

ATTENTION AND EXECUTIVE FUNCTIONING DEFICITS ASSOCIATED WITH
DIMENSIONS OF ANXIETY AND DEPRESSION

BY

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DISSERTATION

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ABSTRACT

Both anxiety and depression have been linked with attention and executive deficits, yet the nature of these deficits and their clinical implications remain unclear. Chapter 1 reviews existing theories and findings from these literatures, along with key limitations of the existing research. Chapter 2 examines relations between worry, Generalized Anxiety Disorder (GAD), and working memory capacity (WMC). Results revealed that reduced WMC was associated with symptoms of GAD and elevated levels of worry, both cross-sectionally and prospectively. In contrast, WMC was not associated with depression. These findings suggest that reduced WMC may play a role in the etiology of excessive worry. Chapter 3 examines executive deficits associated with current and past symptoms of Major Depressive Disorder (MDD). Results revealed that current (MDD) symptoms were associated with deficits in inhibition – nevertheless, these deficits were not unique to depression, but were also associated with measures of state mood and current GAD symptoms, suggesting that they may simply be a by-product of general distress. In contrast, set-shifting deficits were uniquely associated with past MDD symptoms, suggesting that these deficits may reflect an ongoing vulnerability to depression. Chapter 4 examines individual differences in inattention blindness (IB). Across two independent samples, results revealed that attention and executive abilities (e.g., multiple object tracking skills, WMC) did not predict whether participants noticed the unexpected stimulus, but levels of anxiety and depression did. Specifically, results revealed a significant three-way interaction between worry, anxious arousal, and anhedonic depression predicting IB. Chapter 5 discusses a number of important directions for future research on attention and executive deficits associated with dimensions of anxiety and depression.

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CHAPTER 1

INTRODUCTION

Impact and significance of anxiety and depression

It has been estimated that three out of every ten individuals will meet criteria for a DSM-IV anxiety disorder at some point during their lifetime (Kessler et al., 2005). Not surprisingly, the economic cost of these disorders is tremendous (Greenberg et al., 1999; Lepine, 2002; Rice et al., 1998). Anxiety disorders are a major cause of disability and are associated with increased health care utilization (Candilis & Pollack, 1997; Leon et al., 1995; Sanderson & Andrews, 2002). These disorders have even been found to increase risk for the development of a number of chronic medical conditions (Lecrubier, 2001; Wells et al., 1989).

Likewise, DSM-IV unipolar mood disorders (i.e., depressive disorders) are both common and debilitating. Major depressive disorder (MDD) is among the most common mental disorders; the lifetime prevalence for MDD is approximately 17% (Kessler, et al., 2005). Depressive disorders are associated with poor quality of life, which in turn is associated with poor work performance and social adjustment (Goldberg & Harrow, 2005; Rapoport et al., 2005). In fact, unipolar major depression has been deemed the leading cause of disability worldwide (measured in years lived with severe impairment; Lopez & Murray, 1998). Furthermore, the lifetime risk of death by suicide for individuals who meet criteria for MDD is approximately 3.5%, which corresponds to 30% of all completed suicides by some estimates (Blair-West, Cantor, Mellsop, & Eyeson-Annan, 1999).

Given that anxiety and depressive disorders are both common and debilitating, there is an

urgent need for a clear understanding of factors that play a role in the development and/or maintenance of these problems. Once discovered, these factors can play a role in treatment planning and development. Though efficacious treatments for these conditions have been developed (see Roth & Fonagy, 2004 for a detailed review), these treatments are by no means a panacea. For example, many studies exploring the long-term efficacy of interventions for depressive disorders report high rates of relapse (e.g., Fava et al., 1998; Mintz et al., 1992). Furthermore, studies exploring the efficacy of interventions for more chronic anxiety disorders (e.g., generalized anxiety disorder) suggests that only a minority of patients are returned to “well” status (e.g., Ballenger, 1999; Fisher & Dunham, 1999). Thus, it is clear that there is significant room for improvement.

Conceptualization of anxiety and depression

A clear understanding of the nature of anxiety and depression has important implications for research aimed at understanding their causes and impact on functioning, which in turn has implications for the treatment of these conditions. As a result, there has been extensive research exploring the nature of both anxiety and depression.

One critical question addressed in this research involves the distinction between "normal" or "subclinical" anxiety and depression on one hand, and anxiety and depressive disorders on the other. Everyone feels anxious and/or depressed from time to time; so how do these experiences differ from those of individuals who meet diagnostic criteria for clinical disorders? Existing taxometric research suggests that normal and pathological anxiety differ quantitatively rather than qualitatively (e.g., Ruscio, Borkovec & Ruscio, 2001; Ruscio, Ruscio, & Keane, 2002). In other words, the difference seems to be one of degree, rather than kind. Findings for depression

are a bit more complex. The existing evidence suggests that depression in general is dimensional in nature (e.g., Beach & Amir, 2003; Franklin et al., 2002; Ruscio & Ruscio, 2000), though there may be specific subtypes of depression that are taxonic or categorical (e.g., melancholic depression; see Ambrosini et al., 2002, and Haslam & Beck, 1994).

Another critical question addressed in research exploring the nature of anxiety and depression is how these experiences differ *from one another*. In light of the findings discussed above, researchers have attempted to identify critical dimensions of anxiety and depression. Existing research suggests anxiety and depressive disorders share an important dimension in common, which has been described as elevated levels of 'general distress', or 'negative affect' (e.g., Clark & Watson, 1991; Joiner et al., 1996; Watson et al., 1995). In addition to being common to both anxiety and depressive disorders, this dimension is shared with a wide range of other forms of psychopathology (Ormel et al., 2004). Importantly, depressive and anxiety disorders each have unique components; depression is also characterized by decreased levels of positive affect (i.e., anhedonia), whereas anxiety is also characterized by elevated levels of physiological arousal (Clark & Watson, 1991; Joiner et al., 1996; Watson et al., 1995). Finally, there is evidence to support the distinction between two "types" of anxiety: somatic anxiety (i.e., anxious arousal) and cognitive anxiety (i.e., worry) (e.g., Barlow, 1991; Heller et al., 1997; Schwartz, Davidson, & Goleman, 1978). Not only can these two dimensions be distinguished from one another, they can be distinguished from anhedonia and negative affect (Nitschke et al., 2001) and are differentially relevant to specific anxiety disorders (e.g., worry is prominent in generalized anxiety disorder, whereas anxious arousal is characteristic of panic attacks; Barlow, 1991; Nitschke et al., 2000).

To summarize, both anxiety and depression (with the possible exception of some subtypes of depression) can be conceptualized as dimensional in nature. Furthermore, research has delineated four critical dimensions of anxiety and depression: negative affect, anhedonia, anxious arousal, and worry. Taken together, these findings have two important implications for research exploring factors associated with severe anxiety and/or depression. First, these findings suggest that research need not focus on "clinical samples" (i.e., samples that delineate groups on the basis of formal diagnostic status), given that existing diagnostic thresholds are largely arbitrary. Rather, researchers may simply collect data using dimensional measures administered to unselected groups of participants, as long as there is significant variability within the sample. In fact, dimensional conceptualizations of anxiety and depression suggests that novel findings related to the causes and/or impact of severe forms of anxiety and depression could have implications for understanding less severe forms of these phenomena, and vice versa. Second, researchers need to distinguish among worry, anxious arousal, symptoms of depression specific to depressive disorders, and non-specific distress. Ideally, researchers will generate testable hypotheses regarding these specific dimensions and will select measures appropriate to do so. Also, it will generally be valuable in any given study to examine at least two dimensions as a means of assessing whether the hypothesized relationships are specific to one dimension or are common to all dimensions.

Anxiety, attentional control, and executive control

Clinical conceptualizations of anxiety disorders include references to attentional and executive control deficits (APA, 2000). For example, diagnostic criteria for both Generalized Anxiety Disorder (GAD) and Posttraumatic Stress Disorder (PTSD) include 'difficulty

concentrating' as a part of these disorders, and 'hypervigilance' is considered to be a symptom of PTSD. Furthermore, a key feature of both Obsessive-Compulsive Disorder (OCD) and GAD is a reported difficulty controlling intrusive and distressing thoughts (obsessive thoughts in OCD, worries in GAD). Nevertheless, the nature of these deficits remains underspecified.

A large body of literature has examined the association between anxiety and cognitive performance. This literature grew out of interest in the construct of "test anxiety", which emerged from observations that individuals who are anxious tend to perform worse on both aptitude and achievement tests (e.g., Cassady & Johnson, 2002). For some time, the dominant theory in this area of research has been the processing efficiency theory (Eysenck & Calvo, 1992). The basic premise of this theory is that anxiety has a greater effect on "efficiency" than on "effectiveness". In other words, anxiety primarily impairs performance on tasks that require rapid information processing. Nevertheless, anxious individuals may perform just as well on tasks that do not require rapid processing because they can compensate by exerting more effort; thus, they will perform the task comparably, only more slowly. This theory posits that the effect of anxiety on performance is mediated by the effects of worry on working memory.

Despite the popularity of the processing efficiency theory, it has been deemed problematic because it does not clearly specify the cognitive processes that are impaired in anxious individuals. To address these concerns, Eysenck and colleagues (2007) recently introduced a major revision of the processing efficiency theory, which they refer to as the attentional control theory. According to this theory, attentional processes are central to understanding the effects of anxiety on performance. Specifically, anxiety leads to increased influence of the stimulus-driven attentional system (which is "driven" by stimulus salience).

Thus, anxiety impairs performance because it reduces attentional control in the presence of salient distractors. To support this assertion, Eysenck and colleagues cite findings from research involving self-report measures of distractibility (e.g., Broadbent et al., 1982; Derryberry & Reed, 2002), dual-task studies involving salient secondary tasks (e.g., Dusek et al., 1975; Shapiro & Lim, 1989), and tasks involving emotionally-valenced stimuli (see Bar-Haim et al., 2007).

Eysenck and colleagues (2007) argue that these attentional control deficits associated with anxiety emerge because worry specifically impairs the central executive of working memory (see Baddeley, 1986). In support of this, they cite growing evidence to suggest that working memory capacity largely reflects individual differences in “executive attention” (see Engle, 2002), along with findings from numerous studies showing that high levels of anxiety are associated with impaired performance on working memory tasks (e.g., Ashcraft & Kirk, 2001; Darke, 1988; Derakshan & Eysenck, 1998; Eysenck et al., 2005; Hayes et al., 2008; MacLeod & Donnellan, 1993). Nevertheless, there have been some inconsistent findings reported regarding the precise nature of these working memory deficits (e.g., impairment in verbal vs. spatial working memory; see Ikeda et al., 1996 and Shackman et al., 2006 for conflicting findings). Furthermore, some null findings have been reported from studies examining associations between anxiety and performance on tasks commonly used to measure working memory capacity, such as the operation span task (e.g., Santos & Eysenck, 2005). Finally, most of this research focuses on individuals with elevated levels of trait anxiety – thus, the extent to which these findings can be generalized to individuals with anxiety disorders is unclear.

Depression, attentional control, and executive control

Difficulty concentrating and distractibility have long been considered to be hallmark

features of clinical depression (APA, 2000). In line with this notion, depressed individuals show impaired performance on tasks that require sustained attention, even when these tasks involve completely "neutral" or non-emotional stimuli (Mialet et al., 1996; Ottowitz et al., 2002). For example, depressed individuals perform worse than controls on digit-span tasks (e.g., Fossati et al., 1999), the Continuous Performance Task (e.g., Cornblatt et al., 1989), the color-word Stroop task (e.g., Raskin et al., 1982), and negative priming tasks (e.g., MacQueen et al., 2000). Thus, like anxiety, research seems to confirm the clinical impression that depression is associated with attentional control deficits.

However, unlike anxiety, there have not been any comprehensive theories proposed to explain the mechanisms responsible for this deficit in depression (though some fairly basic accounts have been proposed; e.g., Lemelin et al., 1997). Rather, these findings have primarily been considered in the context of the broader literature on cognitive deficits associated with depression. Specifically, depression is also associated with impairments in memory (Burt et al., 1995), visuospatial processing (Elliot et al., 1996), problem solving (Goddard et al., 1996; Marx et al., 1992), and decision making (Conway & Giannopoulos, 1993). On the basis of these findings, some have argued that depression is associated with a general depletion in cognitive resources (Mathews & MacLeod, 1994), which in turn impairs performance on a wide range of cognitive tasks. Nevertheless, when depressed individuals are given appropriate cues or primes, they perform comparably to control participants (e.g., Hertel, 1994; Goddard et al., 2001; Hertel & Gerstle, 2003). As a result, Hertel and colleagues have proposed that depressed individuals have difficulty initiating efficient cognitive strategies.

In line with this notion, Levin and colleagues (2007) have proposed that the wide range

of cognitive deficits associated with depression could be accounted for by a fundamental deficit in executive functioning (EF). EF involves the effortful guidance of behavior toward some sort of goal; these functions are particularly important in nonroutine situations, and seem to rely heavily on the prefrontal cortex (Banich, 2009). Diagnostic criteria for depressive disorder include symptoms that could be construed as reflecting EF deficits (e.g., indecisiveness; APA, 2000), and research has consistently shown that depression is associated with impaired performance on a wide range of EF tasks (for reviews, see Austin et al., 2001; Ottowitz et al., 2002; Fossati et al., 2002; Rogers et al., 2004; and Levin et al., 2007). Though most reviews of research on this topic have concluded that depression is associated with a broad EF deficit, some have concluded that there is stronger evidence for deficits in specific domains of EF (e.g., inhibition, see Fossati et al., 2002; shifting, see Austin et al., 2001). Thus, while there is strong evidence to support the notion that depression is associated with an executive functioning deficit, the precise nature of this deficit is still unclear.

Limitations of Existing Research

As discussed above, research has consistently shown that both anxiety and depression are associated with attentional and executive control deficits. Nevertheless, the existing research has some important limitations. Specifically, most of the existing research in these areas uses: 1) imprecise methods to target specific dimensions of anxiety and depression; 2) imprecise measures of specific attentional and executive control deficits; and 3) cross-sectional designs.

As previously discussed, research has delineated critical dimensions of anxiety and depression. However, the vast majority of the existing research has used: 1) self-report measures of anxiety or depression that primarily measure general distress or negative affect (such as the

Beck Depression Inventory or the Spielberg State-Trait Anxiety Inventory; see Watson & Clark, 1984); 2) contrast groups of individuals that are likely to differ on more than one of these dimensions; or 3) general mood manipulations. In other words, researchers have failed to adopt appropriate methods to distinguish key dimensions of anxiety and depression, and therefore these variables are likely to be confounded in much of the existing research. This problem may help to explain why there is a great deal of overlap in findings from research on cognitive deficits associated with anxiety and depression, as well as inconsistencies in the existing research (including failures to replicate findings). Furthermore, most existing research focuses exclusively on current symptoms. This is particularly problematic in research on depression, since depressive disorders tend to be episodic in nature (APA, 2000; Kessler et al., 1997). As a result, some individuals who will be considered controls (in group comparison studies) or to have low levels of depression (in studies that examine depression dimensionally) will have had major depressive episodes in the past and will likely share vulnerability factors with individuals who are currently more depressed. An alternative approach is to use measures designed to tap specific and distinguishable dimensions of anxiety and depression, and to assess both past and current symptoms.

