EXECUTIVE FUNCTIONING ABILITIES ARE DIFFERENTIALLY ASSOCIATED
WITH ANHEDONIC DEPRESSION AND ANXIOUS AROUSAL

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by

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CHAPTER 1

Introduction

Numerous studies have identified global and diffuse cognitive deficits in individuals with unipolar depression or anxiety disorders (Castaneda et al., 2008; Veiel, 1997). However, it is unclear as to whether these deficits are unique to unipolar depression or anxiety, or merely a function of psychopathology severity (Berenbaum, Kerns, & Taylor, 1990). Determining the extent to which cognitive deficits result from psychopathology or neurologic compromise is common in the practice of clinical neuropsychology, as symptoms of depression and anxiety are reported by as many as half of patients seen for neuropsychological assessments (Kanner, 2005). Furthermore, diminished cognitive functioning has adverse effects on daily functioning and thereby negatively impacts the disease management and convalescence of individuals with medical illnesses (Egede, 2007; Schillerstrom, Horton, & Royall, 2005). Thus, it is important to clarify the relative contribution of psychopathology and neurological dysfunction to cognitive impairment, as this information may have significant treatment and prognostic implications.

Although the cognitive profiles of individuals with unipolar depression or anxiety disorders have not been definitively identified, previous research has shown impaired executive functioning in these groups compared to healthy controls. The executive
functions are a cognitive domain responsible for higher-order abilities such as abstract thinking, planning, inhibition, and coordination of behavior and other cognitive faculties (Lezak, Howieson, Loring, et al. 2004). Converging evidence from the neuroimaging and neuropsychological literature implicate the same neuroanatomical structures underlie impaired executive functions and presence of unipolar depression and anxiety (Rogers et al., 2004), albeit further research has yet to identify functional differences in cognition that would distinguish these disorders from one another.

To this author’s knowledge, there are no published studies that have differentiated unipolar depression from anxiety disorders on specific executive functioning abilities. Previous efforts may have, in part, been limited by the prevailing categorical, syndrome-focused system of diagnosing these disorders (i.e. the Diagnostic and Statistical Manual of Mental Disorders [DSM-IV-TR; APA, 2000]). First, because these disorders frequently co-occur and share many of the same symptoms (Kessler et al., 2003), it is difficult to determine whether the observed cognitive deficits are uniquely associated with unipolar depression or anxiety. Second, the DSM-IV’s diagnostic approach which depends on whether a symptom threshold has been exceeded can prevent the consideration of individuals in the general population who exhibit sub-threshold, but consequential, predispositions toward unipolar depression or anxiety. It is therefore less clear as to whether cognitive deficits are a result of the unique features or severity of the pathology of unipolar depression or anxiety disorders.

Because of these limitations, some researchers have focused their investigations on dimensions underlying unipolar depression and anxiety which account for the
similarities and differences between these disorders. The Tripartite model (Clark & Watson, 1991) is a theoretical model that identifies the unique dimensions of depression (i.e. anhedonic depression) and anxiety (i.e. anxious arousal) on a continuum of severity. The aim of this study is to determine whether these dimensions are differentially associated with performance on various tests of executive functioning in a sample of undergraduates who endorse high levels of anhedonic depression or anxious arousal. To this end, this document will first review previous efforts in identifying executive functioning deficits in unipolar depression and anxiety, and describe how the Tripartite model may contribute to the current understanding of cognition in these psychopathologies.

The Executive Functions

The executive functions have been described as essential to human survival and adaptation, and illustrative of the complexity of our cognitive processes. It is not a unitary concept; rather, a number of moderately correlated abilities comprise the executive functions (Parkin, 1998). According Miyake and colleagues (2000), the functions of mental set-shifting, information updating and monitoring, and inhibition of prepotent responses comprise the latent construct that is executive functioning. Numerous neuropsychological tests measure one or more of these functions to illustrate the various abilities associated with high-order cognitive processing. Specifically, executive functions:

“…allow us to shift our mind set quickly and adapt to diverse situations while at the same time inhibiting inappropriate behaviors. They enable us
to create a *plan, initiate* its execution, and *persevere* on the task at hand until its completion. Executive functions mediate the ability to *organize* our thoughts in a *goal-directed* way…” (Jurado & Rosselli, 2007; italics added)

Executive functioning tests vary in modality (i.e., verbal, non-verbal) and complexity. These independent but related tests measure the ability to maintain and shift cognitive set (e.g. Trails B), generate responses within defined parameters (e.g. COWAT), manipulate increasing amounts of information in one’s working memory (e.g. LNS), suppress automatic responses (e.g. SCWT), disengage from perseveration and adapt to change (e.g. the WCST), engage in complex abstract reasoning (e.g. Matrix Reasoning, Similarities), and demonstrate planning and organizational abilities (e.g. RCFT). Table 1 summarizes each executive functioning measure used in this study, its modality, and specific executive functioning ability measured.

Although the concept of the executive functions was first derived from the deficits observed in patients who suffered frontal lobe lesions, it is largely acknowledged that the frontal lobes and its associations with other cortical regions, subcortical structures, and thalamic pathways are the neural substrate of the expression of executive functioning (Ottowitz, Dougherty, & Savage, 2002). Several neuroanatomical sites and pathways implicated in impaired executive functioning in neurological disorders are also affected in unipolar depression and anxiety. Specifically, converging evidence from neuroimaging and electrophysiological studies have identified dysfunctions in the prefrontal cortex and asymmetrical brain hemispheric activity, respectively. In reviewing
several studies of depressed patients that combined neuropsychological and neuroimaging techniques, Rogers and colleagues (2004) found that executive dysfunction is associated with hypoactivation in the dorsolateral prefrontal and the anterior cingulate cortices, and hyperactivation in the orbitofrontal cortex. This pattern along with decreased activity in the right parietal cortex and increased subcortical (i.e., amygdala and hippocampal) activity is also consistent with compromised executive functions and a concomitant exaggerated response to threat that is frequently observed in anxiety (Levin, Heller, Mohanty, et al. 2007).

Heritability studies investigating cognition and depression lend support to these associations. Twins discordant for unipolar depression and monozygotic twins at high risk for developing a depressive disorder have been shown to exhibit executive functioning deficits in comparison to low-risk monozygotic and dizygotic twins, suggesting that executive functioning may serve as an endophenotype for depressive disorders (Christensen, Kyvik, & Kessing, 2006). Thus, the results of neuroanatomical and genetic research corroborate findings from neuropsychological studies that have identified executive functioning deficits in unipolar depression- and anxiety-disordered individuals.

Unipolar Depression and Anxiety Disorders and Executive Functions

Evidence to the contrary notwithstanding (Crews, Harrison, & Rhodes, 1999; Smitherman et al., 2007), the majority of studies that have investigated cognitive functioning in unipolar depression and anxiety disorders revealed impairments in many of the same executive functioning abilities. However, closer inspection of these findings
reveals unique and differentiable associations between specific executive functioning abilities and these two disorders.

**Unipolar depression and executive functions**

Neuropsychological deficits in depressed individuals consist of global and diffuse impairments across cognitive domains and tests, including deficits in executive functions and memory encoding and retrieval. Notably, as executive functioning deficits have been shown to account for these deficits in memory (Head et al., 2008), it is likely that executive functions are the primary cognitive domain affected in unipolar depression. Indeed, meta-analytic studies have specifically identified executive functioning deficits among individuals with unipolar depression in comparison to healthy controls. For instance, in a pooled sample of depressed patients, Veiel (1997) found the largest effect size differences for measures of mental flexibility or set-shifting (i.e., Trails B) and inhibitory control (i.e., SCWT) as well as medium effects for verbal fluency (i.e., COWAT).

More recent studies corroborate these deficits, such that depressed individuals demonstrate slowed Trails B (Mahurin et al., 2006) and SCWT completion (Markela-Lerenc et al., 2006), and diminished verbal fluency (Zakzanis, Leach, & Kaplan, 1998). Christensen and colleagues (1997) found a large effect size difference between depressed patients and healthy controls in goal formation and planning – deficits akin to findings of increased perseveration, suboptimal response to feedback, and failures to maintain cognitive set on the WCST (Channon, 1996; Degl’Innocenti, et al., 1998; Martin, Oren, & Boone, 1991). Furthermore, deficits in working memory in individuals with
depression have been reported across various measures, wherein patients demonstrate slower reaction times and commit more errors on effortful tasks compared to controls (Moffoot et al., 1994; Rose & Ebmeier, 2006).

However, the deficits in executive function observed in persons with depression may depend on other factors such as the severity or specific features of depression in the samples investigated. A rigorous meta-analysis of verbal fluency deficits in depression identified that deficits in phonemic and semantic fluency are better accounted for by generalized impairment resulting from severe psychopathology (Henry & Crawford, 2005). Similarly, Stordal et al. (2005) investigated a sample of inpatients with a range of Axis I disorders and found that severity of psychopathology better predicted executive dysfunction than a specific diagnosis of depression. Other studies suggest that “melancholic” depression, or the specific type of depression where the inability to experience pleasure is most salient, is more likely to be associated with cognitive dysfunction. More specifically, melancholic patients have been found to perform worse than healthy controls and non-melancholic patients on measures of mental flexibility or set-shifting (Michopoulos et al., 2006), verbal fluency (Naismith et al., 2003), and perseveration (Austin et al., 1999). Thus, psychopathology severity and specific disorder features may impact the type and severity of executive functioning deficits observed in individuals with unipolar depression, though previous research has not explored the extent to which these features are associated with executive functioning in non-clinical groups.
**Anxiety and executive functions**

The cognitive sequelae observed in anxiety disorders are not as thoroughly documented as in the depression literature, though some specific disorders have been directly examined. For instance, studies show that individuals with obsessive-compulsive disorder exhibit deficits in organizational strategies (Greisberg & McKay, 2003), slowed response latencies (Roth et al., 2004), perseverations, and difficulties with utilizing feedback to adapt to change (Olley et al. 2007), particularly in non-verbal (i.e. visuospatially-mediated) tasks (Boldrini, 2005; Bucci, 2007; Savage, 1999). Previous research on cognitive functioning in posttraumatic stress disorder (PTSD) have identified specific deficits in set-shifting (Beckham, Crawford, & Feldman, 1998), concept formation (Kanagatnam & Asbjornin, 2007), working memory, and inhibitory control (Stein, Kennedy, & Twamley, 2002). However, contradictory findings have led researchers to question whether these deficits are associated with comorbid psychopathologies (e.g. depression, substance abuse) or low premorbid intellectual functioning (McNally & Shin, 1995) rather than the independent effects of trauma exposure (Twamley, Hami, & Stein, 2004).

