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Cognitive and Neural Correlates of Coping and Resilience in Depression

Catherine Lee
Loyola University Chicago

LOYOLA UNIVERSITY CHICAGO

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CATHERINE LEE

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ABSTRACT

Depression is one of the most prevalent and devastating psychological disorders, often with a chronic or remitting/reoccurring course. The inability to effectively cope with stress and negative life events has been strongly linked to the development and maintenance of depression symptoms; yet, the cognitive and biological processes underlying the complex and multidimensional behavioral construct of coping are not well understood. Using a combination of self-report measures, computerized cognitive tasks, and scalp electroencephalography (EEG) methodologies, the present study investigated associations between specific executive function abilities (i.e., inhibition and set-shifting), underlying neural activity, coping strategy and flexibility, and depression symptoms. Results did not support the primary study hypotheses predicting coping to mediate the relation between executive dysfunction and depression symptoms. Post-hoc correlational analyses elucidated relations between various components of coping strategy and depression symptomatology, and further demonstrated associations with frontocentral N200/P300 and parietal P300 peak latencies.

CHAPTER ONE

INTRODUCTION

Depression is a pervasive disorder that is chronic and debilitating. It is associated with negative outcomes in various domains, including performance in academics and employment (Kessler et al., 2006; Salmela-Aro, Aunola, & Nurmi, 2008), interpersonal relationships (Kessler, Walters, & Forthofer, 1998), and overall quality of life (Rapaport, Clary, Fayyad, & Endicott, 2005). The national cost of depression (in medical expenses and lost productivity) has been estimated to be $83 billion per year (Greenberg et al., 2003). In order to improve treatment strategies to target potential factors that predict depression, a better understanding of the mechanisms through which these symptoms arise and persist is necessary.

Previous research has demonstrated a strong association between the ability to cope with life stress and the development or exacerbation of psychopathology, including depression (Blalock & Joiner, 2000; Penley, Tomaka, & Wiebe, 2000; Seiffge-Krenke & Klessinger, 2000). Individual differences in coping strategy selection may play an important role in the onset and maintenance of depression. Furthermore, coping *flexibility*, the extent that individuals are able to adjust or adopt different methods of coping across different situations, has been associated with lower levels of depression (Gan, Zhang, Wang, Wang, & Shen, 2006). Research has shown that an inflexible coping style, particularly rigid avoidance, is a strong predictor of the development and

exacerbation of depression symptoms, as well as resistance to treatment (Cronkite, Moos, Twohey, Cohen, & Swindle, 1998; Klein, 2008). However, minimal research has been conducted into the cognitive or biological mechanisms associated with individual differences in the ability to effectively cope with stress.

The present study seeks to investigate potential cognitive and neural correlates of adaptive and maladaptive coping strategies in response to stress, and how these constructs relate to depression symptoms. It aims to extend the large body of literature demonstrating executive function deficits in depression. Executive dysfunction is hypothesized to contribute to ineffective coping strategies, which are theorized to predispose individuals to the development of depression symptoms. Furthermore, deficits in coping ability are predicted to be associated with abnormalities in the frontocingulate brain regions, as these brain networks have been shown to substantiate differences in executive function abilities in individuals with depression (Silton et al., 2011). Electroencephalography (EEG) data was collected while participants complete tasks that evaluate executive functions (i.e., top-down inhibitory control and set-shifting). Depression symptoms and coping behaviors were measured using self-report inventories. A structured clinical interview was administered to assess for depression and anxiety disorders.

This study extends previous work on coping patterns in depression in several notable ways. Specifically, it proposes a novel transactional model for the cognitive and neural mechanisms associated with coping strategy and flexibility, particularly as they relate to depression symptoms. This research will help inform evidenced-based treatment

strategies that uses cognitive training methods to enhance executive function skills in individuals with depression.

CHAPTER TWO

REVIEW OF THE RELEVANT LITERATURE

Background

Major Depressive Disorder (MDD) is a debilitating, widespread and pervasive psychological disorder. It has been associated with life impairment in various domains, including interpersonal relationship difficulties, decreased productivity and performance in academic or work settings, and decreased physical wellbeing (Druss et al., 2009). The yearly cost of depression (in lost productivity and increased medical expenses) is $83 billion per year in the United States (Greenberg et al., 2003). Additionally, suicide is the tenth leading cause of death in the United States (CDC, 2010) and the third leading cause of death in 15- to 24-year-olds (Drum, Brownson, Burton, & Smith, 2009; NIMH, 2008; Schwartz, 2006). More than 90 percent of individuals who die by suicide have depression or other psychological disorders (Moscicki, 2001).

The significant cost associated with depression is likely related to its recurrent nature. That is, 85% of individuals who recover from a Major Depressive Episode (MDE) will experience another episode (Keller & Boland, 1998). Additionally, each successive depressive episode increases the likelihood of recurrence (Boland & Keller, 2009; Kessing, Hansen, Andersen, & Angst, 2004; Mueller et al., 1999). Given the evidence that relapse rates remain very high despite existing pharmacological and psychological treatments (Gotlib, Kurtzman, & Blehar, 1997; Kessler, Chiu, Demler, & Walters, 2005),

recent efforts have been directed to potential factors that predict the onset and relapse of depression. Additional research is needed to investigate the mechanisms and risk factors related to development, maintenance, and recurrence of depression. Advancing understanding regarding the neurobiological processes through which these symptoms arise and persist, will contribute to improving the effectiveness of diagnostic, prevention, and treatment strategies. To this end, the present study explores the relations between cognitive dysfunction, relevant neural correlates implicated in depression, and the ability to cope effectively with stress.

Coping with Stress

A considerable amount of research in contemporary psychology has focused on *resilience* in the face of stress, or variables that buffer individuals against negative outcomes. Within the clinical psychology literature, the primary emphasis of resilience research has been on *coping*, particularly the interacting effect of coping strategies on the association between stressful life events and mental health outcomes (Aldwin & Revenson, 1987; Clarke, 2006; Penley et al., 2002). Coping has been broadly conceptualized as an individual's "constantly changing cognitive and behavioral efforts to manage specific external and internal demands that are appraised as taxing or exceeding the resources of the person" (Lazarus & Folkman, 1984; p. 141). Dysfunctional coping represents an inability to effectively manage these demands, and is considered to be a risk factor for the development of psychopathology. Consistent with this theory, maladaptive coping strategies have been shown to predict negative psychosocial outcomes, including occupational and interpersonal difficulties (e.g., Lussier, Sabourin, & Turgeon, 1997; Montero-Marin, Prado-Abril, Demarzo, Gascon, &

Garcia-Campayo, 2014; Windle & Windle, 1996), decreased physical health and life satisfaction (e.g., Pritchard, Wilson, & Yamnitz, 2007), and an increase in depression symptoms (e.g., Blalock & Joiner, 2000; Beasley, Thompson, & Davidson, 2003; Holahan, Moos, Holahan, Brennan, & Schutte, 2005).