Despite these limitations, there is some evidence to support the notion that separate dimensions of anxiety and depression may be associated with distinct cognitive deficits. Most importantly, preliminary studies conducted in our laboratory suggest that different dimensions of anxiety and depression are associated with different patterns of performance on attention and working memory tasks (Bredemeier et al., 2009; Bredemeier et al., in press; Bredemeier et al., under review). Though our findings warrant replication, there are also several indirect lines of

evidence that serve to bolster our confidence in this general conclusion. First, research involving conceptually relevant constructs lends some support to the notion that different dimensions of anxiety and depression may be associated with distinct cognitive deficits. For example, low levels of positive affect have been linked to general "cognitive inflexibility" and difficulty shifting attention (Ashby et al., 1999; Compton et al., 2004), whereas anhedonia has been linked to deficits in resource allocation (Dubal et al., 2000; Yee & Miller, 1994). Given that low levels of positive affect are associated with depression (but not anxiety), this suggests that anhedonic depression is associated with unique attentional and/or executive control deficits that cannot be accounted for by high levels of negative affect and/or comorbid anxiety. Second, experimental manipulations which (imprecisely) induce different emotional states relevant to depression and anxiety have been shown to have different effects on cognitive performance (e.g., Gray, 2001; Jefferies et al., 2008; Schackman et al., 2006). For example, Jefferies and colleagues (2008) showed that experimentally-induced sadness and anxiety have opposite effects on attentional control. Third, research has shown that different dimensions of anxiety and depression are associated with different patterns of brain activity (e.g., Heller et al., 1995; Keller et al., 2000; Engels et al., 2010; Nitschke et al., 1999). In fact, these patterns of brain activation, when considered in the context of basic research from cognitive neuroscience, may help to explain the nature of the attentional and executive deficits associated with these different dimensions (see Bishop, 2007, Mayberg, 1997, and Levin et al., 2007). Finally, some important differences have emerged from research on cognitive biases associated with anxiety and depression. For example, there is evidence that anxiety is associated with biased orienting toward negatively-valenced stimuli, whereas this does not seem to be the case for depression (e.g., Mogg et al., 2000).

Likewise, depression is associated with explicit memory biases for negative information, but anxiety is not (e.g., Mathews & MacLeod, 2005).

A second key limitation of the existing research on attentional and executive control deficits associated with anxiety and depression concerns the cognitive tasks that have been utilized. Specifically, most of the existing work in these areas of research has not employed appropriate methods to target specific attentional and executive control mechanisms that have been identified in basic cognitive research (see Eysneck et al., 2007 and Miyake et al., 2000 for more detailed discussions of this problem). As a result, performance deficits are often difficult to interpret. For example, in studies using variants of the Stroop task, it is unclear whether deficits occur at the input (i.e., attentional) stage or the output (i.e., response selection) stage (MacLeod, 1991). An alternative is using cutting edge methods from cognitive psychology that were designed to isolate specific attentional and executive control mechanisms, and (when feasible) to use multiple tasks to measure the construct(s) of interest in order to isolate their shared variance (and thus eliminate method variance).

Given that both anxiety and depression are presumed to be associated with distractibility, a logical choice for exploring attentional control deficits associated with anxiety and depression is to use tasks designed to measure "attention capture" (Simons, 2000). Unlike most traditional tasks used to study selective attention (which place an emphasis on concentration), tasks within the attention capture paradigm focus on the extent to which stimuli that participants are supposed to ignore are able to grab or "capture" their attention. Thus, attention capture tasks avoid confounding capture (by distracters) with lapses in attention (i.e., when participants are simply not attending to the task, but are also not attending to distracters). Drawing upon the attention

capture paradigm also brings to bear the distinction between implicit and explicit attention capture (Simons, 2000). In implicit attention capture tasks, evidence for attention capture is based solely upon behavioral effects (i.e., response time differences between conditions). In explicit attention capture tasks, evidence for attention capture is based upon whether participants report noticing the critical distracter. This distinction is important, as implicit and explicit attention capture may involve different processes or mechanisms (see Most & Simons, 2001). However, this distinction has been ignored by prominent theories offered to explain attentional and executive control deficits associated with anxiety and depression. Likewise, while relations between attentional/executive control and implicit attention capture have been examined (e.g., Conway & Kane, 2001; McCabe et al., 2010), relations between attentional/executive control and explicit attention capture have not been sufficiently explored.

A third key limitation of the existing research on attentional and executive control deficits associated with anxiety and depression is that most of this research is cross-sectional, or manipulates participants' mood to examine how this affects cognitive performance. While this is not inherently a limitation, such designs do not permit us to test whether attentional and executive control deficits contribute to the development and/or maintenance of anxiety and depression. While most prominent theories offered to explain these phenomena have been silent on the issue of causality, much of the terminology used in these areas of research strongly implies that these deficits are *the result of* anxiety and depression (e.g., "anxiety *impairs* attentional control...", pg. 338, Eysenck et al., 2007; "depression *impairs* performance in certain cognitive tasks...", pg. 228, Williams et al., 2000). In other words, these deficits seem to be seen as epiphenomena, which may play a role in the impairment associated with these conditions, but

are not likely to be appropriate targets for intervention and will resolve when the disorders are effectively treated. An alternative view is that attentional and executive control deficits play a role in the etiology of anxiety and depression, and thus have important treatment implications. Initial support for this view could be obtained using prospective (i.e., longitudinal) designs.

There is some indirect evidence to suggest that attentional and executive control deficits could play a role in the etiology of these conditions. Specifically, some have argued (e.g., Ochsner & Gross, 2005) that basic cognitive processes play a key role in the generation and regulation of emotions (this perspective is sometimes referred to as the 'common-systems view'). Thus, it may follow that basic cognitive deficits can play a role in the etiology of emotional disturbances. There are some intriguing findings to support this view, including evidence for overlapping neural substrates associated with cognitive control and emotion regulation (Ochsner & Gross, 2005) and evidence that performance on basic cognitive tasks predicts emotional reactions to stressful events (Compton et al., 2008). In line with the common-systems view, there are a variety of ways in which attentional and executive control deficits may play a role in the development and/or maintenance of anxiety and depression. First, these deficits may result in impairments in daily functioning, which in turn can generate various forms of psychological distress, including anxiety and/or depression. Second, attentional and executive control deficits may provide mechanisms through which biased processing of emotional stimuli emerges, which in turn have been implicated in the etiology of anxiety and depression (e.g., Gotlib & Krasnoperova, 1998; MacLeod et al., 2002; Vasey & MacLeod, 2001). For example, attentional control deficits may lead to biased attention capture by negatively-valenced stimuli in anxious individuals (see Derryberry & Reed, 2002). In line with this notion, some have proposed that an

evolutionary function of the stimulus-driven attentional system is to promote detection of possible threats in the external environment (see Johnston & Strayer, 2001; Ohman et al., 2001). Likewise, executive control deficits may lead to memory biases for negative information in depressed individuals. In line with this notion, some evidence suggests that "overgeneral" autobiographical memory (i.e., the tendency to recall categories of events when asked to provide specific instances), as opposed to biased recall of specific negative experiences or events per se, is associated with risk for depression (e.g., Gibbs & Rude, 2004; Williams, 1996). Third, attentional and executive control deficits may influence individuals' ability to deploy certain emotion regulation strategies, which in turn lead to elevated levels of specific dimensions of anxiety and depression. For example, the ability to disengage attention may play a key role in the ability to regulate unpleasant emotions, and thus deficits in this ability may lead to more frequent, enduring, and/or intense unpleasant emotions (see Bredemeier et al., in press). Furthermore, working memory capacity may play a key role in the ability to suppress unwanted thoughts (see Brewin & Beaton, 2002), and thus reduced working memory capacity may lead to higher levels of worry. Finally, executive functions may play a key role in the ability to develop, implement, and follow through with daily plans and goals (see Banich, 2009), as well as our ability to evaluate potentially pleasurable stimuli or activities (see Nuechterlein, 1990). Thus, EF deficits may impair one's ability to initiate and maintain experiences or activities that will promote pleasant emotional states.

Goals of the Present Research

The overarching goal of the present research was to explore attentional and executive control deficits associated with dimensions of anxiety and depression. In Chapter 2, we examine

cross-sectional and longitudinal relations between working memory capacity and worry. In Chapter 3, we examine relations between dimensions of executive functioning (inhibition and set-shifting) and current and past symptoms of Major Depressive Disorder. In Chapter 4, we examine whether working memory capacity, dimensions of executive functioning, and dimensions of anxiety and depression predict explicit attention capture (or its inverse, which is referred to as “inattention blindness”; see Simons, 2000).

CHAPTER 2

WORRY AND WORKING MEMORY CAPACITY

Generalized anxiety disorder (GAD), the core feature of which is excessive worry, has a lifetime prevalence rate of approximately 5% (Kessler, Walters, & Wittchen, 2004). Individuals with GAD typically report a profound dissatisfaction with their quality of life (Turk, Mennin, Fresco, & Heimberg, 2000). Using data from a 15-site World Health Organization collaborative study, Ormel and colleagues (1994) found that of the studied mental health disorders, GAD had one of the strongest associations with disability. In fact, the degree of disability associated with GAD is greater than that associated with some chronic medical disorders, including diabetes and hypertension (Lecrubier, 2001).

Cognitive deficits may play a key role in the impairment associated with GAD. Difficulty concentrating is a diagnostic feature of the disorder (APA, 2000), and there is ample evidence to suggest that anxiety is associated with deficits in cognitive performance (see Eysenck, Derakshan, Santos, & Calvo, 2007, for a recent review). For almost two decades, the prominent theory in this area of research has been the processing efficiency theory (Eysenck & Calvo, 1992). According to this account, worry impairs information processing efficiency by disrupting working memory. Recently, Eysenck and colleagues (2007) introduced a revised version of this theory (referred to as the attentional control theory), in which they assert that worry specifically disrupts the central executive of working memory. This suggests that worry should be associated with impaired performance on a range of tasks that require working memory resources, regardless of the specific nature of the task or content.

Largely inspired by the processing efficiency theory, there is now ample evidence to suggest that anxiety is associated with working memory deficits (e.g., Ashcraft et al, 2001; Darke, 1988; Derakshan & Eysenck, 1998; Elliman et al., 1997; Ikeda et al., 1996; Lavric et al. 2003; MacLeod et al., 1993; Sorg & Whitney, 1992). However, the processing efficiency theory (as well as the attentional control theory) specifically posits that *worry* disrupts working memory. Nevertheless, instead of measuring/manipulating worry, most of these studies have used general anxiety manipulations or have measured trait anxiety (which is related to, but distinguishable from, worry; see Davey, Hampton, Farrell, & Davidson, 1992).

Two recent studies directly tested whether worrying leads to working memory impairments (Hayes et al., 2008, Leigh & Hirsh, 2011). In both cases, instructing participants to worry led to a decrease in residual working memory resources, relative to other cognitive exercises (e.g., thinking positive thoughts, thinking about negative images). However, these effects were only observed in participants with elevated levels of trait worry. Furthermore, worriers also exhibited slightly lower working memory scores in the control conditions. This raises the possibility that worriers have a stable deficit in working memory *capacity* (WMC) that is simply exacerbated when they engage in worry. In fact, WMC, while influenced by state factors, has a strong trait-like component (see Ilkowska & Engle, 2010). Furthermore, reduced WMC has been linked with a wide range of difficulties regulating behavior and cognition (see Feldman-Barrett et al., 2004, for a review), which has led to speculations that reduced WMC might confer risk for psychopathology (e.g., Ilkowska & Engle, 2010; Unsworth et al., 2005).

There are several reasons why reduced WMC might cause people to worry more. Berenbaum (2010) recently proposed a comprehensive two-stage model of worry in which he

argues that worry is initiated by perceptions of threat. There are at least two ways in which reductions in WMC may contribute to enhanced perceptions of threat. First, people with reduced WMC may be more likely to experience negative outcomes (e.g., doing poorly in school) – in essence, they have more reason to worry. Alternatively, these individuals may simply process information in a biased fashion, which in turn leads them to overestimate threat. In line with these ideas, WMC is strongly related to fluid intelligence (Kane et al., 2005) as well as tendencies to make inferences on the basis of limited information (Singer et al., 1992).

Berenbaum (2010) also argued that worry is terminated when the threat is accepted. Again, there are at least two ways in which reductions in WMC may prevent people from terminating their worries. First, people with reduced WMC might have difficulty accepting threat because they are intolerant of uncertainty. Alternatively, these individuals may be willing to accept that a threat exists, but still have difficulty suppressing their worries once they start. In line with these ideas, reduced WMC has been linked with intolerance of ambiguity (MacDonald et al., 1992) and difficulty suppressing unwanted thoughts (Brewin & Beaton, 2002).

Evidence to support the idea that reduced WMC can lead to excessive worry would have important implications for our understanding the etiology of psychopathology, in particular GAD. To our knowledge, no existing research has examined whether GAD is associated with reduced WMC. To provide a preliminary test of this proposal, we examined cross-sectional and longitudinal relations between worry, symptoms of GAD, and WMC. Specifically, college students completed three working memory tasks, as well as self-report measures of worry and a diagnostic interview to assess symptoms of GAD. Furthermore, a subset of these participants completed an online follow-up survey at the end of the academic semester on which they

reported their levels of worry in the past week. In light of evidence suggesting that depression is associated with working memory deficits (e.g., Harvey et al., 2004; Rose & Ebmeier, 2006), we also measured anhedonic depression and current symptoms of Major Depressive Disorder (MDD) to ensure that any findings that emerged could not be accounted for by co-occurring depression or general distress.

Methods

Participants

One hundred ninety eight college students (56% female), ranging in age from 18 to 26 years ($M = 19.6$; $SD = 1.7$), participated in the study¹. Most (51.5%) reported being European American, 19.2% were Asian American, 4.5% were African American, 9.6% were Latino/a, 2.0% were Biracial, and 12.1% selected the descriptor “other.” Of these participants, 163 were recruited through the University of Illinois Psychology participant pool and received course credit in exchange for their participation. The remaining 35 participants were recruited using flyers targeting individuals who have experienced problems with anxiety and/or depression, either recently or in the past. This recruitment strategy was used to obtain better representation of individuals with high levels of anxiety and/or depression in our sample. Participants recruited via these means were paid \$10/hour in exchange for their participation. Only individuals with normal or corrected-to-normal vision were permitted to participate in the study.

¹ Initially, 209 students participated in the study. However, 11 participants reported that they were taking psychotropic medications. Specifically, all of these participants were taking anti-depressants and/or stimulant medications. In light of evidence that these medications can, for better or worse, influence cognitive task performance (e.g., Kempton et al., 1999; McClintock et al., 2010), these participants were excluded from our analyses.