Less well understood are the patterns of cognitive functioning associated with other anxiety disorders. In a population-based study of cognitive impairments in anxiety disorders among adults, Airaksinen et al. (2005) identified slowed cognitive set-shifting among individuals who met criteria for an anxiety disorder in comparison to healthy controls. However, only the groups with panic and obsessive-compulsive disorder exhibited these deficits, and not the groups with social phobia or generalized anxiety. In
a different study, patients with panic disorder and obsessive-compulsive disorder were impaired in visuospatial abilities, which the authors assert are associated with anxiety in general (Boldrini, 2005). However, other studies have concluded that individuals with panic disorder are no different from controls in most neuropsychological domains (Gladsjo et al., 1998; Kaplan et al., 2006), and studies of patients with specific phobia and generalized anxiety disorder have produced similar results (Airaksinen et al., 2005; Graver & White, 2007).

In reviewing these and similar neuropsychological studies of anxiety disorders in young adults, Castaneda and colleagues (2008) assert that too few studies have investigated these associations to identify a cognitive profile associated with anxiety disorders in general. Nonetheless, the extant studies appear to point towards reduced executive functioning, including slowed performance, disorganization, and perseveration that may be more pronounced in non-verbal, visuospatial tasks.

**Comorbid unipolar depression and anxiety, and executive functions**

Unipolar depression, specifically major depressive disorder, shares a high rate of comorbidity with anxiety disorders, with estimates suggesting that almost 60% of patients with primary major depression present with at least one comorbid anxiety disorder (Kessler et al., 2003) and 40% of patients with a primary diagnosis of anxiety disorder meet criteria for major depression (Rodriguez, Weisberg, Pagano, et al., 2004). As such, investigators have suggested that the comorbidity of these disorders account for the inconsistent descriptions of the cognitive phenotypes associated with these disorders (Levin, et al., 2007). Some argue that the deficits reported in previous studies may result
from the compound effects of comorbid depression or anxiety. Specifically, executive
dysfunction has been found to be more prominent in patients with comorbid depression
and anxiety disorders in comparison to depressed or anxious patients who do not have
comorbid disorders.

Basso and colleagues (2007) compared depressed inpatients with and without
comorbid anxiety to a group of controls on a broad battery of neuropsychological
measures. They found that although both depressed groups performed significantly
worse than controls on tests of memory, it was the group with comorbid depression and
anxiety that specifically demonstrated executive functioning deficits and produced more
scores in the impaired range across all the measures used in their study. In studying
panic-disordered patients with and without comorbid major depression, Kaplan and
others (2006) found that the former group had deficits in working memory and longer
latencies in decision-making compared with controls and patients with panic disorder
alone.

Other researchers, however, assert that the presence of anxiety may be associated
with fewer cognitive deficits in depression. Arguing on the basis of extensive brain
electrophysiology evidence, the presence of comorbid anxiety has been shown to cancel
out the effects of depression on neuropsychological performance (Heller, et al., 1995) or
to have a non-additive effect (Keller, Nitschke, Bargava, et al., 2000).

Electroencephalographic studies of normal and psychopathologic samples reveal that
depression and anxiety are associated with contradictory activity in the right parieto-
temporal cortex; sad mood states are associated with lower activity in this region,
whereas anxious individuals show higher blood flow to this area while performing tests of hemispheric bias. In addition, Nitschke and colleagues (2000) suggest that when comorbid anxiety is experimentally and statistically accounted for, executive functioning impairments specific to depression account for the deficits in other cognitive domains such as memory and attention. It is therefore uncertain whether comorbid anxiety eliminates or has an additive effect on executive functioning deficits in depression.

Summary

Executive functioning difficulties in individuals with unipolar depression consist of deficits in mental flexibility or set-shifting, resistance to perseveration, inhibitory control, verbal fluency, working memory, goal formation, and planning. Individuals with anxiety disorders exhibit very similar impairments in set-shifting and resistance to perseveration, but also demonstrate organization deficits and slowed performance, particularly in visuospatially-mediated executive functioning tasks. However, results have varied in identifying the extent to which these deficits are unique to unipolar depression or anxiety, or unaffected by disorder severity, specific features, diagnoses, or comorbidity. Furthermore, only a few studies have explicitly investigated abstract reasoning in relation to unipolar depression or anxiety, though some findings point to deficits in this ability (Austin et al., 1999; Naismith et al., 2003). To address this gap in the literature and to determine the specificity of executive functioning deficits in unipolar depression and anxiety, the current study will employ a model that outlines the underlying dimensions of these disorders to identify the specific executive functioning deficits associated with these unique features.
The Tripartite Model

As previously described, cognitive deficits are often observed in individuals with unipolar depression and/or anxiety disorders. Indeed, the DSM-IV criteria for these disorders include symptoms of diminished concentration and memory, indecisiveness, and intrusive thoughts (APA, 2000). Other symptoms such as sleep difficulties, psychomotor agitation or slowing, hyperarousal, and excessive rumination are also likely to independently impact cognitive performance (Leskin & White, 2007; Philippot & Brutoux, 2008; Redline et al., 1997; White, Myerson, & Hale, 1997). Thus, these overlapping and contributing symptoms further obscure the specific similarities and differences in cognitive functions between these disorders.

The high degree of symptom overlap and non-specific cognitive deficits in unipolar depression and anxiety may result from some limitations of the prevailing categorical, syndrome-focused system of diagnosing these disorders (i.e. DSM-IV-TR; APA, 2000). First, the current classification system reflects a subjective distinction of “apparent” as opposed to “actual” characteristics that comprise each syndrome, which does not directly correspond to observed phenotypic similarities and differences between depression and anxiety demonstrated in empirical research (Watson, 2005). Second, the DSM-IV employs a dichotomous diagnostic approach, wherein an individual meets or does not meet criteria for a diagnosis depending on whether a threshold number or constellation of symptoms has been exceeded. As a result, the majority of individuals who do not meet criteria for a diagnosable syndrome are less frequently subject to clinical neuropsychology research, despite evidence showing that most individuals can exhibit
predispositions (i.e. personality, temperament) toward unipolar depression or anxiety (Tellgen, 1985). Thus, the diminished specificity of the current diagnostic system renders it difficult to identify the cognitive abilities unique to unipolar depression or anxiety, and to identify the degree to which individuals with sub-clinical symptoms have these deficits.

These limitations have led researchers to examine the underlying dimensions of unipolar depression and anxiety and to explore the extent to which these dimensions differentiate these disorders. At the forefront of this line of research is work by Clark, Watson, and colleagues who described and identified dimensions called Negative Affect, Positive Affect, and Physiological Hyperarousal (Clark & Watson, 1991; Watson, Clark, et al., 1995; Watson, Weber, et al., 1995). Specifically, Negative Affect was defined as a non-specific emotional distress factor underlying both depression and anxiety disorders. It represents the extent an individual may feel distressed, upset, angry, guilty, afraid, sad, or scornful, as opposed to feeling calm, relaxed, or unperturbed. Positive Affect is a depression-specific factor that in low levels represents the disorder’s loss of pleasurable engagement, tiredness, or amotivation (Watson, Clark, & Carey, 1988). Further studies that examined these dimensions in both patient and non-patient populations identified another unique dimension, Physiological Hyperarousal, which reflects anxiety-specific somatic and autonomic symptoms including intense fear, racing or pounding heartbeat, sweating, trembling, shortness of breath, and other anxiety-related symptoms (Joiner et al., 1999).

These common and specific features of unipolar depression and anxiety are the bases for their Tripartite model (Clark & Watson, 1991). Specifically, this model
proposes that (a) unipolar depression is characterized by high Negative Affect and low Positive Affect, (b) anxiety is characterized by high Negative Affect and high Physiological Hyperarousal, and (c) the comorbidity of depression and anxiety is characterized by high Negative Affect, low Positive Affect, and high Physiological Hyperarousal (see Figure 1).

In order to fully measure these dimensions and demonstrate their hierarchical structure, the Mood and Anxiety Symptom Questionnaire (MASQ) was developed to assess the general distress/Negative Affect component of unipolar depression and anxiety and included items specific to the low Positive Affect/anhedonia and hyperarousal symptoms that are unique to these disorders, respectively. Their studies show that the MASQ’s disorder-specific scales – Anhedonic Depression and Anxious Arousal – demonstrate better divergent validity than other indices of depressed and anxious mood when contrasting these disorder groups (Buckby et al., 2007; Reidy & Keogh, 1997; Ruth & Mehrotra, 2001). Furthermore, other studies have replicated these dimensions using different affect measures across patient and non-patient samples using confirmatory factor-analytic methods (Brown, Chorpita, & Barlow, 1998; Joiner et al., 1999; Mineka et al., 1998). There is also some evidence that this conceptualization of emotional psychopathology corresponds with brain hemispheric asymmetries identified in experimental research (Voelz et al., 2001). Specifically, low Positive Affect has been associated with hypoactivation of the right parietotemporal and left prefrontal regions, while anxious hyperarousal has been linked to hyperactivation of the right parietotemporal region (Heller, Etienne, & Miller, 1995). The Tripartite model and its
measure, the MASQ, may therefore contribute to the understanding of cognitive functioning in unipolar depression and anxiety.