Coping strategy. Coping is a multidimensional construct that can be conceptualized, classified, and operationalized in numerous ways. A particularly important distinction is between engagement (or approach) coping, wherein the intent is to deal directly with the stressor(s) or related emotions, and disengagement (or avoidance) coping, which aims to escape the stressor or related emotions (Carver & Connor-Smith, 2010; Cronkite & Moos, 1995; Moos & Schaefer, 1993; Skinner, Edge, Altman, & Sherwood, 2003). Engagement coping includes problem-solving strategies that actively address the stressor, as well as emotion-focused coping strategies that seek to manage the related distress. Some examples of these are active support seeking, cognitive restructuring, emotion regulation, and acceptance. Disengagement coping, on the other end of the spectrum, includes strategies such as denial, avoidance, and fantasy. Disengagement coping is often emotion-focused, in that it frequently involves efforts to escape or avoid distress; however, this should be distinguished from emotion-focused *engagement* coping strategies, which seek to address the negative emotions more actively (Carver, Scheier, & Weintraub, 1989; Lazarus & Folkman, 1984).

Disengagement coping is generally ineffective in reducing stress over time, as it ignores the stressor's existence and its eventual consequences. Emotion-focused disengagement coping strategies can decrease distress levels in the short-term, by allowing individuals to disregard the stressor altogether. However, in many situations, the

longer an individual avoids dealing with a stressor, the more difficult the problem becomes to manage and the less time they have to address the issue when they finally decide to confront it. Another potential outcome of disengagement coping is that avoidance and denial can promote a paradoxical increase in intrusive thoughts about the stressor and an increase in negative affect and anxiety (Najmi & Wegner, 2008). Moreover, some behavioral disengagement coping strategies, such as "retail therapy" or substance abuse, may directly result in negative health and/or financial outcomes (Carver & Connor-Smith, 2010). In this way, avoidance coping strategies may have beneficial effects in the short term in that there is a temporary decrease in stress, but may be associated with negative outcomes over the long-term (Nolen-Hoeksema & Morrow, 1993; Robbins & Tanck, 1992).

Research has shown that avoidant or disengagement coping strategies typically result in negative psychological outcomes (Blalock & Joiner, 2000). In particular, employing disengagement strategies to cope with stressors may contribute to, or exacerbate, depression symptoms (Beasley et al., 2003; Steinhardt & Dolbier, 2008). This association has been supported by longitudinal data, demonstrating a significant stable relation between disengagement coping and depression symptoms across time (Seiffge-Krenke & Klessinger, 2000). Specifically, individuals who employed any type of avoidant coping strategy were more likely to report depression symptoms at the end of a four-year period, regardless of whether the disengagement strategies were consistently used. Disengagement coping has been found to predict higher levels of both chronic and acute life stressors (Holahan et al., 2005). Thus, as it serves to maintain and/or intensify levels of stress, disengagement coping is associated with the maintenance and/or

exacerbation of depression symptoms over time. Studies have also shown that depressed individuals who employ rigid disengagement coping strategies are at a much higher risk for partial remission (i.e., with ongoing subthreshold symptoms) and subsequent relapse, and non-remission (Cronkite et al., 1998; Klein, 2008; Nolan-Hoeksema, 2000).

Coping flexibility. Coping flexibility refers not only to the way individuals vary their coping strategies across situations but also to whether such flexible strategy deployment is situation appropriate. Most research on coping flexibility has focused on variability in coping patterns, which is considered more adaptive, in that it often results in positive outcomes, such as decreased anxiety, stress, and depression (Lester, Smart, & Baum, 1994; Mattlin, Wethington, & Kessler, 1990). Within this literature, there have been several proposed approaches to define and assess coping flexibility.

Some researchers have focused on the *repertoire approach*, which focuses on the range of coping strategies available to an individual (Carver et al., 1993; Roussi, Krikeli, Hatzidimitriou, & Koutri, 2007). Others have examined the *variation approach*, defining coping flexibility as the ability to alter a coping strategy across time and situations (Compas, Forsythe, & Wagner, 1988). Still others utilize the *fitness approach*, which involves selecting coping strategies dependent upon changes in the appraisal of a stressor. Cognitive appraisals of the controllability of stressful events may influence an individual's choice of coping strategies (Folkman, Lazarus, Dunkel-Schetter, DeLongis, & Gruen, 1986). Individuals who consistently perceive stressful events as controllable may use more engagement coping strategies. In contrast, individuals who consistently perceive stressful events as uncontrollable may use more disengagement coping strategies (David & Suls, 1999). This group may include those who feel pessimistic, helpless, or

depressed (Aldwin, Sutton, & Lachman, 1996). Alternatively, individuals who are cognitively flexible may vary their behavior according to the perceived nature of different stressful events. However, these approaches and definitions of coping flexibility have been difficult to operationalize and measure, which has resulted in inconsistent findings (Kato, 2012).

The present study defines coping flexibility as the ability to discontinue an ineffective coping strategy and utilize an alternative strategy. This conceptualization presumes an evaluation component (i.e., the ability to assess a situation and gain awareness that one's coping strategy is not effective) and an adaptive component (i.e., the ability to abandon disadvantageous coping behaviors and then create and utilize alternative strategies; Kato, 2012). This operationalization of coping flexibility also incorporates common aspects of other approaches. For example, it presumes the ability to utilize different coping strategies across time and situations, as well as the ability to appraise the situation-coping fit.

Despite the importance of the ability to effectively cope with stress, very little research has investigated the associated cognitive and physiological underpinnings. As with all complex behaviors, the act of coping relies on more fundamental cognitive processes. The current study proposes that effectively coping with stress and/or negative events requires adequate self-regulatory abilities, namely executive function processes such as inhibitory control and set-shifting.

The Role of Executive Functions in Coping and Depression

Executive functions (EFs) generally refer to a group of cognitive processes responsible for the control and regulation of goal-directed and future-oriented behavior

(Alvarez & Emory, 2006). They include processes such as inhibitory control, set-shifting, planning, and working memory (Pennington and Ozonoff, 1996). These cognitive functions are critical for carrying out daily tasks, and also provide the foundation for more complex functions, such as multi-tasking, problem solving, and strategic learning. The present study will focus on the processes of inhibitory control and set-shifting, as these EFs represent the self-regulatory abilities associated with coping, and have been consistently shown to be compromised in individuals with depression (Joormann & Gotlib, 2010; Langenecker et al., 2007; De Lissnyder, Koster, Derakshan, & De Raedt, 2012; Silton et al., 2011).

Inhibitory control. Inhibitory control processes underlie the ability to modulate automated behaviors or dominant responses (i.e., impulse control). They begin to develop at the end of infancy and contribute to the effortful control of behavior, such as delayed gratification and attending to and following instructions (Kochanska, Murray, & Harlan, 2000). Inhibitory control also contributes to emotion regulation skills, specifically the ability to inhibit maladaptive negative emotions and thoughts (Zelazo & Cunningham, 2007; Joormann & Gotlib, 2010).

Previous research has identified early self-regulation abilities to be a robust predictor of adaptive development and well-being. They are typically associated with positive psychosocial outcomes, including academic performance, self-competence, physical health, and lower levels of internalizing symptomatology such as depression and anxiety (Anzman-Frasca, Francis, & Birch, 2015). Dysfunction in these abilities is thought to result in deficient emotional and behavioral functioning (e.g., Anand et al., 2005; Davidson et al., 2002; Rogers et al., 2004; Watkins & Brown, 2002). For example,

impairment in inhibitory control has been associated with the process of rumination, in that the prolonged processing of negative information (as seen in depression and anxiety disorders) is due, at least in part, to the inability to disengage from the thought process (Koster, Lissnyder, Derakshan, & De Raedt, 2011). Ruminative processes have been shown to enhance negative thinking, impair problem-solving efforts, and consistently predict the onset of depression (Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008).

