EXECUTIVE FUNCTION DEFICIT AND DISABILITY IN UNIPOLAR MAJOR DEPRESSION: THE TRANSIENT, THE STABLE, AND THE IMPORTANCE OF EMOTIONAL CONTROL

BY

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THESIS

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Abstract

Executive function impairments associated with unipolar depression contribute heavily to the individual and societal costs of this disorder. Unfortunately, past research on executive function deficits in unipolar depression has not succeeded in providing much detail about the nature of these deficits. Most researchers have used univariate methods to attempt to discern unique patterns of executive function deficits that characterize unipolar depression. To enhance specificity of prediction, the present study used Descriptive Discriminant Analysis, along with an ecologically valid measure of executive function, to reveal a pattern of executive function impairments specifically associated with unipolar depression, including impairments in emotional control, shifting, and planning and organizing. Though each of these deficits predicted current disability, only deficits in emotional control did so after accounting for current depressive and anxious symptoms. Regression analyses also revealed complex relationships between symptoms and executive function deficits in each of three clinical groups (currently depressed, previously depressed, and currently anxious), indicating that although some executive function deficits may resolve as symptoms abate, emotional control may be a more stable predictor of general affective psychopathology (e.g., both anxiety and depression).

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Introduction

Globally, unipolar depression is not only a highly prevalent but also a highly disabling, and therefore costly, condition. For the past 20 years, World Health Organization (WHO) estimates have indicated that depressive disorders are the leading cause of disability in the world (World Health Organization, 2002). Unipolar depression, specifically, is the fourth leading cause of the global burden of disease, surpassed only by perinatal complications, chronic respiratory infection, and HIV/AIDs in terms of its worldwide cost to individuals, governments, and society (Mathers, & Murray, 2004; Ustun, Aysuo-Mateos, Chatterji; World Health Organization, 2002).

In developed regions, where depression accounts for as much as 22% of years of life lost to disability, the costs are even higher (Murray & Lopez, 1997). In Europe, the financial burden of depression now equals approximately 1% of the entire European economy, with more than half of that estimate attributed to the indirect costs of lost productivity, morbidity, and mortality (Sobocki, Johnsson, Angst, & Rehnberg, 2006). In the United Sates, depression is estimated to cost US employers almost $51.5 billion per year (Greenberg, Kessler, Birbaum, Leong, Lowe, Berglund, & Corey-Lisle, 2003), due in large part to so-called "presenteeism," or lost productive time at work (Stewart, Ricci, Chee, Hahn, & Morganstein, 2003). Indeed, when compared to employees with other chronic medical conditions, depressed workers report significantly more difficulty sustaining concentration and focus, as well as more impaired work quality, efficiency, and speed (Wang, Beck, Berglund, McKenas, Pronk, Simon, & Kessler, 2004). Thus, while depression is frequently characterized as a disorder of emotion or mood, the cognitive impairments associated with depression may be the more functionally and economically significant features of the disorder. However, despite decades of research the

pattern of cognitive impairment in depression is far from understood and appears to be dependent upon a number of factors, including the frequency of depressive episodes, co-morbidty (especially co-morbid anxiety and co-morbid medical conditions), severity of current symptoms, depression sub-type, age, gender, and history of treatment (for reviews, see Hammar & Ardal, 2009; Levin et al., 2007).

Cognitive Impairment Versus Executive Function Impairment

Despite use of the term "cognitive impairment," in reality the workplace and epidemiological studies referenced above refer almost exclusively to deficits in executive functions (e.g., inhibiting distraction, task-completion, error-monitoring, shifting between tasks, etc.). Indeed, in the literature to date "cognitive impairment" has often been used as a blanket term that includes abilities and skills typically characterized as "executive functions" in neuropsychological and cognitive neuroscience disciplines. However, using cognitive impairment and executive function impairment interchangeably can obscure important differences between the two (see the definition of executive function, below). Therefore, in the present study cognitive impairment and executive function impairment were assumed to be distinct, though interrelated, constructs. Importantly, only executive functions were measured in the present study.

Cognitive and Executive Function Impairments in Unipolar Depression

Many researchers have posited that, like dementia and schizophrenia, unipolar depression is defined by profuse and often lasting impairments in a variety of cognitive ability domains (Austin et al., 1992). Indeed, the impairment may extend to executive functions as well, though, because they have been shown to be unrelated to the severity of current depressive symptoms, some have contended that these deficits are epiphenomenal (Porter, 2003). However, as Porter

conceded, it is also possible that executive function deficits are a stable marker of unipolar depression *because* they are unrelated to depression severity and are detectable during (and perhaps even after) depressive episodes in people of all ages, severities, and subtypes (Purcell, Maruff, Kyrios, & Pantelis, 1997; Tarbuck & Paykel, 1995) and even in those who are merely dysphoric (Channon, 1996). Thus, an alternative interpretation of the same results would contend that, because memory and learning impairments (e.g., cognitive impairments) fluctuate, they may be unrelated to unipolar depression specifically and instead represent epiphenomena that arise from combinations of illness severity and other individual characteristics (described forthwith). Indeed, though early research suggested that severe cognitive impairments characterized unipolar depression, subsequent investigations have largely indicated that those effects were due to the following: depression subtype (with psychotic depression demonstrating impairments on par with schizophrenia; Basso & Bornstein, 1999; Jest et al., 1993; Schatzberg, 2000); age (no major cognitive impairment in the young, Fossati, Amar, Raoux, Ergis, & Allilaire, 1999; Grant, Thase, & Sweeney, 2001; only elderly show impaired processing, Beats, Sahakian, & Levy, 1996; Tarbuck & Payel, 1995); medication use (SSRI's impair verbal learning; Schmitt et al., 2001); inpatient status/severity (Austin, 1999; Burt, Zembar, & Niederehe, 1995; Veiel, 1997); and co-morbid medical (Hickie & Scott, 1998; Kramer-Ginsberg et al., 1999) and psychological (Basso et al., 2007; Castaneda et al., 2008) conditions.

As with cognitive impairment, disagreement about the existence, nature, and course of executive function impairments in unipolar depression are numerous. A substantial body of research has demonstrated executive function deficits in updating, shifting, and cognitive inhibition within episodes of unipolar depression (Clark, Sarna, & Goodwin, 2005; Gohier et al., 2009; Harvey et al., 2004; Micco, et al., 2009; Ottowitz, Dougherty, & Savage, 2002), but there

is also evidence suggesting that executive function deficits in depression, like general cognitive impairment, are largely a product of age and its concomitant reduction of psychomotor speed (Lockwood, Alexopoulos, & van Gorp, 2002) . The absence of executive function deficits in depressed children (Favre et al., 2009) and in children of parents with mood and anxiety disorders (who thus have a heightened risk of developing these problems; Micco et al., 2009) casts further doubt on the universality of executive function deficits in the disorder.

Some authors assert that depression-related executive function deficits (particularly in shifting) remain after recovery and predict worse outcome (Paradiso, Lamberty, Garvey, & Robinson, 1997), while others disagree, citing naturalistic studies showing roughly normal executive function and processing speed in formerly depressed persons following successful treatment (Gualtieri, Johnson, & Benedict, 2006). Indeed, post-treatment recovery of executive functions has been shown to persist through a 2-year follow-up, even in patients with recurrent unipolar depression, and has been demonstrated to be significantly associated with reductions in depressive symptoms (Biringer et al., 2005).

Combining cognitive-vulnerability theories of depression with the above evidence suggesting "state"-dependent impairment, others have advanced the theory that executive function deficits in depression are, in fact, caused by suboptimal cognitive strategies, like rumination, that characterize the disorder. Indeed, several authors have experimentally demonstrated that rumination can elicit the executive function deficits frequently observed in depression. In a study of induced rumination and induced distraction, rumination was shown to decrease random number generation in depressed patients. Importantly, when rumination was prevented by distraction, depressed patients and controls were equivalent in their levels of both rumination and random number generation (Watkins & Brown, 2002). Similarly, in a study of attention

allocation, depressed patients were observed to perform poorly only in high-interference conditions, and this deficit was highly associated with rumination (Levens, Muhtadie, & Gotlib, 2009). This, too, suggests that the executive function deficits seen in depression may be dynamic and, at the very least, co-vary with depression-typical cognitive strategies. However, a follow up-study examining the effect of rumination on cognitive flexibility and inhibition in the Stroop task revealed a general deficit in flexibility that was more pronounced following rumination but still present across conditions in dysphoric (e.g., highly symptomatic, but not necessarily meeting full criteria for a current major depressive episode) young adults (Philippot & Brutoux, 2008).

In sum, after reviewing the literature one can confidently conclude only that some depressed individuals exhibit some executive function deficits at least some of the time, which, given the enormity of the cost these deficits engender through disability, is hardly reassuring. Interestingly, an alternative approach, advanced by Ravnkilde et al. (2002), is to accept this ambiguity at face value and concede that the lack of any consistent pattern of executive function deficits within unipolar depression indicates the need for sophisticated subgroup analysis. The expediency of this suggestion notwithstanding, it unfortunately fails to explain why no consistent patterns of executive function deficits have emerged from the study of even well-defined subgroups. However, although some investigators have used very sophisticated designs and group selection procedures, their approaches have been limited by one or more of the following: ignoring the effects of anxiety, ignoring severity of current symptoms, ignoring other neuropsychological findings, forgoing more ecologically valid measures of executive function for laboratory tests, and using a non-multivariate approach to compare individual executive functions across groups. Each of these issues will be discussed below.

Anxiety Disorders

Although the purpose of the present study was to examine executive function deficits in unipolar major depression, depression and anxiety disorders are highly co-morbid (Kessler et al., 2003), and both are characterized by high levels of negative affect. Thus, isolating the "pure" effects of one disorder inherently requires isolating and measuring the effects of the other (Clark & Watson, 1991; Mineka, Watson, & Clark, 1998). Unfortunately, executive function deficits in anxiety disorders have been relatively less well studied. Comparing adults with any anxiety disorder to those without, Airaksinen and colleagues found evidence for impairment in shifting but not verbal fluency or processing speed, primarily in those with Obsessive-Compulsive Disorder and Panic Disorder (Airaksinen, Larsson, & Forsell, 2005). Additionally, individuals with a sole diagnosis of General Anxiety Disorder or Specific Phobia showed no appreciable executive function deficits. However, co-morbid depression and alcohol disorders within that sample render the results somewhat difficult to interpret, though others have replicated these negative findings (Asmundson, Stein, Larsen, & Walker, 1994; Gladsjo et al., 1998). Other authors have posited that the executive function deficits and psychomotor slowing frequently observed in depression are actually due to the presence of an anxiety disorder, whereas pure depressed individuals show only memory impairment (Basso et al., 2007). In sum, these data indicate that concurrent measurement of anxiety is essential to identifying a pattern of executive function deficits that uniquely characterizes unipolar depression. Additionally, no study to date has compared depressed, previously depressed, and purely anxious groups' executive functions in a manner that accounts for the high co-morbidity between anxiety and depression. Thus, the present study represents a significant improvement over previous designs in terms of its ability to disentangle a pattern of executive function deficits specific to (pure) unipolar major depression.