Indeed, the Tripartite constructs Anhedonic Depression and Anxious Arousal have been associated with specific dimensions of cognitive functioning, though these studies have largely focused on thought content and the processing of emotion-relevant information (Beck, Benedict, & Winkler, 2003; Beck & Perkins, 2001). These investigations have elaborated on automatic thoughts and cognitive appraisals and how these relate to emotional expression in depression or anxiety. However, the current study focuses on the functional aspects of cognition in order to assess behavioral and neurological functioning, irrespective of the emotional valence of the tasks used to assess these abilities. Nonetheless, because this line of research has identified differences between depressed and anxious groups in the processing of emotionally-valent information, it stands to reason that groups characterized by high levels of Anhedonic Depression and Anxious Arousal may be differentiable in terms of their neuropsychological, executive functioning abilities.

The Present Study

The current study sought to identify the executive functioning abilities associated with the underlying dimensions of unipolar depression and anxiety. First, groups with high scores on Anhedonic Depression (AD), Anxious Arousal (AA), and both (AD&AA) scales of the MASQ will be compared to a control group on multiple measures of executive functioning. Second, these three groups will be directly compared on a comprehensive battery of executive functioning measures with the expectation that they
will demonstrate a different pattern of deficits. Lastly, the classification accuracy of the AD and AA scales to impaired executive functioning test performance will be explored.

**Hypotheses**

**Primary Aim 1.** Individuals with high scores on AD and AA will exhibit poorer executive functioning than healthy controls.

*Hypothesis 1.* The group characterized by high AD will exhibit significantly lower scores on working memory (i.e. LNS), verbal fluency (i.e. COWAT), mental flexibility or cognitive set-shifting (i.e. Trails B), and verbal abstract reasoning (i.e. Similarities) compared to the Controls.

*Hypothesis 2.* The group characterized by high AA will exhibit deficits in mental flexibility or cognitive set-shifting (i.e. Trails B) and non-verbal abstract reasoning (i.e. Matrix Reasoning) compared to healthy controls.

**Primary Aim 2.** Groups with high scores on AD or AA will exhibit differentiable impairments on specific executive functioning abilities.

*Hypothesis 3.* The group characterized by high AD will exhibit significantly lower scores on working memory (i.e. Letter-Number Sequencing), verbal fluency (i.e. COWAT), and inhibitory control (i.e. SCWT Color-Word) compared to the AA group.

*Hypothesis 4.* The group characterized by high AA will exhibit significantly lower scores in organization (i.e. RCFT) and non-verbal executive functioning tasks (i.e. Trails B, WCST Total Number of Errors, WCST Perseverative Responses) in comparison to the AD group.
**Hypothesis 5.** Both groups will demonstrate comparable scores on verbal abstract reasoning (i.e. Similarities), though the AA group will exhibit poorer performance on a non-verbal abstract reasoning task (i.e. Matrix Reasoning) compared to the AD group.

**Primary Aim 3.** The performance of the group with high scores on AD and AA on executive functioning measures will be explored. Due to previous contradictory findings, no specific hypotheses are proposed, except that it is expected they are likely to demonstrate worse performance than the control group.

**Secondary Aim.** The AD and AA groups’ performance will be associated with measures of executive functioning, though these scales may vary in the scores (i.e. level of pathology severity) that would correspond to impairment on executive functioning measures.

Two sets of exploratory analyses will be conducted to test this secondary aim. First, correlations between executive functioning abilities and the AD and AA scores of the total sample will be examined. This step will identify the extent to which executive function measures are associated with AD and AA scores. While the current study seeks to demonstrate that AD and AA are associated with executive dysfunction, the question of what threshold or score corresponds to impaired executive functioning will also be examined. Thus, of the significantly correlated associations between the AD and AA scales and executive functioning measures, the sensitivity and specificity of these scales to scores on executive functioning measures will be calculated. This analysis will identify if a threshold of psychopathology severity corresponds with executive functioning impairments.
CHAPTER 2

Methods

The primary goal of this study was to identify the executive functioning abilities uniquely associated with Anhedonic Depression, Anxious Arousal, and high levels in both Anhedonic Depression and Anxious Arousal compared to a control group.

Undergraduates were recruited to participate in this study if they demonstrated high scores on the AD and AA scales and consented to undergo a comprehensive battery of executive functioning measures in exchange for research credit and/or a chance to receive monetary compensation.

Participants

The participants in this study were composed of university undergraduates 18 years old and older who were recruited from mass testing, the Department of Psychology Psychological Clinic, and through fliers in the Psychological Clinic, Counseling and Human Development Center, and University Psychological Services. Only individuals who met pre-determined inclusion criteria outlined under the Procedures were recruited into this study.

The control group

The data for the control group were collected during the 2007-2008 academic year (Benitez, Gunstad, & Ben-Porath, 2007). Student participants voluntarily took part in
this study in exchange for undergraduate research credits. They provided responses to
the MASQ-SF, health history questionnaire, and were administered a brief
neuropsychological test battery. Of the executive functioning measures included in this
battery, the control group has data for the following tests: Letter-Number Sequencing,
Trails B, COWAT, Similarities, and Matrix Reasoning. In order to better distinguish the
control group from the other groups recruited into this study, only participants whose AD
or AA scores fell at or below the mean (i.e. 50th percentile) of the total control group
sample were included in analyses.

Measures

Mood and Anxiety Symptom Questionnaire – Short Form (MASQ-SF). The
MASQ-SF is a measure of emotional psychopathology that corresponds with the
constructs outlined in the Tripartite model (Watson et al. 1995). The MASQ-SF is an
abbreviated version of the MASQ which omits the items that contribute to the measure’s
General Distress scale and other items that are not scored on any scale. Sixty-two
symptoms of depression and anxiety experienced in the past week were rated on a 5-point
likert scale, ranging from 1 (not at all) to 5 (frequently), and were summed to create a
total score wherein higher scores indicate greater psychopathology. Of interest to this
study were the Anhedonic Depression (AD; 22 items) and Anxious Arousal (AA; 17
items) subscales, which correspond with the Tripartite model’s components of (low)
Positive Affect and (high) Physiological Hyperarousal, respectively. These scales have
demonstrated better discriminant validity in differentiating depressed and anxious groups
than other similar instruments (Reidy & Keogh, 1997; Watson et al., 1995). That is,
whereas many such instruments are saturated with non-specific items that measure general distress (Gotlib & Cane, 1989), the MASQ has been shown to delineate each domain clearly (e.g., Brown, Chorpita, & Barlow, 1998), as will be required in the current study. Good internal consistency was reported by Watson et al. (1995; Cronbach's alpha ranged from .78–.92). See Appendix A for a copy of the measure.

**Health History Questionnaire.** This questionnaire asks participants to provide basic demographic information and identify a history of conditions that may potentially impact cognitive function, including head injury, seizures, and psychiatric disorders (see Appendix B).

**Letter-Number Sequencing (LNS).** In this subtest of the Wechsler Adult Intelligence Scale-III (WAIS-III; Wechsler, 1997), the test administrator reads aloud a group of numbers and letters (e.g. G-1-T) then asks the participant to repeat them after rearranging them with the numbers first, in order from lowest to highest, followed by the letters in alphabetical order. The sequence length is increased until the patient fails three trials of a given length. The score is the total number of correct responses, which is regarded as a measure of working memory.

**Controlled Oral Word Association Test (COWAT).** This test asks individuals to generate as many words as possible to a given letter for 60 seconds, providing a measure of verbal fluency (Benton & Hamsher, 1989). Three letters were provided, and the total word score is the sum of responses across the three trials.

**Stroop Color-Word Test (SCWT; Golden, 1978).** This test is composed of three trials that each participant must complete as quickly as possible in 45 seconds. In the
first trial, the participant is asked to read aloud a list printed in black ink, composed of three color names randomly listed. In the second trial the participant is asked to rapidly name the color of “X’s” printed in colored ink, of which there are three colors. In the third trial, the participant is presented with color-words printed in a color different from the color which it names, and is instructed to name the color of the ink in which the word is printed. This task requires the participant to suppress interfering, automatic responses (i.e. reading out the word as opposed to identifying the color in which the word is printed). A higher score reflects better inhibition.

*Similarities.* This subtest of the WAIS-III requires the participant to describe how two objects or constructs are similar (Wechsler, 1997). The items are arranged in increasing order of difficulty, with higher scores reflective of better abstract verbal reasoning abilities.

*Matrix Reasoning.* This WAIS-III subtest of nonverbal abstract reasoning consists of four types of problems: pattern completion, classification, analogy, and series completion. Participants are presented 35 items in a booklet. They are instructed to look at the presented matrix and choose one item from the five options that completes the problem (Wechsler, 1997).

*Trail Making Test* (Reitan, 1958). Trail Making Test A is similar to the child’s game “Connect the Dots” and measures psychomotor speed and visual scanning. Participants are asked to connect a series of 25 numbered dots in ascending order as quickly as they can (e.g. 1-2-3, etc.). Of interest to this study is the Trail Making Test B (i.e. “Trails B”), which adds a set-shifting component by requiring participants to alternate
between numbers and letters in ascending order (e.g. 1-A-2-B, etc.). A high score indicates slowed cognitive set-shifting abilities.

*Wisconsin Card Sorting Test-64 (WCST-64).* The WCST-64 is a shorter version of the 128-item WCST, which is a test of the ability to conceptualize abstract categories and to disengage from perseveration according to changing contingencies (Heaton, Chelune, Talley, et al., 1993). The administration, scoring procedures, and psychometric properties of the WCST-64 are very comparable to those reported for the WCST (Heaton et al., 1993). Participants are asked to match each test card with one of four stimulus cards with different geometrical shapes. These cards vary in color (red, green, yellow, or blue), shape (triangle, star, plus, or circle), and number (1, 2, 3, 4). The participants are not informed of the rules governing the matching process, but are told by whether they matched correctly or incorrectly. Unbeknownst to the participant, there are three possible categorization rules: color, shape, and number. A total of 10 consecutively correct matches must be obtained before the categorization rule changes. The scores relevant to this study are the Total Number of Errors and Perseverative Responses.