The present study proposes that inhibitory control provides the foundation for adaptive coping strategies. For example, in order to problem-solve or otherwise actively cope with a stressor, individuals must be able to effectively inhibit task-irrelevant information (e.g., maladaptive negative emotions, ruminative thoughts) and/or a dominant response (e.g., the impulse to avoid the stressor). Impairments in inhibitory control may lead to rumination and overwhelming negative affect following a stressful life event (Joormann & Gotlib, 2010; Nolen-Hoeksema, Morrow, & Fredrickson, 1993), hindering the ability to cope actively with the stress or stressor, resulting in increased reliance on disengagement coping strategies and the development or exacerbation of depression symptoms.

Set-shifting. At any given moment, individuals are confronted with an abundance of sensory information that require selective attention. In order to effectively select and process task-relevant information, it is necessary to strategically adapt cognitive systems according to contextual demands (Hawkins et al., 1990; Shiffrin, 1998; Wager, Jonides, & Smith, 2006). However, contextual demands are dynamic and constantly changing, requiring the perpetual reallocation of cognitive resources to certain environmental

stimuli. This process of selectively budgeting and redistributing one's attention is referred to as *set-shifting* (Jersild, 1927; Miyake et al., 2000).

This mechanism is closely related to the concept of coping flexibility, which involves adapting one's coping strategy in response to different stressors or previous unsuccessful attempts at coping. That is, coping flexibility likely depends on the cognitive capacity for efficient set-shifting. In order to cope flexibly (i.e., adapt coping strategies to best address the stressor or related distress), individuals must be able to evaluate their strategy's effectiveness and adopt a new strategy if the first is not successful. To do this, individuals must be able to efficiently shift their attention from one task, stimulus, or thought to another.

Increased levels of cognitive, behavioral, and affective flexibility have been associated with more creative and persistent efforts at problem-solving (Isen & Daubman, 1984), as well as enhanced adaptation to stress and negative life events (Fresco, Williams, & Nugent, 2006; Rosenbaum & Ben-Ari, 1985). Individuals who demonstrate the tendency to approach stressful situations contextually and flexibly are more likely to find adaptive solutions than individuals who tend to respond with a narrow range of coping behaviors. A growing body of research indicates that greater cognitive flexibility and improved creative problem solving is associated with positive affect across a broad range of settings (Isen, 1999). This study proposes that deficits in the capacity for set-shifting will predict decreased coping flexibility.

Executive dysfunction in depression. Over the past few decades, studies have demonstrated a strong association between depression and executive dysfunction (e.g., Austin, Mitchell, & Goodwin, 2001; Biringer et al., 2005; Channon & Green, 1999;

Harvey et al., 2004; Levin et al., 2007; Lyche et al., 2010). One theory is that depression reduces already limited cognitive resources by recruiting them to process negative (often ruminative, task-irrelevant) thoughts and emotions (Joormann & Gotlib, 2010), suggesting that EF deficits are epiphenomena of the disorder. For example, individuals with depression demonstrate an attentional bias to negative stimuli, with subsequent difficulty disengaging from the stimuli (Koster, De Raedt, Goeleven, Franck, & Crombez, 2005). Thus, in order to actively cope with a stressor, these individuals may need to exert extra effort to override a mood- or disorder-induced prepotent bias toward ruminating about negative material, the resulting negative emotions, and the impulse to avoid the stressor.

Alternatively, it has been proposed that a "failure of regulatory systems" (i.e., executive dysfunction) plays a primary role in the development, exacerbation, and persistence of depression symptoms (Langenecker et al., 2007). These regulatory mechanisms have been shown to affect not only attention and information processing, but also cognitive-emotional integration and emotion regulation. For example, individuals with poorer inhibition or set-shifting abilities may be more prone to ruminative patterns (Letkiewicz et al., 2014; Davis & Nolen-Hoeksema, 2000), leading to increased negative emotions when faced with stress or a negative life event (De Lissnyder et al., 2012).

More recently, twin studies have provided insight into the contribution of genetics to executive dysfunction. Hsu and colleagues (2013) investigated monozygotic and dyzogotic twins discordant for depression and found that neuropsychological impairments are more consistent with pre-existing deficits than sequelae of the disorder. Additional studies have found evidence of attentional control dysfunction in families that

are at increased risk for depression (Belleau, Phillips, Birmaher, Axelson, & Ladouceur, 2013; Christensen, Kyvik, & Kessing, 2006), again suggesting that these deficits are hereditary and exist prior to onset of depression.

Together, these studies indicate that executive dysfunction is a vulnerability factor that predisposes individuals to developing depression symptoms and also plays a role in symptom maintenance and reoccurrence, contributing to the disorder's chronic or remitting/reoccurring nature. The current study tests the hypotheses that poor inhibitory control and set-shifting abilities are associated with difficulties adequately coping with stress and negative life events, causing them to be at particular risk for developing depression symptoms.

Brain Circuitry Related to Inhibitory Control and Set-Shifting

Research has shown abnormal activity in prefrontal cortex (PFC), inferior frontal cortex (IFC), anterior cingulate cortex (ACC), and parietal cortex (PC) in individuals with depression (e.g., Davidson & Henriques, 2000; Heller & Nitschke, 1997; Levin et al., 2007; Mayberg et al., 1997; Pizzagalli, Peccoralo, Davidson, & Cohen, 2006; Rogers, Bradshaw, Pantelis, & Phillips, 1998; Rogers et al., 2004; Silton et al., 2011). Consistent with the cognitive sequelae associated with depression symptoms, these brain regions are known to support neural networks that maintain aspects of executive functions (e.g., Cole & Schneider, 2007; Collette et al., 2005; Fassbender et al., 2004; Miller & Cohen, 2001; Rogers et al., 2004; Shackman et al., 2006; Silton et al., 2010; Wagner, Maril, Bjork, & Schacter, 2001). Through their role in regulating top-down attentional control processes, it follows that these networks also support the mechanisms involved in adaptive coping. Thus, the present study explores the hypotheses that abnormalities in these brain circuits

will be associated with decreased coping flexibility and increased use of disengagement coping strategies.The present study used scalp electroencephalography (EEG) to measure the neural correlates of inhibition and set-shifting. This method involves examining event-related potentials (ERP) that are evoked in response to stimuli and provide excellent temporal resolution for examining the time course of neural activity.

Frontocentral N200/P300 complex. Top-down inhibitory control processes recruit frontocingulate brain networks, including PFC and ACC to regulate a dominant response in favor of a competing response (Krawczyk et al., 2008; MacDonald, Cohen, Stenger, & Carter, 2000; Silton et al., 2010). The N200 ERP component is a negative peak at approximately 180-325 milliseconds after stimulus presentation. Research suggests that this component has neural generators in areas of the frontal cortex, specifically the orbitofrontal frontal cortex (Gehring & Willoughby, 2002; Potts, Martin, Burton, & Montague, 2006; Yeung, Botvinick, & Cohen, 2004).

