62</td>
<td>4.59</td>
<td>27.57</td>
<td>0.28</td>
<td>0.14</td>
</tr>
<tr>
<td>Non-Medicated</td>
<td>$M$</td>
<td>5.77</td>
<td>22.73</td>
<td>242.23</td>
<td>0.60</td>
<td>0.23</td>
</tr>
<tr>
<td>Non-Depressed</td>
<td>$SD$</td>
<td>2.02</td>
<td>13.82</td>
<td>68.50</td>
<td>0.85</td>
<td>0.43</td>
</tr>
<tr>
<td>(n = 22)</td>
<td>EMM</td>
<td>5.36</td>
<td>24.94</td>
<td>250.44</td>
<td>0.68</td>
<td>0.25</td>
</tr>
<tr>
<td></td>
<td>SEE</td>
<td>0.54</td>
<td>3.98</td>
<td>23.91</td>
<td>0.24</td>
<td>0.12</td>
</tr>
<tr>
<td>All Groups*</td>
<td>$F$</td>
<td>1.728</td>
<td>1.237</td>
<td>0.836</td>
<td>0.551</td>
<td>0.623</td>
</tr>
<tr>
<td>ANCOVA</td>
<td>$df$</td>
<td>2.48</td>
<td>2.48</td>
<td>2.48</td>
<td>2.48</td>
<td>2.48</td>
</tr>
<tr>
<td>(N = 53)</td>
<td>Partial $\eta^2$</td>
<td>0.07</td>
<td>0.05</td>
<td>0.03</td>
<td>0.02</td>
<td>0.03</td>
</tr>
</tbody>
</table>

**Hypothesis IV**

To test the fourth hypothesis that differences would be found between the medicated depressed, non-medicated depressed, and non-medicated non-depressed groups on the Stroop Color and Word Test (Golden & Freshwater, 2002), a MANCOVA was performed using the four subscores of the Stroop as the dependent variables, participant age and WAIS-III Vocabulary scores as the covariates, and group membership as the independent variables. The overall MANCOVA was significant, Wilkes’ $\Lambda = .645$, $F(8, 90) = 2.76, p = .009$, partial $\eta^2 = .197$. The strength of the relationship between group membership and scores on the Stroop was quite strong as assessed by the partial $\eta^2$.

Analyses of covariance (ANCOVA) were performed on each subscale of the Stroop (Word, Color, Color-Word, & Interference) as follow-up tests to the MANCOVA. Each ANCOVA was tested at the .05 level and all four of the analyses revealed
statistically significant results; Word raw scores, $F(2, 48) = 3.23, p = .048$, partial $\eta^2 = .119$; Color raw score, $F(2, 48) = 5.72, p = .006$, partial $\eta^2 = .192$; Color-Word raw score, $F(2, 48) = 6.72, p = .003$, partial $\eta^2 = .219$; and Interference raw score, $F(2, 48) = 4.06, p = .024$, partial $\eta^2 = .145$, respectively.

Post hoc pairwise analyses to the ANCOVAs (with the participant age and WAIS-III Vocabulary scores serving as covariates) for the four Stroop subscales were performed using the Bonferroni correction with a .013 significance level in order to control for Type I errors. Significant differences were found on the Color raw score subscale between the non-medicated non-depressed group ($M = 82.05, SD = 11.34$) and the medicated depressed group ($M = 67.47, SD = 9.83; p = .005$) and the Color-Word raw score subscale between the non-medicated non-depressed group ($M = 52.10, SD = 13.66$) and the non-medicated depressed group ($M = 40.63, SD = 10.38; p = .004$). No other group comparisons reached statistical significance. Full ANCOVA results are presented in Table 8 comparing the three groups on the subscores of the Stroop as well as the raw score means and standard deviations.

Table 8. Raw Score Means, Standard Deviations, Estimated Marginal Means (EMM), Standard Error of Estimates (SEE), and ANCOVA results for Stroop Color-Word Test.

<table>
<thead>
<tr>
<th>Group</th>
<th>Statistic</th>
<th>Word</th>
<th>Color</th>
<th>Color-Word</th>
<th>Interference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Medicated</td>
<td>$M$</td>
<td>90.07</td>
<td>67.47</td>
<td>39.93</td>
<td>3.13</td>
</tr>
<tr>
<td>Depressed</td>
<td>$SD$</td>
<td>13.92</td>
<td>9.83</td>
<td>8.04</td>
<td>8.30</td>
</tr>
<tr>
<td>(n = 15)</td>
<td>EMM</td>
<td>91.82</td>
<td>68.62</td>
<td>14.34</td>
<td>3.66</td>
</tr>
<tr>
<td></td>
<td>SEE</td>
<td>3.93</td>
<td>2.84</td>
<td>3.12</td>
<td>2.93</td>
</tr>
<tr>
<td>Non-Medicated</td>
<td>$M$</td>
<td>100.94</td>
<td>75.63</td>
<td>40.63</td>
<td>-2.25</td>
</tr>
<tr>
<td>Depressed</td>
<td>$SD$</td>
<td>14.08</td>
<td>8.09</td>
<td>10.38</td>
<td>9.74</td>
</tr>
<tr>
<td>(n = 16)</td>
<td>EMM</td>
<td>100.82</td>
<td>75.22</td>
<td>38.82</td>
<td>-3.54</td>
</tr>
<tr>
<td></td>
<td>SEE</td>
<td>3.69</td>
<td>2.66</td>
<td>2.92</td>
<td>2.74</td>
</tr>
</tbody>
</table>
Table 8 (cont.). Raw Score Means, Standard Deviations, Estimated Marginal Means (EMM), Standard Error of Estimates (SEE), and ANCOVA results for Stroop Color-Word Test.

<table>
<thead>
<tr>
<th>Group</th>
<th>Statistic</th>
<th>Word</th>
<th>Color</th>
<th>Color-Word</th>
<th>Interference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-Medicated</td>
<td>$M$</td>
<td>106.36</td>
<td>82.05</td>
<td>52.10</td>
<td>6.36</td>
</tr>
<tr>
<td>Non-Depressed (n = 22)</td>
<td>$SD$</td>
<td>13.79</td>
<td>11.34</td>
<td>13.66</td>
<td>12.10</td>
</tr>
<tr>
<td></td>
<td>EMM</td>
<td>105.25</td>
<td>81.55</td>
<td>52.45</td>
<td>6.94</td>
</tr>
<tr>
<td></td>
<td>SEE</td>
<td>3.20</td>
<td>2.31</td>
<td>2.53</td>
<td>2.38</td>
</tr>
<tr>
<td>All Groups$^a$</td>
<td>$F$</td>
<td>3.23*</td>
<td>5.72*</td>
<td>6.72*</td>
<td>4.06*</td>
</tr>
<tr>
<td>ANCOVA (N = 53)</td>
<td>$df$</td>
<td>2, 48</td>
<td>2, 48</td>
<td>2.48</td>
<td>2.48</td>
</tr>
<tr>
<td></td>
<td>$Partial \eta^2$</td>
<td>0.12</td>
<td>0.19</td>
<td>0.22</td>
<td>0.15</td>
</tr>
</tbody>
</table>

$^a$ p < .05

**Hypothesis V**

To test the fifth hypothesis that differences would be found between the medicated depressed, non-medicated depressed, and non-medicated non-depressed groups on the Controlled Oral Word Association test (COWA; Spreen & Straus, 1998), a one-way analysis of covariance (ANCOVA) was performed using the COWA as the dependent variable, participant age and WAIS-III Vocabulary scores as the covariates, and group membership as the independent variables. The ANCOVA was not significant, $F(2, 48) = .443, MSE = 109.36, p = .645$, partial $\eta^2 = .018$. The effect of group membership on one’s COWA score was strong as assessed by the partial $\eta^2$. Full ANCOVA results are presented in Table 9 comparing the three groups on the COWA as well as the raw score means and standard deviations.

**Post Hoc Analyses**

While the core of the present study was to examine if depression affects individuals’ executive functions, several multiple linear regressions were also completed.
Table 9. Raw Score Means, Standard Deviations, Estimated Marginal Means (EMM), Standard Error of Estimates (SEE), and ANCOVA results for Controlled Oral Word Association test.