*Rey-Osterrieth Complex Figure Test (RCFT).* A complex geometric form is presented to the participants who are instructed to reproduce the design on a piece of paper as accurately as possible (Knight & Kaplan, 2003). Although it is regarded as a test of visuospatial perception and memory, this study used the Boston Qualitative Scoring System (BQSS; Stern et al., 1999) to assess for planning and organizational abilities. The BQSS’s Organization score indicates the degree to which the participant reproduced the image in an integrated, organized fashion.
WRAT-3 Reading. A subtest of the Wide Range Achievement Test (3rd ed.; Wilkinson, 1993), this test has been shown to be a valid and reliable brief index of premorbid intellectual functioning (Ball, Hart, Stutts, Turf, & Barth, 2007). Participants are required to read a list of words of increasing difficulty, and each correctly pronounced word is given one point.

Procedure

Screening/Recruitment.

Participants were recruited primarily through mass testing sessions of the Fall and Spring semesters of academic year 2008-2009. The students who participated in mass testing were enrolled in lower-division psychology courses such as Introduction to Psychology and Psychology Research Methods. Each student completed the MASQ-SF along with other measures during mass testing and was informed that he or she may be invited to participate in another research project, if he or she consented to be contacted for this in the future. In addition, the MASQ-SF was posted on the undergraduate psychology research website (i.e. Sona Systems-1) to allow students who were not able to participate in mass testing to take the questionnaire online at their own convenience.

Other participants were also similarly recruited through other venues. The MASQ-SF was administered to all new clients that completed the research protocol in the Department of Psychology Psychological Clinic. A consent form that informed the new client about the purpose of the MASQ-SF accompanied the measure and allowed the client to indicate whether he or she agreed to be contacted for the study in the future. Fliers were also posted in the Psychological Clinic, the Counseling and Human
Development Center, and University Psychological Services to invite participants to take the MASQ-SF online (i.e. through Sona Systems-2) for a chance to be recruited into the larger study. Sona Systems-2 was also accessible to other students and psychology majors in upper-division psychology courses.

Potential participants were identified if their Anhedonic Depression (AD) or Anxious Arousal (AA) score was greater than or equal to one standard deviation above the mean scores of a sample of healthy undergraduates \((N=67;\) Benitez et al., 2007), from which the current study’s control group was obtained. Table 2 presents a comparison between AD and AA scores from previous studies with clinical samples and AD and AA scores that are one standard deviation above the mean of the total healthy undergraduate sample. Although a recruitment criterion of \(AD \geq 64.94\) may be less stringent given the higher scores obtained in previous studies with clinical samples, the criterion of \(AA \geq 30.74\) appears comparable to AA scores obtained other studies. It was therefore expected that the mean scores of the participants recruited into this study would be comparable to those reported in previous studies with clinical samples.

For those recruited through mass testing, each participant was invited via telephone or email to participate in this study in exchange for research points. They were also informed that participating in this study would make them eligible for a raffle where one participant each week had a chance to win a $20 gift card. For the participants recruited through other methods, they were informed that they would be compensated with a $20 gift card for their participation.

**Testing.** Testing sessions were conducted by graduate student research assistants,
and the questionnaires and neuropsychological testing took approximately 90 minutes to complete. The MASQ-SF was re-administered at the start of the session to establish the stability of their AD or AA scores. The health history questionnaire was administered prior to cognitive testing. Each research assistant was also instructed to provide behavioral observation ratings for each participant, with a focus on assessing each participants’ level of motivation or effort on all neuropsychological tests administered, as well as their level of cooperation with all the requirements of the study.

**Analyses**

Demographic and medical history information were examined using Analysis of Variance (ANOVA) with post-hoc testing and Chi-square analyses to identify differences between the groups (i.e., Control, AD, AA, AD&AA). Comparisons were also conducted based to determine possible impact of recruitment methods. Participants who identified a primary language other than English, endorsed a history of significant neurologic illness/injury (e.g. head injury, developmental disorder), or who a research assistant indicated was exerting suboptimal effort during testing were excluded from further analyses. Because the stability of MASQ-SF scores has not been reported in previous studies, participants’ scores at recruitment (i.e. mass testing or Sona Systems-1 or -2) were correlated with those obtained on the day of testing to identify the stability of these scores.

To address Primary Aims 1 and 3, executive functioning test performance of the Control group and groups of participants with high scores on AD, AA, and both scales were compared. To limit the number of statistical tests performed on the executive
functioning tests, only when the overall multivariate analysis of covariance (MANCOVA) was statistically significant were the ANOVA and planned comparisons conducted to localize effects outlined in Primary Aim 2. Multivariate analyses included an index of premorbid intellectual functioning as a covariate, as this is the preferred index of cognitive reserve. Cognitive reserve theory proposes that premorbid factors such as level of education, occupational attainment, or intelligence serve as buffers against cognitive impairment, accounting for instances wherein individuals with the same brain disease manifest vastly different cognitive and functional capacities (Stern, 2002). Because of the restricted range of age and education levels in this college undergraduate sample, a premorbid intellectual functioning estimate was selected as a measure of cognitive reserve.

To address the Secondary Aim, bivariate correlations between the AD and AA scales and the scores on the executive functioning measures were conducted. Then, the classification accuracies of the AD and AA scales to impaired performance (i.e. exceeding -1.5 SD) on the executive functioning measures were computed, along with receiver operating characteristic (ROC) curve analyses to evaluate their effectiveness. Estimates based on previous studies indicate that significant proportions of samples of individuals with depressive and/or anxiety pathology demonstrate impaired performance on measures such as Trails B (i.e. 40% ; Airaksinen et al., 2005, based on norms from Tombaugh, Rees, & McIntyre, 1996) and the WCST (50%; Boldrini et al., 2005, based on Kongs et al. 2000 norms). As such, it was expected that participants in the current study would show comparable and prevalent rates of impairment.
For all analyses, raw scores (and not standardized or scaled scores) were used. Though it may have been beneficial to convert all test results to a standard metric, it was deemed that using standardized scores could reduce variability (particularly for scaled scores) and may result in artificially anchoring the scores to a normative sample that is dissimilar from the current sample.
CHAPTER 3

Results

Characteristics of Study Participants

Participants were excluded from analyses if they identified a primary language other than English, exhibited suboptimal effort during testing, or endorsed a history of significant neurologic illness or injury. There were 11 participants excluded based on these criteria, resulting in a total sample size of 107. There were no significant differences between the excluded participants and the current sample on demographic and test variables.

The demographic characteristics of the study participants are presented in Table 3. The Control, AD, AA, and AD&AA groups did not differ in age, years of education, premorbid verbal intellectual functioning (i.e. all in the average range), gender, marital status, ethnic minority status, handedness, or recruitment method. Differences between participants recruited in Fall 2008 and Spring 2009 were examined; due to difficulties with recruiting participants in Fall 2008, the inclusion criteria scores were relaxed from 2 SD (as originally proposed) to 1 SD above the mean for the AD and AA scores (as suggested in the prospectus meeting). Thus, participants recruited in Spring 2009 had significantly lower scores on AD and AA than the Fall 2008 participants, though they did not differ on any demographic characteristics, recruitment method, or test performance.
Participants recruited through mass testing also did not differ from those recruited online on most demographic variables, premorbid intellectual functioning, AD scores at recruitment and testing, or neuropsychological test scores. However, participants who completed the MASQ-SF online on Sona Systems-2 achieved one more year of education (M=14.43, SD=1.20) than those who completed the MASQ-SF online on Sona Systems-1 (M=13.54, SD=0.83; F(2, 79)=4.30, p<.05). The participants recruited through Sona Systems-2 also had significantly higher scores on AA at recruitment (M=38.38, SD=12.05; F(2, 78)=5.31, p<.01) and testing (M=32.26, SD=11.55; F(2, 78)=5.55, p<.01) than the AA scores at recruitment and testing obtained by the groups from mass testing (M=31.15, SD=9.605 and M=25.97, SD=7.57, respectively) and Sona Systems-1 (M=29.42, SD=9.11 and M=24.04, SD=7.69, respectively). These findings were not unexpected, given that the MASQ-SF on Sona Systems-2 was accessible to students in upper-division psychology courses and to clients who potentially had higher levels of psychopathology and were seeking services from one of the three campus mental health facilities in which fliers for this study were posted.

**MASQ-SF Scores Between Recruitment and Testing Among the Study Groups**

Between-group comparisons (Table 4) show that AD and AA scores across the Control, AD, AA, and AD&AA groups showed identical relationships at recruitment and testing. As expected, the AD, AA, and AD&AA groups had significantly higher scores on the scales than the Control group, and the AD&AA groups’ scores were not significantly different from the high scores that defined the AD or AA groups. In addition, the AD scores of the AD group were higher than the AD scores of the AA
group, and conversely, the AA scores were higher in the AA group than the AD group. In summary, the AD and AA scores successfully differentiated the four groups at both recruitment and testing.

Correlations between MASQ scores at recruitment and testing were high for both AD ($r=.73$, $p<.01$) and AA ($r=.71$, $p<.01$), and a smaller correlation was found between AD and AA scores at testing ($r=.19$, $p<.05$). An average of 33.70 (SD=19.02) days elapsed between recruitment and testing, and the groups did not significantly differ on this variable.

Within-group analyses using paired samples t-tests indicate a significant decrease in AD (t (83)=5.93, $p<.001$) and AA (t (83)=6.39, $p<.001$) scores between recruitment and testing for the AD, AA, and AD&AA groups. On average, AD and AA scores decreased by 5.91 (SD=9.12) and 5.33 (SD=7.65) points, respectively. Furthermore, of the participants that were recruited into the AD, AA, and AD&AA groups, only 19, 4, and 12 participants still met criteria for these groups at testing, respectively (see Table 5).

*Group Differences in Executive Functioning Test Performance*

To address Aim 1, Multivariate Analysis of Covariance (MANCOVA) was conducted to compare the executive functioning test performance of the Control, AD, AA, and AD&AA groups, controlling for premorbid intellectual functioning as estimated by the WRAT-3 Reading score. The current study found a significant omnibus difference among the Control, AD, AA, and AD&AA groups on COWAT, Trails B, Similarities, Matrix Reasoning, and Letter-Number Sequencing: $F(15, 270.94)=1.80$, $p<.05$, partial $\eta^2=.084$. Despite this significant result, univariate analyses indicated non-significant
differences on specific tests between groups, although Similarities approached significance (p=.055). See Table 6 for means and standard deviations for each group.