Materials

Self-report questionnaires. Worry was measured using the Penn State Worry Questionnaire (PSWQ; Meyer et al., 1990), which is designed to assess general tendencies to engage in worry, and the Worry Domains Questionnaire, Short Form (WDQ-SF; Stober & Joormann, 2001), which is designed to assess worry about a variety of specific topics. The PSWQ is composed of 16 items (e.g., “My worries overwhelm me”), and past research suggests that it has excellent test-retest reliability and good convergent and discriminant validity in undergraduate and clinical samples (Meyer et al., 1990; Nitschke et al., 2001). Like the PSWQ, the WDQ has been found to have good psychometric properties in undergraduate samples (Stober, 1998). The WDQ-SF was developed by selecting 10 items from the original WDQ - two items from each of five domains (relationships, lack of confidence, aimless future, work, financial; Tallis et al., 1994). This version has been shown to have high internal consistency and a near perfect correlation with the WDQ long form in an undergraduate sample (Stober & Joorman, 2001). For the purposes of the current study, the work-related items from the WDQ-SF were replaced with comparable items pertaining to academics. Furthermore, the instructions of the WDQ-SF were altered slightly. Participants were asked to rate how often they have been worrying about each specific domain *in the past week* (in order to assess recent levels of worry). Past research using suggests that this adaptation provides a reliable and valid measure of recent worry (Stober & Bittencourt, 1998). Internal consistencies (measured using Cronbach’s alpha) for the PSWQ and the WDQ in the present sample were .94 and .87, respectively.

Anhedonic depression was measured using a relevant subscale from the Mood and Anxiety Symptom Questionnaire (MASQ; Watson et al., 1995). On the MASQ, individuals

report how frequently they have experienced a variety of different symptoms during the past week. The anhedonic depression subscale is composed of 22 items related to experiences of pleasant mood and symptoms that are specific to depression (e.g., “felt like nothing was very enjoyable”, “felt really slowed down”). Past research indicates that this subscale has good convergent and discriminant validity in undergraduate, community, and clinical samples (Watson et al., 1995; Nitschke et al., 2001). Internal consistency in the present sample was .93.

Diagnostic Interview. Current symptoms of Generalized Anxiety Disorder and Major Depressive Disorder were assessed using the mood and anxiety modules of the Structured Clinical Interview for DSM-IV Disorders, Nonpatient Edition (SCID-NP; First, Spitzer, Gibbon, & Williams, 2002). These interviews were conducted by the lead author (KB), who is an advanced doctoral student in clinical psychology and has extensive experience conducting diagnostic assessments. In line with SCID guidelines, “current” was operationalized as symptoms experienced in the past month, and each diagnostic criterion was rated on a 3-point scale (0 = absent, 1 = subthreshold, 2 = threshold). Due to time constraints, a subset of the sample did not complete the interview (n = 40), so data from these participants were not included in analyses involving the interview data. Thus, the sample size for these analyses was 158.

Because the rates of current GAD and MDD were fairly low in our sample (6 and 5 individuals, respectively), along with evidence that both anxiety and depression are dimensional in nature (see Brown & Barlow, 2009 and Haslam, 2003), we computed dimensional scores for both GAD and MDD by summing clinical ratings for all of the substantive criteria for these diagnoses. Thus, GAD scores could range from 0 to 18 (given that there are 9 criteria, each of which is rated between 0 and 2), and MDD ratings could range from 0 to 20 (given that there are

10 criteria). To examine interrater reliability, secondary raters (all of whom were graduate students in clinical psychology) listened to 25 randomly selected interviews, and intraclass correlations were computed by treating raters as random effects and the individual rater as the unit of reliability (see Shrout & Fleiss, 1979). The intraclass correlations for the current GAD and MDD dimensional scores were .96 and .91, respectively.

Working Memory Tasks

Automated operation span task. Participants completed the automated version of the operation span task (Unsworth, Heitz, Schrock, & Engle, 2005), which is perhaps the most widely used paradigm in cognitive research on WMC. In this task, participants are presented with a set of arithmetic operations (e.g., " $(6 \times 2) - 5 = ?$ ") which they must solve as quickly as possible, each of which is followed by a letter to remember. At the end of each set of problems, participants are asked to recall the letters that appeared in that set in the proper order, and accuracy feedback is provided. The number of letters in each set varies from three to seven, and participants complete three of each set size. The dependent measure for this task is the OSPAN score, computed by summing all of the perfectly recalled sets. Thus, scores range from 0 to 75. Past research suggests that these scores have excellent test-retest reliability ($r = .83$; Unsworth et al., 2005). The automated OSPAN task is entirely mouse driven, which allows participants to complete the task independently. Furthermore, since letters are used rather than words, performance is less reliant on word knowledge than many other memory span tasks (including the traditional OSPAN task). Nevertheless, scores on the automated OSPAN are highly correlated with scores on the traditional OSPAN ($r = .66$; Unsworth et al., 2005).

N-back tasks. Participants also completed verbal and spatial versions of a two-item n-

back task, which is perhaps the most widely used paradigm in neuroscience research on working memory (see Owen et al., 2005). In this task, participants view a sequence of 20 capital and lower case letters, each of which appears at one of 10 possible spatial locations on a computer screen. When each letter appears, participants are instructed to press the “s” key if the item is the same as the item that appeared two back (not the item immediately before it, but the one before that), or the “d” key if it is different. In the letter version of the task, participants report whether the item is the same letter as the one that appeared two items earlier, or if it is a different letter. In the spatial version, participants report whether the item is in the same spatial location as the one that appeared two items earlier. Each item appears for 500 ms (with a 2000 ms ITI), and nonresponses are treated as errors. Participants completed five blocks of each version, and the first block of each was treated as practice. The dependent measure for this task is percent accuracy.

Procedure

Participants were tested individually. The order of the tasks and questionnaires was counter-balanced across participants, while the clinical interview was always administered at the end of the session. Individuals who reported clinically significant symptoms of psychopathology during the interview were given treatment recommendations and referrals.

Those participants who were undergraduate students, were recruited via the Psychology participant pool, and completed the initial portion of the study during the first half of the academic semester were asked if they were willing to be contacted for a brief online follow-up study during the last two weeks of the same semester. The timing of the follow-up portion was planned to: 1) ensure that there was a sufficient amount of time between measurements; 2)

increase the chances that participants who expressed interest would still be available to contact; and 3) take advantage of the natural stress manipulation that the academic semester entails (based upon the assumption that most undergraduates students experience elevated levels of daily stress at the end of the semester). Those who provided contact information received a notification about the follow-up study via email, along with directions for completing the study. The follow-up study involved completing online versions of the WDQ-SF and the MASQ anhedonic depression scale. Again, for the follow-up study, the directions of the WDQ-SF were altered to assess worry about each specific domain over the past week. Participants received entry into a drawing to win a monetary prize in exchange for their participation.

Out of the 77 participants who were eligible for the follow-up study, 63 (82%) provided contact information; there were no significant differences between those who provided contact information and those who declined (in demographics, scores from the questionnaires, or scores from the tasks). Of the individuals who were contacted, 38 (60% response rate) completed the online questionnaires; there were no significant differences between those who chose to participate in the follow-up and those who declined to participate. On average, participants completed the follow-up survey 64 days after their initial session (range = 36-91).

Statistical analyses

Because each participant completed both the PSWQ and WDQ-SF, we computed worry composite scores by averaging standardized scores from these two measures. Likewise, we computed composite working memory capacity scores by standardizing the scores from all three working memory tasks, then averaging them. Twenty two participants did not score significantly above chance performance (i.e., above 60%) on the 2-back letter task and 28 did not

score significantly above chance on the 2-back spatial task; these data were not included in our calculations². Nevertheless, since every participant had a score on at least one of the working memory tasks, every participant received a score for the working memory composite.

To examine cross-sectional relations between the working memory composite scores and questionnaire scores, we computed zero-order (Pearson) correlations. As expected, the dimensional diagnostic interview symptom variables were highly skewed. Thus, we used non-parametric correlations (Spearman's rho) for analyses involving these variables. Finally, for the longitudinal data, we conducted hierarchical linear regressions predicting scores at follow-up, entering corresponding scores from Time 1 in the first step and the working memory composite score in the second step.

Results

Descriptive statistics for the self-report measures and clinical ratings, along with correlations between these measures, are presented in Table 1. As predicted, there was a significant negative relationship between worry composite scores and working memory composite scores ($r = -.22, p < .01$), suggesting that worriers have reduced working memory capacity. Also as predicted, GAD dimensional scores were negatively correlated with working memory composite scores ($\rho = -.15, p < .05$). In contrast, working memory composite scores were not associated with either anhedonic depression scores ($r = -.02, p = .79$) or current MDD

² On the automated OSPAN task, participants are encouraged to maintain at least 85% accuracy on the arithmetic problems. In light of this, some researchers have endorsed excluding participants who score below this threshold, due to concerns that these participants could have scored higher by ignoring the math problems and focusing on the recall task (e.g., Unsworth et al., 2005). However, we found that arithmetic errors were highly correlated with OSPAN scores ($r = -.45$). Thus, we reasoned that excluding participants on the basis of arithmetic performance might remove important variance from the OSPAN scores. Importantly, all of our participants scored well above chance performance for the arithmetic portion of the task.

dimensional scores ($\rho = .01, p = .95$), suggesting that the relation between worry and working memory capacity cannot be accounted for by co-occurring depression.

Results from the longitudinal analyses are presented in Table 2. Again, as predicted, working memory composite scores predicted levels of worry at follow-up, even after taking into account initial worry ($F\text{-change} = 5.94, p < .05$). Specifically, working memory composite scores were negatively associated with worry at follow-up, suggesting that reduced WMC could lead to increases in worry over time. This association is portrayed graphically in Figure 1. In contrast, working memory composite scores did not predict anhedonic depression at follow-up ($F\text{-change} = 0.84, p = .37$), suggesting that the association between WMC and worry at follow-up cannot be accounted for by an increase in general distress in participants with reduced WMC.

Discussion

Both self-report levels of worry and clinician-rated symptoms of GAD were negatively associated with working memory composite scores, suggesting that people with reduced WMC experience elevated levels of worry. Furthermore, working memory composite scores predicted changes in levels of worry over time, suggesting that a reduction in WMC could confer risk for excessive worry. In contrast, working memory composite scores were not associated with self-reported or clinician-rated symptoms of depression, suggesting that the associations between worry, GAD, and WMC cannot be accounted for by co-occurring depression or general distress.

Based upon these findings, as well as past research showing that worry manipulations lead to a reduction in processing resources, we propose that there is a bi-directional relationship between worry and WMC. In other words, a trait-like reduction in WMC may make people prone to worry. Conversely, this capacity is reduced when people engage in worry, as worrying

occupies some working memory resources. This account would explain why recent studies have found that worry inductions only yield significant decreases in WMC in people with elevated levels of trait worry (Hayes et al., 2008, Leigh & Hirsh, 2011), as evidence suggests that individual differences in trait WMC are more evident when working memory demands are high (see Ilkowska & Engle, 2010). Thus, while our findings provide support for the basic tenets of the attentional control theory (Eysenck et al., 2007), our findings also suggest that this theory may not fully describe the nature of the relation between worry and WMC.

Of course, the longitudinal analyses presented here only provide preliminary evidence for the proposal that reduced WMC can cause excessive worry; these data are by no means sufficient to establish that a causal relationship exists. Rather, stronger longitudinal designs and/or experimental designs should be employed to rule out rival hypotheses that could account for our findings. Additional evidence in support of this proposal would have important implications for our understanding the etiology of GAD, and in turn may have important implications for the treatment of this disorder. To our knowledge, the present study is the first to show that GAD symptoms are associated with reduced WMC. While effective treatments exist for GAD (see Mitte, 2005), they are by no means a panacea. In particular, while many participants benefit from these treatments, only a minority of patients are returned to “well” status (e.g., Fisher & Dunham, 1999). The results of the present study suggest that alternative (or supplementary) interventions could be designed to remediate deficits in WMC as a means of treating GAD. That said, it is important to note that the number of participants who qualified for a full diagnosis of GAD in our sample was low. While existing evidence suggests that worry is dimensional (Ruscio, Borkovec, & Ruscio, 2001), it will be important for future work to explore whether the

link between GAD and WMC can be replicated (in particular, in samples containing more individuals who meet full criteria for the disorder).

In addition to the attention control theory, our findings may have important implications for a number of other prominent theories of GAD. Information processing models (e.g., MacLeod & Rutherford, 2004) suggest that attentional biases for threat-related information play an important role in the etiology of GAD. It is possible that these biases result, at least in part, from working memory deficits. In line with this proposal, WMC is thought to reflect individual differences in attentional control (Engle, 2002), and some work suggests that basic attentional control deficits contribute to attentional biases for threat (e.g., Derryberry & Reed, 2002). Likewise, Dugas and colleagues (Dugas, Marchand, & Ladouceur, 2005) have proposed a cognitive-behavioral model of GAD in which they assert that elevated levels of intolerance of uncertainty lead to excessive worry. Given that WMC has been linked to tolerance of ambiguity (MacDonald et al., 1992), we suspect that reduced WMC could contribute to the development of intolerance of uncertainty. Finally, Wells (2005) proposed a meta-cognitive model of GAD in which he argues that negative beliefs about the utility of worrying lead people to try to suppress their worries, and so individuals who hold such beliefs and have difficulty suppressing unwanted thoughts will worry excessively. Since WMC has been linked with individual differences in thought suppression (Brewin & Beaton, 2002), we propose that reduced WMC could contribute to difficulties suppressing worries once they arise, which, in combination with negative beliefs about worry, leads to excessive worry. Of course, these ideas are merely speculative. Additional research is needed to explore potential mechanisms involved in the relation between worry and

WMC, as well as to explore whether working memory deficits are associated with the initiation of worry, the termination of worry, or both (see Berenbaum, 2010).

The findings from the present study might also help explain some inconsistencies in past research examining relations between working memory and emotional distress. First, some inconsistencies have been reported about the nature of the working memory deficits associated with anxiety (e.g., Ikeda et al., 1996; Lavric et al. 2003), whereas some studies have failed to find any association at all (e.g., Santos & Eysenck, 2005). We propose that these inconsistencies could be accounted for by the fact that these studies have used methods that were not specifically developed to measure/induce worry. In fact, there is some evidence that somatic anxiety (or anxious arousal), which is distinguishable from worry (see Nitschke et al., 2001), selectively disrupts visuospatial working memory (Bredemeier et al., 2009; Shackman et al., 2006). Second, we propose that working memory deficits reported in depressed individuals (e.g., Harvey et al., 2004; Rose & Ebmeier, 2006) could be accounted for by co-occurring worry, given that we did not find that depression was associated with working memory composite scores.

In summary, we found the performance on working memory tasks predicted levels of worry, both cross-sectionally and prospectively. These findings provide evidence to support our proposal that reduced WMC can confer risk for excessive worry (and in turn, GAD). Thus, while some questions remain unanswered, our findings may have important implications for understanding the nature and causes of excessive worry, as well as its treatment.

CHAPTER 3

DEPRESSIVE SYMPTOMS AND EXECUTIVE FUNCTIONING

Major depressive disorder (MDD) is among the most common mental disorders; the lifetime prevalence of MDD is approximately 17% (Kessler, Berglund, Demler, Jin, Merikangas, & Walters, 2005). Unipolar depression is associated with poor quality of life, which in turn is associated with poor work performance and social adjustment (Goldberg & Harrow, 2005; Rapoport et al., 2005). In fact, unipolar major depression has been deemed the leading cause of disability worldwide (measured in years lived with severe impairment; Lopez & Murray, 1998).