<table>
<thead>
<tr>
<th>Group</th>
<th>Statistic</th>
<th>COWA (sec.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Medicated</td>
<td>$M$</td>
<td>33.27</td>
</tr>
<tr>
<td>Depressed</td>
<td>$SD$</td>
<td>10.07</td>
</tr>
<tr>
<td>(n = 15)</td>
<td>EMM</td>
<td>34.33</td>
</tr>
<tr>
<td></td>
<td>SEE</td>
<td>2.94</td>
</tr>
<tr>
<td>Non-Medicated</td>
<td>$M$</td>
<td>36.38</td>
</tr>
<tr>
<td>Depressed</td>
<td>$SD$</td>
<td>11.86</td>
</tr>
<tr>
<td>(n = 16)</td>
<td>EMM</td>
<td>37.73</td>
</tr>
<tr>
<td></td>
<td>SEE</td>
<td>2.75</td>
</tr>
<tr>
<td>Non-Medicated</td>
<td>$M$</td>
<td>39.36</td>
</tr>
<tr>
<td>Non-Depressed</td>
<td>$SD$</td>
<td>10.28</td>
</tr>
<tr>
<td>(n = 22)</td>
<td>EMM</td>
<td>37.65</td>
</tr>
<tr>
<td></td>
<td>SEE</td>
<td>2.39</td>
</tr>
<tr>
<td>All Groups$^a$</td>
<td>$F$</td>
<td>0.44</td>
</tr>
<tr>
<td>ANCOVA</td>
<td>$df$</td>
<td>2.48</td>
</tr>
<tr>
<td>(N = 53)</td>
<td>$Partial \eta^2$</td>
<td>0.02</td>
</tr>
</tbody>
</table>

in order to gain a better understanding of whether or not anxiety, as measured by the SCL-90-R Anxiety dimension, added any predictive ability to the executive function measures. A set of stepwise regression analyses were conducted on the combined groups to evaluate whether raw scores on the second independent variable, the Anxiety dimension of the SCL-90-R, predicted performance on the measures of executive functioning above and beyond that of the BDI-II.

Indeed, the Anxiety dimension accounted for a statistically significant proportion of the variance after controlling for the effects of the BDI-II score on the following measures of executive function: WCST Total Errors ($\Delta R^2 = .066, p = .049$), WCST Perseverative Responses ($\Delta R^2 = 0.93, p = .018$), WCST Perseverative Errors ($\Delta R^2 = .099$).
$p = .015$), WCST Categories Completed ($\Delta R^2 = .080, p = .033$), WCST Learning to Learn ($\Delta R^2 = .077, p = .045$), Stroop Word ($\Delta R^2 = .109, p = .009$), and the Stroop Color ($\Delta R^2 = .113, p = .003$). Such results indicate that anxiety scores on the SCL-90-R, in several cases, accounted for significant amounts of variance in EF above and beyond that of scores on the BDI-II. Table 10 presents the data from the regression analyses.

Table 10. Regression Coefficients of the Statistically Significant Executive Function Measures with the BDI-II (Step 1) and SCL-90-R (Step 2) as Independent Variables.

<table>
<thead>
<tr>
<th>Criterion</th>
<th>$R^2$</th>
<th>$\Delta R^2$</th>
<th>$\Delta F$</th>
<th>$df$s</th>
<th>$p$</th>
</tr>
</thead>
<tbody>
<tr>
<td>WCST</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trials Administered</td>
<td>.506</td>
<td>.056</td>
<td>3.78</td>
<td>1, 50</td>
<td>.057</td>
</tr>
<tr>
<td>Total Correct</td>
<td>.109</td>
<td>.001</td>
<td>0.07</td>
<td>1, 50</td>
<td>.794</td>
</tr>
<tr>
<td>Total Errors</td>
<td>.187</td>
<td>.066</td>
<td>4.06</td>
<td>1, 50</td>
<td>.049*</td>
</tr>
<tr>
<td>Perseverative Responses</td>
<td>.224</td>
<td>.093</td>
<td>5.97</td>
<td>1, 50</td>
<td>.018*</td>
</tr>
<tr>
<td>Perseverative Errors</td>
<td>.225</td>
<td>.099</td>
<td>6.41</td>
<td>1, 50</td>
<td>.015*</td>
</tr>
<tr>
<td>Nonperseverative Errors</td>
<td>.142</td>
<td>.039</td>
<td>2.30</td>
<td>1, 50</td>
<td>.136</td>
</tr>
<tr>
<td>Conceptual Level Response</td>
<td>.030</td>
<td>.023</td>
<td>1.18</td>
<td>1, 50</td>
<td>.283</td>
</tr>
<tr>
<td>Categories Completed</td>
<td>.173</td>
<td>.080</td>
<td>4.83</td>
<td>1, 50</td>
<td>.033*</td>
</tr>
<tr>
<td>Trials to Complete 1st Category</td>
<td>.123</td>
<td>.061</td>
<td>3.46</td>
<td>1, 50</td>
<td>.069</td>
</tr>
<tr>
<td>Failure to Maintain Set</td>
<td>.201</td>
<td>.017</td>
<td>1.04</td>
<td>1, 50</td>
<td>.313</td>
</tr>
<tr>
<td>Learning to Learn</td>
<td>.086</td>
<td>.077</td>
<td>4.22</td>
<td>1, 50</td>
<td>.045*</td>
</tr>
<tr>
<td>Trail Making Test</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Part A</td>
<td>.201</td>
<td>.027</td>
<td>1.66</td>
<td>1, 50</td>
<td>.203</td>
</tr>
<tr>
<td>Part B</td>
<td>.247</td>
<td>.052</td>
<td>3.44</td>
<td>1, 50</td>
<td>.070</td>
</tr>
<tr>
<td>Tower of London $^{DX}$</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total Correct</td>
<td>.117</td>
<td>.005</td>
<td>0.29</td>
<td>1, 50</td>
<td>.592</td>
</tr>
<tr>
<td>Total Moves</td>
<td>.063</td>
<td>.001</td>
<td>0.03</td>
<td>1, 50</td>
<td>.854</td>
</tr>
<tr>
<td>Total Time</td>
<td>.127</td>
<td>.056</td>
<td>3.18</td>
<td>1, 50</td>
<td>.081</td>
</tr>
<tr>
<td>Time Violation</td>
<td>.084</td>
<td>.067</td>
<td>3.65</td>
<td>1, 50</td>
<td>.062</td>
</tr>
<tr>
<td>Rule Violation</td>
<td>.084</td>
<td>.073</td>
<td>3.97</td>
<td>1, 50</td>
<td>.052</td>
</tr>
</tbody>
</table>
Table 10 (cont.). Regression Coefficients of the Statistically Significant Executive Function Measures with the BDI-II (Step 1) and SCL-90-R (Step 2) as Independent Variables.

<table>
<thead>
<tr>
<th>Criterion</th>
<th>$R^2$</th>
<th>$\Delta R^2$</th>
<th>$\Delta F$</th>
<th>dfs</th>
<th>$p$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Controlled Oral Word</td>
<td>.070</td>
<td>.021</td>
<td>1.11</td>
<td>1, 50</td>
<td>.298</td>
</tr>
<tr>
<td><strong>Stroop</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Word</td>
<td>.277</td>
<td>.107</td>
<td>7.42</td>
<td>1, 50</td>
<td>.009*</td>
</tr>
<tr>
<td>Color</td>
<td>.413</td>
<td>.113</td>
<td>9.66</td>
<td>1, 50</td>
<td>.003*</td>
</tr>
<tr>
<td>Color-Word</td>
<td>.176</td>
<td>.001</td>
<td>0.04</td>
<td>1, 50</td>
<td>.840</td>
</tr>
<tr>
<td>Interference</td>
<td>.047</td>
<td>.017</td>
<td>0.88</td>
<td>1, 50</td>
<td>.352</td>
</tr>
</tbody>
</table>

* = Statistically Significant

Of particular note is the regression analysis on the dependent variable of WCST Learning to Learn and the independent variable, the BDI-II. This was not significant; however, the second step using the Anxiety dimension of the SCL-90-R was statistically significant, $R^2$ change = .077, $F(1, 50)$ = 4.224, $p$ = .045. This suggests that while depression, as scored on the BDI-II, does not predict scores on the WCST Learning to Learn subscale at a statistically significant level, Anxiety, as scored on the SCL-90-R, does. This is noteworthy as the Anxiety dimension of the SCL-90-R is only able to account for variance that has been left over from depression, as measured by the BDI-II. Thus, this data suggests that anxiety has more predictive ability regarding the WCST Learning to Learn subscale than depression.
CHAPTER V
DISCUSSION

Depression is a psychological condition that afflicts approximately 121 million people worldwide (WHO, 2001) and has been estimated to cost employers over $33 billion dollars a year due to missed work and decreased productivity (Quinn, 2000). The deleterious effects of depression are typically not limited to one painful experience. Barlow and Durand (1999) have reported that as many as 80% of depressed individuals experience additional, and often more severe, depressive episodes later on in their lives.