Severity and Current Symptoms

As noted above, illness severity has frequently been associated with greater impairment (Austin, 1999; Burt, Zembar, & Niederehe, 1995; Veiel, 1997). Thus, in the present study measures of anxious and depressive symptoms were used to assess the effect of symptom severity on executive functions.

Other Neuropsychological Findings

A large body of evidence from lesion and stroke patients has demonstrated that the right parietal area of the brain is necessary not only for the recognition of emotional stimuli (Adolphs, Damasio, Tranel, & Damasio, 1996; Borod, Koff, Lorch, & Nichols, 1986) but also for the regulation of arousal in response to emotional stimuli (Schrandt, Tranel, & Damasio, 1989). Synthesizing this information with models of frontal brain regions' involvement in emotional experience, Heller (1993) proposed a model of emotional function whereby the frontal areas of the brain are involved in modulating emotional valence and the right posterior brain is involved in processing emotional information and mediating autonomic arousal. Predictions regarding activity and under-activity of the right parietal regions in various forms of psychopathology directly follow from this model and have received considerable support.

Although not perfectly correlated with brain activity (Green, Morris, Epstein, West, & Engler, 1992; Kim & Levine, 1991), perceptual asymmetries, as measured by the strength of preferred or improved performance when stimuli are presented to one side of the body (one ear, one half of the visual field, etc.), have frequently been used to approximate underlying hemispheric brain activity. Though they may not perfectly mirror brain activity, perceptual asymmetries are nevertheless highly relevant, as right-ear/left-hemisphere biases in dichotic listening tasks predict response to both cognitive behavior therapy and antidepressant medication but do not change following either intervention (Bruder, 1996, 1997).

The Chimeric Faces Task (CFT; Heller & Levy, 1981) is a well-established method of eliciting and measuring perceptual asymmetries related to emotional processing. Substantial testing has indicated that healthy, right-handed people perceive emotional facial expressions presented to the left hemi-space (e.g., the right hemisphere of the brain) as more salient on the CFT (Levy, Heller, Banich, Burton, 1983). Consistent with Jaeger et al (1987), Heller and colleagues demonstrated that reduced left-hemispatial bias on the CFT is associated with depression. Further, as Heller's model would predict, anxiety (specifically anxious arousal) has been shown to be associated with an opposite pattern of right posterior activity and is related to an increase in left-hemispatial bias on the CFT (Heller, Etienne, & Miller, 1995; Keller et al., 2000).

Remarkably, the strength of left-hemispatial bias on the CFT can also be used to predict the development of depressive and anxious symptoms. In a longitudinal study of undergraduate women Voelz et al. (2001) found that increased left-hemispatial bias on the CFT predicted anxiety symptoms (specifically, anxious arousal symptoms) 6 weeks later, while decreased left-hemispatial bias predicted decreased positive affectivity (e.g., increased anhedonic depression) during the same interval. A recent longitudinal study of youth revealed that not only does reduced left-hemispatial bias predict later depressive symptoms, this link is mediated by maladaptive responses to interpersonal stress and is unique to the onset of depressive symptoms (as opposed to anxiety or misbehavior) (Flynn & Rudolph, 2007; Flynn & Rudolph, 2010).

What neuropsychological measures like the CFT have suggested, psychophysiological measures have confirmed, both across imaging modalities and within multiple populations. For example, in an EEG study of war veterans with Post-Traumatic Stress Disorder (PTSD), anxious arousal symptoms were associated with increased right parietal activation, just as Heller et al.

(1993) and Keller et al. (2000) would predict, and the combination of anxious arousal and depressive symptoms accounted for nearly 25% of variance in brain activity over the right parietal region (Metzger et al., 2004). An MEG study of depressed, non-anxious patients and non-depressed controls found that, although both groups demonstrated increased activity in the dorsal visual stream in response to neutral and emotional photos, only controls showed increased activity in the right tempo-parietal area in response to the emotional pictures (Moratti, Rubio, Campo, Keil, & Ortiz, 2008). This supports the assertion that depression is associated with hypo-activation of the right parietal region, and that this, in turn, may be related to the lack of arousal (low-positive affect/high anhedonia) that is characteristic of depression. Indeed, the association between depression and reduced right posterior hemisphere activity has been well established in both depressed and subclinical samples (Henriques & Davidson, 1997; Rabe, Debener, Brocke, & Beauducel, 2005) as well as formerly depressed patients (Henriques & Davidson, 1990) and even the children and grandchildren of depressed patients (Bruder, Tenke, Warner, & Weissman, 2007). Moreover, individual differences in perceptual asymmetries on the CFT have been shown to be remarkably stable (Levy et al., 1983) and to exist independently of current mood state (Compton, Fisher, Koenig, McKeown, & Munoz, 2003).

The robust finding of reduced right parietal activity in depression may be associated with impaired executive functioning in several ways. First, as an indication of an impaired ability to perform visuospatial tasks (Asthana, Mandal, Khurana, & Haque-Nizamie, 1998; Elliott et al., 1996), it may be associated with specific deficits in aspects of planning and organizing. Secondly, as a vulnerability marker reflecting a reduced capacity to process emotional information, it may be associated with specific deficits in emotional control. Lastly, as Flynn and Rudolph have shown, as a vulnerability marker interacting with interpersonal stress, it may result

in increased negative affect/anhedonic depression symptoms (Bruder, 1995) which in turn has been shown to be uniquely associated with deficits in shifting and selective attention (Austin et al., 1999).

Measuring Executive Function

Though executive function as a construct is far from perfect (for an excellent review of this problem, see Dick & Overton, 2010) and various definitions of executive function exist, executive functions are typically defined as a set of higher-order cognitive abilities that direct more molecular capacities like attention and memory to orchestrate complex, goal-oriented behaviors (Stuss & Levine, 2002; Welsh & Pennington, 1988). Executive functions are necessary and crucial, then, when circumstances require that these lower-order capacities be deployed in a non-routine or non-habitual manner. Following from this, conceptualizations of executive function typically include inhibition, shifting, planning, cognitive and behavioral fluency, and working memory (Pennington & Ozonoff, 1996).

Importantly, though the studies cited thus far refer almost exclusively to relationships between depression and executive function deficits as measured by formal neuropsychological tests, it is not uncommon even for individuals with known brain injuries and physical head trauma to perform normally on formal tests but nonetheless manifest significant executive function impairment in real-world situations (Stuss & Buckle, 1992). In a study of severely depressed inpatients awaiting electroconvulsive therapy, self ratings of cognitive disability were not particularly strongly associated with neuropsychological tests of cognitive ability (except for memory) but were better at predicting ratings of well-being and physical disability than formal test measurements (Naismith, Longley, Scott, & Hickie, 2007). Thus, the literature assessing executive function deficits in unipolar depression may underestimate the impairment associated

with the disorder because ecologically valid measures of real-life impairment are infrequently, if ever, used. Indeed, the only known study to use an ecologically valid measure demonstrated that global cognitive impairment (not age nor illness severity) was associated with problems in instrumental activities of daily living (handling finances, employment, etc.; McCall & Dunn, 2003). The present study, therefore, employed a measure of executive functions that was designed to be ecologically valid and measure everyday real-world deficits (Gioia & Isquith, 2004).

A Multivariate Approach

Although most researchers have indicated that identification of a pattern of deficits is their primary objective, hardly any have used statistical techniques well-suited for this purpose, especially given the high inter-correlation among individual executive functions. When multivariate approaches have been used, rather than interpret the omnibus findings using follow-up techniques that preserve a multivariate approach (for example, through descriptive discriminant analysis), many researchers have settled for multiple univariate tests, an approach that has been widely criticized by statisticians on both conceptual (why do the multivariate test in the first place?) and technical grounds (because of increased likelihood of type-I error) (Enders, 2003; Grice & Iwasaki, 2007; Huberty & Morris, 1989). Thus, the present study adopted a systematically multivariate approach, using descriptive discriminant analysis (DDA) to discern a pattern of deficits that optimally differentiated unipolar depression from anxiety, remitted depression, and individuals without a history of mental disorder. Lastly, the results of the DDA were used to guide regression analyses to determine whether deficits in executive function predicted current disability, as measured by a rating system frequently used by insurance companies to decide disability claims.

Method

Participants

The present study utilized two sources of participants in a retrospective, cross-sectional design. Both sets of participants were recruited over several years for a separate neuroimaging study. The recruiting process for each source differed slightly, as described below.

Psychology students from a large Midwestern university were recruited from a larger pool (N = 2, 637) of students who completed questionnaires as partial fulfillment of a class research requirement. In service of the fMRI study mentioned above, students were selected based upon their profile of scores on a trio of measures assessing worry, anxious arousal, and anhedonic depression, respectively. Following established guidelines (Nitschke, Heller, Imig, McDonald, & Miller, 2001), the 80th percentile was used to demarcate elevated scores and the 50th percentile was used to demarcate normal-range scores on the aforementioned measures of symptoms. Thus, students were recruited and subsequently utilized in the present study if they met criteria for one of the following profiles: elevated on all three measures, elevated on one of the measures and normal on the two other measures, or normal on all three measures. This process resulted in 103 student participants (62 female, 41 male), aged 18 to 22 (M = 19.02, SD = 1.04). Importantly, the specific profile of scores associated with any particular participant was concealed from investigators to prevent bias. The questionnaire data used to recruit participants were not analyzed in the present study. To ensure stability of symptoms and to standardize when participants' symptoms were assessed, student participants completed symptom measures a second time, and only these data were used in the present analyses.

To enhance variability in age, education, symptom severity, and co-morbidity, a second source of participants was included in the present study. A total of 149 participants (95 female,

54 male), aged 19 to 51 (M = 34.62, SD = 9.23), were recruited from the community through electronic and newspaper advertisements, word of mouth, and a local mental health center. These participants were also recruited for a separate neuroimaging study. Although community participants, unlike students, were recruited without advance knowledge of their current pattern of symptoms, the number of individuals meeting provisional or full criteria for one or more diagnoses was roughly equivalent in each sample (104 of 149 and 44 of 103 in the community and student groups, respectively). Participants were screened for left-handedness, impaired vision or hearing, pregnancy, history of serious head injury or prolonged loss of consciousness, past receipt of electroconvulsive therapy, metal embedded in the body (due to restrictions in the neuroimaging study), recent substance use, and English as a second language. Participants were financially compensated for their participation. The two samples were combined for further analysis, except where noted below.