Cognitive deficits may play a key role in understanding the impairment associated with depression. Difficulty concentrating is a diagnostic feature of Major Depressive Disorder (APA, 2000), and research has shown that people experiencing depression display a wide range of deficits in cognitive performance, including difficulties with attention, memory, and problem solving (see Levin, Heller, Mohanty, Herrington, & Miller, 2007 and Hammar & Ardal, 2009 for recent reviews). Given the breadth of these deficits, some have argued that depression involves a general depletion in cognitive resources (e.g., Mathews & MacLeod, 1994). However, the results of numerous studies suggest that people with depression do have sufficient resources, but simply have difficulty initiating efficient cognitive strategies (e.g., Hertel & Gerstle, 2003; Marx, Claridge, & Williams, 1992; see Hertel, 1994) and/or appropriately allocating these resources (e.g., Levens, Muhtadie, & Gotlib, 2009; Yee & Miller, 1994; see Ellis & Ashbrook, 1989).

Executive functioning (EF) involves the effortful guidance of behavior towards some sort of goal; these functions are particularly important in nonroutine situations, and seem to rely

heavily on the prefrontal cortex (Banich, 2009). In light the findings discussed above, along with evidence for structural and functional abnormalities in prefrontal cortex associated with depression, some have argued that cognitive deficits in depressed individuals could result from deficits in EF (e.g., Levin et al., 2007; Davidson, Pizzagalli, Nitschke, & Putnam, 2002). In line with this proposal, there is now ample evidence to suggest that depressed individuals show impaired performance tasks that require EF (see Austin, Mitchell, & Goodwin, 2001, Fossati, Ergis, & Allilaire, 2002, Ottowitz, Tondo, Dougherty, & Savage, 2002, and Rogers, Kasai, Koji, Fukuda, Iwanami, Nakagome et al., 2004, for reviews). However, recent research suggests that the construct of EF is multi-dimensional (e.g., Miyake, Friedman, Emerson, Witzki, & Howerter, 2000). In the context of such findings, the nature of the EF deficits associated with depression remains unclear. Some reviews have concluded that these impairments might be unique to inhibition (e.g., Fossati et al., 2002), some have concluded that they might be unique to set-shifting (e.g., Austin et al., 2001), and some have concluded that EF is broadly impaired in depressed individuals (e.g., Rogers et al., 2004).

There are a number of potential reasons for this lack of clarity. First, most of the existing work in this area uses classic EF paradigms, such as the Wisconsin Card Sorting task. While most of these tasks are well-validated, they generally require the use of multiple aspects of EF (see Miyake et al., 2000). As a result, impaired performance on these tasks can be difficult to interpret. An alternative approach is to use tasks developed to tap specific dimensions of executive functioning (e.g., inhibition, set-shifting). Second, most of the existing work in this area compares a group of diagnosed individuals with “healthy” controls. Not only does this approach involve contrasting groups at the extreme ends of a continuum, but it also introduces a

number of potential confounds. In particular, depressed individuals often experience co-occurring difficulties with anxiety, which in turn have been linked with EF deficits (see Eysenck et al. 2007). However, most existing research in this area fails to take into account co-occurring anxiety (see Levin et al., 2007). Finally, most of the existing work in this area has focused on individuals who are currently depressed. Nevertheless, depression tends to be an episodic phenomenon (Kessler, Zhao, Blazer, & Swartz, 1997). Thus, it is important to consider whether or not participants have experienced depression in the past as well, as this may be indicative of an underlying vulnerability to depression. This approach might prove particularly fruitful in discovering EF deficits that confer such risk, as opposed to deficits that are simply a by-product of participants' current mood state. In fact, there is some evidence to suggest that EF deficits associated with depression can persist even after depressed mood has remitted (e.g. Paelecke-Habermann, Pohl, & Lelow, 2005). Nevertheless, the existing work in this area has yielded inconsistent findings (see DeBattista, 2005), which could be a reflection of some of the methodological issues discussed above (e.g., co-occurring anxiety), or could simply suggest that some EF deficits persist after remission, while others do not.

In the present study, we examined EF deficits associated with current and past MDD symptoms. To do so, we employed tasks designed specifically to measure inhibition and set-shifting, both of which have been identified as possible aspects of EF that are particularly impaired in depressed individuals. In order to examine specificity, we also measured state mood and symptoms of Generalized Anxiety Disorder (GAD), which frequently co-occurs with MDD (Kessler, Chiu, Demler, & Walters, 2005; Kessler et al., 1997). Finally, we measured processing and motor speed in order to examine whether impaired performance on the EF tasks could be

accounted for by psychomotor slowing.

Methods

Participants

One hundred and sixty two college students (57% female), ranging in age from 18 to 26 years ($M = 19.7$; $SD = 1.8$), participated in the study³. Most (58%) reported being European American, 17% were Asian American, 5% were African American, 11% were Latino/a, 3% were Biracial, and 5% selected the descriptor “other.” Of these participants, 129 (79%) were recruited through the University of Illinois Psychology participant pool and received course credit in exchange for their participation. The remaining 34 participants were recruited using flyers targeting individuals who have experienced problems with depression and/or anxiety, either recently or in the past. This recruitment strategy was used to obtain better representation of individuals with elevated levels of distress in the sample. Participants recruited via these means were paid \$10/hour in exchange for their participation. Only individuals with normal or corrected-to-normal vision and no significant hearing problems were permitted to participate.

Materials

Diagnostic Interview

Current and past symptoms of a Major Depressive Episode (MDE) and current symptoms

³ Initially, 209 students participated in the study. However, 11 participants reported that they were taking psychotropic medications. Specifically, all of these participants were taking anti-depressants and/or stimulant medications. In light of evidence that these medications can, for better or worse, influence cognitive task performance (e.g., Kempton et al., 1999; McClintock et al., 2010), these participants were excluded from our analyses. Also, 47 participants did not complete the diagnostic interview.

of Generalized Anxiety Disorder (GAD)⁴ were assessed using the mood and anxiety modules of the Structured Clinical Interview for DSM-IV Disorders, Nonpatient Edition (SCID-NP; First, Spitzer, Gibbon, & Williams, 2002). These interviews were conducted by the lead author (KB), who is an advanced doctoral student in clinical psychology and has extensive experience conducting diagnostic assessments. In line with SCID guidelines, “current” was operationalized as symptoms experienced in the past month, and each diagnostic criterion was rated on a 3-point scale (0 = absent, 1 = subthreshold, 2 = threshold).

In our sample, 5 participants met full diagnostic criteria for a current MDE, 29 met full diagnostic criteria for a past MDE, and 6 met full diagnostic criteria for current GAD. Because the rates of current MDE and GAD were fairly low, along with evidence that both depression and anxiety exist on a continuum of severity (Brown & Barlow, 2009; Haslam, 2003), we computed dimensional symptom scores for current MDE, past MDE, and current GAD by summing clinical ratings for all of the substantive criteria for these diagnoses. Thus, current and past MDE scores could range from 0 to 20 (given that there are 10 criteria, each of which is rated between 0 and 2), and GAD ratings could range from 0 to 18 (given that there are 9 criteria). To examine interrater reliability, secondary raters (all of who where graduate students in clinical psychology) listened to 25 randomly selected interviews, and intraclass correlations were computed by treating raters as random effects and the individual rater as the unit of reliability (see Shrout & Fleiss, 1979). The intraclass correlations for current MDE, past MDE, and current GAD were .91, .92 and .96, respectively.

⁴ Past GAD symptoms were also assessed, but very few participants reported experiencing more severe symptoms in the past, relative to symptoms their in the past month. This is consistent with evidence that, unlike MDD, GAD tends to be chronic rather than episodic (Wittchen & Hoyer, 2001).

State mood

State positive affect (PA) and negative affect (NA) were measured using the Positive Affect and Negative Affect Schedule (PANAS; Watson et al., 1988). The PA scale consists of 10 pleasant emotion words (e.g., active, strong, proud), and the NA scale consists of 10 unpleasant emotion words (e.g., jittery, guilty, ashamed). Both scales were supplemented with 5 additional low arousal emotion words (e.g., content, proud, bored, ashamed) to provide better coverage of the full range of positive and negative affect states. State mood was assessed by asking participants to rate each item based upon how they felt “at the moment.” Because the testing session was fairly long, the PANAS was administered twice, and scores from the two administrations were averaged. Past research has shown that the PANAS is a reliable and valid measure of state mood in undergraduate and community samples (Watson et al., 1988; Crawford & Henry, 2004). Average internal consistencies (measured in Cronbach’s alpha) for PA and NA in the present sample were .87 and .84, respectively.

EF Tasks

Stop-signal task. To measure inhibition, participants completed the STOP-IT task (Verbruggen, Logan, & Stevens, 2008), a novel variant of the classic stop-signal paradigm (Logan, 1994). In this task, participants must categorize shapes as either a square or a circle as quickly and accurately as possible. On some trials (25%), an auditory beep occurs after the shape appears on the screen. This sound serves as a “stop-signal”, and participants are told to try not to respond (or to inhibit their response) when they hear a beep. The task consists of a practice block of 32 trials, followed by three experimental blocks of 64 trials each. After each block, performance feedback is provided. The primary dependent measure on this task is stop-

signal reaction time (the estimated time that it takes the “stopping process” to finish – thus, higher scores suggest worse inhibition), which is determined using a tracking method. Specifically, the delay between the onset of the shape and the beep is 250 ms on the first stop trial. Each time the participant is able to successfully inhibit their response, the beep occurs 50 ms later on the next stop trial; otherwise, the beep occurs 50 ms earlier on the next stop trial. Participants are told that they should only be able to stop on approximately half of the stop trials. Mean RT on no-signal trials from this task were also examined as an index of processing speed (higher scores reflect slower processing).

Plus-minus task. To measure set-shifting, participants completed the plus-minus task (Jersild, 1927; Spector & Biederman, 1976). This task consists of three lists of 30 two-digit numbers (prerandomized without replacement) on separate sheets of paper. For the first list, participants are asked to add 3 to each number and write down their answers. For the second list, participants are asked to subtract 3 from each number. For the third list, the participants are asked to alternate between adding 3 to and subtracting 3 (i.e., add 3 to the first number, subtract 3 from the second number, and so on). Participants are instructed to complete each list as quickly and accurately as possible. The dependent measure for this task is shifting costs, which was computed by taking the difference between the time each participant takes to complete the third list and the average of the times they take to complete the first two lists. Thus, higher scores reflect larger switch costs, and thus worse set-shifting. Error data for this task were also analyzed to examine possible speed-accuracy tradeoffs.

Finger tapping test. The Halstead-Reitan finger tapping test (Halstead, 1947; Reitan, 1979) was administered to measure motor speed. In this task, participants place their dominant

hand on a table, palm down and fingers extended, with their index finger resting on a lever that is attached to a counting device. They are instructed to tap their finger as quickly as possible for 10 seconds, keeping their hand and arm stationary. Each participant completed four trials, which were timed using a stopwatch. The dependent measure for this task is the average number of taps across the four trials (higher scores reflect greater motor speed).

Procedures & Statistical Analyses

Participants were tested individually. The order of the tasks and self-report questionnaires were counter-balanced across participants, while the clinical interview was always administered at the end of the session. Individuals who reported clinically significant symptoms of psychopathology during the interview were given treatment referrals.

As expected, the dimensional symptom variables, as well as error scores from the plus-minus task, were highly skewed. Thus, we used non-parametric correlations (Spearman's rho) for analyses involving these variables. For the remaining analyses, we computed zero-order Pearson correlations. Participants who met full diagnostic criteria for a current MDE were excluded from analyses involving past MDE dimensional scores⁵.

Sixteen participants stopped significantly more or less than 50% of the time on stop trials for the stop-signal task, and thus were excluded from analyses involving performance indices for this task. Likewise, 5 participants did not follow instructions properly on the plus-minus task, and thus were excluded from analyses for this task. In addition, 1 participant was missing data for each of these tasks, 1 participant was missing data for the MDE dimensional variables, and 4

⁵ The findings reported for past MDE symptoms did not change when participants who met full diagnostic criteria for a current MDE were included in the analyses.

were missing data for the GAD dimensional variable.

Results

Descriptive statistics for all of the measures are presented in Table 3, and correlations between symptoms, state mood, and performance indices from the cognitive tasks are presented in Table 4. There was a significant association between current (but not past) MDE symptoms and stop-signal RT. This finding is consistent with evidence that depression is associated with deficits in inhibition (e.g., Fossati et al., 2002). However, current GAD symptoms were also significantly associated with stop-signal RT, as was state positive affect (and there was a trend for state negative affect). This suggests that the association between current depression and inhibition may be driven by current distress, rather than being specific to depression.

In contrast, only past MDE symptoms were significantly associated with switch costs on the plus-minus task. This finding is consistent with evidence that depression is associated with deficits in set-shifting (e.g., Austin et al., 2001). While current MDE symptoms were not significantly associated with switch costs, they were significantly associated with errors on the plus-minus task, which may suggest that individuals experiencing current depressive symptoms exhibited a speed-accuracy tradeoff.

None of these variables was significantly associated with mean RT on no-signal trials from the stop-signal task, and only state positive affect was associated with performance on the finger tapping task. This suggests that the observed associations between depressive symptoms and EF are unlikely to be accounted for by deficits in motor or processing speed.

Discussion

We found that current MDE symptoms were associated with deficits in inhibition, whereas past MDE symptoms were associated with deficits in set-shifting. These findings highlight three potentially important methodological considerations for research examining EF associated with depression.

First, our findings highlight the utility of employing tasks that are designed to tap specific dimensions of EF. Whereas some reviews have concluded that depression is associated with broad deficits in EF (e.g., Rogers et al., 2004), this conclusion may be the result of an overreliance on classic paradigms (e.g., the Wisconsin Card Sorting task) that require the use of multiple aspects of EF. By employing relatively simple tasks designed to tap two critical dimensions of EF (inhibition and set-shifting), we found evidence for relatively distinct correlates of each. Specifically, current MDE and GAD symptoms were associated with deficits in inhibition, as was state mood. In contrast, only past MDE symptoms were associated with deficits in set-shifting. Future research should employ this approach when examining EF deficits associated with psychopathology, which can be strengthened by employing multiple tasks to measure each dimension (see Miyake et al., 2000) as well as tasks designed to tap other potentially important dimensions of EF (e.g., fluency, planning, updating).

Second, our findings highlight the potential utility of measuring symptoms of anxiety and state mood in research on EF and depression. While current MDE symptoms were associated with impaired inhibition, current GAD symptoms were as well, along with state positive affect (and to a lesser extent, state negative affect). In fact, deficits in inhibition have been implicated in a range of mental disorders (see Nigg, 2000). Thus, inhibition deficits associated with current

depressive symptoms may be function of current distress, rather than being unique to depression. Additional evidence to support this hypothesis could be obtained by using experimental designs to examine the effects of general stress/mood manipulations on inhibitory processes.