While several areas of science have investigated the etiology of depression, WHO (2001) asserted that there is a combination of biological, genetic, and psychological underpinnings at the core of depression. The effects of depression on various aspects of human functioning have traditionally been a major area of research. The brain and effects depression has upon brain functioning have also been major areas of interest to scientific investigation. More specifically, the frontal lobes, the area of the brain believed to house higher-order regulatory and supervisory functions in human beings (Zillmer et al., 2001), have recently seen increased interest. However, the effects of psychotropic medications and differences in various age ranges have left this area of investigation with many questions unanswered.

Thus, the purpose of the current study was to assess young adult individuals on measures of executive functioning in a depressed and non-depressed sample.
Furthermore, medication status was addressed by separating the depressed group into two separate groups, medicated and non-medicated. Additionally, participants were required to be between the ages of 19 and 40. These restrictions allowed for a purer understanding of the effects of depression on the executive functions of depressed young adults. The following discusses the present study’s findings.

Preliminary Analyses

The preliminary analyses revealed that there were statistically significant differences between the three groups in regards to intelligence, as measured by the Vocabulary subtest of the WAIS-III (Wechsler, 1997). Furthermore, there were statistically significant age differences between the three groups. This suggests that the three groups differed enough in terms of age and estimated intelligence that these variables were used as covariates in the present study. This statistical procedure allows one to compare groups on a variable of interest by simultaneously controlling for the effect of a confounding variable. In this case, it allowed for the examination of executive functioning, while simultaneously controlling for age and estimated intelligence differences between the three groups.

The preliminary analyses also revealed that there were statistically significant group differences on the Global Severity Index (GSI) of the SCL-90-R, which was used to evaluate overall psychological well-being. As hypothesized, there were statistically significant differences between the non-medicated non-depressed group and the two depressed groups suggesting that the individuals in the non-medicated non-depressed group possessed better overall psychological well-being than the two depressed groups.
Furthermore, the GSI revealed no statistically significant differences between the two depressed groups.

The analyses revealed no statistically significant differences between the two depressed groups on any of the SCL-90-R subscales even beyond the GSI. There were some subscales that failed to reveal statistically significant differences between the non-depressed and the depressed groups. For example, the non-medicated non-depressed group and the non-medicated depressed group failed to reveal statistically significant differences on the Interpersonal Sensitivity, Anxiety, and Paranoid subscales and neither of the depressed groups differed from the non-medicated non-depressed group on the Phobic Anxiety and Hostility subscales at a statistically significant level.

That there were no statistically significant differences between the two depressed groups suggests that they are similar in their level of psychological distress. However, this does not indicate or suggest that there depressive symptomatology is similar. While there are similarities between depressive symptoms, there are different ways in which one can meet criteria for the clinical diagnosis. For example, one may be suicidal and another individual may not be; yet both can still obtain similar scores on measures of depressive symptomatology.

Main Hypotheses

The main hypotheses of the current study were that medicated depressed, non-medicated depressed, and non-medicated non-depressed participants would perform differently on tests of executive functioning at a statistically significant level.
Hypothesis I

Hypothesis I stated that statistically significant differences would be found between the medicated depressed, non-medicated depressed, and non-medicated non-depressed groups on the Wisconsin Card Sorting Test (WCST). The overall omnibus test was not significant. These findings are contrary to much of the research literature in this area (Channon, 1996; Grant et al., 2001; & Merriam et al., 1999). Although the omnibus test indicated that there were no statistically significant differences between the three groups on their overall WCST performance, there were statistically significant differences on two subscales, Trials Administered and Failure to Maintain Set.

The Trials Administered score is simply the total number of cards used in the administration of the WCST. Little research has been completed investigating the Trials Administered scale's effect upon the overall WCST. However, conceptually, an increased number of trials administered score is likely to affect other scale scores (e.g., Total Number Correct, Total Number of Errors, etc.). The other subscale that revealed statistically significant differences between groups was the Failure to Maintain Set subscale. A failure to maintain a set is achieved on the WCST (Heaton et al., 1993) when one "makes five or more consecutive correct matches but then makes an error before successfully completing the category" (p. 12). Therefore, while there may appear to be some understanding involved of the task, errors made suggest that the examinee was accidentally sorting the cards correctly based on an inaccurate understanding of the sorting principle.

Although overall group differences were found on these two subscales (Trials Administered and Failure to Maintain Set), no specific group differences reached
statistical significance. This suggests that group differences are present when all groups are considered together, but not when individual groups are compared with one another.

The WCST manual offers no information on how one should interpret the Trials Administered subscale or if one should at all. One study that reported data from the Trials Administered scale (Fey, 1951) found statistically significant differences between young adult schizophrenics and healthy controls. Another (Channon, 1996) found statistically significant differences between dysphoric and non-dysphoric participants on the Trials Administered subscale. However, neither study discussed the implications of the finding, thus, providing little insight into the meaning of Trials Administered performance. Furthermore, that the pairwise comparisons failed to reveal statistically significant individual group differences, the current study is unable to interpret the effects of depression on Trials Administered performance.

The current finding that statistically significant overall group differences were also found on the Failure to Maintain Set subscale is contrary to some previous research (Merriam et al., 1999). However, this finding is congruent with others (e.g., Channon, 1996). As with the Trials Administered subscale, no individual group differences reached statistical significance in regards to the Failure to Maintain Set subscale. Although the Failure to Maintain Set subscale measures one’s ability to shift and maintain a set as well as one’s ability to make use of feedback (Heaton et al., 1993), the current data do not allow for a discussion of which group is more affected in this area than another. Due to the lack of pairwise follow-up significance between any of the three groups, it can only be speculated as to which group outperformed the others. As such speculation is unacceptable in scientific research the need for further research to shed light into such a
Hypothesis II stated that statistically significant differences would be found between the medicated depressed, non-medicated depressed, and non-medicated non-depressed groups on the Trail Making Test (TMT) parts A and B (Reitan, 1993). Tests of between-subjects effects revealed statistically significant group differences on the TMT Part B, but not Part A. Follow-up pairwise comparisons on TMT Part B revealed that the non-medicated non-depressed group outperformed the medicated depressed group at a statistically significant level while no statistically significant differences were found between the two depressed groups nor the non-medicated non-depressed and non-medicated depressed groups.

The TMT (Parts A & B) are considered to be “tests of speed for attention, mental flexibility, and of visual search and motor function” (Spreen & Strauss, 1998). Heilbronner et al. (1991) reported that parts A and B might measure different functions, which highlights the importance of understanding what Part B is believed to actually measure. The primary difference between Parts A and B is that there are numbers and letters that one must alternate between while completing Part B. On TMT Part B, in particular, one must analyze the stimulus material and then integrate the numbers and letters into a single sequence (Reitan & Wolfson, 1993). Additionally, Part B requires more visual perceptual processing, psychomotor ability, and mental flexibility (Golden et al., 2000; Woodruff, Mendoza, Dickson, Blanchard, & Christenberry, 1995). Anderson (1994) noted that Part B assesses number and letter recognition, visual scanning,
cognitive flexibility, visual-spatial functioning, and visuomotor coordination. Moreover, as noted previously, Part B is considered to be "one of the best general indicators of cerebral dysfunction" (Thomas, 2000, p. 171).

The current findings are consistent with the results reported by others such as Paradiso et al. (1997) and Reitan and Wolfson (1993). However, the present data are contrary to others who have found no such differences on overall TMT performance (e.g., Grant et al., 2001; Landrø et al., 2001). Of particular interest is the current finding that the medicated depressed group obtained higher average times to complete Part B than the non-medicated non-depressed group. The fact that no differences were found between the non-medicated depressed and the non-medicated non-depressed groups might initially suggest that medication status may affect performance on this measure. However, the lack of statistically significant differences between the two depressed groups cast doubt on such an assertion.