To address Aim 2, a MANCOVA was conducted to investigate the differences between the AD, AA, and AD&AA groups on the aforementioned executive functioning tests in addition to SCWT Color-Word, WCST Total Number of Errors, WCST Perseverative Responses, and RCFT Organization. No significant omnibus difference was found: F(18, 138)=1.12, p=.344, partial $\eta^2=.127$. However, univariate analyses indicated a significant difference between the groups on SCWT Color-Word test, wherein the AA group ($M=42.72$, S.E.=2.46) named fewer Color-Word pairs than the AD group ($M=50.85$, S.E.=1.65; F(2, 77)=3.74, p<.05, partial $\eta^2=.09$). The means and standard deviations of the raw scores of the executive functioning measures for the Control, AD, AA, and AD&AA groups are presented in Table 6. In addition, the proportions of participants that exhibited impaired performance on these tests are presented. Comparison of rates of impairments across groups revealed the AA group had more participants with impaired performance on Trails B than the other groups. It is notable that the AD&AA group did not have more participants with impaired performance than either the AD or AA groups.

Classification Accuracy of the AD and AA scales in Predicting Executive Functioning Deficits

Bivariate correlations between the AD and AA scores at recruitment and testing and all executive functioning measures were conducted (see Table 7). Two executive functioning measures (i.e. Letter-Number Sequencing and SCWT Color-Word)
demonstrated consistent, significant relationships with AD and AA scores during recruitment and testing. Consistent with study hypotheses, small correlations were observed between Letter-Number Sequencing performance and AD scores both at recruitment and testing. An unexpected but similarly modest correlation was observed between SCWT Color-Word and AA scores at recruitment and testing, as this was expected to be more associated with AD scores. A correlation emerged between COWAT and AA scores at recruitment; because these data were collected at different time points and are marginally correlated, this association will not be interpreted.

To address the secondary aim of this study, the classification accuracies of the AD and AA scores in predicting impairment on executive functioning measures were examined (see Table 8). Because of the very low number of participants with impaired performance on Letter-Number Sequencing and SCWT Color-Word, the classification accuracies of AD and AA to impaired performance on other measures with higher base rates of impairment (i.e. COWAT, Trails B, Rey-CFT Organization) were explored. Area under the ROC curve (AUC) provided an index of discriminative power. As the results indicate, neither the AD nor the AA scores were useful in predicting impaired neuropsychological test performance (i.e. beyond -1.5 SD).
CHAPTER 4

Discussion

This study examined whether individuals with unique depression or anxiety pathology exhibited differentiable impairments on executive functioning tests and whether these groups could be distinguished from control and comorbid depression/anxiety groups. Contrary to expectations, very few between-group differences were observed across Control, AD, AA, and AD&AA groups. Correlations revealed some expected and unexpected associations, though these were relatively modest. Despite these relationships, neither the AD nor the AA scale emerged as a good predictor of executive functioning deficits.

In spite of these limited findings, the current study makes several important contributions to the existing literature. First, the current study extends past work by employing a more extensive collection of executive function measures (i.e. including Rey-CFT Organization, Similarities, Matrix Reasoning) than those used in previous studies with non-pathological samples. Despite the importance of organization and abstract reasoning to executive functioning, no association to affective or anxiety pathology emerged in the current sample. Future research may benefit from exploring these aspects of executive functioning in more pathological samples, as there may be more variability in these abilities in more impaired populations. For instance, Austin and
colleagues (1999) demonstrated that depressed outpatients produced worse scores on Similarities than controls, particularly the non-melancholic subgroup. More exploration into the differential associations between dimensions of psychopathology and their cognitive correlates, particularly with non-traditional measures, may yield further information.

Second, the current findings suggest that AD and AA scores are uniquely associated with different aspects of executive functioning. The presence of anhedonic depression was associated with working memory deficits (i.e. Letter-Number Sequencing). Prior research has identified that depressed individuals not only struggle with processing affectively arousing information, but have a primary deficit in the effortful processing of complex stimuli, regardless of its emotional valence (Rose & Ebmeier, 2006). Anxious arousal, on the other hand, was associated with difficulties with inhibiting prepotent responses (i.e. SCWT Color-Word), which is more typically associated with depression. However, considering that anxiety in this study was indexed by propensity towards increased physiological hyperarousal, it is possible that this correlation reflects the sympathetic arousal component of anxiety, wherein neuroendocrine disturbances lead to disruption of ability to suppress automatic, involuntary responses (McFarlane, 2000). Interestingly, a similar pattern of deficits is found in persons with PTSD (Stein, Kennedy, & Twamley, 2002), suggesting a possible mechanism for neuropsychological impairment in that population.

Third, the current study encourages the future use of the dimensional perspective to better understand the effects of comorbidity on neuropsychological function.
Dimensional models are well-suited to identifying the relative contributions of multiple disorders, as these allow researchers to partial out extraneous confounds (i.e. general distress) and focus on the independent effects of distinct, underlying pathologies. In the current study, the group with high scores on both the AD and AA scales did not exhibit a greater rate of cognitive impairment than the AD or AA groups. Contrary to the assumption that individuals with comorbid depression and anxiety should have more severe psychopathology and, as a result, a greater likelihood of cognitive impairment, it appears that depression and anxiety demonstrate a non-additive effect on cognition. A similar result has been mentioned in previous work (e.g. Keller et al., 2000), where it was demonstrated that depression and anxiety were associated with decreases and increases in activity in a specific brain region, respectively, suggesting that these counteractive effects explain the discrepant findings across studies that did or did not account for comorbidity. Although the modest findings in the current study only provide limited support for this hypothesis, future researchers are encouraged to assess the unique components of these psychopathologies and to consider that comorbid disorders may not necessarily correspond with greater cognitive dysfunction.

Limitations

There are several features of this study that may account for the lack of supported hypotheses. First, the participants in the current study had lower rates of impairment on measures of executive function than those in past studies, despite similar MASQ scores (See Tables 2 and 4). It is possible that recruitment of a college student sample included persons who were higher functioning than community-based sample, thereby potentially
decreasing the variability in executive functioning performance. Also, due to significant
difficulties with data collection, the inclusion criteria were relaxed from 2 SD above the
mean on AD or AA to 1 SD during the second semester of data collection, which may
have resulted in a sample with less severe forms of psychopathology. However, analyses
showed that participants that were recruited from the Fall and Spring semesters did not
deriff on executive function test performance.

Given these findings, it is likely that other characteristics unique to current sample
account for the minimal differences between the groups. Despite the potential effects of
neurologic compromise seen in high levels of psychopathology, individuals who are
successfully enrolled in college may possess adequate levels of cognitive reserve and
personal resources to compensate for potential difficulties. Cognitive reserve refers to
the mind’s resilience to neuropathological damage, in that factors such as level of
education or intelligence serve as buffers against cognitive impairment (Stern, 2002).
The participants in this study are predominantly in the average range of intelligence and
are likely to be cognitive resilient despite moderate levels of psychopathology.
Furthermore, several of the participants in the study had taken psychology courses and
may have had some knowledge of test mechanics, particularly the SCWT and WCST.
Exploring these variables in populations with more functional impairment and who have
a less sophisticated understanding of cognitive measures may capture greater variability.

A second potential limitation is that the unique features of depression and anxiety
as outlined in the Tripartite Model may be less related to neurocognitive deficits than
believed. Although the MASQ-SF scales demonstrated stable relationships between
recruitment and testing, these scales may be limited in their ability to predict cognitive
effects of affective or anxiety psychopathology. For instance, Keller and others (2000)
found no correlations between AD and AA scores and indices of hemispheric asymmetry
unique to depression or anxiety, but demonstrated significant relationships with scores
from the Beck Depression Inventory (BDI; Beck & Steer, 1993a) and State-Trait Anxiety
Inventory (STAI; Spielberger, 1983). In their discussion they explain that the AD and
AA scales may not be tapping into pathologies related to the cognitive abilities that are
driven by the specific hemispheric regions assessed in this study. However, the
researchers did not address a crucial difference between these scales and measures like
the BDI and STAI; the latter measures are saturated with a general distress factor
common to both depression and anxiety, which account for these measures’ limited
ability to discriminate between the disorders. For instance, Steer and colleagues (1995)
factor analyzed responses to the BDI (BDI; Beck & Steer, 1993a) and Beck Anxiety
Inventory (BAI; Beck & Steer, 1993b) and found that a general distress factor accounted
for the majority of the variance common to both measures, while depression- and
anxiety-specific factors composed of low positive affect/ anhedonia BDI items and
physiological BAI items, respectively, were identified. Considering this information in
light of the lack of supported hypotheses in the current study, it is possible that the
cognitive deficits in depression or anxiety identified in previous research are more closely
associated with general levels of distress rather than unique features of each disorder.
Future researchers are encouraged to examine this possibility through projects involving
multiple measures of affective and anxiety psychopathology.
A third important consideration is that an extraneous variable may moderate the relationship between executive functioning measures and psychopathology. In investigating the ecological validity of common executive functioning measures (i.e. COWAT, Trails B, WCST) with neuropsychology outpatients, Chaytor and colleagues (2007) identified that assessing cognitive compensation strategy use and environmental demands helped predict adaptive daily functioning. That is, cognitive test performance better predicted adaptive daily functioning after accounting for reported compensation strategy use and the frequency and impact of executive functioning-type problems in daily life, than cognitive test performance alone. It may be possible that executive functioning deficits can be overcome through additional effort in persons with psychopathology. The high functioning participants in this study may have identified effective compensatory strategies and employed these throughout testing to maximize their test performance. However, even with intact performance on testing, these individuals may display poor executive function on more complex, real-world tasks. Although this is speculative, it would be useful for future studies to consider the influence of self-reported cognitive problems and compensatory cognitive strategies, particularly in high functioning populations.