The N200 is enhanced during tasks requiring monitoring of conflicting information and response options, and it is thought to signal the effort required to employ attentional control (Nieuwenhuis, Yeung, van den Wildenberg, & Ridderinkhof, 2003; van Veen & Carter, 2002). The frontocentral P300a ERP component is a positive peak at approximately 250-500 milliseconds after stimulus presentation (e.g., Bruder, Kayser, & Tenke, 2012; Polich, 2007; Polich, 2010), and neural generators in the ACC and motor/premotor cortices have been identified (Pires, Leitão, Guerrini, & Simões, 2014). Its amplitude is sensitive to the amount of attentional resources engaged during cognitive tasks (Pires et al., 2014), particularly with regard to tasks that require motor inhibition (Enriquez-Geppert, Konrad, Pantev, & Huster, 2010).

When individuals are asked to inhibit a dominant response, various midfrontal ERPs are observed, including N200 and P300 components (Badzakova-Trajkov Barnett, Waldie, & Kirk, 2009; Kiefer, Marzinzik, Weisbrod, Scherg, & Spitzer, 1998). The N200/P300 complex is considered to reflect brain activity associated with inhibitory control (Enriquez-Geppert et al., 2010; Falkenstein, Hoormann, & Hohnsbein, 1999). The complex is also observed during tasks that require emotional control, indicating the recruitment of top-down attentional control to inhibit task-irrelevant negative emotion. That is, when individuals attempt to modulate their emotions, they recruit resources from frontal networks, and the N200/P300 can represent the amount of cognitive effort applied (Chiu, Holmes, & Pizzagalli, 2008; Stewart, Coan, Towers, & Allen, 2011).

Individuals with depression have been shown to display frontocingulate abnormalities when asked to process conflicting information (Silton et al., 2011), and this seems to be particularly true when the conflict is emotionally laden (De Lissnyder et al., 2010). Research has demonstrated that individuals with depression require greater neural activation in frontal, limbic, and temporal regions to perform as well as non-depressed controls on inhibition tasks (Langenecker et al., 2007). The greater prefrontal activation may reflect a "cortical inefficiency" (Wagner et al., 2006), such that individuals with depression may need to recruit additional resources to successfully complete inhibition tasks (Silton et al., 2011).

The present research examined the frontocentral N200/P300 during a response inhibition and implicit emotion regulation task. Larger mean amplitude of the N200/P300 complex is thought to represent the recruitment of more cognitive resources. The present study tests the hypothesis that individuals who demonstrate increased neural activity

during this task of inhibitory control will report greater utilization of disengagement strategies, as they likely have more difficulty coping actively with stressors. Additionally, larger N200/P300 amplitude and greater reliance upon disengagement coping strategies are hypothesized to be associated with more depression symptoms.

Parietal P300. As an index of cognitive control, set-shifting is presumed to be substantiated by PFC (Brass, Ullsperger, Knoesche, Von Cramon, & Phillips, 2005; Nakahara, Hayashi, Konishi, & Miyashita, 2002; Ravizza & Ciranni, 2002). However, research has found mixed results regarding a one-to-one relationship between shifting and PFC activity (Alvarez & Emory, 2006). The lesion literature has shown that patients with frontal focal lesions demonstrate impairments on a common set-shifting task; however, studies have also documented similar impairments in patients with posterior lesions (Anderson, Damasio, Jones, & Tranel, 1991; Barceló & Santome-Calleja, 2000). Similarly, in neuroimaging research, set-shifting tasks have been found to elicit increased activity in both frontal and parietal regions (Brass et al., 2005; Collette et al., 2005). Furthermore, a commonly reported ERP associated with set-shifting is observed as a late positivity over the parietal scalp (Barceló, 2003; Karayanidis, Coltheart, Michie, & Murphy, 2003; Kieffaber & Hetrick, 2005; Nicholson, Karayanidis, Poboka, Heathcote, & Michie, 2005; Rushworth, Passingham, & Nobre, 2002; Swainson et al., 2003, 2006).

This can be explained by the dual components of the P300 ERP. The P300 is comprised of two distinct components, commonly referred to as P300a and P300b. P300a, introduced in the previous section, is considered to reflect frontal attention or orienting mechanisms (Knight, 1996; Luck, 2012). P300b has a peak latency ranging from 300-500 ms and is maximal over midline parietal scalp sites (Bruder et al., 2012). It is thought to

reflect temporal-parietal brain activity associated with context updating and memory processing (Polich, 2007).

It has been proposed that during set-shifting tasks, stimulus evaluation engages focal attention (P300a) to facilitate context updating (P300b), which is associated with memory operations (Hartikainen & Knight, 2003; Kok, 2001; Polich, 2003). These mechanisms are critical to coping flexibility, as individuals must constantly incorporate and utilize new information, in order to respond adaptively. An individual lacking the ability to shift his or her attention to a novel situation or update his or her mental representation with new information cannot respond flexibly in a given situation.

Given that individuals with depression demonstrate relative deficits in the fundamental cognitive operations of attention allocation and working memory required in set-shifting, this population likely has abnormalities in the associated brain regions (Polich, 2010). Furthermore, as the literature demonstrates that rigidity in coping style predicts negative psychological outcomes (Cronkite et al., 1998; Klein, 2008; Nolen-Hoeksema, 2000), the present study tests the hypothesis that set-shifting deficits and associated brain circuit dysfunction play a critical role in predicting less coping flexibility and more depression symptoms.

Previous research supports this hypothesis, as the most commonly reported ERP finding in depression is a reduction of P300 amplitude (e.g., Ancy, Gangadhar, & Janakiramaiah, 1996; Kawasaki, Tanaka, Wang, Hokama, & Hiramatsu, 2004; Röschke & Wagner, 2003; Urretavizcaya et al., 2003; Yanai, Fujikawa, Osada, Yamawaki, & Touhouda, 1997). However, the vast majority of this research has focused on the parietal P300 in an auditory oddball task (Bruder et al., 2012). Studies have also shown a

hereditary component of brain circuit abnormalities. Individuals with a family history of depression, who are at increased risk for developing depression symptoms, show smaller P300b amplitudes over temporal-parietal regions compared to controls (Zhang, Hauser, Conty, Emrich, & Dietrich, 2007). Thus, reduced amplitude of the parietal P300 may be considered a trait marker of vulnerability to depression (Bruder et al., 2012). This present project proposes to investigate the P300b as a measure of set-shifting involving parietal resources.

Research Overview: Model and Hypotheses

The primary goal of the present study was to examine how executive function and associated neural correlates are related to maladaptive coping strategies that predict the exacerbation of depression symptoms. The study tested the following four mediation models investigating proposed associations between inhibitory control, utilization of disengagement coping strategies, and depression symptoms (Figure 1), and set-shifting abilities, coping flexibility, and depression symptoms (Figure 2):

Model 1. This model proposes that utilization of disengagement coping strategies mediates the association between deficits in inhibitory control and depression symptoms, wherein difficulties with response inhibition are measured by task performance.

Hypothesis 1a. Difficulties with response inhibition are associated with higher levels of depression symptoms.

Hypothesis 1b. Difficulties with response inhibition are associated with higher levels of self-reported use of disengagement coping strategies.

Hypothesis 1c. Higher levels of self-reported use of disengagement coping strategies are associated with higher levels of depression symptoms.

Hypothesis 1d. Difficulties with response inhibition are associated with higher levels of depression symptoms, at least in part, due to their association with increased utilization of disengagement strategies.