Finally, our findings highlight the potential utility of measuring past as well as current symptoms. In doing so, we found that past MDE symptoms were uniquely associated with shift costs, suggesting that people who have experienced elevated levels of depression in the past display a unique deficit in set-shifting that persists even after their symptoms have remitted. As a result, these deficits are unlikely to be mere by-products of mood disturbances, and thus may represent an enduring vulnerability to depression. Consistent with this idea, dopaminergic functioning has been implicated in set-shifting (O'Reilly, 2006) as well as the etiology of depression (Nestler & Carlezon, 2006). The finding that set-shifting but not inhibition was associated with past depressive symptoms could help to explain inconsistent findings about the persistence of EF deficits in depression (DeBattista, 2005) – in other words, our findings suggest that some of these deficits may persist, while others do not.

Of course, given that the present study used a college sample, caution must be exercised in generalizing from these findings. While our sampling strategy has a number of potential strengths relative to alternatives (e.g., limited number of participants receiving treatment, greater variability in symptoms, less concerns about age-related declines in EF), it also has some potential drawbacks (e.g., relatively low rates of current MDE and GAD, many participants not yet through the age of risk for MDD). Thus, our findings warrant replication in more diverse samples. Furthermore, our measure of past MDE symptoms was retrospective, and thus is subject to recall bias (see Henry et al., 1994). Ideally, future research should use prospective

designs. On a related note, since the data from this study is cross-sectional, we cannot conclude whether the observed relations between depression and EF are causal. For example, while it is possible that set-shifting deficits reflect an ongoing vulnerability to depression, it is also possible that these deficits are merely a by-product of other variables that are associated with risk for depression, such as rumination. In fact, rumination has been linked with both risk for depression (e.g., Nolen-Hoeksema, 2000) and deficits in set-shifting (Altimorano et al., 2010). These questions could also be addressed through the use of longitudinal designs, as well as through the use of cleverly designed experiments examining laboratory analogues of depression.

In conclusion, the findings from the present study provide useful insights into the nature of EF deficits associated with depression, and in turn highlight a number of important methodological considerations for future research on this topic. We found that current MDE symptoms, current GAD symptoms, and state mood were associated with inhibition, whereas set-shifting was uniquely associated with past MDE symptoms. Based upon these findings, we propose that set-shifting deficits cause vulnerability to depression, whereas inhibition deficits are a by-product of current distress. This proposal should be tested in future research.

CHAPTER 4

INDIVIDUAL DIFFERENCE IN INATTENTIONAL BLINDNESS

Inattentional blindness (IB) is the failure to notice a visible but unexpected object when attention is focused elsewhere (Mack & Rock, 1998; Neisser, 1979; Simons & Chabris, 1999). Most existing literature on this topic has used experimental designs to examine factors that influence rates of IB (or conversely, "explicit attention capture"; see Simons, 2000). Based upon this research, a number of important factors that influence rates of IB have been identified, including the difficulty of the task that participants are engaged in (e.g., Simons & Jensen, 2009) and the degree of similarity between the unexpected object and what participants are supposed to attend to or ignore (e.g., Most, Simons, Scholl, Jimenez, Clifford, & Chabris, 2001). However, in virtually all cases, there is some variability across participants – in other words, some people notice the unexpected object and others do not. While it is possible that these differences result from variability in the effectiveness of the manipulation (e.g., some participants didn't understand the instructions) or are simply due to chance (see Simons & Jensen, 2009 for a discussion of this issue), it is also possible that they result from stable individual differences that are related to cognitive performance.

Very few studies have attempted to examine individual differences in IB, and those that have focus on whether cognitive abilities related to attention predict noticing. For example, influenced by the literature suggesting that expertise can influence attentional performance (e.g., Boot et al., 2008), Simons and Jensen (2009) examined whether individual differences in performance on the primary task (i.e., multiple object tracking) predicts rates of IB. Despite the fact that increasing the difficulty of the task led to increased rates of IB, participants who were

better at performing the task were not more likely to notice the unexpected object. More recently, based upon evidence that working memory capacity reflects individual differences in executive attention (see Engle, 2002), Richards and colleagues (Hannon & Richards, 2010; Richards, Hannon, & Derakshan, 2010) examined whether working memory capacity predicts rates of IB. They found that participants with lower working memory capacity were less likely to notice the unexpected object (or conversely, more likely to exhibit IB).

Given the challenges inherent in studying the relation between individual differences and IB (e.g., since there is only one “critical” trial, there is no way to examine reliability), these previously reported findings warrant replication. Furthermore, we hypothesized that a participants’ current level of emotional distress will influence their likelihood of noticing an unexpected object. Specifically, both anxiety and depression have been linked with attentional control deficits (see Eysenck et al., 2007 and Mialet et al., 1996), but to our knowledge no previous research has examined whether anxiety or depression are associated with IB⁶.

Study 1

Both anxiety and depression are often conceptualized (and operationalized) in categorical terms. However, existing evidence suggests that both are better conceptualized as a continuum of severity (see Brown & Barlow, 2009 and Haslam, 2003). Thus, an alternative approach is to examine specific and distinguishable *dimensions* of emotional distress.

Whereas both anxiety and depressive disorders are characterized by elevated levels of general distress, depression (but not anxiety) is associated with diminished motivation and

⁶ One previous study (Lee & Telch, 2008) examined whether socially anxious individuals are more likely to notice an unexpected face with a negative expression, but did not necessarily explore whether social anxiety is associated with the general phenomenon of IB.

pleasant mood (i.e., anhedonic depression), and anxiety (but not depression) is associated with increased physiological arousal (see Clark & Watson, 1991). Furthermore, anxiety can be divided into two types: somatic anxiety (i.e., anxious arousal) and cognitive anxiety (i.e., worry) (see Barlow, 1991, Heller, Nitschke, Etienne, & Miller, 1997 and Schwartz, Davidson, & Goleman, 1978), which are distinct from one another and from anhedonic depression (Nitschke, Heller, Imig, McDonald, & Miller, 2001). These three dimensions of emotional distress—worry, anxious arousal, and anhedonic depression—have different biological and behavioral correlates (e.g., Engels et al., 2010; Heller et al., 1997; Larson, Nitschke, & Davidson, 2007). Thus, in the present study, we administered instruments specifically designed to tap these three constructs.

Methods

Participants

One hundred thirty four college students (58% female), ranging in age from 18 to 22 years ($M = 19.0$; $SD = 1.0$), participated in the study for course credit. Only individuals who reported having normal or corrected-to-normal vision were permitted to participate.

Materials

Cognitive Tasks

Inattentional blindness task. Participants completed a sustained attention task, with a single critical trial to measure IB. This task will be based upon Most and colleagues (2001); a schematic of the task is shown in Figure 2. In this task, participants completed successive trials of a dynamic multiple object tracking task, in which they were instructed to monitor the movement of four white shapes but ignore the four black shapes. For both colors, two of the

shapes were capital Ls and two of the shapes were capital Ts. Participants were instructed to count the total number of times any of the white shapes touch the sides of a 640 x 480 gray window centered on the screen. Each trial lasted approximately 12 seconds, after which participants were prompted to type the total number of times the white shapes touched the sides of the window. Participants completed four trials on which all of the items move at a standard speed, followed by one critical trial. On this critical trial, after 4 seconds of object motion, a gray cross entered the display vertically centered on the right, moved linearly across the display from right to left, and exited the display at the vertical midpoint on the left side. The gray cross was visible for 4 seconds, moving at the same rate as the rest of the items in the display. After the cross exited the display, the objects continued moving for 4 seconds. After reporting the number of touches, participants were asked “Did you notice anything other than the Ls and Ts on that last trial?” If they respond affirmatively, they were asked to describe what they saw. For these two questions, participants typed their responses into a text field on the display. Those who were able to accurately describe at least one feature of the expected stimulus (shape, color, direction of motion) were considered to have noticed it, while those who reported that they did not notice anything unexpected were considered to have experienced inattention blindness. Tracking accuracy was computed in a relatively conservative fashion; counts within 20% above or below the actual number of bounces (rounding up) were treated as correct. Percent accuracy for the first four non-critical trials was computed as an index of primary task performance⁷.

N-back tasks. Participants also completed verbal and spatial versions of a two-item n-back task (Owen et al., 2005). In this task, participants view a sequence of 20 capital and lower

⁷ The results did not change when other approaches for computing tracking accuracy were used (e.g., absolute accuracy, relative accuracy).

case letters, each of which appears at one of 10 possible spatial locations on a computer screen. When each letter appears, participants are instructed to press the “s” key if the item is the same as the item that appeared two items back (i.e., not the item immediately before it, but the one before that), or the “d” key if the item is different. In the letter version of the task, participants report whether the item is the same letter as the one that appeared two items earlier, or if it is a different letter. In the spatial version, participants report whether the item is in the same spatial location as the one that appeared two items earlier. Each item appears for 500 ms (with a 2000 ms ITI), and nonresponses are treated as errors. Participants completed five blocks of each version, and the first block for each was treated as practice. The dependent measure for this task is percent accuracy. Since each participant completed both versions, scores were standardized and summed as an index of working memory capacity.

Self-report Questionnaires

Worry was measured using the Penn State Worry Questionnaire (PSWQ; Meyer et al., 1990). The PSWQ is composed of 16 items (e.g., “My worries overwhelm me”). Past research has shown that the PSWQ has excellent test-retest reliability and good convergent and discriminant validity in undergraduate and clinical samples (Meyer et al., 1990; Nitschke et al., 2001). Internal consistency (measured using Cronbach’s alpha) for the PSWQ in the present sample was .93.

Anxious arousal was measured using the relevant subscale from the Mood and Anxiety Symptom Questionnaire (MASQ; Watson et al., 1995). On the MASQ, individuals indicate how frequently they have experienced a variety of different symptoms during the past week. The anxious arousal subscale is composed of 17 items related to somatic tension and sympathetic

hyperarousal (e.g., “hands were shaky”, “startled easily”). Past research indicates that the anxious arousal subscale of the MASQ has good convergent and discriminant validity in undergraduate, community, and clinical samples (Watson et al., 1995; Nitschke et al., 2001). Internal consistency in the present sample was .84.

Anhedonic depression was also measured using the relevant subscale from the MASQ. The anhedonic depression subscale is composed of 22 items related to experiences of pleasant mood and symptoms that are specific to depression (e.g., “felt like nothing was very enjoyable”, “felt really slowed down”). Like the anxious arousal subscale, past research indicates that the anhedonic depression subscale has good convergent and discriminant validity in undergraduate, community, and clinical samples (Watson et al., 1995; Nitschke et al., 2001). Internal consistency in the present sample was .90.

Procedures

Participants were tested individually. All participants completed the questionnaires first, followed by the n-back tasks (in counterbalanced order), followed by the inattention blindness task. Twenty seven participants did not score significantly above chance performance (i.e., above 60%) on one or both versions of the n-back task, and thus were excluded from analyses for working memory capacity.

Results and Discussion

The top half of Table 5 contains descriptive statistics for the self-report measures of worry, anxious arousal, and anhedonic depression. In this sample, 88 (70.4%) participants

reported noticing the unexpected stimuli and provided an adequate description⁸.

The top half of Table 6 contains means on all of the performance indices for participants who did and did not notice the unexpected stimulus. To test whether the performance indices predicted which individuals noticed the unexpected stimulus, we conducted a separate logistic regression analysis for each predictor⁹. The Wald statistics and p-values from these analyses are presented in Table 6. Consistent with Simons and Jensen (2009), tracking accuracy on the non-critical trials did not significantly predict IB. Inconsistent with findings reported by Richards and colleagues (Hannon & Richards, 2010; Richards et al., 2010), working memory capacity was also not a significant predictor of IB.

Worry, anxious arousal, and anhedonic depression did not significantly predict IB when these variables were considered separately. However, exploratory analyses revealed a significant three-way interaction between these variables predicting IB. The statistics from this regression analysis are presented in the top half of Table 7, and a graphical illustration of the nature of this interaction is presented in the top panel of Figure 3. As shown in Figure 3, there was a cross-over interaction between worry and anxious arousal predicting IB in participants reporting elevated levels of anhedonic depression. Specifically, among individuals with high anhedonic depression, greater worry was associated with more noticing among individuals low in anxious arousal, whereas greater worry was associated with less noticing among individuals high in

⁸ Eight participants reported noticing something unexpected on the last trial but did not accurately describe the unexpected stimulus, and thus were excluded from analyses. The results were virtually identical when these individuals were treated as non-noticers, rather than excluding them.

⁹ Logistic regression was used for these analyses (as opposed to independent sample t-tests) since the number of participants who noticed the unexpected stimulus and the number who did not notice were unequal. Nevertheless, our findings did not differ when t-tests were conducted instead of logistic regression analyses. Likewise, the results were quite comparable when the predictors were entered simultaneously in the regression analyses (rather than separately).

anxious arousal. In contrast, this pattern was not present in participants with diminished levels of anhedonic depression. Rather, for these participants, elevated levels of anxious arousal were associated with slightly higher rates of noticing, regardless of their reported levels of worry.

In summary, multiple object tracking ability did not predict IB, supporting the notion that individuals who notice the unexpected stimuli are not simply performing the primary task worse (or better). Working memory capacity also did not predict IB, suggesting that individual differences in executive attention, while related to measures of implicit attention capture (Conway & Kane, 2001), do not predict explicit attention capture (see Most & Simons, 2001). Finally, anxiety and depression did not predict IB when analyzed alone, but did so interactively. This finding is consistent with evidence showing that anxiety and depression are associated with deficits in attentional control (e.g., Eysenck et al., 2007, Mialet et al., 1996), and suggest that these deficits extend to the phenomenon of explicit attention capture. However, our findings suggest that an individual's pattern of anxiety and depressive symptoms may be particularly important, rather than their levels on each dimension alone.

Study 2

In Study 1, participants completed 4 trials of the multiple object tracking task, followed by a critical trial which contained an unexpected stimulus. Thus, given that participants received very little exposure to the primary task before the critical trial, it is not too surprising that rates of IB were somewhat low in this sample, which is not ideal for examining individual differences in IB. At the same time, this design did not provide a particularly strong measure of tracking performance. To address these concerns, in Study 2, we employed a multiple object tracking task that was comparable to the task used in Study 1, but using some methodological

modifications developed by Simons and Jensen (2009). These modifications were designed to provide a better index of primary task performance, while at the same time providing participants with more experience performing the task.

In Study 1 we found that working memory capacity did not predict IB. This result is inconsistent with recent findings reported by Richards and colleagues (Hannon & Richards, 2010; Richards et al., 2010). One potential explanation for this discrepancy is that Richards and colleagues used a different measure of working memory capacity - the operation span task. Thus, in Study 2, we explored whether our findings from Study 1 would replicate in a larger sample using the same working memory task as Richards and colleagues (in addition to the n-back tasks). In light of recent evidence that working memory and executive functioning tasks tap a common executive attention construct (see McCabe et al., 2010), we also administered tasks to measure other dimensions of executive functioning (i.e., response inhibition, set-shifting) to see whether these abilities predict IB, as well as to examine whether reported links between IB and working memory capacity might be accounted for by other executive deficits.

Likewise, the observed interaction between dimensions of anxiety and depression was not predicted. Thus, it is important to examine whether this finding will replicate in a larger sample with more participants who are experiencing elevated levels of anxiety and/or depression.

Finally, a potential limitation of Study 1 is that we did not collect information about participants' familiarity with the concept of IB. A common practice in IB research is to exclude participants with knowledge of IB from analyses (e.g., Most et al., 2001; Simons & Jensen, 2008; Richards et al., 2010), due to concerns that they may be less likely to exhibit IB as a result of this familiarity. Thus, failing to exclude these participants could invalidate findings.