Although medication status cannot completely account for the difference between groups in the current study, important information can be gleaned from these results. As noted previously, Part B assesses several important areas of executive functioning that are necessary for adequate daily functioning (e.g., number and letter recognition, visual scanning, and cognitive flexibility [Anderson, 1994]). Thus, the current data suggest that depressed individuals taking antidepressant medications may very well experience greater difficulties in regards to the aforementioned areas than others.

**Hypothesis III**

Hypothesis III stated that statistically significant differences would be found between the medicated depressed, non-medicated depressed, and non-medicated non-
depressed groups on the Tower of London, Drexel University: 2nd Edition (TOL^DX; Culbertson & Zillmer, 2005). The overall omnibus test was not statistically significant indicating that participants in the three groups did not perform differently on these measures.

The TOL^DX was designed to assess one's high-order problem solving abilities (Culbertson & Zillmer, 2005), such as "abstraction, reasoning, judgment, analysis ..." (Lezak et al., 2004, p. 30) and ability to synthesize information (Lezak et al.). More specifically, the TOL^DX was developed in order to assess executive planning abilities. As no statistically significant differences were found between any of the three groups of participants, the current data suggests that neither depression nor medication status affects planning abilities as measured by the TOL^DX.

The current findings correspond with those of Grant et al. (2001), Porter et al. (2003), and Purcell et al. (1997). Porter et al. (2003) and Purcell et al. (1997) both failed to find statistically significant differences on the Tower of London planning task comparing depressed younger adults against healthy comparisons. Although there are subtle differences between the original Tower of London task (see Shallice, 1982) and the edition used in the current study (TOL^DX), one's ability to plan and carry out a task is assessed by both.

Additionally, the work of Grant et al. (2001) utilized a different measure of planning (the Stockings of Cambridge) that was based on the original Tower of London test. Grant et al. found statistically significant group differences on one subscale of the Stockings of Cambridge in the area of Initial Thinking Time relative to a certain number of moves. However the TOL^DX does not assess this and thus does not allow for direct
comparison. Nonetheless, the overall lack of evidence supporting the effect depression or medication status on one’s ability to plan is growing as evidenced by this and other studies (Grant et al., 2001, Porter et al., 2003; Purcell et al., 1997) and may be an area of executive functioning that is spared from depression and/or antidepressant medication effects.

*Hypothesis IV*

Hypothesis IV stated that statistically significant differences would be found between the medicated depressed, non-medicated depressed, and non-medicated non-depressed groups on the Stroop Color and Word test (Stroop; Golden & Freshwater, 2002). The overall omnibus test was statistically significant indicating that participants in the three groups performed differently on these measures at a statistically significant level. Furthermore, subsequent analyses revealed that each subtest of the Stroop also revealed statistically significant differences between the three groups. More specifically, the non-medicated non-depressed group outperformed the medicated depressed group at a statistically significant level on the Color subscale of the Stroop. Additionally, the non-medicated non-depressed group outperformed the non-medicated depressed group on the Color-Word subscale. Interestingly, no differences reached statistical significance on the remaining two subscales of the Stroop (Word or Interference) at the group pairwise level.

One particularly confusing finding is the pairwise comparisons of the Color-Word subscale. As previously noted, the non-medicated non-depressed group outperformed the non-medicated depressed group at a statistically significant level, but not the medicated depressed group. This is noteworthy since the difference between mean scores on this subscale between the two depressed groups was only 0.7. However, this may be the result
of the one participant difference between the two depressed groups (medicated depressed, \( n = 15 \); non-medicated depressed, \( n = 16 \)).

Several different issues that are relevant to the current study can affect Color subscale scores. For example, psychiatric patients may have low scores because of emotional rather than cognitive reactions to the colors. Alternatively, these effects are also hypothesized to be the result of low effort and or low motivation (Golden & Freshwater, 2002).

The current study’s findings are in line with the idea named above to some degree—that is—the non-medicated non-depressed group may have performed better than the medicated depressed group based on the aforementioned emotional influences or due to low effort. However, this would not account for the lack of statistical significance between the non-medicated non-depressed group and the non-medicated depressed group. Such findings suggest a medication component to the effects. However, these findings also contrast Paradiso et al. (1997) who found no such differences between their groups.

Low Color scores on the Stroop (Golden & Freshwater, 2002), in the absence of color-blindness, are sometimes seen in the presence of psychiatric conditions (e.g., depression) as the colors may arouse emotional reactions rather than cognitive ones. However, Golden and Freshwater noted that such scores are also seen as a result of low effort rather than any impairment in cognitive functioning. As just detailed, the importance of statistically significant differences between the non-medicated non-depressed group and only one of the depressed groups (medicated depressed), suggests that emotionality is not necessarily the reason for the lack of performance in the medicated depressed group as the non-medicated depressed group’s scores did not differ.
from the non-medicated non-depressed group’s scores at a statistically significant level. Thus, it appears, based on the current findings that medication status may account for difficulties encountered on the Stroop Color subscale.

There were also statistically significant differences found between the non-medicated non-depressed group and the non-medicated depressed group on the Color-Word subscale, but no statistically significant differences between the two depressed groups or the non-medicated non-depressed and medicated depressed groups. The Color-Word subscale is often said to reveal pre-frontal lobe brain pathology (in which case the dominant word naming cannot be inhibited) through low scores (Golden & Freshwater, 2002). However, a major caveat is needed in that emotional turmoil, again, has been implicated in obtaining low scores on this subscale.

While it was easier to identify likely primary involvement of either medication status or depression status in the previously described Color subscale, this current finding does not lend itself to such simple interpretation. The fact that the non-medicated non-depressed group outperformed the non-medicated depressed group at a statistically significant level initially suggests that medication effects were at play. However, that the medicated depressed group was not found to differ from the non-medicated non-depressed group at a statistically significant level takes such a hypothesis away and leaves one struggling to understand what may be at the root of such a difference. Furthermore, the lack of a statistically significant difference between the two depressed groups further complicates this finding. This reveals an interesting difference that must be investigated further as the quasi-experimental nature of the current study limits the ability to speculate what is at the root of such a finding. Although, the unequal number of
participants in each of the depressed groups may have affected the statistical analyses enough to result in one group achieving statistically significant differences while the other depressed group revealed none.

The Interference subscale of the Stroop can identify individuals with frontal lobe dysfunction when viewed in the context of certain scoring patterns with other Stroop subscales (Golden et al., 2000). Low Interference scores can also suggest problems with cognitive flexibility and one’s ability to respond to job demands (Golden et al.). On this scale, no statistically significant individual group differences were found suggesting that although groups were affected by depression in regards to the construct measured by the Interference subscale, individual groups cannot be contrasted to reveal which groups outperformed others.

Paradiso et al. (1997) reported statistically significant differences between depressed individuals and non-depressed individuals on neurocognitive tests, including some executive functioning measures. The current study is consistent with Paradiso et al.’s finding that depressed participants showed weaker performances than non-depressed participants. With the Stroop test commonly used as a measure of frontal lobe dysfunction (Golden et al., 2000), these findings, in combination with findings from Paradiso et al. (1997), suggest that depression affects the area of the frontal lobes and one’s ability to suppress automatic word-reading response, separate word- and color-naming stimuli, shift perceptual set to conform with changing demands, and attention that are assessed by the Stroop Color-Word test (Anderson, 1994).

In day-to-day functioning this may affect an individual’s ability to inhibit an action or thought when confronted with multiple stimuli. Furthermore, as task demands
change, one will likely show difficulties adjusting to the changes adequately. For example, work difficulties could present themselves if one works in an area where tasks are dynamic and one must evaluate situations and adjust accordingly.

Hypothesis V

Hypothesis V stated that statistically significant differences would be found between the medicated depressed, non-medicated depressed, and non-medicated non-depressed groups on the Controlled Oral Word Association test (COWA; Spreen & Strauss, 1998). The overall omnibus test was not statistically significant indicating that participants in the three groups did not perform differently on this measure at a statistically significant level.