Self-Report Measures of Symptoms

As current level of symptoms and illness severity may affect executive functions (Austin, 1999; Burt, Zembar, & Niederehe, 1995; Veiel, 1997), both depressive and anxious symptoms were assessed in the present study via three scales on two self-report measures.

Worry. Worry, as a measure of anxious apprehension in Heller's (1993) model, has been shown to affect brain activity in areas of the frontal lobes known to be involved in implementing executive functions (Engels et al., 2007, 2010). Therefore, the Penn State Worry Questionnaire (PSWQ; Meyer, Miller, Metzger, & Borkovec, 1990; Molina & Borkovec, 1994) was used to assess anxious apprehension in the present study. This 16-item questionnaire asked participants to rate how well a variety of statements characterized them, such as "my worries overwhelm me," from 1 (not at all) to 5 (very typical). The PSWQ had a very high reliability in this sample,

Cronbach's α = .96.

Anxious arousal. The Mood and Anxiety Symptom Questionnaire (MASQ; Watson & Clark, et al., 1995; Watson & Weber, et al., 1995) is a 77-item self-report measure designed around the Tripartite Model of Affect and, as such, assesses general distress as well as anxiety-specific (Anxious Arousal; MASQ-AA) and depression-specific (Anhedonic Depression; MASQ-AD) symptoms. Because the intent of the present study was to differentiate disorders, the General Distress Subscale, as a measure of what is largely shared by anxiety and depression, was not used.

Because increased right-hemisphere asymmetry has primarily been associated with the anxious arousal dimension of anxiety (Heller & Nitschke, 1998; Metzger et al., 2004), the MASQ-AA was used to obtain a measure of anxious arousal. The MASQ-AA scale asks participants to indicate how much they have experienced a symptom (such as "startled easily" and "was trembling or shaking") during the previous week, from 1 (not all) to 5 (extremely). Higher scores reflect more symptoms. The Anxious Arousal subscale of the MASQ had high reliability in this sample, Cronbach's α = .81.

Anhedonic depression. Because melancholic depression (which is characterized by loss of pleasure and diminished response to rewarding stimuli; American Psychiatric Association, 2000) has been shown to be uniquely associated with deficits in shifting and selective attention (Austin et al., 1999), the Anhedonic Depression (AD) scale of the MASQ was selected to measure current depressive symptoms. The MASQ-AD scale uses the same rating system as the MASQ-AA scale but asks participants to consider symptoms such as "felt withdrawn from other people" and "felt like nothing was very enjoyable." Previous research (Nitschke et al., 2001) has shown that the AD scale can be further reduced into subcomponents that roughly correspond to

Loss of Interest (LI) and High Positive Affect (HP) as identified by Watson, Clark, et al. (1995). Recent research has suggested that, compared to the full AD scale or the reverse-scored HP scale the 8-item LI scale better distinguishes current depression from past-depression and no depression in non-patients (Bredemeier et al., in press). Thus, only the 8-item LI scale (MASQ-AD8) was used as a measure of anhedonic depressive symptoms in the present study. The 8 item Anhedonic Depression subscale of the MASQ had a high reliability in this sample, Cronbach's α = .82.

Neuropsychological Measures

Executive function. The Behavior Rating Inventory of Executive Function- Self-Report Version (BRIEF-SR; Guy, Isquith, & Gioia, 1996) was used to obtain measures of current executive function. As discussed above, the BRIEF-SR was designed to capture everyday problems with executive functions that formal neuropsychological testing, with its necessarily highly structured assessments, may miss (Gioia & Isquith, 2004). Participants were asked to indicate how frequently (Never, Sometimes, or Often) they had had problems with a list of behaviors (such as "having a short attention span") in the last 6 months. The BRIEF-SR is composed of 80 items that can be reduced to 8 non-overlapping scales which are described in Table 1.

The Inhibit, Shift, Emotional Control, and Monitor scales are combined to produce a Behavior Regulation Index (BRI), which is an overall measure of executive functioning as it pertains to regulating everyday behavior. Similarly, the Working Memory, Plan/Organize, Organization of Materials, and Task Completion scales are combined to produce a Metacognition Index (MCI), which is an overall measure of problem-solving ability via planning and organizational skills. Finally, the BRI and MCI can be combined to produce a Global

Executive Composite (GEC). Higher scores indicate greater deficits in executive functions. The BRIEF-SR has been shown to have high (.96 for the GEC) to moderate (.72 for the scales with fewer items) internal consistency and correlates moderately (.56 for the GEC) with well-validated informant versions on which it was based (Gioia, Isquith, Guy, & Kenworthy, 2000). In this sample, the reliabilities for the BRI, MCI, and GEC were all very high, with Chronbach's α's equal to .93, .94, and .96, respectively. On the individual subscales the reliability varied from a low of .76 for the Organization of Materials subscale to a high of .91 for the Emotional Control subscale. As expected, the subscales with the lowest reliability (Organization of Materials and Monitor) were also the subscales with the fewest items (with only 7 and 5 items, respectively). Only the Organization of Materials and Monitor subscales had Chronbach α's lower than the recommended .08, and neither had a Chronbach α lower than .76.

Importantly, the BRIEF-SR also contains scales to assess how inconsistently and negatively participants rate themselves. Per the recommendation of the manual, participants with inconsistency scales greater than or equal to 7 were excluded from further analysis. Thus, all of the 149 community participants and all of the 104 student participants had inconsistency scores less than or equal to 6. Lastly, although an adult version of the BRIEF-SR now exists, at the time of data collection only an adolescent version was available. Careful inspection of the items on the adult and adolescent measures revealed that the differences between the two are slight and likely insignificant. For example, in the adult version Task Completion is called Task Monitor, and, as with the child version of the BRIEF, an Initiate scale was added. The versions are approximately the same length (80 versus 75 questions), and both yield BRI, MCI, and GEC composite subscales.

Perceptual/hemispheric asymmetry. The Chimeric Faces Task (CFT; Levy, Heller,

Banich, & Burton, 1983) was used to assess asymmetric hemispheric activity. The CFT is a free-vision task comprised of a 36-page booklet. Each page displays two chimeric (split) faces with one half of the face portraying a smile and the other half of the face portraying a neutral expression. The chimeric faces are arranged vertically, the two chimeras on a single page are of the same person, and each face is a mirror image of the other. Participants were asked to decide which face looked happier, the top or the bottom. Responses indicating a preference for the smile being in the left visuospatial field (e.g., indicating right-hemisphere activity) were scored as -1, and responses indicating a preference for the smile being in the right visuospatial field were scored as 1. A mean perceptual asymmetry score was calculated by adding the individual scores for each pair of chimeric faces and dividing by 36. Importantly, higher (e.g., more positive) scores on the measure reflect reduced right-hemisphere bias. The mean and standard deviation for the entire sample (N = 252) was M = -.46 (SD = .44), which is comparable to those found by Levy et al. (1983; M = -.30, SD = .44) and Heller et al. (1995; M = -.38, SD = .50). The CFT had a very high reliability in this sample, Cronbach's α = .92.

Diagnostic Interview

Participants were assessed for Axis I psychopathology using the Structured Clinical Interview for DSM-IV Disorders, Non-patient Edition (SCID-IV-NP; First, Spitzer, Gibbon, & Williams, 2002). Doctoral-student clinicians with at least a year of inpatient SCID experience conducted the interviews and arrived at diagnoses for each participant after consultation with other interviewers and a licensed clinical psychologist with more than twenty years experience. Interviews were audio-recorded to ensure fidelity.

Information gathered during the interview is summarized using a four-point scale for each diagnosis. A rating of four indicates that full clinical criteria for that diagnosis have been

met; three indicates a provisional diagnosis or the absence of one or two needed criteria to meet clinical thresholds; two indicates the presence of only a few symptoms; and one indicates the absence or near absence of symptoms for that diagnosis. For the purposes of classification in this study, participants were considered to have sufficiently met criteria for a disorder if they were given a rating of a three (provisional) or a four (certain). Diagnosis was then used to select participants for inclusion in study groups and to exclude participants from further analysis. Using these standards, data from participants who met criteria for the following DSM-IV diagnoses were excluded: past mania, current mania, hypomania, Bipolar Not-Otherwise-Specified, Bipolar-I, Bipolar-II, Cyclothymia, Mood Disorder Caused by a General Medical Condition, Substance Induced Mood Disorder, Schizophrenia, Schizoaffective Disorder, Obsessive-Compulsive Disorder, Anxiety Disorder due to a General Medical Condition, Substance-Induced Anxiety Disorder, Somatization Disorder, Conversion Disorder, Pain Disorder, Hypochondriasis, Body Dysmorphic Disorder, Anorexia Nervosa, Bulimia Nervosa, Eating Disorder Not-Otherwise-Specified, Substance Abuse, Substance Dependence, and Lifetime Substance Abuse. Though these exclusions may have weakened the generalizability of the data (e.g., many of the excluded disorders are frequently co-morbid with anxiety and depression), the selection criteria were designed to limit confounding influences on executive function impairment as well as to create more homogeneity within diagnostic groups to better isolate the constructs of interest (the executive function impairments associated specifically with unipolar depression). The exclusion of Obsessive-Compulsive Disorder (OCD) - the only major anxiety disorder to be excluded - was motivated by correlational and factor-analytic work on diagnostic taxonomy suggesting that OCD is not optimally characterized by common factors extracted from anxiety and mood disorders (Watson, 2005), as well as neurological evidence suggesting that OCD is on a

continuum of movement and tic disorders that are conceptually distinct from anxiety (Rapoport, 1990). In principle, OCD could have been treated as its own diagnostic category and analyzed accordingly. However, too few participants met criteria for this disorder (N = 7, 2 of whom had current problematic alcohol issues) to make such an analysis feasible. Therefore, OCD was excluded from further consideration.

Due to a cohort effect of heavier drinking among young adults (22% of all participants aged 18 to 29 and 16.5% of students met criteria for Alcohol Abuse or Dependence), alcohol use was assessed in more detail before decisions about exclusion were made. After completing the self-report measures, participants completed a short questionnaire concerning their current alcohol-use habits. Composed of four items from the Alcohol Use Disorders Identification Test (AUDIT; Saunders & Aasland, 1987; Saunders, Aasland, Babor, De La Fuerte, & Grant, 1993), this questionnaire asked participants to indicate how frequently (seldom, once a month, twice a month, weekly, or daily) they drank more than 5 drinks in a 24-hour period, drank more than 4 drinks in a 24-hour period, missed class (or work) due to drinking, or experienced periods of memory loss or blackout after drinking ("forgotten where I was or what I did"). To ensure current alcohol use was at a level that would not reasonably be expected to affect current cognitive functioning, participants were excluded from further analysis if they indicated that they drank 5 or more drinks in a 24-hour period more than twice a month, drank 4 or more drinks in a 24-hour period more than once a week, more than seldom missed class (or work) due to drinking, or more than seldom experienced memory loss after drinking. Twenty-five participants did not complete the assessment for current alcohol use. Instead, the narrative summary of their SCID-IV-NP interview was reviewed to determine their current alcohol-use habits. Using the above standards, 7 of the 149 community participants and 35 of the 103 students met criteria for current

problematic alcohol use; their data were excluded from subsequent analysis. Notably, though the intent of the alcohol-use measure was to reduce unnecessary data loss due to oversensitivity of the SCID-IV-NP alcohol abuse criteria in younger participants, the net result slightly increased loss in the student sample, as 18 students who did not meet diagnostic criteria for an alcohol disorder did meet criteria for current problematic alcohol use. However, the classification system did succeed in reducing the amount of data lost in the community group, saving 53 participants from unnecessary removal.