Methods

Participants

One hundred ninety six college students (55% female), ranging in age from 18 to 26 years ($M = 19.6$; $SD = 1.7$), participated in the study¹⁰. Of these participants, 161 were recruited through the University of Illinois Psychology participant pool and received course credit in exchange for their participation. The remaining 35 participants were recruited using flyers targeting individuals who had experienced problems with anxiety or depression, either recently or in the past. This recruitment strategy was used to obtain better representation of individuals with elevated levels of anxiety and depression in the sample. Participants recruited via these means were paid \$10/hour in exchange for their participation. Only individuals with normal or corrected-to-normal vision and no significant hearing problems were permitted to participate.

Materials

Cognitive Tasks

Inattentive blindness task. As in Study 1, participants completed successive trials involving a dynamic multiple object tracking task, in which they were instructed to monitor the movement of four white letters but ignore the four black letters. After 5 practice trials, participants complete a block of trials in which the speed of the objects in the display was adjusted based upon counting accuracy. Counts were considered accurate if the response is within 20% of the correct total, rounding up. Following an accurate count, the speed of the

¹⁰ Initially, 207 students participated in the study. However, 11 participants reported that they were taking psychotropic medications. Specifically, all of these participants were taking anti-depressants and/or stimulant medications. In light of evidence that these medications can, for better or worse, influence cognitive task performance (e.g., Kempton et al., 1999; McClintock et al., 2010), these participants were excluded from our analyses.

objects was increased, and following an inaccurate count, it was decreases. In this manner, the object speeds were adjusted for each individual in order to achieve a consistent level of accuracy across participants. In this case, the accuracy threshold was set at 75%. The dependent measure for this portion of the task is threshold tracking speed (i.e., how fast a participant could track the objects and still achieve 75% accuracy), measured in pixels/second. Immediately after determination of the threshold speed, participants completed four trials on which all of the items move at a standard speed (4.32°/second), followed by one critical trial.

During the debriefing portion of the study, each participant was asked: 1) whether they had heard of the concept of ‘inattention blindness’ (and if so, to describe what it means in their own words); and 2) whether they had ever seen any famous IB demonstrations (e.g., ‘gorillas in our midst’; Simons & Chabris, 1999). Follow-up (clarification) questions were asked as needed.

N-back tasks. As in Study 1, verbal and spatial versions of a two-item n-back task were administered, and standardized accuracy scores from these two tasks were summed to provide an index working memory capacity.

Automated operation span task. Participants also completed an automated version of the operation span task (Unsworth, Heitz, Schrock, & Engle, 2005). In this task, participants are presented with a set of arithmetic operations (e.g., "(6 x 2) - 5 = ??") which they must solve as quickly as possible, each of which is followed by a letter to remember. At the end of each set of problems, participants are asked to recall the letters that appeared in the proper order, and accuracy feedback is provided. The number of letters in each set varies from three to seven, and participants complete three of each set size. The dependent measure for this task is the OSPAN score, computed by summing all of the perfectly recalled sets. Thus, possible scores range from

0 to 75. Past research suggests that these scores have excellent test-retest reliability ($r = .83$; Unsworth et al., 2005). The automated OSPAN task is entirely mouse driven, and thus allows participants to complete the task independently. Furthermore, since letters are used rather than words, performance is less reliant on word knowledge than many other memory span tasks (including the traditional OSPAN task). Nevertheless, scores on the automated OSPAN are highly correlated with scores on the traditional OSPAN ($r = .66$), suggesting that this task adequately taps working memory capacity (Unsworth et al., 2005).

Stop-signal task. To measure response inhibition, participants completed the STOP-IT task (Verbruggen, Logan, & Stevens, 2008), a novel variant of the classic stop-signal paradigm (Logan, 1994). In this task, participants must categorize shapes as either a square or a circle as quickly and accurately as possible. On some trials (25%), an auditory beep occurs after the shape appears on the screen. This sound serves as a “stop-signal”, and participants are told to try not to respond (or to inhibit their response) when they hear a beep. The task consists of a practice block of 32 trials, followed by three experimental blocks of 64 trials. After each block, visual performance feedback is provided. The primary dependent measure on this task is stop-signal reaction time (the estimated time that it takes the “stopping process” to finish), which is determined using a tracking method. Specifically, the delay between the onset of the shape and the beep is 250 ms on the first stop trial. Each time the participant is able to successfully inhibit their response, the beep occurs 50 ms later on the next stop trial; otherwise, the beep occurs 50 ms earlier on the next stop trial. Participants are informed that they should only be able to stop on approximately half of the stop trials.

Plus-minus task. To measure set-shifting, participants complete the plus-minus task

(Jersild, 1927; Spector & Biederman, 1976). This task consists of three lists of 30 two-digit numbers (prerandomized without replacement) on separate sheets of paper. For the first list, participants are asked to add 3 to each number and write down their answers. For the second list, participants are asked to subtract 3 from each number. For the third list, the participants are asked to alternate between adding 3 to and subtracting 3 (i.e., add 3 to the first number, subtract 3 from the second number, and so on). Participants are instructed to complete each list as quickly and accurately as possible. The dependent measure for this task is shifting costs, which was computed by taking the difference between the time each participant takes to complete the third list and the average of the times they take to complete the first two lists.

Self-Report Questionnaires

Worry, anxious arousal, and anhedonic depression were measured using the same self-report questionnaires as in Study 1. Internal consistencies for the PSWQ, MASQ anxious arousal subscale, and MASQ anhedonic depression subscale were .94, .79, and .93, respectively.

Procedure

Participants were tested individually. The order of all of the questionnaires and cognitive tasks was randomized to address potential concerns about fatigue and/or task order effects interacting with the individual difference variables of interest (e.g., working memory capacity, anxiety). Thirty four participants did not score significantly above chance performance (i.e., above 60%) on one or both versions of the n-back task. Likewise, 19 participants stopped significantly more or less than 50% of the time on stop trials for the stop-signal task, and 5 participants did not follow instructions properly on the plus-minus task. Data for these participants was not included in analyses for these tasks.

Results and Discussion

The bottom half of Table 5 contains descriptive statistics from the self-report measures. In this sample, 50 (27.2%) participants reported noticing the unexpected stimuli and provided an adequate description¹¹. A large percentage of our participants reported having previous knowledge of IB during the debriefing (knowledge of IB concept: 26%; familiarity with IB demos: 53%). Thus, rather than simply excluding a large portion of our sample from the analyses, we conducted cross-tabulation analyses to determine whether rates of IB actually varied as a function of previous knowledge of IB. We found that rates of noticing did not significantly differ in those who were familiar with the IB concept (28.0%) from those who were not (22.4%; $\chi^2 = .57, p = .45$). Likewise, rates of noticing did not differ in those who have previously seen IB demonstrations (27.1%) relative to those who had not (27.1%; $\chi^2 = .00, p = .99$). In light of these findings, we did not exclude any participants from analyses based upon their responses to these questions.

The bottom half of Table 6 contains means on all of the performance indices for participants who did and did not notice the unexpected stimulus. Again, to test whether the performance indices predicted which individuals noticed the unexpected stimulus, we conducted a separate logistic regression analysis for each predictor. Consistent with Simons and Jensen (2009) and with Study 1, tracking thresholds did not significantly predict IB. Inconsistent with findings reported by Richards and colleagues (Hannon & Richards, 2010; Richards et al., 2010) but consistent with our findings from Study 1, working memory capacity (measured using the n-back tasks and the OSPAN) did not significantly predict IB. In fact, although there was a trend

¹¹ Twelve participants reported noticing something unexpected on the last trial but did not accurately describe it. In both samples, the results were virtually identical when these individuals were treated as non-noticers, rather than excluding them.

for OSPAN scores, it is important to note that this trend was in the opposite direction to those observed by Richards and colleagues (i.e., participants who experienced IB had *higher* scores). Performance indices from the other executive functioning tasks also did not significantly predict noticing, which replicates and extends findings reported by Richards et al. (2010) showing that performance on an inhibition task is not associated with IB. Collectively, these findings suggest the individual differences in executive functioning do not predict explicit attention capture.

Again, worry, anxious arousal, and anhedonic depression did not predict IB when considered in isolation. However, consistent with Study 1, there was a significant three-way interaction between these variables predicting IB. The statistics from this regression analysis are presented in bottom half of Table 7, and a graphical illustration of the nature of this interaction is presented in the bottom panel of Figure 3. Importantly, even though rates of IB were much higher in this sample, the pattern of this interaction was quite comparable to the pattern observed in Study 1 (except that the main effect of anxious arousal was not as strong).

In summary, neither familiarity with the concept of IB nor popular IB demonstrations predicted whether or not participants notice the unexpected stimuli, suggesting that it is not necessary to exclude participants with previous knowledge of IB due to concerns that rates of IB will be lower in these participants. Likewise, participants' ability to perform the primary (tracking) task did not predict IB, replicating our findings from Study 1, as well as previous research (Simons & Jensen, 2009).

As in Study 1, working memory capacity did not predict IB, nor did other aspects of executive functioning. Again, these findings suggest that individual differences in executive attention (which has been proposed as a common component of executive functioning and

working memory capacity; McCabe et al., 2010) do not play a prominent role in explicit attention capture. In contrast, the interaction of worry, anxious arousal, and anhedonic depression did predict IB, thus replicating our finding from Study 1. These findings suggest that anxiety and depression are associated with explicit attention capture, but in an interactive rather than additive fashion. In other words, it is the pattern of emotional distress that an individual is experiencing that matters, rather than their levels on any particular distress dimension alone.

As predicted, giving people more experience with the tracking task led to increased rates of IB in this sample (relative to Study 1). Our rationale for predicting that this would be the case was fairly simple – when participants are still getting used to performing the task, they may be more vigilant for things that are unexpected (as a means of monitoring whether or not they are performing the task correctly). However, it is important to note that this effect is inconsistent with the recent finding that “training” on the tracking task leads to lower rates of IB (Richards et al., 2010). This inconsistency may suggest that the relation between experience with the primary task and IB is complex. For example, this relation may be non-linear, in that more experience initially leads to lower rates of IB (e.g., as participants become comfortable with the task instructions), but beyond a certain point, more experience leads to higher rates of IB (e.g., as participants get bored with the task). Researchers should take this issue into consideration when planning studies to examine individual differences in IB, in order to maximize variance (and in turn, statistical power).

General Discussion

Using two different methods to gauge primary task performance (accuracy on standard speed trials in Study 1 and tracking speed thresholds in Study 2), we found that individual

differences in how well people were able to perform the primary tracking task do not predict IB. These findings replicate previous reports (Simons & Jensen, 2009), and thus lend strong support to the claim that, while task demands do influence rates of IB, individuals' ability to handle these demands do not. Interestingly, we also found that performance on (other) tasks designed to measure aspects of executive functioning did not predict IB. These findings replicate and extend previous reports (Richards et al., 2010), and again support the claim that participants' ability to handle task demands do not predict whether or not they will exhibit IB. Likewise, these findings suggest that individual differences in executive attention do not play a prominent role in explicit attention capture.

Importantly, across multiple samples and measures, we did not find that working memory capacity predicted IB, which is inconsistent with findings recently reported by Richards and colleagues (Hannon & Richards, 2010; Richards et al., 2010). There are a number of possible explanations for this inconsistency. First, Richards and colleagues used samples that included a large age range, whereas both of our samples consisted entirely of college students. Including older participants may be problematic given evidence of cognitive declines associated with aging, including declines in working memory capacity (see Salthouse, 1994) and increases in IB (Graham & Burke, 2011). While Richards and colleagues did show that age did not predict IB in their samples, they did not report whether they examined possible interactions between age and working memory capacity. Another possibility is that the participants tested by Richards and colleagues were particularly good at performing the tracking task, and thus those participants who also had "residual" resources were more likely to notice the unexpected stimulus. In line with this idea, there is some very recent evidence to suggest that working memory capacity only

predicts IB in participants who perform the primary task perfectly (Seegmiller, Watson, & Strayer, in press). On the other hand, there is no particular reason to suspect that the participants tested by Richards and colleagues were particularly skilled at tracking multiple objects, and exploratory analyses in our data (examining whether working memory capacity predicted IB in participants who did particularly well on the tracking task) did not support this speculation. Third, both studies conducted by Richards and colleagues used a single-trial design to measure IB (as did the recent study by Seegmiller and colleagues). When participants are not provided with an opportunity to practice the tracking task, those with lower working memory capacity may need to devote relatively more cognitive resources to the process of understanding the task, rendering them less likely to notice the unexpected stimulus. However, when participants are given more experience with the task, this effect goes away. In line with this idea, Richards et al., (2010) showed that even very limited practice with the tracking task greatly decreases rates of IB. Though they did not find a significant interaction between working memory capacity and task exposure, it is possible that more practice is required for the effect to disappear (plus, this study may have been significantly underpowered to detect such an interaction). Finally, Richards and colleagues used a task in which the unexpected stimulus was physically salient (i.e., a red cross amongst white and black letters), whereas we used a task in which the unexpected stimulus was designed to be equally similar to both the attended and ignored stimuli (i.e., a gray cross amongst white and black letters). Thus, it is possible that working memory capacity only predicts IB when the unexpected stimulus is physically salient (for evidence that colored objects are more likely to be noticed, see Koivisto, Hyona, & Revonsuo, 2004). While

additional research is needed to test these ideas, our findings suggest that if working memory capacity is related to IB, it is likely that this relationship is strongly moderated by other factors.

Interestingly, while performance on working memory and executive tasks did not predict IB, patterns of emotional distress did. Specifically, we found a significant three-way interaction between levels of worry, anxious arousal, and anhedonic depression predicting IB in both of our samples. Importantly, these findings support the general conclusion that variability in IB is not simply a random phenomenon. In fact, these findings raise the possibility that other stable individual differences variables (e.g., personality traits) might predict IB as well, given that anxiety and depression are associated with personality (particularly in those experiencing both, see Krueger et al., 1996). Furthermore, these findings suggest that attention control deficits associated with anxiety and depression extend to the phenomenon of explicit attention capture. In light of evidence that IB has important implication for problems outside of the laboratory (e.g., contributions to automobile accidents; see Strayer & Drews, 2007), these findings may in turn have important implications for understanding how anxiety and depression influence daily functioning. These findings also suggest that it is the *pattern* of symptoms that the individual is experiencing that matters, at least in terms of IB. While we did not initially predict that this would be the case, researchers have argued that the relation between depression and cognitive performance is likely to be moderated by co-occurring symptoms of anxiety, and vice versa (e.g., Levin et al., 2007). Furthermore, there is some evidence showing that interactions between worry, anxious arousal, and anhedonic depression predict cognitive processing and performance (e.g., Engels et al., 2010). Nevertheless, additional research is needed to understand why these variables interact in the manner that they do to predict IB.