While various studies have reported statistically significant differences between depressed participants and non-depressed participants (Landro et al., 2001; Porter et al., 2003), the current study adds to the research described by those such as Grant et al (2001) contradicting such assertions. Grant et al. found no differences between a sample of depressed participants and a healthy comparison group. The COWA assesses an individual’s word fluency, speech, and verbal comprehension. It is also known to be sensitive to dysfunction in the left frontal lobe of the brain where lesions tend to negatively impact oral word fluency (Anderson, 1994).

According to Lezak et al. (2004), the COWA requires generation of words, which has proven to be sensitive to brain dysfunction associated with the frontal lobes (regardless of hemisphere). As frontal lobe lesions have repeatedly revealed decreased verbal fluency scores, the lack of such support evidenced by the current study and Grant
et al. (2001) indicates that verbal fluency and word production are unhampered by depression and/or medication status.

Summary of Hypotheses

A major trend that was revealed through the current analyses is that the non-medicated non-depressed group outperformed both depressed groups in all aspects of tests of executive functioning. Not all differences reached statistical significance, but several did. The two most impressive differences occurred on the TMT Part B and the Stroop Color and Color-Word tests.

As noted previously, the TMT Part B is considered one of the most sensitive measures to any type of cerebral disruption (Reitan & Wolfson, 1993). This measure also serves to highlight what are believed to be more specific frontal lobe functions such as effortful attention, mental flexibility, visual scanning, fine motor control, and alphanumerical recognition (Reitan & Wolfson). This measure does not count errors against an examinee. However, more errors lead to higher overall times and thus do count against the individual in this capacity.

The Stroop Color and Color-Word subscales also revealed pronounced differences between the groups. While the non-medicated non-depressed group outperformed the medicated depressed group and the non-medicated depressed group at a statistically significant level, it was on two different subscales. The Color subscale appears to implicate medication use as the non-medicated depressed group did not perform differently from the non-medicated non-depressed group at a statistically significant level. However, the Color-Word subscale only revealed statistically significant differences between the non-medicated non-depressed group and the non-
medicated depressed group. This finding is complicated, as neither medication status nor depression status is readily identifiable as the reason for the affected performance on this subscale.

The Stroop and the TMT Part B have revealed areas of differential functioning between non-medicated non-depressed and medicated and non-medicated depressed individuals (e.g., planning, distractibility, lack of insight, poor decision-making, perseveration, impulsivity, and an inability to inhibit responses; Burgess, 2003). Both measures utilize these aspects of executive functioning, and typically not in isolation. This highlights Ricco's (2006) assertion that the study of the executive functions is complicated by the lack of being able to assess a function (e.g., inhibition) in isolation. That is, to complete a measure, one typically visually scans a page and then inhibits the automatic desire to impulsively react. Inhibiting such impulses, potentially limits incorrect responses. This small example highlights the fact that several areas of executive functioning are used simultaneously when completing certain tasks. Thus, while science can implicate areas of dysfunction, localizing such dysfunction to specific brain areas or isolated executive functions have been elusive.

The Color and Color-Word subscales of the Stroop and TMT Part B have been identified as problematic areas of functioning for individuals with depression (either medication free or those taking medication) in the current study as well as previous research (Paradiso et al., 1997; Reitan & Wolfson, 1993; Ridout et al., 2007). However, such findings have also been contradicted in previous studies where no such differences have been displayed (e.g., Grant et al., 2001; Landrø et al., 2001). Furthermore, the overall results of the present study have also failed to show deficiencies in depressed
individuals that have typically been a consistent finding in previous research (e.g., WCST, COWA; Grant et al., 2001; Landrø et al., 2001; Porter et al., 2003).

The main overall consistent finding in the current study is one of inconsistency in performance on tests of executive functions. This inconsistency is in line with previous research (see; Channon, 1996; Channon & Green, 1999; Grant et al., 2001; Landrø et al., 2001; Merriam et al., 1999; Paradiso et al., 1997; Porter et al., 2003; Purcell et al., 1997). An important question of whether these differences represent a static or dynamic disruption in cerebral and frontal lobe dysfunction cannot be answered by the current study. However, the present data are trending in a direction that suggests dysfunction is present in depressed individuals with a slight emphasis toward medication effects, as demonstrated by the TMT Part B and the Color subscale of the Stroop. Such findings have valuable implications in that research is now accumulating that reveals a trend toward certain executive function deficits in depressed individuals and that medication status affects functioning as well (Channon, 1996; Channon & Green, 1999; Grant et al., 2001; Landrø et al., 2001; Merriam et al., 1999; Paradiso et al., 1997; Porter et al., 2003, Purcell et al., 1997).

The present study has not been able to definitively implicate depression, medication status, or one particular area of executive functioning that is at the root of the broad research findings in this area of study. However, clearer understanding of the interaction between depression and executive functioning has resulted.

The present study has revealed deficits in executive function domains. Although not specific, a generalization of areas that may provide difficulties to depressed individuals has been an accomplishment of the present study. Depression and medication
status appear to deleteriously affect certain areas of executive functioning (e.g., planning, distractibility, lack of insight, poor decision-making, mental flexibility, perseveration, inability to make use of feedback, impulsivity, and an inability to inhibit responses) while sparing others (e.g., planning, higher order problem solving, mental flexibility, and inhibition).

Researchers have detailed the difficulties of studying the executive functions (Burgess, 1997; Riccio, 2006). The present study found deficient performances in certain areas of executive functions (e.g., planning and mental flexibility), yet found no such deficiencies on other measures of the same faculty. Riccio (2006) asserted that this is where studies of executive functioning suggest that there may be other areas of the brain and/or associations that are not yet identified or understood. Future research will be needed in order to fully flesh out such inconsistencies.

Post Hoc Analyses

The post hoc regression analyses revealed several interesting findings. Most importantly was that Anxiety, as measured by the SCL-90-R Anxiety dimension, accounted for a statistically significant amount of the variance on several subscales of the WCST and the Stroop after taking out the variance accounting for depression as measured by the BDI-II.

Anxiety accounted for a statistically significant amount of the variance on the WCST’s Total Errors, Perseverative Responses, Perseverative Errors, and Learning to Learn subscales. Heaton et al. (1993) instructed that an error occurs when one’s response does not match “the correct sorting principle in effect at the time the response is made” (pp. 7-8). Therefore, the total score subscale does not provide a great deal of information
in that one still does not know what type of errors were made (e.g., perseverative versus nonperseverative). However, this finding suggests that one who scores high on a measure of anxiety may make more errors on complex problem solving tasks such as the WCST.

The finding that individuals who obtained higher scores on an anxiety measure also made more perseverative responses and perseverative errors on the WCST suggests that anxiety may be a condition that affects ones ability to shift sets and inhibit one’s actions. This is an interesting finding and worthy of further research in order to deduce the affects of anxiety on this executive function domain. Similarly, the Categories Completed subscale was also better predicted by scores on a measure of anxiety. This subscale simply informs one to how many categories (color, form, or number) an individual has completed over the course of the test. This finding suggests that ones level of anxiety affects how many categories one will complete on the WCST. Thus, these findings are significant for clinical situations (e.g., anxious individuals may have greater difficulty moving from one situation to the next and may very well be ruminating over past situations rather than moving on and dealing with situations as they happen) and deserve further investigation to better decipher their impact.

The Learning to Learn subscale of the WCST is another interesting finding in that this score “reflects the client’s average change in conceptual efficiency across the consecutive categories” (Heaton et al., 1993, p. 13). Therefore, a positive score indicates improved efficiency of learning across the categories, while a negative score suggests no learning or slower learning. Overall, this means that higher scores on an anxiety measure predict lower efficiency in learning across the consecutive categories on the WCST.
In regards to the Stroop, higher scores on a measure of anxiety predicted lower scores on the Word and the Color subscales. The Word subscale is presumed to be a measure of basic reading speed and may reflect motor-speech problems, poorly developed reading skills, and/or poor dominance for reading skills as is the case in many learning disabled individuals (Golden et al., 2000, Golden & Freshwater, 2002). Golden and Freshwater (2002) noted that lower scores on the Word subscale, and resulting difficulties (as just described), are suggestive of disorders of the posterior left hemisphere. The word reading score is not indicative of an executive functioning skill, yet in the myriad interconnections within the brain, this finding is important in the overall effort to better understand psychiatric disorders and their effects upon neurocognitive performance.