Model 2. This model proposes that utilization of disengagement coping strategies mediates the association between deficits in inhibitory control and depression symptoms, wherein difficulties with response inhibition are measured by neural indicators of cognitive effort. The study examined the mean amplitude of the frontocentral N200/P300 ERP during "stop" trials of the Stop-Signal Task as an indicator of the cognitive effort required for response inhibition.

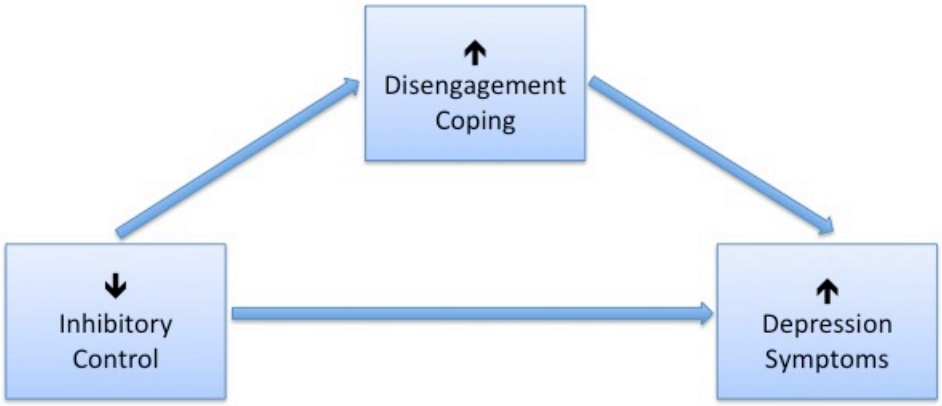
Hypothesis 2a. Greater frontocentral N200/P300 amplitude during a response inhibition task is associated with higher levels of depression symptoms.

Hypothesis 2b. Greater N200/P300 amplitude during a response inhibition task is associated with higher levels of reported disengagement coping utilization.

Hypothesis 2c. Higher levels of self-reported utilization of disengagement coping strategies are associated with higher levels of depression symptoms.

Hypothesis 2d. More cognitive effort required for inhibitory control (as indicated by greater N200/P300 amplitude during response inhibition) is associated with higher levels of depression symptoms, at least in part, due to its association with increased utilization of disengagement strategies.

Figure 1. Conceptual diagram: Utilization of disengagement coping strategies mediating the relation between inhibitory control and depression symptoms



Model 3. This model proposes that coping flexibility mediates the association between set-shifting abilities and depression symptoms, wherein set-shifting is measured by task performance.

Hypothesis 3a. Set-shifting deficits (poor task performance) are associated with higher levels of depression symptoms.

Hypothesis 3b. Set-shifting deficits (poor task performance) are associated with less self-reported coping flexibility.

Hypothesis 3c. Less coping flexibility is associated with higher levels of depression symptoms.

Hypothesis 3d. Difficulties with set-shifting (as indicated by poor task performance) are associated with higher levels of depression symptoms, at least in part, due to their association with lower levels of coping flexibility.

Model 4. This model proposes that coping flexibility mediates the association between deficits in set-shifting ability and depression symptoms, wherein difficulties

with set-shifting are measured by neural indicators of cognitive resource recruitment. The study examined the mean amplitude of the grand average parietal P300 ERP during correct "switch" trials of the Color-Word Stroop as an indicator of the cognitive resources recruited for set-shifting.

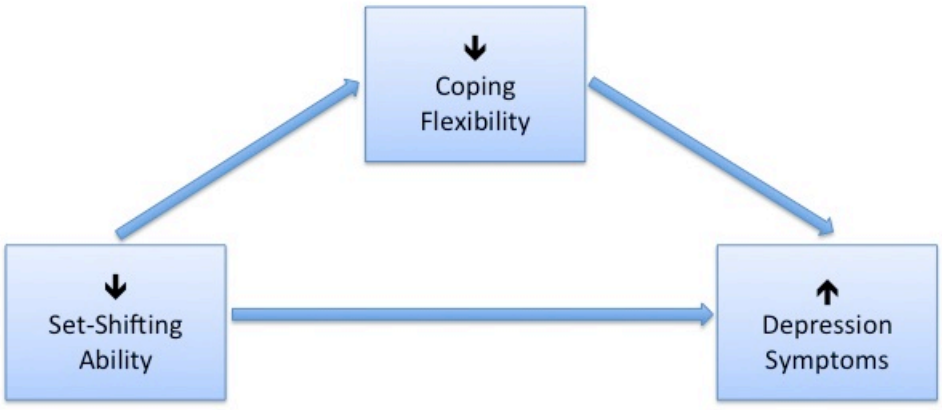
Hypothesis 4a. Decreased neural activity in the parietal lobe (as measured by P300b amplitude) during a set-shifting task is associated with higher levels of depression symptoms.

Hypothesis 4b. Lower P300b amplitude during a set-shifting task is with lower levels of coping flexibility.

Hypothesis 4c. Lower levels of coping flexibility are associated with higher levels of depression symptoms.

Hypothesis 4d. Difficulties with set-shifting (as indicated by decreased P300b amplitude) are associated with higher levels of depression symptoms, at least in part, due to their association with decreased coping flexibility.

Figure 2. Conceptual diagram: Coping flexibility mediating the relation between set-shifting ability and depression symptoms



CHAPTER THREE

METHOD

Study Overview

The present research study used a combination of experimental and self-report measures. After participants were deemed eligible for inclusion, they were invited for three laboratory visits, during which they completed informed consent, a semi-structured clinical interview, EEG and behavioral tasks, and self-report measures.

Participants

Participants in this study were native English-speaking undergraduate students from an urban, midwestern university. They were recruited from a university-based participant registry maintained by the Cognitive and Affective Neuroscience Lab. The study employed a high-risk recruitment paradigm. Participants were screened for the proposed study using the following criteria (available via participant registry): 1) current depression symptoms as evidenced by a Patient Health Questionnaire (PHQ-9; Kroenke, Spitzer, & Williams, 2001) score of greater than 10; 2) healthy control participants with no depression symptoms as defined by a PHQ-9 score of less than 5. These cutoffs were determined from validity work conducted on the PHQ-9 in primary care settings (Kroenke et al., 2001). This recruitment strategy has been shown to ensure a representative range (normal distribution) of depression symptom severity (Silton et al., 2011). Particularly within a high functioning population such as undergraduate students,

it is necessary to specifically recruit individuals who score high on depression measures to avoid a skewed distribution and to ensure that individuals with depression are included in the sample.