Some practical, design-related issues may also have influenced the current findings. First, many of the participants that met criteria for either of the three groups based on their AD and AA scores at the time of recruitment no longer met criteria on the day of testing. Although it would have been preferable to only include the participants that met inclusion criteria at both recruitment and testing, this would require several years
of data collection as participants’ scores tended to regress to the mean.

Second, the relatively small sample sizes may have obscured potential findings. However, given the small effect sizes observed for many of the executive functioning measures, an inordinate number of participant data would be needed to reach statistical significance. For example, post-hoc power analyses indicate that 232 participants would be needed to reach significance for the between-group difference in Similarities from the first MANCOVA (p. 29), while 17,200 participants would be needed to reach significance for Rey-O CFT Organization described in the second MANCOVA.

Third, the current study did not control for whether participants were taking medication for their depression or anxiety symptoms, which may have attenuated the cognitive deficits observed. Fourth, the test battery did not include neuropsychological measures that exclusively measure cognitive effort, thereby precluding the option to exclude participants based on a set, pre-identified score that indicates insufficient effort. Although less than ideal, the research assistants of this study were tasked to identify participants who did not exert sufficient effort, and 5 protocols were excluded based on this criterion. However, the very low rates of neuropsychological impairment suggest that participants gave reasonable effort, though some mitigation of performance cannot be fully excluded.

Lastly, in terms of methodology, the order in which the tests were administered was fixed and not randomized. Although counterbalancing the test measures may have minimized any potential impact of participant fatigue, the tests relevant to the current study were a subset of measures administered as part of a larger study.
neuropsychological test battery of the larger study included tests of memory, which required a less flexible ordering of measures to ensure that sufficient time elapsed between learning and delayed recall trials. Furthermore, the test battery was relatively short compared to those used in many clinical and research settings, thus minimizing likely impact of fatigue.

**Clinical Implications**

Despite the aforementioned limitations, this study provides some information that would be of clinical utility, particularly since a large proportion of individuals who seek neuropsychological evaluations also report symptoms of depression and anxiety (Kanner, 2005). Affective and anxiety symptoms should be regarded in relation to executive functioning and how these might influence the effectiveness of a given form of intervention. For instance, individuals with high levels of anhedonic depression may benefit from having information conveyed in discrete and straightforward increments so as to prevent the adverse impact of compromised working memory abilities. In the same way, as anxious arousal was associated with poorer inhibition, interventions could focus on developing mindfulness-based response-prevention strategies in distressing situations to minimize anxious behavior such as rumination or hypervigilance. Although other indices of executive functioning were not found to be significantly effective in differentiating these psychopathologies in this study, these measures are likely to contribute additional information regarding the unique cognitive characteristics of each individual and should thus be regarded in conjunction with other cognitive strengths and weaknesses.
In conclusion, the current study demonstrated that executive functioning abilities are only modestly associated with unique features of depression and anxiety in this sample of high functioning, healthy young adults. Future studies examining populations with more severe psychopathology and functional impairment are needed to clarify the contribution of affective and anxiety disorders to impaired neuropsychological function. Prospective studies that include serial cognitive assessment and neuroimaging are particularly needed.
REFERENCES


Benitez, A., Gunstad, J., & Ben-Porath, Y. S. (2007, November). Attention and memory are associated with MMPI-2 Restructured Clinical (RC) scales in healthy undergraduates. Poster accepted for presentation at the annual meeting of the National Academy of Neuropsychology, Scottsdale, AZ.


Figure 1. The Tripartite Model

Negative Affect

$\uparrow$NA and $\downarrow$PA $=$ Depression

$\uparrow$NA, $\downarrow$PA, and $\uparrow$AA $=$ Comorbid Depression and Anxiety

$\uparrow$NA and $\uparrow$AA $=$ Anxiety

Positive Affect

Anxious Arousal

NA, PA, and AA

Comorbid Depression and Anxiety

Depression

Anxiety
<table>
<thead>
<tr>
<th>Test</th>
<th>Modality</th>
<th>Specific Ability</th>
</tr>
</thead>
<tbody>
<tr>
<td>Letter-Number Sequencing (LNS; Wechsler, 1997)</td>
<td>Verbal</td>
<td>Working memory; Ability to maintain &amp; manipulate a limited set of information</td>
</tr>
<tr>
<td>Controlled Oral Word Association Test (COWAT; Benton &amp; Hamsher, 1989)</td>
<td>Verbal</td>
<td>Verbal fluency, or the ease with which words can be produced given fixed criteria</td>
</tr>
<tr>
<td>Stroop Color-Word Test (SCWT; Golden, 1978)</td>
<td>Verbal</td>
<td>Inhibition or suppression of automatic responses</td>
</tr>
<tr>
<td>Similarities (Wechsler, 1997)</td>
<td>Verbal</td>
<td>Verbal abstract reasoning</td>
</tr>
<tr>
<td>Matrix Reasoning (Wechsler, 1997)</td>
<td>Non-verbal</td>
<td>Non-verbal abstract reasoning</td>
</tr>
<tr>
<td>Trail Making Test (Trails B; Reitan, 1958)</td>
<td>Non-verbal</td>
<td>Cognitive set-shifting, or ability to switch between competing cognitive sets</td>
</tr>
<tr>
<td>Wisconsin Card Sorting Test (WCST; Heaton, Chelune, Talley, et al., 1993)</td>
<td>Non-verbal</td>
<td>Concept-formation, and ability to disengage from perseveration and adapt to change</td>
</tr>
<tr>
<td>Rey-Osterrieth Complex Figure Test (RCFT; Knight &amp; Kaplan, 2003)</td>
<td>Non-verbal</td>
<td>Organization and planning as applied to the reproduction of a complex figure</td>
</tr>
</tbody>
</table>
Table 2. Means and Standard Deviations of Raw Scores on AD and AA From Clinical Studies

<table>
<thead>
<tr>
<th>Study</th>
<th>Patient diagnoses</th>
<th>AD Mean (SD)</th>
<th>AA Mean (SD)</th>
</tr>
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<tbody>
<tr>
<td>Benitez et al. (2007)</td>
<td>Healthy undergraduates (N=67)</td>
<td>64.94\textsuperscript{a}</td>
<td>30.74\textsuperscript{b}</td>
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<tr>
<td>Buckby et al. (2007)</td>
<td>Depressive disorder (N=29)</td>
<td>82.82 (17.13)</td>
<td>38.11 (16.38)</td>
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<td>Buckby et al. (2007)</td>
<td>Anxiety disorder (N=22)</td>
<td>67.18 (17.13)</td>
<td>30.63 (11.18)</td>
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<tr>
<td>Chiu &amp; Deldin (2007)</td>
<td>Depressive disorder (N=140)</td>
<td>86.38 (8.48)</td>
<td>29.67 (10.01)</td>
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<td>Clark et al. (1998)</td>
<td>Depressive disorder (N=21)</td>
<td>81.33 (11.22)</td>
<td>37.33 (11.40)</td>
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<tr>
<td>Hughes et al. (2006)</td>
<td>Social phobia (N=148)</td>
<td>67.48 (14.53)</td>
<td>25.35 (8.09)</td>
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<td>Lee et al. (2005)</td>
<td>Depressive disorder (N=37)</td>
<td>Not reported</td>
<td>37.70 (12.60)</td>
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<tr>
<td>Norton &amp; Hope (2005)</td>
<td>Anxiety disorder (N=23)</td>
<td>Not reported</td>
<td>25.71 (4.08)</td>
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<td>Yung et al. (2007)</td>
<td>Depressive disorder (N=105)</td>
<td>76.11 (17.28)</td>
<td>Not reported</td>
</tr>
</tbody>
</table>

\textsuperscript{a} Raw score that is one standard deviation above the group mean for AD (X=51.51, SD=13.43)

\textsuperscript{b} Raw score that is one standard deviations above the group mean for AA (X=23.93, SD=6.81)
### Table 3. Demographic Characteristics of the Control, AD, AA, and AD&AA groups

<table>
<thead>
<tr>
<th>Index</th>
<th>Controls (n=26)</th>
<th>AD (n=34)</th>
<th>AA (n=15)</th>
<th>AD&amp;AA (n=32)</th>
<th>F / $\chi^2$</th>
<th>P</th>
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</thead>
<tbody>
<tr>
<td>Age</td>
<td>19.00 ± 1.20</td>
<td>19.91 ± 2.14</td>
<td>19.13 ± 1.51</td>
<td>20.22 ± 2.70</td>
<td>2.14</td>
<td>.100</td>
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<tr>
<td>Education</td>
<td>13.38 ± .75</td>
<td>13.85 ± 1.11</td>
<td>13.80 ± 1.15</td>
<td>14.00 ± 1.16</td>
<td>1.73</td>
<td>.165</td>
</tr>
<tr>
<td>WRAT-3</td>
<td>48.42 ± 3.23</td>
<td>48.94 ± 4.26</td>
<td>47.07 ± 3.22</td>
<td>46.91 ± 5.10</td>
<td>1.63</td>
<td>.188</td>
</tr>
<tr>
<td>Reading</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female %</td>
<td>61.54</td>
<td>82.35</td>
<td>73.33</td>
<td>90.63</td>
<td>7.76</td>
<td>.051</td>
</tr>
<tr>
<td>Single %</td>
<td>100</td>
<td>97.06</td>
<td>100</td>
<td>96.88</td>
<td>1.27</td>
<td>.736</td>
</tr>
<tr>
<td>White %</td>
<td>100</td>
<td>91.18</td>
<td>73.33</td>
<td>90.63</td>
<td>16.08</td>
<td>.065</td>
</tr>
<tr>
<td>Right hand %</td>
<td>92.31</td>
<td>82.35</td>
<td>86.67</td>
<td>93.75</td>
<td>5.46</td>
<td>.487</td>
</tr>
<tr>
<td>Mass Testing</td>
<td>n.a.</td>
<td>41.18</td>
<td>53.33</td>
<td>37.50</td>
<td>7.21</td>
<td>.302</td>
</tr>
<tr>
<td>Recruit %</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Table 4. AD and AA Scores at Recruitment and Testing for the Control, AD, AA, AD&AA Groups