Low color scores on the Stroop, in the absence of color-blindness, are sometimes seen in the presence of psychiatric conditions (e.g., depression or anxiety). Thus, this findings is somewhat anticipated. However, the fact that anxiety accounted for a statistically significant amount of the variance after depression is interesting. Why symptoms of anxiety would affect an individual’s ability to name a word or the color of ink printed on a page is currently unknown and not easily understood. With lower Word scores suggestive of posterior left hemisphere dysfunction, it complicates the earlier finding of all three experimental groups performing differently at a statistically significant level on all four Stroop subscales. This study viewed the overall Stroop as a test of frontal lobe functioning (executive functioning). Such a finding highlights Riccio’s
(1996) assertion that while the executive functions may be viewed as being related to the frontal lobes, associations running throughout the brain are likely impaired in areas other than strictly the frontal lobes.

Numerous studies have investigated anxiety's affect on various aspects of cognitive functioning (see Derakshan & Eysenck, 1998; Dutke & Stöber, 2001; Eysenck, 1979, 1985; Kusche, Cook, & Greenberg, 1993). Dutke and Stöber (2001) investigated the effects of test anxiety on working memory and cognitive performance. They noted that anxious individuals worry and grow concerned about irrelevant things rather than focusing on a task. Highly anxious individuals tend to self-evaluate, which occupies aspects of one's working memory capacity (Dutke & Stöber). "In easy tasks, the remaining memory capacity may suffice to fulfill task requirements. In complex tasks, however, it may not" (pp. 382-383). As a result of this, highly anxious individuals tend to show deficits in task performance.

Similarly, Kusche et al. (1993) investigated the neuropsychological effects of anxiety in school-aged children. In their study, anxious participants had lower levels of academic achievement, yet obtained superior scores on nonverbal tasks over verbal tasks. Additionally, anxious participants obtained lower scores on analogies and three-dimensional block designs implicating right-temporal parietal dysfunction. On measures associated with the executive functions, anxious participants exhibited poorer performances on the TMT Part B, which is consistent with the current study.

Overall, Kusche et al. (1993) asserted that the anxious school-aged participants in their study possessed cognitive processing deficits. Interestingly, they pointed out that most of these children were "good" kids and thus go unnoticed by their parents and/or
teachers because they do not exhibit behavioral problems. Findings such as the current study and these just described indicate that there is a need for further research in the area of anxiety and its affect on cognitive functioning. Furthermore, an emphasis on the executive functions may add more specific information to an area that appears to be both an issue for younger school-aged children as well as adults. These "good" kids will grow up and if their cognitive deficits have gone unnoticed by their parents and teachers, they will undoubtedly have difficulties throughout their adolescence, young adulthood, and-potentially-adult years.

Clinical Implications

Depression. The current study has added another layer of understanding and data to the expanding research base investigating the effects of depression upon younger adult executive functioning. While some measures and subscales revealed statistically significant differences between the medicated depressed, non-medicated depressed, and non-medicated non-depressed groups, others uncovered no such evidence. This is critically important for those whose clinical practice focuses not only on neuropsychological assessment, but also those whose primary clinical work is with individuals experiencing affective disorders encompassing depression.

The research literature discussing depression and executive functions appears to be limited to assessment of deficits. A recent literature search of PsycINFO yielded no articles that discussed counseling/psychotherapy with individuals experiencing executive/cerebral/cognitive dysfunction and depression. Thus, at the present time, psychotherapists are without direction as research is indicating that depression and medication status are affecting certain aspects of executive functioning in their clients,
yet no research is presently available to them to assist in treatment. Recommendations made in the following text are therefore speculative at best and will serve to engender future research in the area of executive dysfunction, depression, and psychotherapy outcomes.

In an individual’s daily life there are countless times when one uses aspects of executive functions. Which route will be chosen in order to avoid traffic, changing lanes while driving on an interstate or expressway, and deciding what to say and what not to say to a friend or colleague are all examples of day-to-day tasks that involve the executive functions. Take, for example, the last situation described. If one’s boss is being unreasonable and an individual is depressed and displaying executive functions deficits, there is a strong chance that executive dysfunction symptoms will hinder not only interpersonal communications with colleagues, but also job performance (Quinn, 2000).

This example is multilayered as the symptoms of depression and the symptoms of executive dysfunction are identified. The low mood (e.g., feeling sad and/or empty inside), finding no interest or pleasure in one’s activities, weight fluctuations, difficulty sleeping, fatigue, feelings of worthlessness, and-potentially-thoughts of death and/or suicide (DSM-IV-TR, 2000) deleteriously affect an individual without argument. Additionally, if one also experiences cognitive processing difficulties such as planning, distractibility, lack of insight, poor decision-making, apathy, perseveration, impulsivity, Know-Do dissociations, poor abstract thinking, and an inability to inhibit responses, the difficulties experienced simply as a result of the depression may be amplified and exacerbated and will likely profoundly add to the depressed individual’s difficulties.
Those whose clinical practice focuses on psychotherapeutic interventions with depressed individuals will need to consider such dysfunctions (on the part of their clients) in the context of their clinical work. Based on the results of the current study, individual clients may present with difficulties related to a lack of ability to shift and maintain a set or make use of feedback (from WCST Failure to Maintain Set; Heaton et al., 1993), problems with slow cognitive processing speed, visual scanning deficits, decreased motivation/effort, and decreased psychomotor deficits (TMT Part B), as well as reading, motivation, and inhibition (Stroop Color-Word test). Normalizing these issues for the individual experiencing depression may grant clients some measure of relief.

Additionally, psychotherapeutic interventions should be evaluated in the context of how they are to be carried out. For example, in the context of the present study, a depressed individual who is experiencing difficulties with his/her ability to shift thoughts and/or pay attention may benefit from interventions that help assuage the impact of such a deficit. This information is important in that some deficits may present as brain damage, only to have been attributable to the individuals’ affective state. A psychotherapist without knowledge of the effects of depression upon the executive functions could send his/her client for medical or neuropsychological evaluation and begin a lengthy and expensive process of ruling out non-affectively related symptom clusters only to, in the end, work on alleviating depressive symptoms and assisting the client to adapt more productively to his/her situation.

Depressed individuals experiencing executive functioning deficits who are engaged in psychotherapy will not likely respond well to an inflexible unidimensional approach. Due to the client’s own inflexibility, they may appear as refractory and
interventions will likely need to be symptom specific. As with most psychotherapeutic interventions, what may work for one, may not work for another (see Wampold, 2001), thus necessitating a flexible, yet highly structured, approach.

Moreover, those whose clinical work emphasizes neuropsychological evaluation need to keep in mind the growing evidence that those experiencing depression may have deficits in areas of executive functioning. Therefore, patients presenting with a history of depression should alert the clinical neuropsychologist to deficits that will likely be revealed upon examination. Only then can proper diagnosis and treatment planning occur.

Another clinical implication resulting from the current study is in regards to medication status. Some measures of executive functioning revealed statistically significant differences between medicated depressed and non-medicated depressed individuals. Although many of the measures and subscales failed to reach statistical significance, data trends indicated that the non-medicated depressed individuals consistently performed better than medicated depressed individuals.

This is cause for attention as antidepressant medications continue to be the standard of treatment for depression (Hollon et al., 1993), thus a large percentage of depressed individuals takes some type of antidepressant medication. Antidepressant medications play a larger role in executive functioning than many believe (Vasko & Gutierrez, 2003). Some may argue that this is not a result of the medication, but an issue of depressive severity (i.e., more severely depressed individuals take antidepressants and thus score lower on tests of executive functioning due to increased depressive symptoms). Yet, the current study failed to reveal statistically significant differences
between the two depressed groups (medicated depressed and non-medicated depressed) based on BDI-II (Beck et al., 1996) scores and thus casts doubt upon a severity argument. Although the current study cannot specifically implicate medication status as a sole cause of decreased performance, the data can serve as a launch pad pointing out the clear necessity of further research into the question of how exactly antidepressant medications do and do not affect cognitive functioning, and more specifically, the executive functions.

**Anxiety.** The post-hoc analyses results regarding anxiety and executive functioning also have implications for clinical work. While the link between depression and anxiety is well known (Hunt, Slade, Andrews, 2004; also see Lenze et al., 2001), these two disorders have been studied separately in the literature in the context of neurocognitive functioning. Several studies investigating anxiety’s affect upon neurocognitive functioning have displayed significant deficits (Dutke & Stöber, 2001; Eysenck, Payne, & Derakshan, 2005; Kusche et al., 1993).