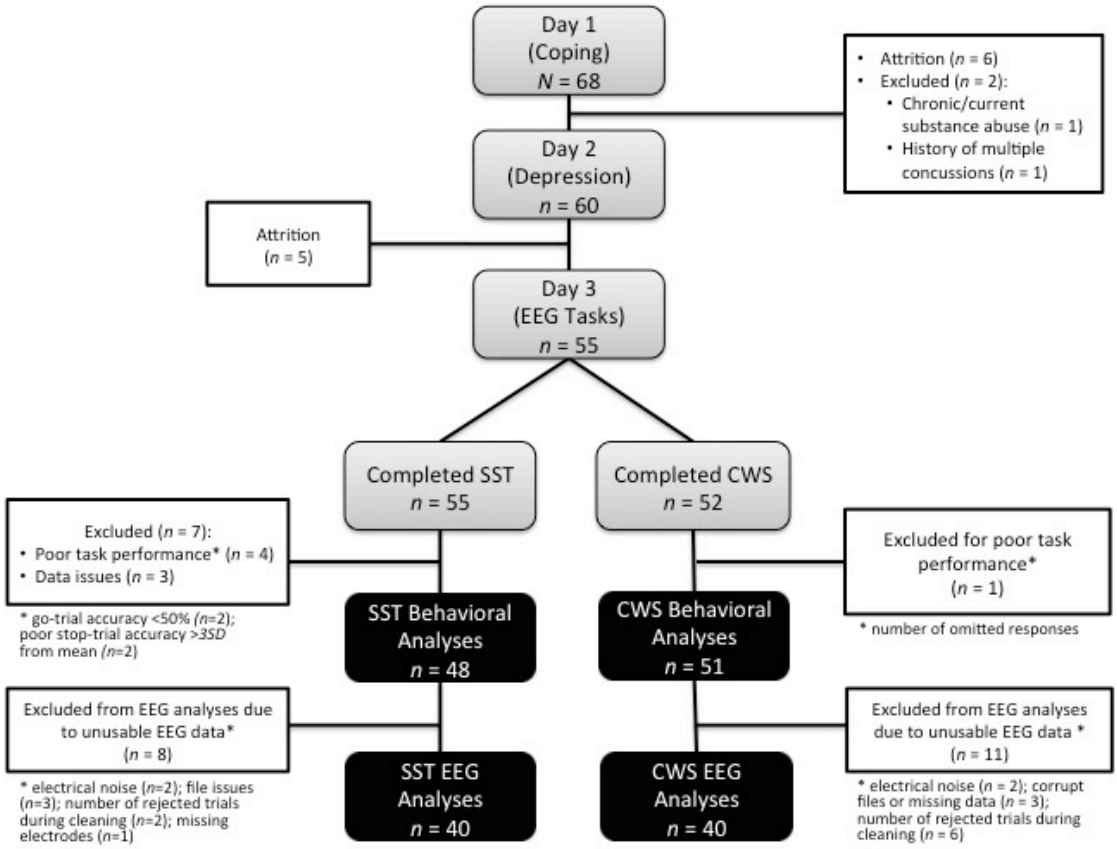
Participants were excluded from the study if they met the following criteria, which are known to alter EEG recording: 1) colorblindness, 2) known neurological medical condition, 3) a visual, hearing, voice, or motor impairment that would prevent completion of study procedures, 4) regular substance use, and 5) left handedness. Participants reporting past and/or current psychiatric treatment (medication or psychotherapy) were included in the study.

Sixty-eight undergraduate students were paid $15 per hour for their participation in the study. Fifty-five participants completed all three laboratory visits (age M = 19.65, SD = 1.32, range = 18-24; 67.3% female; 61.8% White, 27.3% Asian American, 3.6% African American, 3.6% Hispanic/Latino, 3.6% Other). These participants did not differ significantly from those who did not complete the study in age (M = 19.54, t(66) = -.30, p = .77), gender ($\chi^2(1) = 0.83$, p = .36), year in school ($\chi^2(4) = 2.47$, p = .65), reported cumulative GPA (t(65) = -1.35, p = .18), psychiatric treatment history ($\chi^2(1) = .018$, p = .89), or current psychiatric treatment ($\chi^2(1) = 0.12$, p = .73). However, study completers, compared to noncompleters, were more likely to be White ($\chi^2(6) = 12.86$, p = .045).

Seven participants were eliminated from further analysis for the Stop-Signal Task due to poor task performance (i.e., <50% accuracy on go-trials or abnormally low accuracy (>3 SD from the mean) on stop-trials); eight were additionally excluded from EEG analyses for the Stop-Signal Task due to unusable data (e.g., number of rejected trials during cleaning, electrical noise). One participant was excluded from Color-Word

Stroop analyses due to poor task performance (i.e., high frequency of non-responding), and 11 were additionally excluded due to unusable data. The final sample included 48 participants for the Stop-Signal Task (40 with EEG data) and 51 for the Color-Word Stroop (40 with EEG data). The university Institutional Review Board approved all recruiting and experimental methods.

Figure 3. Flow of participants through each stage of the study



Procedure

The current study consisted of self-report questionnaires, a clinical interview, and computer tasks with EEG monitoring, completed over three laboratory visits. All

questionnaire and demographic (e.g., age, gender, year in school, handedness, ethnicity) data were collected online via SurveyMonkey, administered on an Apple iPad® in the lab.

Demographics. Participants provided information regarding their age, gender, year in school, handedness, and ethnicity on surveys they are asked to complete.

Depression symptoms. Participants completed the Anhedonic Depression subscale of the Mood and Anxiety Symptom Questionnaire (MASQ-AD; Watson & Clark, 1991). The MASQ-AD asks individuals to indicate how frequently they have experienced a variety of symptoms during the past week (e.g., "Felt like nothing was very enjoyable," "Felt really slowed down"). It is comprised of 22 items and demonstrates psychometric properties similar to the full Mood and Anxiety Symptom Questionnaire (MASQ; Clark & Watson, 1991), including a strong factor structure, good convergent and discriminant validity, and good internal consistency (i.e., Cronbach's alpha in the mid .80s; Casillas & Clark, 2000). The MASQ-AD has demonstrated acceptable reliability and validity both within clinical and non-clinical samples, and is highly correlated with the PHQ-9 (Bredemeier et al., 2010). In the current sample, internal consistency for the 22-item Anhedonic Depression subscale was $\alpha = .95$.

Coping strategy. The Coping Response Inventory-Adult Form (CRI; Moos, 1993) was used to measure the utilization of various coping styles. The CRI is a 48-item self-report measure that assesses an individual's cognitive appraisal and coping strategies in response to a "specific, recent stressful life event." Participants indicated the extent to which they utilize each coping strategy on a 4-point Likert scale from 1 (*not at all*) to 4 (*fairly often*).

This measure classifies coping strategies into four domains, each of which has two 6-item subscales. *Cognitive approach* coping includes Logical Analysis (e.g., "Did you think of different ways to deal with the problem?") and Positive Reappraisal (e.g., "Did you try to see the good side of the situation?"). *Behavioral approach* coping includes Support Seeking (e.g., "Did you seek help from persons or groups with the same type of problem?") and Problem Solving (e.g., Did you seek help from persons or groups with the same type of problem?"). *Cognitive avoidance* coping includes Cognitive Avoidance (e.g., "Did you try to put off thinking about the situation, even though you knew you would have to at some point?") and Resigned Acceptance ("Did you accept it; nothing could be done?"). *Behavioral avoidance* coping includes Seeking Alternate Rewards (e.g., "Did you turn to work or other activities to help you manage things?") and Emotional Discharge (e.g., "Did you take it out on other people when you felt angry or depressed?"). The CRI has good reliability, stability, and convergent and predictive validity (Holahan & Moos, 1991; Holahan, Moos, Holahan, & Cronkite, 1999). Disengagement coping will be assessed by combining the *cognitive avoidance* and *behavioral avoidance* domains. The internal consistency for the disengagement (avoidance) coping and engagement (approach) coping items in the present sample were $\alpha = .80$ and $\alpha = .89$, respectively; the internal consistency of the encompassed domains ranged from $\alpha = .72$ to $\alpha = .85$.