Initially, substance use was similarly re-assessed to reduce data loss. However, a review of the SCID-IV-NP summaries for participants meeting criteria for a substance disorder revealed that, although most participants were not currently abusing substances, many participants had been poly-substance users for variable periods of time, including within months of participation. Therefore, in order to prevent the introduction of systematic error in cognitive ability, no additional measures were employed to reduce data loss from substance abusing and substance dependent participants: any participant who met criteria for substance abuse or dependence (even lifetime abuse or dependence) were excluded from further analysis.

Use of the SCID-IV-NP also provided a measure of general disability via the Global Assessment of Functioning (GAF), a 100-point rating scale designed to capture overall illness severity and general functional impairment. A rating of 100 on the GAF indicates superior functioning across life-domains (work, social, family, etc.); a rating of 50 indicates severe symptoms such as suicidal ideations or chronic unemployment; and a rating of 10 indicates persistent danger of hurting self or others, inability to maintain minimal personal hygiene, or a serious suicidal act with a clear expectation of death (American Psychiatric Association, 2000). Although the GAF has questionable reliability and validity (Soderberg, Tungstrom, & Armelius,

2005), insurance companies heavily weight GAF ratings in decisions concerning insurance eligibility and disability status (Bilsker, Wiseman, & Gilbert, 2006). Thus, for the purposes of the present study it served as an ecologically relevant measure of disability.

Based on the SCID, remaining participants were classified into four groups defined by the following: past depression (N = 41; met criteria for at least one past depressive episode and no current depression with or without comorbid anxiety); current depression (N = 9; met criteria for a major depressive episode at the time of the study with or without comorbid anxiety); anxiety (N = 30; met criteria for an anxiety disorder other than OCD and did not meet criteria for any depressive disorder); and control (62; did not meet criteria for any diagnosis, past nor present). Ideally, the depression groups could have been further subdivided based on the presence or absence of co-morbid anxiety; however, only 5 participants met criteria for a current major depressive episode and only 4 met criteria for a current major depressive episode combined with comorbid anxiety. Therefore, to maximize power and to preserve the ability to perform meaningful analyses, the groups were collapsed as described above (e.g., co-morbid anxiety was mixed with "pure" depression in both the current and past depressed groups). Importantly, the two depression groups contained approximately equal proportions of individuals who additionally met criteria for a comorbid anxiety disorder (4 of 9 in the currently depressed group, and 20 of 41 in the previously depressed group, respectively). DDA is designed to downplay group similarities (Betz, 1987). Thus, because all three psychopathology groups contain anxiety, and the two depression groups contain it in approximately equal proportions, the discriminant function(s) derived from comparison of the four diagnostic groups should largely reflect the influence of the presence or absence of depression. In other words, because it is unlikely that the pattern of deficits caused exclusively by anxiety will optimally discriminate

between three groups that contain anxiety, the pattern associated with depression, which *does* discriminate between *all* groups, can still be expected to emerge from the DDA despite co-morbidity within the depression groups.

Procedure

Participants completed questionnaire booklets containing the CFT, the MASQ-AA, the MASQ-AD, the BRIEF, and other measures not analyzed in the present study while alone in a laboratory room following completion of a general orientation to the previously mentioned neuroimaging study. The PSWQ, along with other measures, formed part of a questionnaire packet that participants could take home to finish if they did not complete all of the questionnaires in the time allotted (approximately an hour). The diagnostic interviews were conducted in a private office two to four weeks after completion of the written questionnaires and the CFT.

Results

Data Analysis

The data were examined for outliers. Participants with scores greater than 2.5 standard deviations (SD) from the group mean on any measure were excluded from subsequent analysis. The dependent measures were tested, by group, for non-normality using the Shapiro-Wilks test. Additionally, skewness and kurtosis values were converted to z-scores and evaluated for significance. Measures with distributions characterized by significant skewness or kurtosis and significant non-normality as indicated by the Shapiro-Wilks test were excluded from further analysis. Thus, the Task Completion scale on the BRIEF was not analyzed singly. However, the Task Completion scale is a component of the Meta Cognition Index (MCI), and the distribution of the MCI was approximately normal for each diagnostic group. Therefore, important differences in Task Completion, if they exist, were captured indirectly in other analyses. All analyses were performed using SPSS, Version 17.0.

First, diagnostic groups (including the diagnosis-free group) were compared for equivalence in age and years of education using univariate analysis of variance (ANOVA). Then, zero-order correlations were computed to examine the general pattern of association between current symptoms, disability, and the neuropsychological measures. Although a Bonferroni correction was not applied to avoid being overly conservative, significance was determined at $p < .01$ to reduce the risk of Type I error (Perneger, 1998). Significant correlations were then used to guide selection of independent variables for DDA, which was used to determine whether a specific pattern of executive function deficits characterized each diagnostic group. Lastly, the results of the DDA were used to guide regression analyses to determine whether the specific pattern(s) of executive function deficits detected could be used to predict current functional

disability as measured by Global Assessment of Functioning (GAF) scores.

Group Characteristics

Data on the highest level of education achieved was available for 93 participants (25 past depressed, 8 currently depressed, 19 with anxiety, and 41 controls). To estimate whether level of education differed significantly across diagnostic groups, a one-way analysis of variance (ANOVA) was computed using data from this subset of 93. Levene's statistic was non-significant, indicating homogeneity of variances across the subset. The groups were found to differ in their level of education, $F(3, 89) = 3.11$, $p = .03$. However, the Hochberg GT2 test (selected because of the large differences between sample sizes across groups; Hochberg, 1974) did not reveal any significant differences between the groups, although the difference between the past depression and the control groups approached significance, $\Delta M = 1.75$, $p = .07$, ns. Notably, the mean number of years of education attained for the past depression group was 16.7 ($SD = 3.5$), while the mean for the current depression group was 14.1 ($SD = 1.1$). This equals the difference between being finished and being half-way through college. Since almost half the sample was composed of college students, this difference, while statistically significant, is in practical terms likely unimportant.

To determine whether the groups differed in their level of current disability, a one-way ANOVA was computed using GAF scores as the dependent measure. Levene's statistic was found to be significant. Thus, in place of the typical F statistic, the Welch and Brown-Forsythe statistics were computed. Both tests indicated that GAF scores were unequal across groups, with $F(3, 30.55) = 27.75$, $p < .01$ and $F(3, 53.68) = 21.57$, $p < .01$, respectively. The Games-Howell post hoc test revealed the following: GAF scores were lower in the current depressed group than in all other groups, and GAF scores in the past depressed and anxiety groups were lower than in

the control group but not significantly different from each other (all *p*s < .05).

Table 2 presents demographic characteristics for the 4 study groups and for the entire sample. Not unexpectedly, women were over-represented in the diagnostic groups, but roughly equally so. A one-way ANOVA was performed to assess whether the groups differed in age. Levene's statistic was non-significant, indicating homogeneity of variances across groups. Results indicated that no group was older or younger than another, F (3, 138) = 2.27, $p = .08$, ns.

Table 3 presents the means and standard deviations for all measures used in the analyses. Visual inspection of the means suggested a trend of heightened impairment across executive function domains in the current depression group, followed by similar levels of impairment in the past depression group and anxiety group. It appeared, as well, that the past depression group was different than (e.g., more impaired than) the control group. As expected, the current depression group had the most positive score on the CFT, whereas the anxiety group had the most negative. However, a one-way ANOVA revealed that these differences were non-significant, F (3, 138) = 1.75, $p = .16$, ns, though when considered as a one-tailed test, the trend approached significance ($p = .08$).

Correlations

Tables 4 and 5 present the correlations among the variables for each group, with Table 4 displaying the correlations within the past and currently depressed groups, and Table 5 displaying the correlations within the anxiety and control groups. Table 6 presents the correlations among the variables within the sample as a whole. As described above, to adjust for the number of comparisons performed, significance was determined at $p < .01$. The BRIEF subscales were moderately correlated with each other across groups. Thus, they were well-suited for DDA, which works best with moderately correlated independent variables (Sherry, 2006).

Surprisingly, the CFT was uncorrelated with any other measure within any of the groups. Therefore, based on the recommendations of Finch (2010), it was not entered into the DDA. Similarly, GAF was significantly associated with a measure of executive functioning only in the Anxiety group and the full sample, the Monitor subscale was associated with a symptom measure only in the full sample, and the Inhibit and Organization of Materials subscales of the BRIEF were not found to be associated with any symptom measures in any group. Therefore, the GAF, Monitor, Inhibit, and Organization of Materials variables were also withheld from the DDA. Very few measures were significantly associated with any other variables in the current depression group. This was likely due to the very small sample size of this group. Not unexpectedly, across groups symptoms were significantly associated with the various subscales of the BRIEF (except the few mentioned above). Thus, the symptom measures were also included in the DDA below.

Descriptive Discriminant Analysis (DDA)

As described above, executive functions have been conceptualized as a set of correlated skills. Because univariate analyses are, in effect, blind to correlations between dependent measures, executive functions are therefore best suited for analysis in a multivariate context (Grice & Iwasaki, 2007). However, unlike the multivariate technique of Multivariate Analysis of Variance (MANOVA), DDA can be used to determine not only whether groups differ on a selection of correlated variables but how they differ (Sherry, 2006). Thus, a DDA was performed to examine the differences between diagnostic groups on the selected BRIEF subscales (Huberty, 1984). As previous research and the above correlation analyses have indicated, current symptoms influence executive functions in significant ways. Therefore, the three symptom measures (the PSWQ, the MASQ-AA, and the MASQ-AD8) and the Shift, Emotional Control, Working

Memory, and Plan/Organization subscales of the BRIEF were entered as independent variables in the DDA.