As was the case with depression, those whose clinical practice focuses on psychotherapy with individuals experiencing the symptom constellation associated with the construct of anxiety will need to consider the executive function deficits that appear to coexist with anxiety. Based on the results of the present study, individuals may present to psychotherapy with difficulties related to ones ability to shift sets, inhibit actions, and efficiently understand concepts in the presence of changing situations (Heaton et al., 1993). As previously noted, an anxious individual may have greater difficulty moving from one situation to the next due to his/her ruminating over past events instead of dealing with situations as they occur. This is important for psychotherapists to remember as they apply their chosen theoretical orientation to a client’s treatment. Just as in
working with depressed individuals, normalizing such difficulties may provide a measure of relief to clients presenting to psychotherapy with symptoms of anxiety.

Anxious individuals experiencing executive functioning deficits who are attempting to assuage the deleterious experiences associated with anxiety may benefit from structured psychotherapy emphasizing the decrease of ruminative thoughts. Barlow and Durand (1999) described how challenging such behaviors and dealing with fear provoking stimuli “head on” and in the moment have proven to be beneficial.

As well, those whose clinical work focuses on neuropsychological evaluation need to understand the effects of anxiety upon cognitive functioning in those experiencing anxiety. As previously detailed regarding depression, a history or current diagnosis of anxiety (or symptoms consistent with anxiety during the testing session) should be considered “red flags” and alert the clinical neuropsychologist to deficits that are consistent in individuals experiencing anxiety. This will serve to facilitate proper and accurate diagnosis as well as assist in treatment planning.

Overall, the current study, as well as previous literature in the area of anxiety and cognitive functioning, highlights the need for further research that may identify specific areas of cognitive deficits as well as potential interventions that could help assuage and ameliorate such effects.

Limitations of the Current Study

The limitations of the current study are noted here and discussed in an attempt to aid future research in avoiding such limitations. The first, and primary, limitation of the current study is in regards to the quasi-experimental design implemented. The hallmark
of a quasi-experimental design is the lack of random assignment of participants to experimental groups (Cook & Campbell, 1979; Heppner et al., 1999).

The lack of random assignment of participants to groups represents a threat to internal validity, as one is not able to identify fully causal reasons for changes among and/or between groups (Dunn, 2005). For example, are the differences in the current study between the non-medicated depressed group and the medicated depressed group due to medication effects? Or, do these two groups represent selection effects that predisposed one group to perform differently on tests of executive functioning? That is, are there differences in those that are more willing to take antidepressant medications and those who prefer to treat their depression without pharmacological agents? Such questions cannot be answered using a quasi-experimental design due to the lack of random assignment of participants to groups (Cook & Campbell, 1979; Dunn, 2005; Heppner et al., 1999). Such a limitation restricts how much one is able to generalize in regards to the current findings and represents a need for further, more controlled, laboratory experiments.

However, having pointed out this limitation, Heppner et al. (1999) noted the usefulness of quasi-experimental designs in contemporary applied clinical research. As a result of the intent of the current study, random assignment of participants was not an option. However, as recommended by Heppner et al., a sound attempt was made to understand the basis on which the groups were formed and the applied setting from which they came in order to better understand the somewhat naturally occurring groups that were used (i.e., medicated depressed, non-medicated depressed, & non-medicated non-depressed). Such efforts assisted in the understanding of these groups that resulted in
better controlling of preexisting issues (e.g., using estimated IQ as a covariate). While this is not the preferred manner in which to reveal cause and effect relationships in psychological research, such a design has allowed for understanding and interpretation at a clinical level which is an important component of the struggle to take laboratory research to applied settings (Heppner et al.).

A second and equally important limitation is related to the total number of participants in the current study. Although great amounts of time and effort were made to obtain a higher number of participants for the two depressed groups, the non-medicated non-depressed (healthy comparison) group was the only group that reached the desired group size \((n = 22)\). Obtaining desired participant numbers for the two depressed groups proved challenging. Despite utilizing several facilities as recruiting sites, the two depressed group numbers fell short and thus limits the generalizability of the current data. A replication study that obtains the desired number of participants based on a power analysis would eliminate this limitation as well as add to the research literature in this area of study.

In this same vein, the ratio of men to women in the current study is not as reflective of the prevalence rates of the DSM-IV-TR (2000) as was desired. The DSM-IV-TR reported prevalence rates of MDD in community samples as between 10% and 25% for women and between 5% and 12% for men, or roughly a ratio of 2:1. The current study's sample had a ratio of nearly 5 women to every 1 man. Although this ratio equates to the high estimate for women and low estimate for men, a 2:1 ratio was desired and believed to be a better representation of the disorder's occurrence. A replication study investigating only males or only females would be beneficial to the overall understanding
Additionally, the racial and ethnic make-up of the current study’s sample is another limitation. Given the region where the research study was carried out, the generalizations of the data must be restricted to similar individuals. Upper Midwestern Caucasians were the largest single group of individuals who participated (over 90% of the sample). Such a homogenous sample presents a major difficulty in the generalizability of the study’s results to ethnically and racially diverse populations. Research utilizing a more diverse sample would be beneficial.

Recommendations for Future Research

The current research has added to the burgeoning literature base investigating the effects of depression on executive functioning. However, several questions have been created as a result. For example, previous research (e.g., Channon & Green, 1999; Landrø et al., 2001; Paradiso et al., 1997), as well as the present study, has attempted to manage the group sampling in such a way that group differences could explain executive functioning deficits found in different populations. Future investigations should attempt to entirely control medication status through experimental design in order to fully understand if and how medication status influences executive functioning. Such control would resolve the problems associated with a quasi-experimental design (i.e., random assignment of participants to groups) and enable one to more easily identify a causal relationship between medication status and any cognitive functioning deficits that may be found.
Moreover, in an attempt to better explain the inconsistent findings of research looking into depression and the executive functions (e.g., Landrø et al., 2001; Paradiso et al., 1997), research that further reduced the age range and limited the degree of depressive symptoms experienced by potential research participants would add needed clarity. This would serve to focus the information gained to a better-defined group of individuals (based on a smaller age range and narrower scope of depression). As smaller groups become better understood, such findings could be brought together to better explain the inconsistent findings of research looking into depression and the executive functions.

Another suggestion for future research is in regards to the instruments used to assess executive functions. There is no dearth of measures and instruments available to study the executive functions. However, this makes it difficult to compare research findings across several studies (e.g., Grant et al., 2001; Landrø et al., 2001; Purcell et al., 1997). A meta-analytic review may be quite helpful in this regard and would surely enable a more standardized, reliable, and valid battery of measures to assess executive functioning in order to carry out the aforementioned research. Consequently, instrument development and validation is a vital component of any research line in this area.

Lastly, future research implementing neuroimaging and biofeedback could greatly advance the understanding of what is exactly happening on a neurochemical and neurophysiological level. Although neuroimaging is not perfect and cannot fully reveal all happenings of the brain, attempts could be made to identify and localize areas of the brain that become activated while depressed individuals are attempting tasks associated with tests of executive functioning (Rogers et al., 2004). Moreover,
Regarding anxiety, future research should address the shared variance that is well known in the psychological community between depression and anxiety. Completing laboratory research with individuals experiencing anxiety and not depression will allow for a purer understanding of the effects anxiety has upon cognitive functioning and, more specifically, the executive functions. Although research such as Dutke and Stöber (2001) have pronounced that cognitive deficits among anxious individuals are well established, delineating specific differences between the effects of anxiety versus depression are needed.

Furthermore, behavioral interventions focused on both the deleterious effects of anxiety symptoms as well as decreased cognitive functioning are needed. While psychotherapeutic work with anxious individuals is well established and researched (see Barlow & Durand, 1999), the effects of cognitive dysfunction and whether or not such deficits remain upon anxiety symptom remittance are less well known.

Lastly, anxiolytic medications are typically sedative hypnotics and are known to affect cognitive capacities (DiMicco & Gutierrez, 2003). While cognitive deficits are expected with such medications, it would be helpful to fully understand the severity and specific domains affected. Such information would undoubtedly aid clinicians in their work to increase treatment compliance. Such efforts would also likely improve the overall educational needs and desires of the mental health care consumer of the 21st century.
Conclusion

The current study attempted to further the research literature base that has revealed equivocal findings regarding the effect of depression upon younger adult individuals’ executive functions. A major intent of the current study was to utilize a methodology that would increase understanding of psychotropic medication effects upon executive functioning as well as compare executive functioning of depressed individuals against non-depressed individuals.