Coping flexibility. The Coping Flexibility Scale (CFS; Kato, 2012) is a 10-item, self-report measure of coping flexibility. Participants will be instructed to indicate the extent to which they endorsed statements of coping awareness and adaptation (e.g., "If I feel that I have failed to cope with stress, I change the way in which I deal with stress"

and "When stressed, I use several ways to cope and make the situation better") on a 4-point Likert scale from 1 (*not applicable*) to 4 (*very applicable*). The CFS consists of two 5-item subscales: an Evaluation Coping subscale and an Adaptive Coping subscale. Higher scores on eight of the items indicate higher levels of coping flexibility, while higher scores on the other two (items 2 and 7) indicate lower levels of coping flexibility. These two items will be reverse-scored so that, in the overall analyses, higher scores indicate higher levels of coping flexibility. The internal consistency for the CFS in the present sample was $\alpha = .77$.

Response inhibition in an emotional context. The *Stop-Signal Task* (Sagaspe, Schwartz, & Vuilleumier, 2011) is an implicit emotion regulation task that involves using top-down attentional control to inhibit an automatic response in an emotional context. During the task, emotional faces (sad or happy expressions) were presented. Participants were asked to identify the gender of the faces, disregarding the emotional content. A striped box flashed momentarily around the stimuli for certain trials (Figure 4B), and this cued the participant to withhold or inhibit their response (per instructions). Individuals with deficits in inhibitory control will likely require more cognitive effort to inhibit a response; these individuals will either be more likely to respond (contrary to instructions), which will be coded as an incorrect response, or they will exhibit neural activity indicating greater cognitive effort in their attempt to correctly inhibit a response.

Stimuli. Participants were shown a series of grayscale photographs of male and female human faces from the Karolinska Directed Emotional Faces (KDEF) dataset (Lundqvist, Flykt, & Öhman, 1998). The KDEF database consists of 490 (JPEG images) displaying faces with emotional expressions (angry, fearful, disgusted, sad, happy,

surprised, and neutral) from 70 individuals (35 men and 35 women) at five different angles. All individuals were between 20 and 30 years of age and were excluded if they displayed beards, mustaches, earrings, eyeglasses, and visible make-up. All faces are facing forward, with the hairline removed, so that the emotional content is more distinct. The final images were selected using ratings from Goeleven, De Raedt, Leyman, and Verschuere (2008). Half of the faces had happy expressions, while the other half had sad expressions. The selected happy and sad faces have been matched on levels of arousal (Goeleven et al., 2008). They have been normed to ensure easy identification of gender, and that happy faces do not differ from sad faces in level of difficulty.

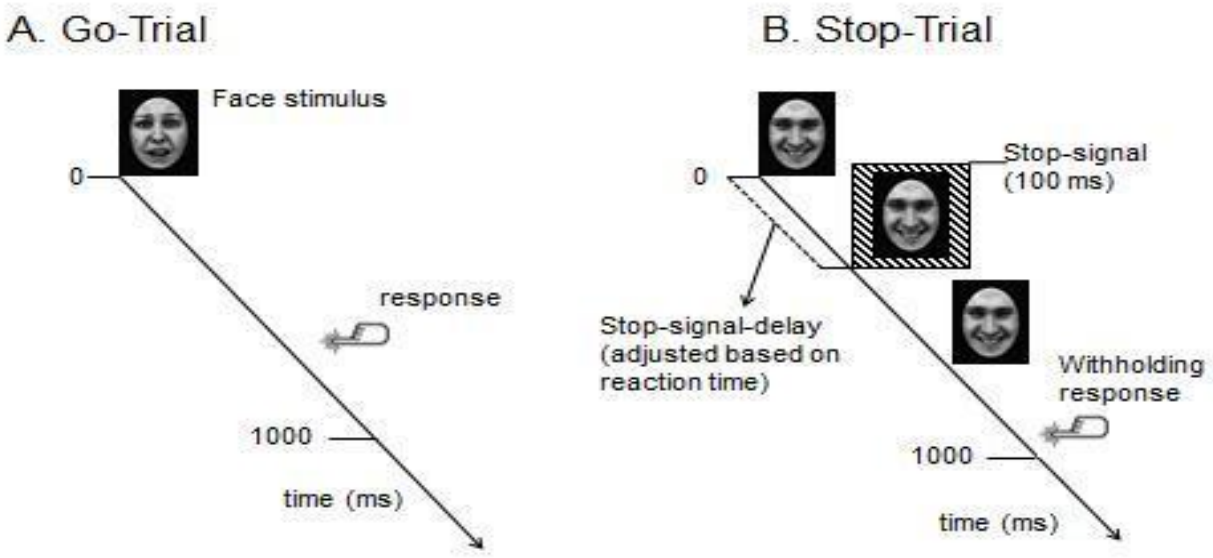
Task design. Participants were asked to identify the gender of each face (male or female) by pressing one of two buttons on an electronic response box as quickly and accurately as possible. On half of the trials, a striped rectangular mask surrounded the photo for 100 to 300 milliseconds after the face was presented. This mask was the "stop-signal," and on these trials, the participant was instructed to withhold their button press. Thus, half of trials were "go" trials and the other half were "stop" trials, randomly intermixed within each block of trials. "Go" trials began with a fixation screen, followed by a face presented for 1 second while participants responded.

Participants were able to respond any time after stimulus onset, for up to 1 second. A black screen followed stimulus presentation. In order to adjust the task to correct for test-retest biases, the lag of the stop-signal varied according to the participant's ability to inhibit their response during previous "stop" trials. The stop-signal delay was adjusted based on the participant's behavior using the following rule: if a participant was successful in withholding their responses on the two previous "stop"

trials, the next "stop" trial had a 20 ms longer stop delay (making the task more challenging). The better the participant performed at withholding their responses, the longer the delay became, and vice versa (i.e., becoming shorter with incorrect responses), varying according to participant performance. In lieu of response reaction time, given that a correct response is achieved by withholding a response, the average stop-signal delay was calculated to represent response inhibition task performance.

Each condition ("stop" and "go") included equal presentations of the two facial expressions (happy and sad). The task consisted of 60 trials per block with twelve blocks, for a total of 720 trials (360 male faces and 360 female faces). Each block was followed by a 20-second break. Stop-signal tasks have been shown to produce a reliable N200/P300 ERP (Verbruggen & Logan, 2008).

Figure 4. Stop-Signal Task trials, displaying one Go (A) and one Stop (B) trial



Set-shifting. To assess set-shifting abilities, participants completed a computerized *Color-Word Stroop* task, based upon the Delis-Kaplan Executive Function System (D-KEFS) Color-Word Interference task (Delis, Kaplan, & Kramer, 2001). During this task, color words (i.e., "green," "yellow," "red," and "blue") were presented to participants on a computer screen, one at a time, in colored (green, yellow, red, or blue) text. Each visual stimulus had two facets to consider: a word stimulus and a color stimulus, and participants were asked to either read the word or name the color of the text. Certain conditions paired conflicting (incongruent) stimuli (e.g., the word "green" printed in red). One segment of the task required participants to switch back and forth between identifying ink color and word reading.