Box's M test was non-significant using an alpha level of .001, (p = .003), indicating that the assumption of homogeneity of variances between groups was not violated. Table 7 displays the canonical correlations, tests of significance, and effect sizes for the three discriminant functions produced in the DDA. Examining the canonical discriminant functions, there was a modest canonical correlation (.544) for Function 1, a modest canonical correlation (.422) for Function 2, and a small canonical correlation (.276) for Function 3. The tests of the full model (Functions 1-3) and a reduced model (Functions 2-3) were statistically significant at $p < .01$. Function 3, tested on its own, approached significance (p = .056). Therefore, it was also interpreted with Functions 1 and 2, below.

Table 8 displays the standardized discriminant function coefficients and structure coefficients for all analyses. In DDA, function coefficients are analogous to beta weights in regression, and structure coefficients represent the correlation between the latent discriminant function variable and the observed variables. Although both types of coefficients can be used to determine the relative importance of each variable in a given discriminant function, because function coefficients can be highly affected by multicollinearity (Courville & Thompson, 2001), structure coefficients are frequently given more weight in this determination. Using the cutoff value recommended by Tabachnick and Fidell (2001) and demonstrated to provide optimal power by Finch (2010), structure coefficients greater than 0.3 were considered significant contributors to a given function and, therefore, worthy of interpretation. As shown in Table 8, the Anehdonic Depression scale, the Anxious Arousal scale, and the Emotional Control subscale of the BRIEF contributed heavily to group differences on Function 1. The Shifting and

Planning/Organization subscales of the BRIEF and the Anxious Apprehension (Worry) subscale also contributed to group differences for the first function. For Function 2, Anxious Apprehension (Worry) contributed heavily, followed by the Emotional Control, Shifting, and Working Memory subscales from the BRIEF. Lastly, for Function 3, the Anhedonic Depression measure was again a very large, but negative, contributor to group differences, followed by the Emotional Control subscale and the Anxious Apprehension (worry) scale.

As is evident in the group centroids displayed in Table 9, the current depression group scored much higher on the first function than did any of the three other groups. Thus, the first function was interpreted as characterizing current depression. For ease of interpretation, variables found to distinguish groups on each function will always be listed in descending order by the magnitude of the absolute value of the variable's structure coefficient. Therefore, from the first function it was determined that current depression could be distinguished from anxiety and past depression due to elevated anhedonic depression symptoms, increased impairment in emotional control, elevated anxious arousal and anxious apprehension symptoms, and increased impairment in planning and organizing as well as shifting.

In the same fashion, the group centroids for the second function revealed that the past depression group scored highest on this function, followed by the anxiety group, whereas the currently depressed and control groups scored similarly to each other but in the opposite direction of the past depression and anxiety groups. Thus, the second function largely characterized the past depression group, which was distinguishable from the other groups due to elevated worry (anxious apprehension) symptoms and increased impairment in emotional control, shifting, and working memory.

Finally, the group centroids on the third function indicated that the pure anxiety group

was best distinguished from the other groups by low anhedonic depression symptoms, increased impairment in emotional control, and elevated worry (anxious apprehension) symptoms. Considered collectively, the three functions indicated that problems with emotional control differentiated the clinical groups from the control group, whereas impairments in shifting uniquely characterized depression (current and past) from both the anxiety and the control groups. Additionally, the three functions suggested that problems with planning and organizing uniquely characterize current depression, whereas problems with working memory uniquely characterized past depression.

Lastly, univariate analysis of variance (ANOVA) was used to determine whether the discriminant scores for each diagnostic group were significantly different for each function. Where statistics robust against violation of homogeneity of variances were not required, Hochberg's GT2 test was used for post-hoc analysis of significant ANOVA due to the large differences in samples sizes across groups (Hochberg, 1974). For Function 1, Levene's statistic was found to be significant. Thus, the Welch and Brown-Forsythe statistics were computed in place of the typical F statistic. Both robust tests indicated that discriminant scores significantly differed across groups, with $F(3, 30.42) = 7.56$, $p < .01$, and $F(3, 24.79) = 12.27$, $p < .01$, respectively. The Games-Howell post hoc test revealed that the current depression group scored higher on the first function than the control group, the past depression group, and the anxiety group (all ps $< .05$), who scored approximately the same. For Function 2, Levene's statistic was non-significant; thus, the typical F test was computed, which revealed significant differences between groups on the second function, $F(3, 138) = 9.90$, $p < .001$. Using Hochberg's GT2 post-hoc test, it was determined that both the past depression (who scored highest) and the anxiety groups scored significantly higher than the control group on the second function (all ps $< .001$).

The differences between the current depression group and the other groups on Function 2 were not significant, though the current depressives scored most similarly to the control group on this function (ΔM = -.19, SD = .36). Finally, Levene's statistic was significant for Function 3. The Welch and Brown-Forsythe statistics revealed significant differences between groups, Welch F (3, 32.12) = 5.03, $p < .05$. The Games-Howell statistic computed post-hoc indicated that the anxiety group scored significantly higher on the third function than both the control ($p < .05$) and the past depression groups ($p < .01$), who scored similarly to each other and to the current depression group. Table 10 summarizes the results of the DDA.

Regression Analyses

A series of post-hoc, exploratory hierarchical multiple regression analyses were conducted to examine whether the executive function deficits found to characterize and differentiate groups in the DDA could be used to predict current functional disability as indicated by scores on the Global Assessment of Functioning (GAF) scale from the diagnostic interview. As described above, the DDA demonstrated that combinations of the four BRIEF subscales could be used to significantly differentiate between groups. Specifically, emotional control differentiated the clinical groups from the control group, shifting differentiated both depression groups from the other groups, planning/organization differentiated the current depression group from the other groups, and working memory differentiated the past depression group from the other groups.

First, to determine whether each of the four subscales predicted disability in the full sample (N = 142), four hierarchical multiple regressions were performed. One BRIEF subscale per regression was entered alone in the first step of each of the multiple regressions. Because past research and the correlational and DDA analyses above indicated that current symptoms

affect both executive functioning and current level of disability, anhedonic depression and anxious apprehension symptoms were entered simultaneously in a second step for each multiple regression. As displayed in Table 11, all four of the full regression models (BRIEF subscale plus symptom measures) predicting GAF were significant, with problems in each area of executive functioning emerging as negative predictors of global functioning (e.g., positive predictors of disability). Specifically, the full model that included emotional control accounted for 20%, the full model that included shifting accounted for 14%, the full model that included planning and organization accounted for 14%, and the full model that included working memory accounted for 16 % of the variance in general functioning (e.g., disability) across the sample. However, the associations between general functioning and shifting, planning/organization, and working memory, respectively, reduced to non-significance after the symptom measures were included in the second step. As the t-tests indicate, only emotional control continued to significantly predict GAF once the symptom measures were added to the model. Thus, the effect of shifting, working memory, and planning and organizing on global functioning was attributable to the effect of current symptoms, while most of the effect of emotional control on global functioning was not attributable to symptoms. Further confirming this, follow-up analyses indicated that although anhedonic depression and anxious apprehension (worry) negatively predicted GAF, this association reduced to non-significance after emotional control was included in the model, R^2 = .20, F(3, 138) = 11.30, $p < .001$; $t(138) = -1.77$, $p = .08$, ns and $t(138) = -1.04$, $p = .30$, ns, for depression and anxiety, respectively.

Second, to explore whether the four subscales predicted disability in each clinical group specifically, four hierarchical multiple regressions were performed per group for the anxiety (N = 30), current depression (N = 9), and past depression groups (N = 21). Importantly, because the

sample sizes in each group were far below what could be considered appropriate for regressions with three or four predictors, the within-group regressions were performed solely to explore whether the trends observed in the full sample could be replicated within the clinical groups (the depression groups specifically) in less than ideal statistical conditions. They were therefore interpreted with caution.

As described above, the current and past depression groups were composed of equal proportions of individuals with "pure" depression and individuals with co-morbid depression and anxiety to increase power for the DDA. However, this heterogeneity of diagnoses was problematic for the group-specific regression analyses because of the confounding potential of the co-morbid anxiety. To reduce the effect of co-morbid anxiety in the past depression group, individuals with co-morbidity were excluded from all regression analyses within this group. Thus, for the group specific regression analyses the past-depression group contained only 21 participants instead of 41. Unfortunately, due to the small size of the current depression group this same strategy could not be similarly used within this group to "un-confound" anxiety and current depression. Instead, the interaction between anhedonic depression and anxious apprehension was entered as a predictor in a third step for all hierarchical regression analyses within this group. Thus, rather than remove the effect of comorbid anxiety, the effect of co-morbidity was modeled in the current depression group. Otherwise, the regression analyses for the three clinical groups were conducted in the exact same fashion as for the full sample above.

As displayed in Table 12, emotional control significantly predicted GAF in the anxious group in both the full and the reduced models, $F(3,26) = 3.29$, $p < .05$ (full model). In the current depression group, emotional control approached significance as a predictor in the full model that included the interaction between anhedonic depression and anxious apprehension, as displayed in

Table 13. Lastly, in the past depression group, emotional control approached significance as a predictor in the reduced model, as displayed in Table 14. As with the full sample, problems with emotional control generally emerged as a negative predictor of global functioning (e.g., a positive predictor of disability) across groups, though in the current depression group this trend was reversed, with impaired emotional control associated with better functioning, $t(4) = 2.61$, $p < .10$, ns. The Shifting, Planning/Organization, and Working Memory subscales of the BRIEF did not emerge as significant predictors of global functioning within any of the clinical groups. With the exception of anxious apprehension (which emerged as a negative predictor of global functioning in the anxiety group, $R^2 = .16$, $F(1, 28) = 5.23$, $p < .05$), current anhedonic depression and anxious apprehension symptoms also did not significantly predict global functioning within the clinical groups.

Finally, within each clinical group exploratory multiple regression analyses were conducted to further examine the relationship between current anxious apprehension and anhedonic depression symptoms and impairment on the four subscales of the BRIEF. First, the four BRIEF subscales were entered simultaneously in multiple regressions predicting symptoms within the three clinical groups. Non-significant predictors, if any, were removed, and the multiple regression analyses were repeated (with predictors, again, entered simultaneously).

In the anxiety group, a model including problems with emotional control and planning and organizing significantly predicted anhedonic depression symptoms, $R^2 = .55$, $F(2, 27) = 16.23$, $p < .001$. As indicated by the beta values, both emerged as positive predictors of anhedonic symptoms, $ß = 0.39$, $t(27) = 2.91$, $p < .01$ and $ß = 0.55$, $t(27) = 4.09$, $p < .001$ respectively. A model including emotional control, shifting, and planning and organization significantly predicted anxious apprehension symptoms in the anxiety group, $R^2 = .42$, $F(3, 26) =$

6.34, $p < .01$. Emotional control emerged as a positive predictor of anxious apprehension symptoms, while planning and organizing emerged as a negative predictor of anxious apprehension symptoms, $ß = 0.38$, $t(26) = 2.32$, $p < .001$ and $ß = -0.56$, $t(26) = -3.46$, $p < .01$. Shifting approached significance as a positive predictor of anxious apprehension symptoms, $ß = 0.33$, $t(26) = 1.93$, $p = .065$, ns.