<table>
<thead>
<tr>
<th>Score</th>
<th>Control (n=26)</th>
<th>AD (n=34)</th>
<th>AA (n=15)</th>
<th>AD&amp;AA (n=32)</th>
<th>M</th>
<th>S.D.</th>
<th>M</th>
<th>S.D.</th>
<th>M</th>
<th>S.D.</th>
<th>M</th>
<th>S.D.</th>
<th>F / χ²</th>
<th>Tukey &lt;sup&gt;a&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>AD</td>
<td>42.81</td>
<td>7.26</td>
<td>75.71</td>
<td>8.34</td>
<td>52.87</td>
<td>9.42</td>
<td>77.44</td>
<td>8.96</td>
<td>111.63</td>
<td>1&lt;2, 1&lt;3, 1&lt;4, 3&lt;2, 3&lt;4</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AA</td>
<td>19.54</td>
<td>1.70</td>
<td>23.09</td>
<td>4.32</td>
<td>38.93</td>
<td>7.92</td>
<td>40.03</td>
<td>8.65</td>
<td>78.35*</td>
<td>1&lt;3, 1&lt;4, 2&lt;3, 2&lt;4</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AD</td>
<td>42.81</td>
<td>7.26</td>
<td>67.68</td>
<td>9.59</td>
<td>53.67</td>
<td>13.28</td>
<td>70.09</td>
<td>10.74</td>
<td>44.69*</td>
<td>1&lt;2, 1&lt;3, 1&lt;4, 3&lt;4</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AA</td>
<td>19.54</td>
<td>1.70</td>
<td>22.91</td>
<td>5.22</td>
<td>30.60</td>
<td>10.99</td>
<td>30.13</td>
<td>10.47</td>
<td>12.59*</td>
<td>1&lt;3, 1&lt;4, 2&lt;3, 2&lt;4</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**p<.001; <sup>a</sup> In Tukey column, 1=Control, 2=AD, 3=AA, 4=AD&AA, < indicates significant difference (p<0.01); <sup>b</sup> The values for the Control group are the same at Recruitment and Testing.
Table 5. Percentages of Each Group that Met or Did Not Meet Cut-off Criteria at Testing

<table>
<thead>
<tr>
<th>Group</th>
<th>AD (n=34)</th>
<th>AA (n=15)</th>
<th>AD&amp;AA (n=32)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Met AD criteria at Testing</td>
<td>55.88</td>
<td>6.67</td>
<td>43.75</td>
</tr>
<tr>
<td>Met AA criteria at Testing</td>
<td>5.88</td>
<td>26.67</td>
<td>6.25</td>
</tr>
<tr>
<td>Met both AD and AA criteria</td>
<td>2.94</td>
<td>20.00</td>
<td>37.50</td>
</tr>
<tr>
<td>at Testing</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Did not meet criteria for</td>
<td>35.29</td>
<td>46.67</td>
<td>12.50</td>
</tr>
<tr>
<td>both AD and AA at Testing</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Table 6. Executive Functioning Test Performances of Control, AD, AA, and AD&AA Groups

<table>
<thead>
<tr>
<th>Measure</th>
<th>Controls (n=26)</th>
<th>AD (n=34)</th>
<th>AA (n=15)</th>
<th>AD&amp;AA (n=32)</th>
<th>F / χ²</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>S.D.</td>
<td>M</td>
<td>S.D.</td>
<td>M</td>
<td>S.D.</td>
</tr>
<tr>
<td>LNS</td>
<td>12.65</td>
<td>3.12</td>
<td>11.35</td>
<td>3.47</td>
<td>11.13</td>
<td>1.89</td>
</tr>
<tr>
<td>Imp. %</td>
<td>0</td>
<td>5.9</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>COWAT</td>
<td>36.62</td>
<td>8.63</td>
<td>36.56</td>
<td>10.66</td>
<td>41.07</td>
<td>12.29</td>
</tr>
<tr>
<td>Imp. %</td>
<td>15.4</td>
<td>26.5</td>
<td>13.3</td>
<td>18.8</td>
<td>1.70</td>
<td>.64</td>
</tr>
<tr>
<td>SCWT</td>
<td>51.47</td>
<td>8.63</td>
<td>42.33</td>
<td>9.46</td>
<td>47.31</td>
<td>10.89</td>
</tr>
<tr>
<td>Color-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Word</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Imp. %</td>
<td>n.a.</td>
<td>2.9</td>
<td>6.7</td>
<td>9.4</td>
<td>1.19</td>
<td>.55</td>
</tr>
<tr>
<td>Similar.</td>
<td>24.69</td>
<td>3.51</td>
<td>22.71</td>
<td>4.93</td>
<td>21.40</td>
<td>4.93</td>
</tr>
<tr>
<td>Imp. %</td>
<td>0</td>
<td>5.9</td>
<td>13.3</td>
<td>0</td>
<td>6.52</td>
<td>.09</td>
</tr>
<tr>
<td>Reason.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Imp. %</td>
<td>3.8</td>
<td>2.9</td>
<td>0</td>
<td>3.1</td>
<td>.55</td>
<td>.91</td>
</tr>
<tr>
<td>Trails B</td>
<td>58.30</td>
<td>16.22</td>
<td>52.18</td>
<td>17.68</td>
<td>60.87</td>
<td>26.66</td>
</tr>
<tr>
<td>Imp. %</td>
<td>19.2</td>
<td>5.9</td>
<td>33.3</td>
<td>3.1</td>
<td>11.2</td>
<td>.01</td>
</tr>
<tr>
<td>WCST</td>
<td>n.a.</td>
<td>14.32</td>
<td>6.45</td>
<td>12.67</td>
<td>7.70</td>
<td>12.25</td>
</tr>
</tbody>
</table>
Total # of

Errors

<table>
<thead>
<tr>
<th></th>
<th>Imp. %</th>
<th>WCST</th>
<th>Respons.</th>
<th>RCFT</th>
<th>Org</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n.a.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2.9</td>
<td>6.7</td>
<td>3.1</td>
<td>.46</td>
<td>.80</td>
<td>n.a.</td>
</tr>
<tr>
<td>5.9</td>
<td>6.7</td>
<td>0</td>
<td>2.05</td>
<td>.36</td>
<td>n.a.</td>
</tr>
<tr>
<td>32.4</td>
<td>40.0</td>
<td>46.9</td>
<td>1.46</td>
<td>.48</td>
<td></td>
</tr>
</tbody>
</table>

Note: Imp. %: Percentage of participants with impaired performance defined by a score 2 standard deviations below the mean.
Table 7. Bivariate Correlations Between AD and AA Scores and Executive Functioning Measures

<table>
<thead>
<tr>
<th>Measure</th>
<th>AD (Rec)</th>
<th>AA (Rec)</th>
<th>AD (Test)</th>
<th>AA (Test)</th>
</tr>
</thead>
<tbody>
<tr>
<td>LNS&lt;sup&gt;a&lt;/sup&gt;</td>
<td>-.21*</td>
<td>-.10</td>
<td>-.19*</td>
<td>-.12</td>
</tr>
<tr>
<td>COWAT&lt;sup&gt;a&lt;/sup&gt;</td>
<td>.04</td>
<td>.19*</td>
<td>.10</td>
<td>.09</td>
</tr>
<tr>
<td>SCWT Color-Word&lt;sup&gt;b&lt;/sup&gt;</td>
<td>.18</td>
<td>-.29**</td>
<td>.14</td>
<td>-.27*</td>
</tr>
<tr>
<td>Similarities&lt;sup&gt;a&lt;/sup&gt;</td>
<td>-.09</td>
<td>-.01</td>
<td>-.10</td>
<td>-.12</td>
</tr>
<tr>
<td>Matrix Reasoning&lt;sup&gt;a&lt;/sup&gt;</td>
<td>.15</td>
<td>-.03</td>
<td>.12</td>
<td>-.05</td>
</tr>
<tr>
<td>Trails B Time&lt;sup&gt;a&lt;/sup&gt;</td>
<td>-.14</td>
<td>.06</td>
<td>-.07</td>
<td>.16</td>
</tr>
<tr>
<td>WCST Total Number of Errors&lt;sup&gt;b&lt;/sup&gt;</td>
<td>.00</td>
<td>-.14</td>
<td>-.09</td>
<td>-.05</td>
</tr>
<tr>
<td>WCST Perseverative Responses&lt;sup&gt;b&lt;/sup&gt;</td>
<td>.01</td>
<td>-.11</td>
<td>-.10</td>
<td>-.09</td>
</tr>
<tr>
<td>RCFT Organization&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-.05</td>
<td>.02</td>
<td>-.04</td>
<td>-.01</td>
</tr>
</tbody>
</table>

* p<.05, **p<.01; <sup>a</sup>N=107; <sup>b</sup>n=81
Table 8. Classification Accuracy for AD and AA Scales Across Select Executive Functioning Tests