In Study 1, we did not collect information about participants' previous knowledge of IB. While this could be problematic, in Study 2 we found that such knowledge did not predict IB. While we suspect that previous knowledge of IB would influence noticing in the same or a highly similar task (for evidence to support this, see Simons, 2010), our findings suggest that the impact of such knowledge does not generalize to unfamiliar tasks. In line with this idea, research on problem-solving has consistently shown that exposure to problem solutions often does not generalize to similar problems, particularly when the problems only share structural features in common and the problem-solver lacks expertise in the problem area (e.g., Gick & Holyoak, 1980; Novick, 1988). Importantly, these findings suggest that there is no need to exclude participants with previous knowledge about IB in research on this topic. Nevertheless, additional research is needed to ensure that correlates of IB do not differ in those with such knowledge, relative to those without.

Of course, the current research has some important limitations. First, since both of our samples consisted entirely of college students, caution needs to be exercised in attempting to generalize our findings. While this sampling strategy does have potential advantages (e.g., less concerns about age related declines in cognitive functioning), our findings warrant replication in more diverse samples (including samples which include more individuals experiencing high levels of emotional distress). Second, we used the same basic paradigm to measure IB in both studies. Thus, future research should examine whether our findings will replicate using other IB paradigms (e.g., Mack & Rock, 1998). Third, we measured symptoms of anxiety and depression using self-report questionnaires, which may be subject to reporting biases. Thus, our findings for anxiety and depression warrant replication using other assessment methods. Finally, since the

data from this research is correlational, we cannot conclude whether any of the observed relations are causal. For example, it is possible that symptoms of distress cause people to be more or less likely to exhibit IB, whereas it is also possible that other factors (e.g., lack of motivation, attention control deficits) account for this link. Additional research using longitudinal and/or experimental designs are needed to explore this.

In conclusion, we found that individual differences in IB were not related to participants' ability to handle task demands (measured using their performance on the primary task at hand, as well as working memory and executive tasks). On the other hand, participants reported levels of anxiety and depression did predict IB, suggesting that individual differences in IB are not simply random. Furthermore, these findings suggest that attention control deficits associated with anxiety and depression may extend to the phenomenon of explicit attention capture, but that the nature of these relations depends upon participants' levels of each.

CHAPTER 5

FUTURE DIRECTIONS

The present research yielded a number of intriguing findings that help shed light on the nature of attentional and executive control deficits associated with dimensions of anxiety and depression. In Chapter 2, we found that working memory capacity was negatively associated with worry and GAD symptoms, and predicted changes in levels of worry over time. In Chapter 3, we found that current depressive symptoms were associated with inhibition deficits, whereas past depressive symptoms were associated with shifting deficits. In Chapter 4, we found that cognitive abilities (e.g., multiple object tracking, working memory capacity) did not predict inattentive blindness, but anxiety and depression did. These findings, when considered in the context of existing research, highlight a number of important directions for future research examining attentional and executive control deficits associated with anxiety and depression.

First, future research should employ a number of the strategies utilized in the present research (e.g., using measures designed to tap specific dimensions of anxiety and depression, using tasks designed to tap specific dimensions of EF) to examine attentional and executive control deficits in larger and more diverse samples, in particular samples which would be expected include more individuals with diagnosable disorders (e.g., treatment seeking samples). If research is able to clearly document specific attentional and executive control deficits, then such findings could be used to inform the revision of diagnostic criteria for future editions of the Diagnostic and Statistical Manual of Mental Disorders. In doing so, it would also be valuable to include more extensive measures of other forms of psychopathology (e.g., substance use disorders, ADHD). This would help rule out alternative accounts for findings, provide stronger

evidence of specificity, and ultimately inform theories about common and unique pathways to different forms of psychopathology.

Future research should also aim to develop and utilize more advanced methods for measuring cognitive deficits. There are a variety of ways this can be achieved. First, researchers can continue to adapt cutting edge tasks developed by cognitive psychologists as a means of exploring individual differences. Nevertheless, in doing so, it is important to explore the psychometric properties of these tasks (e.g., test-retest reliability) to ensure that they are appropriate to use for such purposes. Second, it can be advantageous to employ multiple measures of constructs of interest. In doing so, statistical techniques such as factor analysis and latent variable analysis can be employed to explore the critical abilities that impact task performance, as well as to isolate shared variance (or conversely, remove method variance). Third, traditional (explicit) task performance indices can be supplemented with additional measures of cognitive processing and performance, including implicit measures (e.g., eye tracking) and psychophysiological measures (e.g., neuroimaging). This approach can help circumvent some of the inherent limitations of traditional performance measures, in particular that lack of differences does not necessarily entail equivalent performance (i.e., equifinality), and that equivalent performance deficits can result from a number of different mechanisms (i.e., multifinality). It might also be interesting to explore self-report methods of assessing task performance, in particular as a way to explore individual differences in factors such as strategy use, comprehension of task instructions, and task engagement. In doing so, it would be important to thoroughly examine the validity of this approach by examining convergence with other methods. That said, there is some evidence to suggest that self-report indices of cognitive

processing and performance can be fruitful, including the use of this methodology to assess awareness of unexpected stimuli in IB research (including in the present research) and awareness of mistakes in error monitoring research (e.g., Nieuwenhuis et al., 2001).

While some findings from the present research provide evidence to support our proposal that attentional and executive control deficits play a role in the etiology of anxiety and depression, our findings are by no means sufficient to establish causal relations. Thus, future research should continue to explore causal models of these phenomena. First and foremost, stronger longitudinal designs should be employed to test our predictions and rule out rival hypotheses. Likewise, experimental designs can be used in a creative fashion to examine laboratory analogues of anxiety and depressive disorders. Second, future research should explore possible mechanisms that account for these relations by measuring proposed mechanisms (e.g., difficulty suppressing unwanted thoughts), particularly in the context of longitudinal and experimental designs. Third, research should explore causal antecedents of attentional and executive control deficits (e.g., genes), as well as how these deficits may function in conjunction with other known causal factors. For example, attentional and executive control deficits may serve as ‘endophenotypes’ (see Gottesman & Gould, 2003) which, when combined with other factors (e.g., life stress), confer risk for anxiety and/or depression.

If attentional and executive deficits are found to play a role in the etiology of anxiety and depression, these findings could have important treatment implications that should be investigated in future research. First, if some individuals who meet criteria for anxiety and/or depressive disorders exhibit these deficits but others do not, research could investigate whether these deficits make some clients less likely to respond to traditional interventions (and thus

contribute to the maintenance of these problems). For example, current approaches to the treatment of anxiety disorders are dominated by exposure-based techniques. Based on evidence suggesting that attentional focus during exposure sessions significantly influences long-term habituation (Foa & Kozak, 1986; see Grayson et al., 1982, Johnstone & Page, 2004, and Rodriguez & Craske, 1993), it seems quite plausible that attentional control deficits might compromise one's ability to benefit from exposure. Likewise, cognitive-based interventions have been developed to treat both anxiety and depressive disorders which involve helping clients identify and change maladaptive thoughts and/or beliefs. Again, it seems quite plausible that cognitive deficits could interfere with one's ability to benefit from these interventions. In line with this notion, there is evidence that individuals with more self-reported "cognitive dysfunction" are less likely to benefit from cognitive therapy (Elkin, 1994). If attentional and/or executive control do moderate responses to existing interventions, then assessment of these deficits could prove quite useful for informing treatment decisions.

Perhaps more importantly, findings to support the notion that attentional and executive control deficits play a role in the etiology of anxiety and/or depression would suggest that these deficits may be an appropriate target for interventions. In fact, cognitive remediation strategies are growing in popularity as interventions for chronic mental disorders, including schizophrenia (e.g., Silverstein, 2000) and attention-deficit/hyperactivity disorder (e.g., Klingberg et al., 2002). Interestingly, some of these interventions have targeted deficits similar to those found to be associated with dimensions of anxiety and depression (e.g., working memory deficits; Bell et al., 2003). Thus, similar approaches, either alone or in combination with traditional techniques, might be useful for treating anxiety and depressive disorders – particularly those with chronic

courses for which traditional treatment approaches are less successful (e.g., GAD, recurrent MDD). In addition to traditional cognitive remediation strategies, mindfulness-based interventions may be another intervention that can be used to "remediate" attentional and executive control deficits associated with anxiety and depression. Current conceptualizations of "mindfulness" suggest that mindfulness-based exercises (including, but not limited to, meditation) can help individuals develop the ability to intentionally regulate their attention (see Baer, 2003 and Kabat-Zinn, 1982). Interestingly, there has been growing interest in the use of mindfulness-based interventions to treat chronic forms of anxiety and depression, coupled with evidence to support the efficacy of these interventions (e.g., Miller et al., 1995; Roemer & Orsillo, 2002; Segal et al., 2002). Furthermore, there is evidence that mindfulness training can improve performance on cognitive tasks, including working memory and sustained attention tasks (e.g., Chambers et al., 2008). In short, research should be conducted to evaluate the efficacy of interventions designed to remediate attentional and executive control deficits associated with anxiety and depression, as well as to explore whether improvements in these domains mediate changes resulting from both new and existing interventions.

Finally, future research should examine the potential implications of research on attentional and executive control deficits associated with anxiety and depression for other areas of research. For example, research has shown that anxiety and depression are associated with different types of emotion regulation problems (e.g., Mennin et al., 2007). In line with the common-systems view (discussed in Chapter 1), these emotion regulation problems may arise for attentional and/or executive control deficits. Likewise, findings from this area of research might prove useful in helping us to understand emotional sequelae of problems that have a well-

established impact on cognitive functioning, such as traumatic brain injuries and multiple sclerosis. While it is often assumed that these emotional problems are a natural result of individuals' reactions to changes in their cognitive abilities (e.g., people with MS become depressed because a perceived loss of cognitive function; see Arnett et al., 1999), it is possible that these deficits lead to emotional problems for other reasons (e.g., they cause deficits in emotion regulation). Finally, findings from this area of research can and should be used to inform theories and research on basic cognitive phenomena. For example, our finding that symptoms of anxiety and depression predict inattention blindness could help shed new light on the nature of this phenomenon. In short, insights from research on attentional and executive control deficits associated with anxiety and depression could be used to generate novel hypotheses about topics such as the origins of emotional regulation deficits, the emotional consequences of cognitive disorders, and the mechanisms of explicit attention capture.

TABLES

Table 1. Descriptive statistics and correlations between the worry and depression measures.

		M	SD	Min	Max	1	2	3	4	5	6
1.	PSWQ	49.9	13.7	18	80	--					
2.	WDQ-SF	23.1	8.7	10	47	.54	--				
3.	MASQ-AD	55.3	14.9	26	90	.47	.64	--			
4.	GAD Sx count	3.2	4.4	0	17	.70	.49	.37	--		
5.	MDD Sx count	2.0	4.5	0	18	.29	.32	.34	.49	--	
6.	FU WDQ, SF	22.3	7.8	10	40	.45	.68	.44	.30	.11	--
7.	FU MASQ-AD	56.2	14.2	27	85	.38	.47	.57	.15	-.05	.71

Table 2. Hierarchical regression analyses for the follow-up data.

DV = Time 2 WDQ-SF	Beta	ΔR^2
Step 1		
Time 1 PSWQ	.03	
Time 1 WDQ	.66**	.46
Step 2		
Time 1 WM composite	-.29*	.08

DV = MASQ-AD, time 2	Beta	ΔR^2
Step 1		
Time 1 MASQ-AD	.57**	.32
Step 2		
Time 1 WM composite	-.13	.02

NOTE: * = $p > .05$, ** = $p > .01$

Table 3. Descriptive statistics and correlations between the measures.

	M	SD	Min	Max	1	2	3	4
1. Current MDE Sx	1.7	4.3	0	18	--			
2. Current GAD Sx	2.9	4.3	0	17	.48	--		
3. State PA	2.9	0.6	1.7	5.0	-.09	-.15	--	
4. State NA	1.5	0.4	1.0	3.7	.37	.24	-.18	--
5. Past MDE Sx	5.7	6.7	0	20	.10	.18	.07	-.05

	M	SD	Min	Max	1	2	3	4
1. Stop-signal RT	270.4	40.7	186.0	442.4	--			
2. Mean RT, no-signal trials	613.2	151.3	376.1	1013.9	-.17	--		
3. PM switch cost	15.9	11.6	-5.1	56.9	.11	.09	--	
4. PM errors	2.2	2.9	0	20	.07	.09	.11	--
5. Finger tapping	45.0	6.2	29.5	58.75	-.10	-.01	-.17	-.02

Table 4. Correlations between symptoms/mood and performance on the cognitive tasks.

	Stop-Signal RT	Mean RT on No-signal trials	Plus-minus switch cost	Plus-minus task errors	Finger tapping
Current MDE Sx	.23**	.04	.00	.22**	.02
Current GAD Sx	.19*	-.05	.01	.12	-.08
State PA	-.20*	.12	.01	.03	.19*
State NA	.15 ^t	-.09	-.06	.19*	.06
Past MDE Sx	-.03	.07	.18*	.00	-.05

NOTE: $t = p > .10$, $* = p > .05$, $** = p > .01$

Table 5. Descriptive statistics for the self-report questionnaires.

	Mean	SD	Min	Max
<i>Study 1</i>				
Worry	49.9	13.2	19	80
Anxious Arousal	25.3	7.6	17	69
Anhedonic Depression	52.9	14.9	28	98
<i>Study 2</i>				
Worry	49.6	13.7	18	80
Anxious Arousal	26.7	8.2	16	67
Anhedonic Depression	55.4	15.0	26	90

Table 6. Means (and standard errors) for participants who did and did not notice the unexpected stimulus, along with statistics from the logistic regression analyses.

	Did not notice	Noticed	Wald Statistic	P-value
<i>Study 1</i>				
Tracking accuracy	.62 (.05)	.59 (.04)	.226	.63
WM capacity	.39 (.32)	.00 (.20)	1.076	.30
<i>Study 2</i>				
MOT tracking threshold	137.1 (2.0)	136.9 (2.9)	.000	.99
WM capacity – N-back tasks	-.09 (.16)	.08 (.28)	.087	.77
WM capacity - OSPAN	43.7 (1.4)	39.7 (2.6)	2.116	.15
Response inhibition	269.6 (3.9)	273.0 (5.6)	.218	.64
Set-switching	14.7 (1.1)	16.6 (1.4)	.89	.35

Table 7. Emotional distress predicting IB.