In the current depression group, a model including problems with emotional control, planning and organizing, and shifting approached significance in predicting anhedonic depression, $R^2 = .75$, $F(3, 5) = 4.97$, $p = .058$. Specifically, in this model, problems with emotional control and planning and organizing emerged as positive predictors of anhedonic depression symptoms, $ß = 0.96$, $t(5) = 3.13$, $p < .05$ and $ß = 1.45$, $t(5) = 3.45$, $p < .05$, respectively. However, problems with shifting emerged as a negative predictor of anhedonic depression in the currently depressed group, $ß = -1.42$, $t(5) = 2.9$, $p < .05$. Problems with emotional control emerged as a significant positive predictor of anxious apprehension in the currently depressed group, $ß = 0.70$, $R^2 = .48$, $F(1, 7) = 6.55$, $p < .05$.

In the past depression-only group (i.e., the group with co-morbid anxiety participants removed), problems with shifting emerged as a positive predictor, $ß = 0.66$, $t(18) = 3.00$, $p < .01$, and problems with working memory emerged as a negative predictor, $ß = -0.51$, $t(18) = -2.33$, $p < .05$, of anhedonic depression symptoms, $R^2 = .35$, $F(2, 18) = 4.92$, $p < .05$. Emotional control and planning/organization did not emerge as significant predictors of anhedonic depression symptoms in the past depression-only group. None of the four BRIEF subscales significantly predicted anxious apprehension in the past depression-only group.

Discussion

Though numerous sources of evidence have indicated that impairments in executive functions are responsible for the lion's share of the economic burden associated with unipolar major depression, decades of research on this topic have produced more questions than answers. Departing from previous research, the present study adopted a systematically multivariate approach to examining executive function deficits in unipolar depression, using descriptive discriminant analysis (DDA) and multiple regression to discern a pattern of ecologically-valid deficits characteristic of depression and predictive of current global functioning, as measured by a rating system frequently used by insurance companies to decide disability claims.

Considered collectively, the three functions that emerged from the DDA suggest that, in the realm of executive functioning, unipolar depression (both current and past) is characterized by an increased difficulty with shifting. Further, current unipolar depression is specifically associated with difficulty planning and organizing, whereas past unipolar depression is specifically associated with impairment in working memory. Finally, affective disorders (past depression, current depression, and anxiety) appear to share impairment in emotional control, which is most pronounced in current unipolar depression.

Thus, the results of the DDA are generally supportive of the assertion that current depression is associated with executive function impairment. Research has found evidence of deficits in updating, shifting, and cognitive inhibition (Clark, Sarna, & Goodwin, 2005; Gohier et al., 2009; Harvey et al., 2004; Levin et al., 2007; Micco, et al., 2009; Ottowitz, Dougherty, & Savage, 2002). The present study's observation of elevated problems in shifting, emotional control (which requires inhibition of emotional responses and emotionally upsetting thoughts), and planning and organizing (which requires updating plans to fit changing circumstances)

roughly corresponds to this expected pattern of deficits. This harmony of conclusions across modes of measurement (including the real-world manifestations of executive dysfunction measured in the current study) suggests that the deficits associated with unipolar depression are not epiphenomenal. Instead, they appear to represent key features of the disorder that have complex and, in the case of emotional control, possibly mediational relationships with symptoms and exert considerable influence over global life functioning and disability. Further, though some have asserted that the deficits are largely the result of age (and subsequent psychomotor slowing), inpatient status, and comorbid psychological and medical conditions, the depressed individuals who participated in the present study were young (the average age for the group was 26, with six individuals under the age of 23), ambulatory, medically healthy, carefully psychologically screened non-patients. Thus, these characteristics (age, severity, comorbidity) are unlikely to explain present results.

Age may, however, partially explain the somewhat surprising result of increased impairment in working memory in the past but not currently depressed group. Though the average age of each group was not significantly different, the past depressed group contained 9 individuals (22% of the group) over age 42, while the current depressed group contained only 2 individuals over the age of 40 (also 22% of the group) and no individuals over age 42. Though controversial, previous research has suggested a relationship between the total number of depressive episodes experienced across the life-span and cognitive impairment (Beats, Sahakian, & Levy, 1996). Because the past depression group contained more middle-aged individuals, it may have also contained more individuals who have experienced multiple depressive episodes (purely as a result of having had more years of life in which to do so). Thus, this may explain why working memory was found to characterize past depression and not current depression. It is

also likely that, with only nine participants, the statistical power was simply too low to detect the importance of working memory in the currently depressed group. Alternatively, the working memory deficit may be a spurious result unrelated to a history of depression. Indeed, the symptom regression analyses support this explanation, as working memory was found to be negatively related to current anhedonic depression symptoms. Future research will have to clarify this relationship using larger samples.

In agreement with previous research (Paradiso, Lamberty, Garvey, & Robinson, 1997), the results of the DDA are supportive of the assertion that executive function impairments persist even after a depressive episode has receded, as difficulties with shifting differentiated both current and past depression from anxiety and the lifelong absence of an Axis I disorder. Age-appropriate norms were not available for the BRIEF. However, it is important to remember that while more impaired scores on the shifting subscale were shown to characterize current and past depression, the means for both groups (16.89 and 15.83, respectively, on a 30 point scale) suggest that neither group scored in a clinically impaired range. Thus, the deficit in shifting that characterizes current and past depression is likely subtle, perhaps exerting a negative effect on performance only in more complex real-world situations. This may explain the inconsistency that has resulted from studies relying solely on formal neuropsychological testing to examine executive function deficits, as such testing would likely miss this important difference in shifting ability.

Importantly, the results of the DDA additionally suggest that a common deficit in emotional control characterizes anxiety, current depression, and past depression. Emotional control was also the only executive function deficit uniquely associated with impaired global functioning in regression analyses (i.e., above and beyond what could be attributed to current

anhedonic depression and anxiety symptoms). Thus, while the effects of impairment in shifting, planning and organizing, and working memory on functional disability appear to be largely mediated by current depressive and anxious symptoms, difficulty using cognitive processes to control emotional reactions may represent a more stable, and perhaps trait-like, predictor of affective disorders. Further, since the predictive ability of symptoms disappeared after accounting for impairment in emotional control, difficulty governing emotional reactions may mediate the relationship between symptoms and general disability, suggesting a point of divergence between isolated symptoms (which are high base-rate phenomena) and diagnosable psychopathology (which is not). Although emotional control was not predictive of depression or anxiety symptoms in the previously depressed group without co-morbid anxiety, this is likely due to the fact that current symptoms in this group were quite low. Indeed, follow-up analyses revealed that anhedonic depression and anxious apprehension symptoms in the previously depressed group without co-morbid anxiety were no different than those observed in participants without a current or previous Axis I diagnosis, $t(27.46) = 1.45$, $p = .16$, ns, equal variances not assumed and $t(81) = 1.82$, $p = .07$, ns, respectively.

Surprisingly, problems with emotional control were associated with better global functioning in the currently depressed group. Given the small sample size of the currently depressed group, this seemingly contradictory result could be the result of sampling error. However, this result may also stem from the fact that only ahedonic depression was measured in the present study. As previous research has shown, melancholic depression (which is characterized by anhedonia, e.g., a lack of pleasurable response) is highly associated with impairment (Austin et al., 1999). Thus, a depressed person who retains the ability to feel emotions strongly and has a tendency toward emotional outbursts (which characterize impaired

emotional control) may be exhibiting less anhedonia and, therefore, would be expected to manifest improved functioning and reduced global impairment. However, impaired emotional control was associated with higher anhedonic depression symptoms in current depression (as well as anxiety), suggesting that the relationship between anhedonic depression, emotional control, and global functioning is even more complex, resulting, perhaps, from the improved interpersonal functioning that can accompany increased emotional expression in depressed individuals (Gurtman, 1987; Paddock & Nowicki, 1986).

Interestingly, the results of the exploratory regression analyses suggest that in current and past depression difficulty shifting is related to anhedonic depression symptoms in opposing ways, with impairment associated with increased symptoms in the past depressed-only group but associated with decreased symptoms in the currently depressed group. This may be an artifact of the depression measure used in the present study, which, as described in the Methods section, was composed of a sub-selection of items from MASQ-AD scale that excluded items measuring positive affect. Low positive affect, however, has been specifically associated with impaired attentional set shifting (Compton, Wirtz, Pajoumand, Claus, & Heller, 2004). Further, if difficulties in shifting can be viewed as a defining feature of rumination, this result supports that of a recent meta-analysis that concluded that rumination predicts the onset of a major-depressive episode but appears unrelated to the duration of a depressive episode once it has begun (Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008).

The present study has several important limitations. First, small sample size, particularly in the currently depressed group, limits the generalizability of the findings. Moreover, while DDA is suitable as long as the sample size of the smallest group is larger than the number of continuous variables under consideration (Tabachnick & Fidell, 2001), regression analyses

perform better with larger samples than those tested here. Therefore, the regression analyses in the present study should be considered preliminary and interpreted with particular caution. This is especially true of the group-level regression analyses, as noted above. Additionally, though medication usage was recorded by the experimenters, participants were, understandably, not always forthcoming about their use of psychotropic medications. Thus, data on psychotropic medication was largely incomplete and unsuitable for analysis. Non-medicated depressed patients show impairment across cognitive abilities, including attention, learning, memory, and executive function (Porter et al., 2003). However, certain kinds of anti-depressants (and other psychotropic drugs) are also known to cause executive function impairments (Schmitt et al., 2001). Thus, medication status is an important, yet unmeasured, potential contributor to the results of the study. Thirdly, although the use of a self-report measure of everyday executive functions was intended to capture elements of executive dysfunction that go unnoticed in formal testing situations, due to self-report bias it may also have artificially inflated evidence of such dysfunction (Lahr, Beblo, & Hartje, 2007). Indeed, as has been recently shown, depressed individuals may be particularly poor at gauging their actual level of executive function impairment, at least when the results of formal testing are equated with their actual level of impairment (which, as described above, may a problematic assumption) (Naismith, Longley, Scott, & Hickie, 2007). Additionally, though the inclusion of the perceptual bias measurement had a strong theoretical rationale, it is likely that the usefulness of this information was substantially limited by the necessity of collapsing the "pure" depression groups into "mixed' groups that also contained comorbid anxiety. Since anxious apprehension and depression are known to produce opposite patterns in the Chimeric Faces Task, creating groups equally mixed for both depression and anxiety symptoms, though acceptable for the DDA, likely cancelled out

any effect that could be discerned for the CFT using this analysis. Therefore, future research should re-examine the influence of perceptual biases/hemispheric asymmetries on executive function in unipolar depression using larger samples of anxiety free currently and previously depressed individuals. Lastly, as a correlational study employing a retrospective, cross-sectional design, it is impossible for the present research to discern the direction of causality between executive function impairments and unipolar depression. Indeed, as the literature has indicated, the relationship between unipolar depression, disability, deficits in executive function, age, physical health, cognitive style, and life-experience is unlikely to be simple or uni-directional. Although only prospective and experimental studies can definitively shed light on causal relationships, as the present study has demonstrated, future studies would do well to incorporate ecologically valid measurements of executive function as the deficits detected in the present study were typically sub-clinical in severity but still influenced global functioning, even more so in conjunction with elevated symptoms. Further, future studies would greatly benefit from using multivariate statistics. Supporting this, follow-up analyses revealed that, using the same dataset, an analysis strategy that relied exclusively upon multiple univariate ANOVAs would have completely missed the effects on both working memory and planning and organizing.