<table>
<thead>
<tr>
<th>Scale</th>
<th>Measure</th>
<th>BR</th>
<th>Sens.</th>
<th>Spec.</th>
<th>HR</th>
<th>PPP</th>
<th>NPP</th>
<th>AUC</th>
</tr>
</thead>
<tbody>
<tr>
<td>AD</td>
<td>LNS</td>
<td>.02</td>
<td>.50</td>
<td>.77</td>
<td>.77</td>
<td>.04</td>
<td>.99</td>
<td>.46</td>
</tr>
<tr>
<td>AA</td>
<td>LNS</td>
<td>.02</td>
<td>.50</td>
<td>.83</td>
<td>.82</td>
<td>.05</td>
<td>.99</td>
<td>.48</td>
</tr>
<tr>
<td>AD</td>
<td>SCWT-CW</td>
<td>.06</td>
<td>.00</td>
<td>.67</td>
<td>.63</td>
<td>.00</td>
<td>.91</td>
<td>.53</td>
</tr>
<tr>
<td>AA</td>
<td>SCWT-CW</td>
<td>.06</td>
<td>.40</td>
<td>.78</td>
<td>.75</td>
<td>.11</td>
<td>.95</td>
<td>.61</td>
</tr>
<tr>
<td>AD</td>
<td>COWAT</td>
<td>.20</td>
<td>.29</td>
<td>.78</td>
<td>.68</td>
<td>.24</td>
<td>.82</td>
<td>.52</td>
</tr>
<tr>
<td>AA</td>
<td>COWAT</td>
<td>.20</td>
<td>.14</td>
<td>.81</td>
<td>.68</td>
<td>.16</td>
<td>.80</td>
<td>.50</td>
</tr>
<tr>
<td>AD</td>
<td>Trails-B</td>
<td>.12</td>
<td>.15</td>
<td>.76</td>
<td>.68</td>
<td>.08</td>
<td>.87</td>
<td>.54</td>
</tr>
<tr>
<td>AA</td>
<td>Trails B</td>
<td>.12</td>
<td>.23</td>
<td>.83</td>
<td>.76</td>
<td>.16</td>
<td>.89</td>
<td>.46</td>
</tr>
<tr>
<td>AD</td>
<td>RCFT Org</td>
<td>.40</td>
<td>.44</td>
<td>.78</td>
<td>.64</td>
<td>.56</td>
<td>.68</td>
<td>.38</td>
</tr>
<tr>
<td>AA</td>
<td>RCFT Org</td>
<td>.40</td>
<td>.22</td>
<td>.76</td>
<td>.54</td>
<td>.37</td>
<td>.60</td>
<td>.59</td>
</tr>
</tbody>
</table>

Note: BR=Base Rate, Sens.=Sensitivity, Spec.=Specificity, HR=Hit Rate, PPP=Positive Predictive Power, NPP=Negative Predictive Power, AUC=Area Under the Curve.
APPENDIX A

Mood and Anxiety Questionnaire – Short Form

Below is a list of feelings, sensations, problems, and experiences that people sometimes have. Read each item and then mark the appropriate choice in the space next to that item. Use the choice that best describes how much you have felt or experienced things this way during the past week, including today.

<table>
<thead>
<tr>
<th></th>
<th>Not at all</th>
<th>A little bit</th>
<th>Moderately</th>
<th>Quite a bit</th>
<th>Extremely</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Felt sad</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>2. Startled easily</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>3. Felt cheerful</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>4. Felt afraid</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>5. Felt discouraged</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>6. Hands were shaky</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>7. Felt optimistic</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>8. Had diarrhea</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>9. Felt worthless</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>10. Felt really happy</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>11. Felt nervous</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>12. Felt depressed</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>13. Was short of breath</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>14. Felt uneasy</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>15. Was proud of myself</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>16. Had a lump in my throat</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>17. Felt faint</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>18. Felt unattractive</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>19. Had hot or cold spells</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>20. Had an upset stomach</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>21. Felt like a failure</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>22. Felt like I was having a lot of fun</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>23. Blamed myself for a lot of things</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>24. Hands were cold or sweaty</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>25. Felt withdrawn from other people</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Not at all</td>
<td>A little bit</td>
<td>Moderately</td>
<td>Quite a bit</td>
</tr>
<tr>
<td>---</td>
<td>---</td>
<td>------------</td>
<td>--------------</td>
<td>------------</td>
<td>-------------</td>
</tr>
<tr>
<td>26. Felt keyed up, &quot;on edge&quot;</td>
<td></td>
<td>1 2 3 4 5</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>27. Felt like I had a lot of energy</td>
<td></td>
<td>1 2 3 4 5</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>28. Was trembling or shaking</td>
<td></td>
<td>1 2 3 4 5</td>
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<td>29. Felt inferior to others</td>
<td></td>
<td>1 2 3 4 5</td>
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<tr>
<td>30. Had trouble swallowing</td>
<td></td>
<td>1 2 3 4 5</td>
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<td>31. Felt like crying</td>
<td></td>
<td>1 2 3 4 5</td>
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<td>32. Was unable to relax</td>
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<td>1 2 3 4 5</td>
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<td>33. Felt really slowed down</td>
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<td>1 2 3 4 5</td>
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<td>34. Was disappointed in myself</td>
<td></td>
<td>1 2 3 4 5</td>
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<td>35. Felt nauseous</td>
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<td>1 2 3 4 5</td>
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<td>36. Felt hopeless</td>
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<td>1 2 3 4 5</td>
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<td>37. Felt dizzy or lightheaded</td>
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<td>1 2 3 4 5</td>
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<td>38. Felt sluggish or tired</td>
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<td>1 2 3 4 5</td>
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<td>39. Felt really “up” or lively</td>
<td></td>
<td>1 2 3 4 5</td>
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<td>40. Had pain in my chest</td>
<td></td>
<td>1 2 3 4 5</td>
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<tr>
<td>41. Felt really good</td>
<td></td>
<td>1 2 3 4 5</td>
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<tr>
<td>42. Felt like I was choking</td>
<td></td>
<td>1 2 3 4 5</td>
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<tr>
<td>43. Looked forward to things with enjoyment</td>
<td></td>
<td>1 2 3 4 5</td>
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<tr>
<td>44. Muscles twitched or trembled</td>
<td></td>
<td>1 2 3 4 5</td>
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<tr>
<td>45. Felt pessimistic about the future</td>
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<td>1 2 3 4 5</td>
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<td>46. Had a very dry mouth</td>
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<td>1 2 3 4 5</td>
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<tr>
<td>47. Felt like I had a lot of interesting things to do</td>
<td></td>
<td>1 2 3 4 5</td>
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<tr>
<td>48. Was afraid I was going to die</td>
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<td>1 2 3 4 5</td>
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<tr>
<td>49. Felt like I had accomplished a lot</td>
<td></td>
<td>1 2 3 4 5</td>
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<tr>
<td>50. Felt like it took extra effort to get started</td>
<td></td>
<td>1 2 3 4 5</td>
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<tr>
<td>51. Felt like nothing was very enjoyable</td>
<td></td>
<td>1 2 3 4 5</td>
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<tr>
<td>52. Heart was racing or pounding</td>
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<td>1 2 3 4 5</td>
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<tr>
<td>53. Felt like I had a lot to look forward to</td>
<td></td>
<td>1 2 3 4 5</td>
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<td>54. Felt numbness or tingling in my body</td>
<td></td>
<td>1 2 3 4 5</td>
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<td>55. Felt tense or “high-strung”</td>
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<td>1 2 3 4 5</td>
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<tr>
<td>56. Felt hopeful about the future</td>
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<td>1 2 3 4 5</td>
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<tr>
<td>57. Felt like there wasn’t anything interesting or fun to do</td>
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<td>1 2 3 4 5</td>
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<td>58. Seemed to move quickly and easily</td>
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<td>1 2 3 4 5</td>
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<tr>
<td>59. Muscles were tense or sore</td>
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<td>1 2 3 4 5</td>
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<tr>
<td>60. Felt really good about myself</td>
<td></td>
<td>1 2 3 4 5</td>
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<td>61. Thought about death or suicide</td>
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<td>1 2 3 4 5</td>
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<td>62. Had to urinate frequently</td>
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<td>1 2 3 4 5</td>
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APPENDIX B

Health History Questionnaire

A. Demographic Information

1. Age ________ 2. Date of Birth ________

3. Race/Ethn.: Caucasian/European American □ Asian-American/Pacific Islander □
African-American □ Other (Specify): □
Hispanic/Latino □ __________________________
American Indian/Alaskan Native □

(please check all that apply)

4. Primary Language: English □
Other (Specify): □

5. Handedness: Right □
Left □
Mixed □

6. Height Feet ________ Inches ________

7. Weight Pounds______

8. Year in School (please encircle) Freshman Sophomore Junior Senior

B. Family History

Have your biological parents or grandparents ever had the following:

1. Attention Deficit Hyperactivity Disorder (ADHD) Yes □ No □
2. Learning Disorder (e.g. dyslexia) □ □
3. Alzheimer’s Disease □ □
4. Stroke □ □
5. Parkinson’s Disease □ □

6. Vascular Dementia □ □
7. Multiple Sclerosis □ □
8. Epilepsy or Seizures □ □
9. High Blood Pressure □ □
10. Heart Attack or Heart Surgery □ □
C. Medical History

Have you ever had the following:

1. Alcoholism or Drug Abuse ☐ Yes, currently ☐ Yes, past ☐ No
2. Anorexia Nervosa ☐ ☐ ☐
3. Anxiety problems ☐ ☐ ☐
4. Asthma ☐ ☐ ☐
5. Attention Deficit Hyperactivity Disorder (ADHD) ☐ ☐ ☐
6. Being held back in school/Failing a grade ☐ ☐ ☐
7. Binge-Eating ☐ ☐ ☐
8. Birth Defects ☐ ☐ ☐
9. Bulimia Nervosa ☐ ☐ ☐
10. Cancer or Leukemia ☐ ☐ ☐
11. Deafness or Hearing problems ☐ ☐ ☐
12. Depression ☐ ☐ ☐
13. Developmental Delays ☐ ☐ ☐
14. Diabetes ☐ ☐ ☐
15. Epilepsy or Seizures ☐ ☐ ☐
16. Head Injury w/ >10min loss of consciousness ☐ ☐ ☐
17. Heart Disease or Surgery ☐ ☐ ☐
18. Insomnia (problems falling or staying asleep) ☐ ☐ ☐
19. Kidney Disease ☐ ☐ ☐
20. Learning Disorder (e.g. dyslexia) ☐ ☐ ☐
21. Lyme Disease ☐ ☐ ☐
22. Manic Depression (Bipolar Disorder) ☐ ☐ ☐
23. Migraine or Problem Headaches ☐ ☐ ☐
24. Brain Diseases (e.g. Multiple Sclerosis) ☐ ☐ ☐
25. Pain problems ☐ ☐ ☐
26. Problems learning to talk or walk as a child ☐ ☐ ☐
27. Problems learning to read as a child ☐ ☐ ☐
28. Thyroid Disease ☐ ☐ ☐

D. Smoking History

1. Have you smoked at least 100 cigarettes in your entire life? ☐ Yes ☐ No
2. Have you smoked at least part of a cigarette in the past 7 days? ☐ Yes ☐ No
3. During a typical week, how many cigarettes do you smoke? _______