	B (SE)	P-value
<i>Study 1</i>		
Worry	-.27 (.25)	.28
AA	.67 (.33)	.04
AD	.10 (.29)	.74
Worry x AA	-.70 (.35)	.05
Worry x AD	-.01 (.32)	.97
AA x AD	-.16 (.28)	.58
Worry x AA x AD	-.95 (.44)	.03
<i>Study 2</i>		
Worry	.15 (.20)	.47
AA	.04 (.21)	.85
AD	-.06 (.19)	.77
Worry x AA	-.31 (.19)	.10
Worry x AD	.07 (.17)	.66
AA x AD	-.06 (.22)	.78
Worry x AA x AD	-.40 (.21)	.05

FIGURES

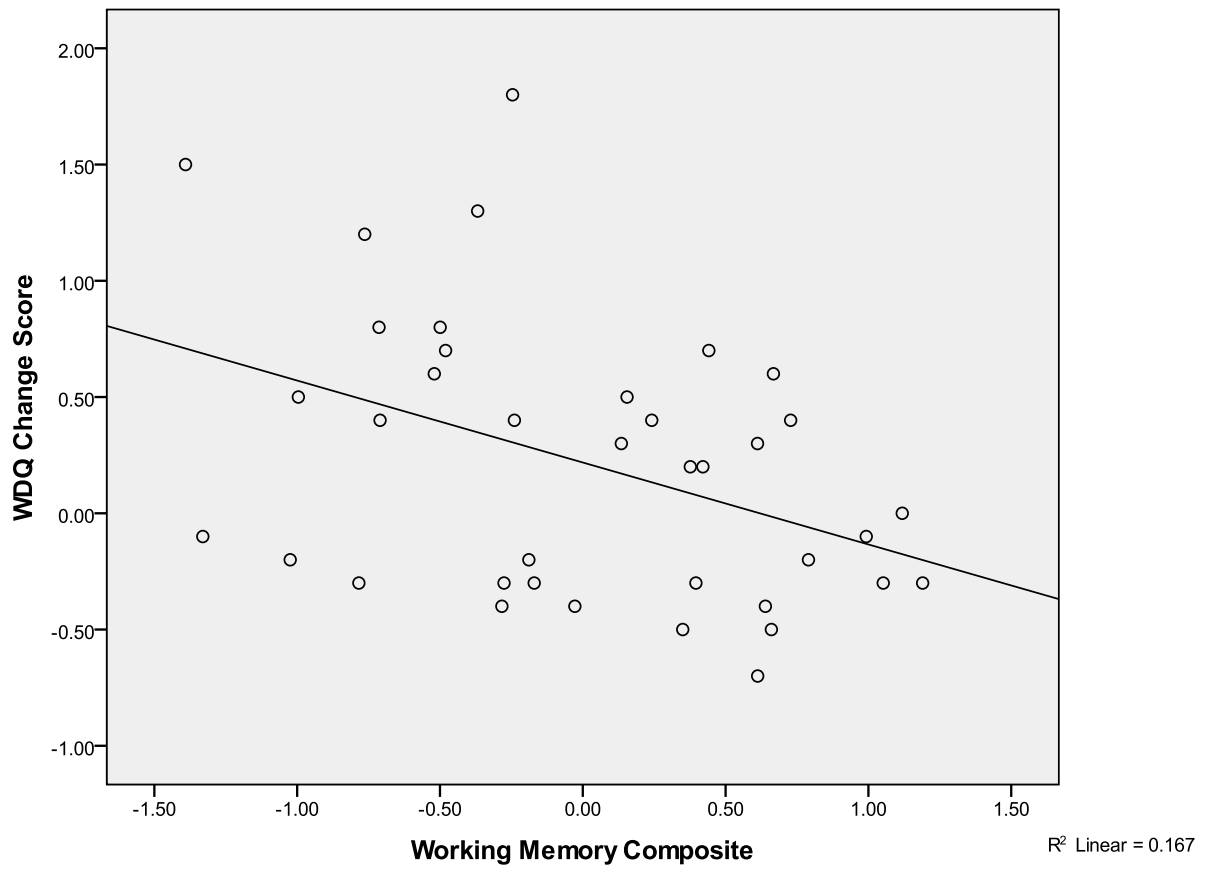
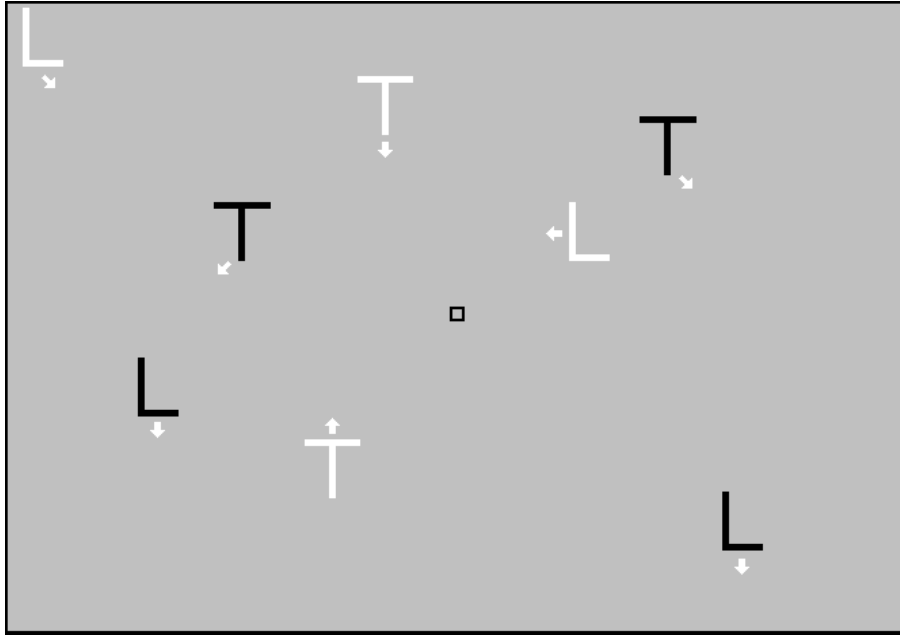


Figure 1. Scatterplot showing the association between working memory composite scores and changes in levels of worry from Time 1 to Time 2.

a)



b)

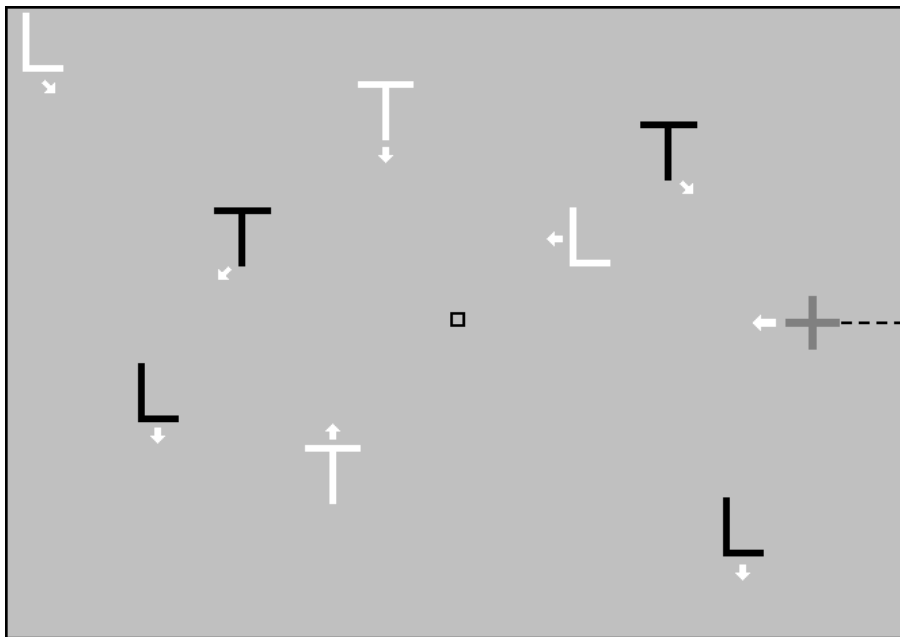


Figure 2. Schematics of the non-critical (multiple object tracking) trials (a) and the critical (inattentional blindness) trial (b).

a)

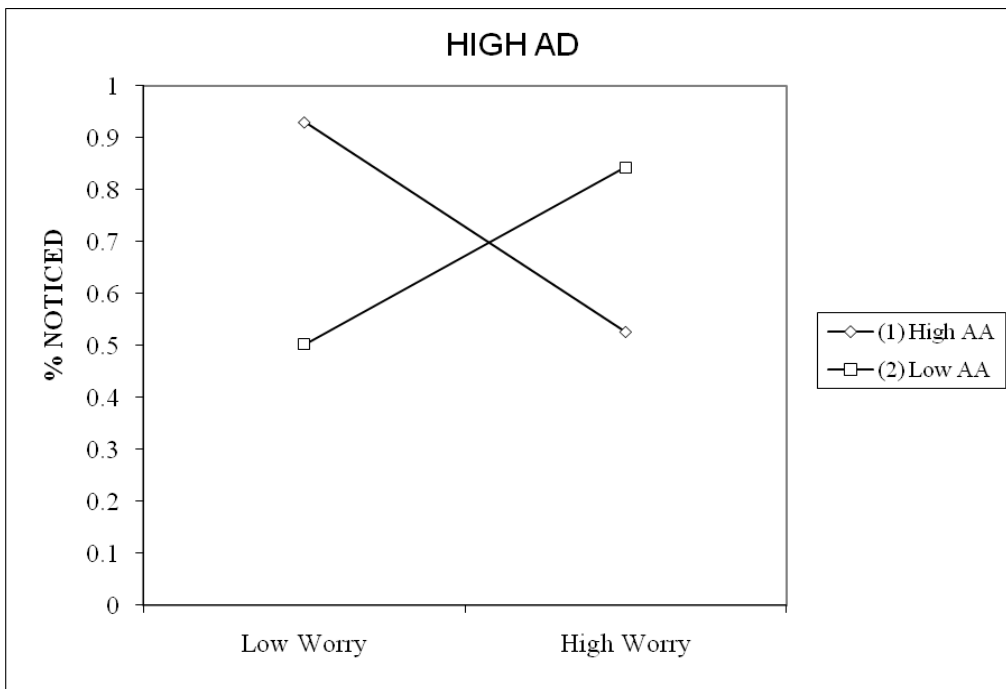
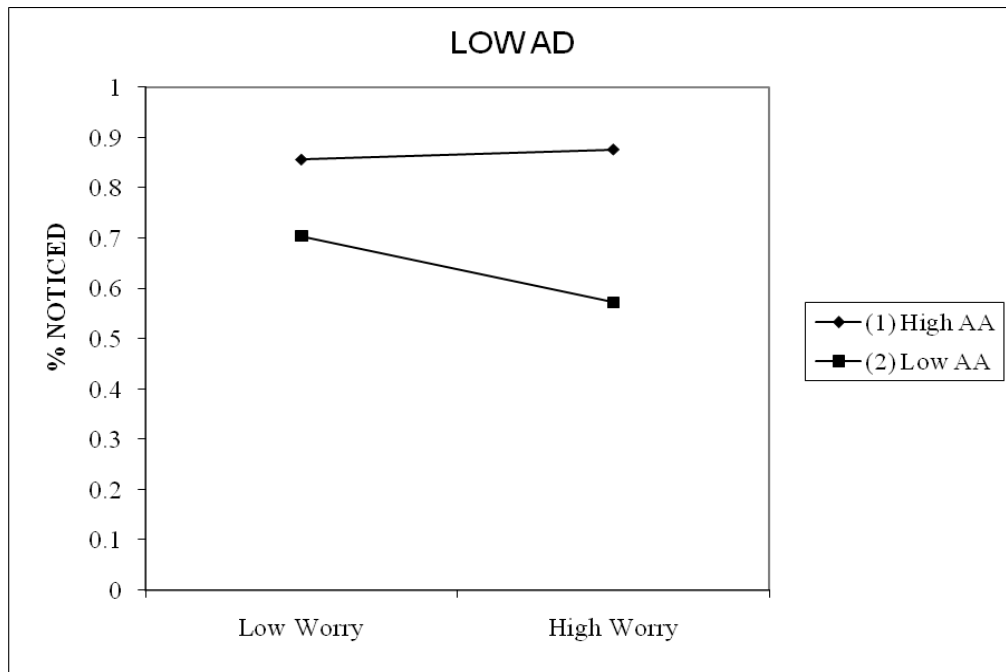


Figure 3. Interactions between worry, anxious arousal, and anhedonic depression predicting IB from Study 1 (a) and Study 2 (b).

b)

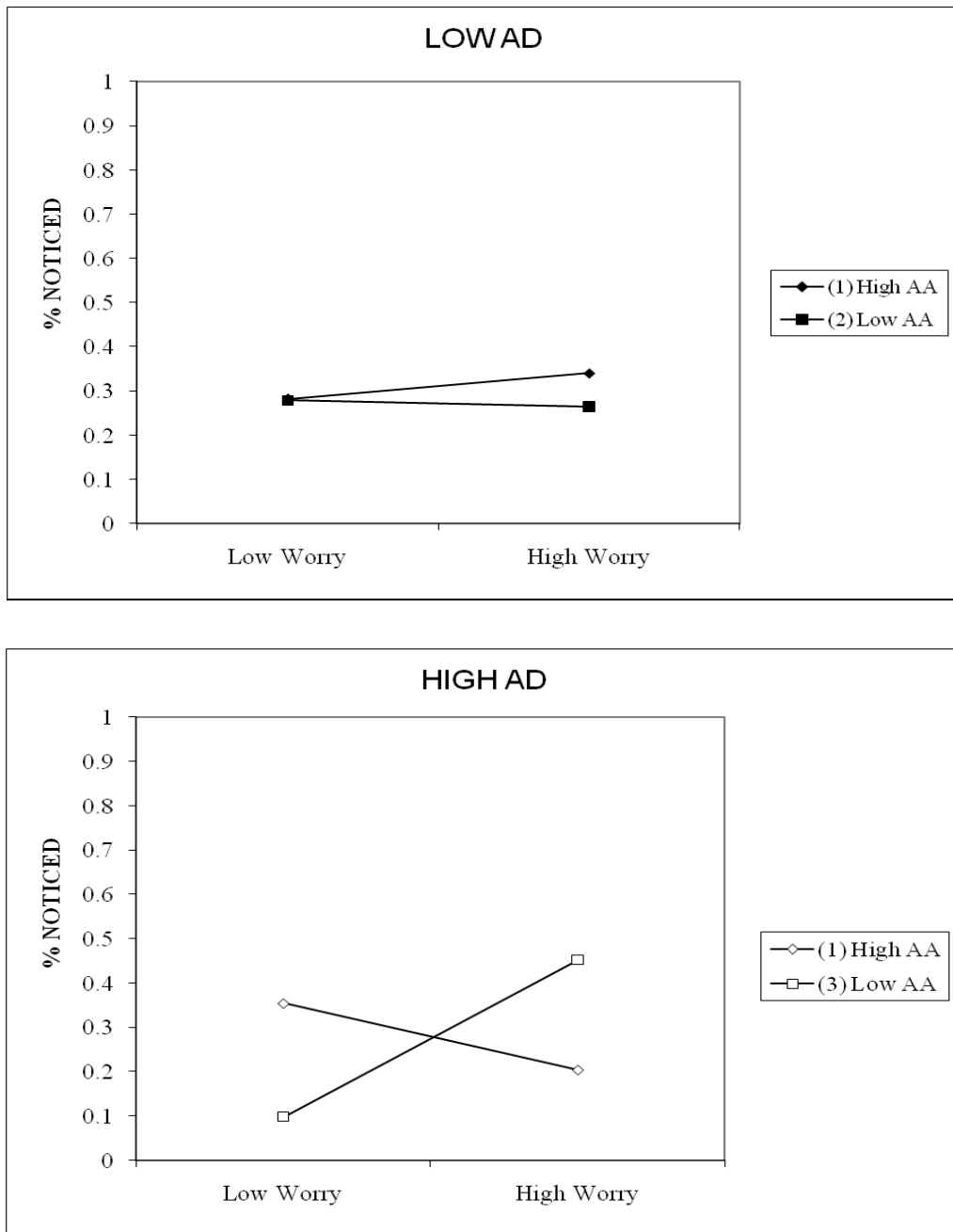


Figure 3 (cont.).

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