The results of the current study have added information to the research base in various areas. However, several findings will ultimately fuel future research. The results of the current study lends support to the hypothesis that there are differences in executive functioning between medicated depressed, non-medicated depressed, and non-medicated non-depressed individuals in different areas related to executive functioning. This is especially true in regards to one’s ability to shift and maintain a set or make use of feedback (from WCST Failure to Maintain Set; Heaton et al., 1993), problems with slow cognitive processing speed, visual scanning deficits, decreased motivation/effort, and decreased psychomotor deficits (TMT Part B), as well as reading, motivation, and inhibition (Stroop Color Word test) where non-medicated non-depressed participants outperformed participants in the two depressed groups. Furthermore, there was a trend toward the non-medicated depressed group also outperforming the medicated depressed group. However, definitive answers have not resulted from the current study. Such answers will have to come from future research (as detailed previously).

Burgess (2003) recently noted the executive functions are likely the newest area of neuropsychological investigation. Thus, this and other such investigations are clearly
in their infancy; much more will be learned as more research is carried out. The aim of
the current study was to add a foundational piece to the burgeoning base of literature
attempting to explain the effects of depression on executive functioning. Clearly,
disorders such as depression (and potentially anxiety) interact with our executive
functions, and evidence from this study suggests that it does so differently depending on
the presence or absence of antidepressant medication. Future research must build on these
findings in order to advance our understanding of executive functioning in depressed
younger adults.
APPENDICES
Appendix A

Informed Consent Depressed Groups

My name is Michael Ransom; I am a Counseling Psychology doctoral student under the advisement of Kara B. Wettersten, Ph.D. in the Department of Counseling at the University of North Dakota. I am conducting a research study looking at cognitive functioning in depressed and non-depressed individuals.

You are invited to participate in this research study which will require you to meet with a research team member and complete a demographic questionnaire and some tasks assessing depression and cognitive functioning (e.g., attention, planning, memory, etc.) which will take approximately 1 and one-half hours to complete. Your participation in this study is completely voluntary and anonymous and your confidentiality is extremely important. Therefore, while the results of this study may be published, neither your name nor any other identifiable information will be used. Furthermore, upon completed data collection, the anonymous data will be stored in a secure location. All data obtained will be retained for at least three years following the completion of this study and kept in a secure location where only Michael Ransom, Dr. Wettersten, and IRB auditing personnel will have access to it.

There is a possibility that you may experience some psychological discomfort in completing these measures. Should you have any concerns regarding your performance or any other aspect of this research study, you are encouraged to let the principle investigator know so your questions can be answered or provided with an appropriate referral. If you should choose to withdraw your participation from this research study please inform the individual administering the assessments to you (i.e., Michael Ransom, M.A.). Furthermore, there will be no penalty or repercussions should you choose not to participate or choose to withdraw from the research study. While there is no financial or monetary compensation provided for participation in this study, the benefits of you participation include an opportunity to participate in the advancement of scientific and clinical knowledge regarding the diagnosis and treatment of clinical depression. Additionally, there is no cost to you to participate in the present study or receive the free depression evaluation. However, if you choose to seek any treatment based on the findings of the free depression evaluation, the cost will be your sole responsibility. There is a possibility that you may be excused from the research study if you are taking certain medications or have a medical diagnosis that may impact your test taking abilities. However, if this is the case, the depression evaluation will still be completed free of charge.

It is required by law that I must obtain assistance for individuals who are imminently suicidal. If you are in immediate danger or harming yourself or others, I will contact the referral source or other emergency personnel.

If you have any questions or concerns regarding this research study, please contact myself at michael.ransom@und.edu or Dr. Wettersten at kara_wettersten@und.edu, both of whom are at the University of North Dakota, Department of Counseling (701-777-2729). If you have any other questions or concerns, please call Research Development and Compliance at (701) 777-4279.

Thank you very much for your time and help in this research project,

Michael Ransom, M.A.

I have read and understand that above information regarding this research study. I also have been given a chance to ask any questions. A copy of this form has been provided to me for my records. By signing below, I agree to participate and understand that I may stop my participation at any time without repercussion for doing so.

Name (please print) ____________________________ Date ____________

Signature ____________________________
Informed Consent Non-Depressed Group

My name is Michael Ransom; I am a Counseling Psychology doctoral student under the advisement of Kara B. Wettersten, Ph.D. in the Department of Counseling at the University of North Dakota. I am conducting a research study looking at cognitive functioning in depressed and non-depressed individuals.

You are invited to participate in this research study which will require you to meet with a research team member and complete a demographic questionnaire and some tasks assessing depression and cognitive functioning (e.g., attention, planning, memory, etc.) which will take approximately 1 and one-half hours to complete. Your participation in this study is completely voluntary and anonymous and your confidentiality is extremely important. Therefore, while the results of this study may be published, neither your name nor any other identifiable information will be used. Furthermore, upon completed data collection, the anonymous data will be stored in a secure location. All data obtained will be retained for at least three years following the completion of this study and kept in a secure location where only Michael Ransom, Dr. Wettersten, and IRB auditing personnel will have access to it.

There is a possibility that you may experience some psychological discomfort in completing these measures. Should you have any concerns regarding your performance or any other aspect of this research study, you are encouraged to let the principle investigator know so your questions can be answered or provided with an appropriate referral. If you should choose to withdraw your participation from this research study please inform the individual administering the assessments to you (i.e., Michael Ransom, M.A.). Furthermore, there will be no penalty or repercussions should you choose not to participate or choose to withdraw from the research study. Your participation will help in the advancement of scientific and clinical knowledge regarding the diagnosis and treatment of clinical depression and be rewarded with a $5 gift card to a local eating establishment or discount store. Additionally, there is no cost to you to participate in the present study or receive the free depression evaluation. However, if you choose to seek any treatment based on the findings of the free depression evaluation, the cost will be your sole responsibility. There is a possibility that you may be excused from the research study if you are taking certain medications or have a medical diagnosis that may impact your test taking abilities. However, if this is the case, the depression evaluation will still be completed free of charge.

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Thank you very much for your time and help in this research project,

Michael Ransom, M.A.

I have read and understand that above information regarding this research study. I also have been given a chance to ask any questions. A copy of this form has been provided to me for my records. By signing below, I agree to participate and understand that I may stop my participation at any time without repercussion for doing so.

Name (please print)                                         Date

Signature
Appendix B

Demographic Questionnaire

1. Age (please specify): 

2. Sex (circle one): Female Male

3. Race/Ethnicity (please specify): 

4. Relationship Status (check one):
   Single (never married) 
   Married 
   Partnered 
   Divorced 
   Widowed 
   Remarried 
   Separated 
   Other (please specify): 

5. Yearly household income (check one):
   $0 to $25,000 
   $25,000 to $50,000 
   $50,000 to $75,000 
   $75,000 to $100,000 
   $100,000 to $150,000 
   $150,000 to $200,000 
   Greater than $200,000
Appendix C

Interview Questions

1. Are you:  
   Right Handed _____  
   Left Handed _____  
   Ambidextrous _____  

2. How many years of education have you completed:___________

3. Have you ever been diagnosed with depression (check one):  
   Yes _____  
   No _____  
   If yes, age of first onset: _____  
   (If no, skip to question 8)  
   Number of total depressive episodes: _____  
   If individual is currently receiving counseling, how many sessions have been completed: _____  
   Have you ever received Electroconvulsive Therapy (ECT):  
      Yes _____  
      No _____  

4. Have you ever been diagnosed with Attention Deficit Hyperactivity Disorder (ADHD, also known as ADD) (check one):  
   Yes _____  
   No _____  
   If yes, age of diagnosis: _____  

5. Have you ever received a formal mental health diagnosis:  
   Yes _____  
   No _____  
   If yes, what (please list): ____________________________

6. Have you ever been hospitalized for a psychiatric condition:  
   Yes _____  
   No _____  
   If yes, how many times: ____________

7. Have you ever sustained an injury that resulted in a loss of consciousness (knocked out):  
   Yes _____  
   No _____
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