In aggregate, the present study indicates that unipolar depression is associated with executive function impairments, specifically impairments in emotional control, shifting, and planning and organizing. These impairments may fall below clinical significance and, therefore, be difficult to detect in formal testing situations, yet they nevertheless predict current disability (as measured by GAF). However, only deficits in emotional control appear to do so in the absence of depressive and anxious symptoms. Thus, the relationship between symptoms and executive function deficits is complex. Although deficits in shifting seem to persist into the

remitted state of unipolar depression, they also appear to have opposite relationships to anhedonic depression symptoms depending on whether or not one is currently experiencing a major depressive episode. So, while some executive function deficits may resolve as symptoms abate, emotional control appears to be a more stable predictor of affective psychopathology (e.g., anxiety and depression). As executive function training is increasingly considered as an adjunctive treatment for psychological disorders (Watkins, 2009), emotional control appears to be a worthwhile, yet previously unstudied, target for intervention.

Tables

Table 1

Summary of BRIEF Subscales

Scale	*# of items*	*Description of Ability*	*Example of Deficit*
Inhibit	13	stop behavior; delay impulses	easily distracted; act without thinking
Shift	10	switch activities; think flexibly	difficulty changing topic; difficulty adapting
Emotional Control	10	use of executive functions to regulate emotions	emotional outbursts; over-reactivity
Monitor	5	track effect of behavior on Others	thoughtlessness; inconsiderateness
Working Memory	12	hold information temporarily in mind	inability to follow directions; forgetfulness
Plan/ Organize	13	anticipate events & complete steps needed for future goals	not allowing enough time; overwhelmed by large tasks
Organization of Materials	7	keep workspace & livingspace organized	frequent loss of items; messiness
Task Completion	10	finish tasks in timely manner; complete tasks correctly	missing deadlines; making numerous errors

Note. BRIEF = Behavior Rating Inventory of Executive Function

Table 2

Demographic Characteristics

Characteristic	pMDE (n = 41)	cMDE (n = 9)	Anxiety (n = 30)	Control (n = 62)	Total (N =142)
Age (in years)					
M	32.51	26.11	30.23	27.5	29.44
SD	10.51	10.12	11.08	10.08	10.57
Gender (%)					
Male	29.27	11.11	13.33	51.61	34.51
Female	70.73	88.89	86.67	48.39	65.49

Note. pMDE = past depression group, cMDE = current depression group, anxiety = anxiety group, and control = comparison group with no diagnoses

Table 3

Descriptive Statistics

	pMDE	cMDE	Anxiety	Control	Total
Measure	*M(SD)*	*M(SD)*	*M(SD)*	*M(SD)*	*M(SD)*
1. GAF	77.39(9.96)	65.89(9.14)	76.23(10.00)	86.73(5.38)	80.49(10.17)
2. Inhibit	16.85(2.61)	18.11(2.89)	17.20(2.28)	17.15(2.96)	17.13(2.71)
3. Shift	15.63(3.77)	16.89(4.01)	15.87(2.79)	13.90(2.97)	15.00(3.38)
4. Emco	15.46(4.28)	19.78(6.01)	16.43(4.38)	13.19(3.00)	14.95(4.30)
5. Monitor	6.63(1.62)	7.33(1.58)	6.40(1.77)	6.00(1.25)	6.35(1.54)
6. Wkmem	17.41(3.80)	16.89(2.52)	17.17(3.32)	15.90(2.87)	16.67(3.28)
7. PlanOrg	18.05(3.29)	21.89(6.29)	18.70(3.91)	17.95(3.67)	18.39(3.90)
8. OrgMat	10.10(2.58)	9.89(2.15)	9.77(2.42)	9.95(1.87)	9.95(2.21)
9. BRI	54.59(10.21)	62.11(10.60)	55.90(7.75)	50.24(7.86)	53.44(9.27)
10. MCI	59.73(10.61)	64.78(12.16)	59.17(9.80)	57.08(8.91)	58.77(9.91)
11. GEC	114.32(18.66)	126.89(21.02)	115.07(15.20)	107.32(15.02)	112.22(17.21)
12. PSWQ	48.61(13.12)	53.11(22.00)	51.30(12.11)	39.98(13.96)	45.70(14.76)
13. MASQ-AA	21.90(3.51)	29.00(7.82)	23.03(4.82)	22.50(4.58)	22.85(4.85)
14. MASQ-AD8	15.37(4.55)	22.44(5.70)	13.93(3.18)	13.51(3.44)	14.70(4.43)
15. CFT	-0.41(0.45)	-0.36(0.58)	-0.60(0.36)	-0.52(0.33)	-0.50(0.40)

Note. pMDE = past major depressive episode; cMDE = current major depressive episode; GAF = Global Assessment of Functioning; Emco = Emotional Control; Wkmem = Working Memory; PlanOrg = Plan/Organization; OrgMat = Organization of Materials; BRI = Behavior Regulation Index; MCI = Metacognition Index; GEC = Global Executive Composite; PSWQ = Penn State Worry Questionnaire; MASQ-AA = Anxious Arousal subscale of Mood and Anxiety Symptoms Questionnaire; MASQ-AD8 = Loss of Interest items on Anhedonic Depression subscale of the Mood and Anxiety Symptom Questionnaire; and CFT = Chimeric Faces Task

Table 4

Correlations Among the Variables, pMDE vs cMDE

Measure	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1. GAF	(+)	-.01	-.08	.24	-.10	-.24	.05	-.06	-.15	-.07	-.12	-.17	-.25	-.09	-.06
2. Inhibit	-.14	(+)	.61*	.45*	.59*	.42*	.53*	.30	.76*	.48*	.69*	.24	.13	.13	.03
3. Shift	.22	.50	(+)	.68*	.47*	.44*	.68*	.24	.88*	.57*	.81*	.50*	.46*	.46*	-.10
4. Emco	.32	-.01	.50	(+)	.55*	.44*	.45*	.26	.87*	.45*	.73*	.60*	.38	.38	-.14
5. Monitor	-.49	.54	.52	.11	(+)	.43*	.59*	.32	.71*	.53*	.69*	.23	.15	.20	-.04
6. Wkmem	.06	-.19	.53	.61	-.02	(+)	.56*	.53*	.52*	.86*	.77*	.33	.19	.06	.16
7. PlanOrg	-.21	.49	.78	.10	.77	.27	(+)	.49*	.67*	.83*	.83*	.34	.32	.37	.03
8. OrgMat	-.01	.32	.59	.38	.45	.16	.65	(+)	.32	.75*	.60*	.19	-.15	.17	.27
9. BRI	.16	.54	.88*	.77	.56	.49	.60	.59	(+)	.61*	.89*	.53*	.39	.39	-.09
10. MCI	-.11	.53	.87*	.23	.67	.44	.97*	.66	.70	(+)	.90*	.31	.12	.22	.23
11. GEC	.02	.58	.95*	.52	.67	.50	.86*	.68*	.91*	.93*	(+)	.47*	.28	.34	.08
12. PSWQ	.14	-.05	.32	.70	-.03	.46	.03	.66	.50	.18	.35	(+)	.35	.39	-.16
13. AA	-.33	-.09	.38	.61	.03	.30	.15	.66	.47	.18	.34	.68	(+)	.59*	-.13
14. AD8	-.33	.16	.19	.39	.68	.04	.44	.57	.44	.39	.45	.40	.14	(+)	-.28
15. CFT	.22	-.60	-.25	.50	-.27	.13	-.57	-.29	-.01	.57	-.34	.22	.37	-.05	(+)

Note. Past depression group is above the diagonal, and current depression group is below the diagonal; GAF = Global Assessment of Functioning; Emco = Emotional Control subscale; Wkmem = Working Memory subscale; PlanOrg = Plan/Organization subscale; OrgMat = Organization of Materials subscale; BRI = Behavior Regulation Index; MCI = Metacognition Index; GEC = Global Executive Composite; PSWQ = Penn State Worry Questionnaire; AA = Anxious Arousal subscale of the Mood and Anxiety Symptoms Questionnaire; AD8 = the Loss of Interest items on the Anhedonic Depression subscale of the Mood and Anxiety Symptom Questionnaire; and CFT = Chimeric Faces Task

*$p < .01$ (2-tailed).

Table 5

Correlations Among the Variables, Anxiety vs Control

Measure	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1. GAF	(+)	-.24	.09	-.47*	-.18	-.12	.06	-.16	-.34	-.07	-.22	-.40	-.22	-.16	-.04
2. Inhb	-.08	(+)	-.01	.26	.46	.21	.42	.26	.53*	.38	.51*	-.19	-.03	.13	-.22
3. Shift	-.07	.39*	(+)	.38	.31	.30	.36	.04	.63*	.39	.57*	.27	.22	.33	.16
4. Emco	-.11	.51*	.49*	(+)	.28	.23	.23	.27	.84*	.34	.65*	.37	.19	.52*	.04
5. Montr	-.04	.52*	.38*	.30	(+)	.10	.37	-.05	.63*	.22	.46*	.12	-.03	.18	-.26
6. Wkmem	-.12	.60*	.39*	.44*	.42*	(+)	.47*	.33	.32	.78*	.66*	.08	.56*	.43	-.28
7. PlanOrg	-.09	.36*	.43*	.33*	.35*	.56*	(+)	.30	.47*	.85*	.79*	-.35	.13	.64*	-.30
8. OrgMat	-.01	.41*	.28	.34*	.18	.43*	.53*	(+)	.23	.60*	.50*	-.09	.09	.07	-.15
9. BRI	-.10	.80*	.78*	.81*	.61*	.61*	.48*	.42*	(+)	.49*	.83*	.28	.17	.49*	-.04
10. MCI	-.05	.48*	.48*	.45*	.43*	.77*	.90*	.65*	.60*	(+)	.90*	-.18	.33	.59*	-.31
11. GEC	-.08	.70*	.69*	.69*	.58*	.78*	.79*	.60*	.88*	.91*	(+)	.03	.30	.63*	-.22
12. PSWQ	-.03	.04	.27	.30*	.04	.10	.02	-.04	.24	.03	.14	(+)	.33	-.03	.16
13. AA	-.07	.24	.25	.04	.18	.19	.04	.13	.23	.12	.19	.13	(+)	.40	-.14
14. AD8	-.02	-.02	.29	.06	-.02	-.02	-.02	-.03	.12	-.01	.06	.38*	.29*	(+)	-.12
15. CFT	-.30	.03	.01	-.02	.03	.06	-.08	-.18	.01	-.11	-.06	-.06	-.12	.13	(+)

Note. The anxiety group is above the diagonal, and the control group is below the diagonal; GAF = Global Assessment of Functioning; Inhb = Inhibit subscale; Emco = Emotional Control subscale; Montr = Monitor subscale; Wkmem = Working Memory subscale; PlanOrg = Plan/Organization subscale; OrgMat = Organization of Materials subscale; BRI = Behavior Regulation Index; MCI = Metacognition Index; GEC = Global Executive Composite; PSWQ = Penn State Worry Questionnaire; AA = Anxious Arousal subscale of the Mood and Anxiety Symptoms Questionnaire; AD8 = the Loss of Interest items on the Anhedonic Depression subscale of the Mood and Anxiety Symptom Questionnaire; and CFT = Chimeric Faces Task

*$p < .01$ (2-tailed).

Table 6

Correlations Among the Variables, Total Sample

Measure	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1. GAF	(+)														
2. Inhibit	-.10	(+)													
3. Shift	-.19	.39*	(+)												
4. EmoC	-.41*	.36*	.58*	(+)											
5. Monitor	-.24*	.51*	.44*	.41*	(+)										
6. Wkmem	-.23*	.40*	.43*	.41*	.34*	(+)									
7. PlanOrg	-.12	.43*	.53*	.35*	.47*	.48*	(+)								
8. OrgMat	-.05	.32*	.22*	.26*	.18	.43*	.44*	(+)							
9. BRI	-.33*	.68*	.82*	.85*	.66*	.52*	.56*	.32*	(+)						
10. MCI	-.17	.46*	.55*	.43*	.45*	.78*	.86*	.66*	.61*	(+)					
11. GEC	-.27*	.63*	.76*	.71*	.62*	.73*	.80*	.55*	.89*	.90*	(+)				
12. PSWQ	-.30*	.05	.41*	.52*	.17	.24*	.06	.08	.43*	.14	.32*	(+)			
13. AA	-.22*	.16	.32*	.31*	.14	.25*	.20	.08	.33*	.21*	.30*	.30*	(+)		
14. AD8	-.31*	.09	.39*	.41*	.24*	.13	.34*	.10	.40*	.28*	.38*	.36*	.43*	(+)	
15. CFT	-.11	-.07	-.01	-.04	-.05	.05	-.14	.00	-.02	-.05	-.04	-.00	-.03	-.02	(+)

Note. GAF = Global Assessment of Functioning; Inhb = Inhibit subscale; Emco = Emotional Control subscale; Montr = Monitor subscale; Wkmem = Working Memory subscale; PlanOrg = Plan/Organization subscale; OrgMat = Organization of Materials subscale; BRI = Behavior Regulation Index; MCI = Metacognition Index; GEC = Global Executive Composite; PSWQ = Penn State Worry Questionnaire; AA = Anxious Arousal subscale of the Mood and Anxiety Symptoms Questionnaire; AD8 = the Loss of Interest items on the Anhedonic Depression subscale of the Mood and Anxiety Symptom Questionnaire; and CFT = Chimeric Faces Task

*$p < .01$ (2-tailed).

Table 7

Wilks's Lambda and Canonical Correlations for Diagnostic Groups

Function	Wilks's Λ	χ^2	*df*	p	R_c	R^2_c
1 to 3	.534	85.908	21	.000	.544	29.59%
2 to 3	.759	38.385	12	.000	.422	17.81%
3	.924	10.754	5	.056	.276	7.62%

Table 8

Standardized Discriminant Function and Structure Coefficients

Measure	*Function Coefficient*	*Structure Coefficient (r_s)*	r^2_s
Function 1			
Shift	-.340	.303	9.18%
Emco	.537	.581	33.76%
Wkmem	-.309	.081	0.656%
PlanOrg	.313	.381	14.51%
PSWQ	-.070	.302	9.12%
MASQ-AA	.342	.534	28.52%
MASQ-AD8	.631	.833	69.39%
Function 2			
Shift	.477	.520	27.04%
Emco	.178	.570	32.49%
Wkmem	.504	.447	19.98%
PlanOrg	-.505	-.002	0.00%
PSWQ	.465	.656	43.03%
MASQ-AA	-.605	-.169	2.86%
MASQ-AD8	.104	.177	3.13%
Function 3			
Shift	-.131	.171	2.92%
Emco	.472	.388	15.10%
Wkmem	-.556	-.018	0.03%
PlanOrg	.603	.198	3.92%
PSWQ	.450	.345	11.90%
MASQ-AA	.462	.255	6.50%
MASQ-AD8	-.964	-.453	20.52%

Note. Emco = Emotional Control; Wkmem = Working Memory; PlanOrg = Planning/Organization; PSWQ = Penn State Worry Questionnaire; MASQ-AA = Anxious Arousal subscale of MASQ; MASQ-AD8 = the Loss of Interest items on Anhedonic Depression subscale of MASQ

Table 9

Group Centroids

Group	*Function 1*	*Function 2*	*Function 3*
pMDE	-.074	.515	-.309
cMDE	2.422	-.286	-.036
Anxiety	-.046	.372	.497
Control	-.280	-.479	-.030

Note. pMDE = past depression group, cMDE = current depression group, Anxiety = anxiety group, and Control = comparison group with no current or past diagnoses

Table 10

Summary of DDA Results

Measure	*pMDE*	*cMDE*	*Anxiety*
MASQ-AD8	+[2]	+[1]	-
PSWQ	+[3]	+[1]	+[2]
MASQ-AA		+	
Emco	+[3]	+[1]	+[2]
Shift	+[2]	+[1]	
Wkmem	+		
Plan/Org		+	

Note. pMDE = past depression group, cMDE = current depression group, and Anxiety = anxiety group; + = elevated symptoms or increased impairment; - = lower symptoms or decreased impairment; where a single measure characterized multiple groups, the superscript represents the rank of the group mean compared to the mean score of other groups, with lower numbers indicating a higher mean score

Table 11

Regression Analyses Predicting GAF (N = 142)

	Variable	*β*	*t*	ΔR^2	*F*
Emotional Control					
Step 1	Emco	-.41	-5.31***	.17***	$F(1,140) = 28.20$***
Step 2				.03	$F(3,138) = 11.30$***
	Emco	-.30	-3.22**		
	MASQ_AD8	-.15	-1.76		
	PSWQ	-.10	-1.04		
Shifting					
Step 1	Shift	-.189	-2.28*	.04*	$F(1,140) = 5.20$*
Step 2				.14***	$F(3,138) = 7.30$***
	Shift	-.01	-0.12		
	MASQ_AD8	-.22	-2.51*		
	PSWQ	-.22	-2.47*		
Planning/Organizing					
Step 1	PlanOrg	-.12	-1.46	.02	$F(1,140) = 2.14$
Step 2				.14***	$F(3,138) = 7.38$***
	PlanOrg	-.04	-0.46		
	MASQ_AD8	-.21	-2.35*		
	PSWQ	-.23	-2.67**		
Working Memory					
Step 1	Wkmem	-.23	-2.75**	.05**	$F(1,140) = 7.54$**
Step 2				.16***	$F(3,138) = 8.67$***
	Wkmem	-.15	-1.89		
	MASQ_AD8	-.22	-2.60*		
	PSWQ	-.19	-2.23*		

Note. ΔR^2 = percent variance accounted for at each step; GAF = Global Assessment of Functioning; Emco = Emotional Control; PlanOrg = Planning/Organization; Wkmem = Working Memory; MASQ-AD8 = Loss of Interest Items of MASQ; PSWQ = Penn State Worry Questionnaire. *$p < .05$; **$p < .01$; ***$p < .001$.

Table 12

Regression Analyses Predicting GAF, Anxiety Group Only (N = 30)

	Variable	β	t	ΔR^2	F
Emotional Control					
Step 1	Emco	-0.47	-2.78*	.27*	$F(1,28) = 7.70$*
Step 2				.06	$F(3,26) = 3.29$*
	Emco	-0.39	-1.80^		
	MASQ_AD8	0.04	0.20		
	PSWQ	-0.25	-1.34		

Note. ΔR^2 represents percent variance accounted for at each step; GAF = Global Assessment of Functioning; Emco = Emotional Control; MASQ-AD8 = Loss of Interest Items, Anhedonic Depression Subscale of MASQ; PSWQ = Penn State Worry Questionnaire. ^$p < .10$; *$p < .05$.

Table 13

Regression Analyses Predicting GAF, Current Depression Group Only (N = 9)

	Variable	*β*	*t*	ΔR^2	*F*
Emotional Control					
Step 1	Emco	0.32	0.91	.11	$F(1,7) = 0.82$
Step 2				.25	$F(3,5) = 0.91$
	Emco	0.55	1.09		
	MASQ_AD8	-0.54	-1.35		
	PSWQ	-0.03	-0.05		
Step 3				.47*	$F(4,4) = 4.69$^
	Emco	0.80	2.61^		
	MASQ_AD8	-2.10	-3.95		
	PSWQ	-3.61	-3.18		
	Intx	4.33	3.27*		

Note. ΔR^2 represents percent variance accounted for at each step; GAF = Global Assessment of Functioning; Emco = Emotional Control; MASQ-AD8 = Loss of Interest Items, Anhedonic Depression Subscale of MASQ; PSWQ = Penn State Worry Questionnaire; Intx = the interaction of PSWQ and the MASQ_AD8. ^$p < .10$; *$p < .05$.

Table 14

Regression Analyses Predicting GAF, Past Depression Only (No Anxiety) (N = 21)

	Variable	*β*	*t*	ΔR^2	*F*
Emotional Control					
Step 1	Emco	-0.41	-1.96^	.17^	$F(1,19) = 3.85$^
Step 2				.02	$F(3,17) = 1.33$
	Emco	-0.46	-1.99^		
	MASQ_AD8	-0.03	-0.12		
	PSWQ	0.16	0.67		

Note. ΔR^2 represents percent variance accounted for at each step; GAF = Global Assessment of Functioning; Emco = Emotional Control; MASQ-AD8 = Loss of Interest Items, Anhedonic Depression Subscale of MASQ; PSWQ = Penn State Worry Questionnaire; Intx = the interaction of PSWQ and the MASQ_AD8. ^$p < .10$.

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