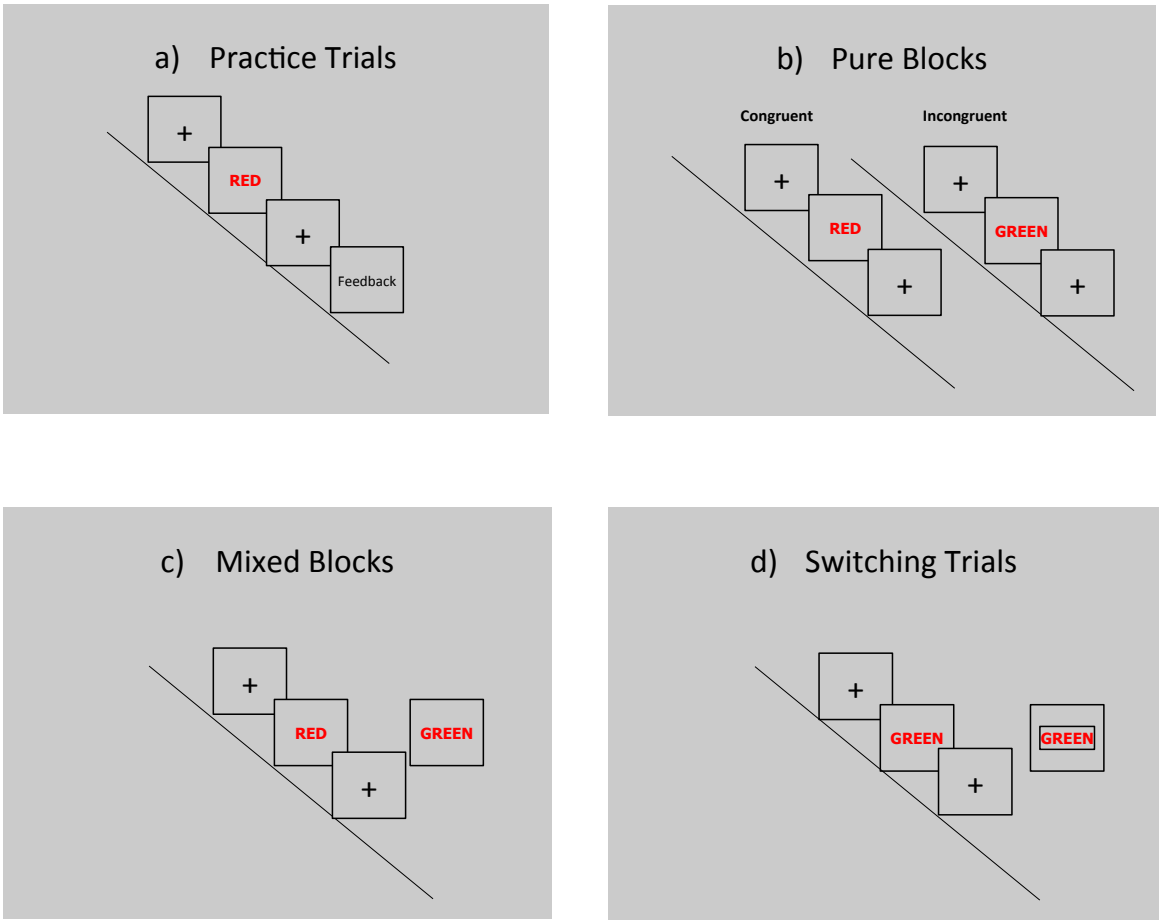
Task design. Stimulus presentation times were based upon Stroop tasks found in studies by Compton and colleagues (2011) and Lansbergen & Kenemans (2008). Each trial will begin with a fixation cross in the center of the screen, the presentation of which will vary between 500-1100 ms to minimize potential habituation to the stimuli presentation format. The stimulus will then be presented for 150 ms, followed by a blank screen, during which participants will have 1500 ms to respond before the next trial. On each trial, participants will indicate their response (blue, green, red, yellow) via a button press. The four response options are mapped onto a Cedrus RB-830 response box (http://www.cedrus.com/responsepads/rb830.htm), which has color-coded keys. First, participants will complete a practice set of 16 color identification trials with accuracy feedback. Sixteen practice trials with accuracy feedback will also precede each of the main trial blocks; twenty-four practice trials will precede the switching trials. Feedback will not be included in the main blocks of trials.

Word reading: Participants will complete two blocks of a word reading task; one block will consist of 96 congruent trials, and the other will consist of 96 incongruent trials. There will be a 10-second break in between blocks.

Color naming: Participants will complete six blocks of color identification. This will consist of 96 congruent trials, a 10-second break, and 96 incongruent trials. This is followed by a 90 second break and then four blocks of mixed trials; each block will consist of 48 trials, with a 10-second break in between each block. The order of pure congruent and pure incongruent blocks will be counterbalanced, to control for potential order effects.

Switching: Participants completed four blocks of 48 trials; color- and word identification trials will be randomized within each block, participants will be required to switch between cognitive tasks (i.e., read the word when there is a black box around the stimulus, and identify the color when the stimulus is presented without the box). There will be a 10-second break in between each block. To obtain an index of general set shifting ability, performance on shift trials (i.e., from color identification to word reading, and vice versa) will be compared with performance on repeat trials (i.e., color identification trial to another color identification trial, word reading trial to another word reading trial); the behavioral "switch cost" will be the increase in response time on "switch" trials compared to "non-switch" trials (Wylie & Allport, 2000). Set-shifting tasks such as this version of the Stroop have been shown to produce reliable P300 ERPs during the switch (Barcelo, Munoz-Cespedes, Pozo, & Rubia, 2000; Rushworth et al., 2002; Swainson et al., 2003).

Figure 5. Color-Word Stroop task trials



(a) Participants completed practice trials with accuracy feedback (i.e., correct, incorrect) prior to each task. (b) For both word-reading (read the words on the screen, ignoring the color of the print) and color-identification (identify the color of the text, ignoring the word that is printed) tasks, there will be blocks of pure congruent and pure incongruent trials. Congruent trials are those in which the word and color stimuli match (i.e., the word RED in red lettering). Incongruent trials are those in which the word and color stimuli do not match (i.e., the word GREEN in red lettering). (c) Mixed blocks included both congruent and incongruent trials randomized within each block. (d) Participants were instructed to identify the color of the text, *unless* they saw a box around the word, in which case they were instructed to read the word.

EEG measurement. Scalp EEG was measured while participants completed the Color-Word Stroop and Stop-Signal tasks. Participants were seated in a comfortable chair, approximately 100 cm from a 21-inch CRT monitor in a quiet room. They were

monitored by a task administrator in a nearby room and received task instructions by intercom. EEG data was recorded using a Biosemi Active2 EEG system. Custom-designed Falk Minow 64-channel cap with equidistantly spaced BioSemi active Ag and AgCl electrodes were used for data collection. CMS/DRL were placed near the vertex. Two electrodes were located on the mastoid bones. After placement of the electrode cap, electrode positions were digitized for later topographic and source localization analyses. An additional electrode was placed on the inferior edge of the orbit of each eye to monitor vertical eye movements; nearby electrodes in the cap (lateral to each eye) monitored horizontal eye movements. Data were collected with without applied filters at a sampling rate of 512 Hz.

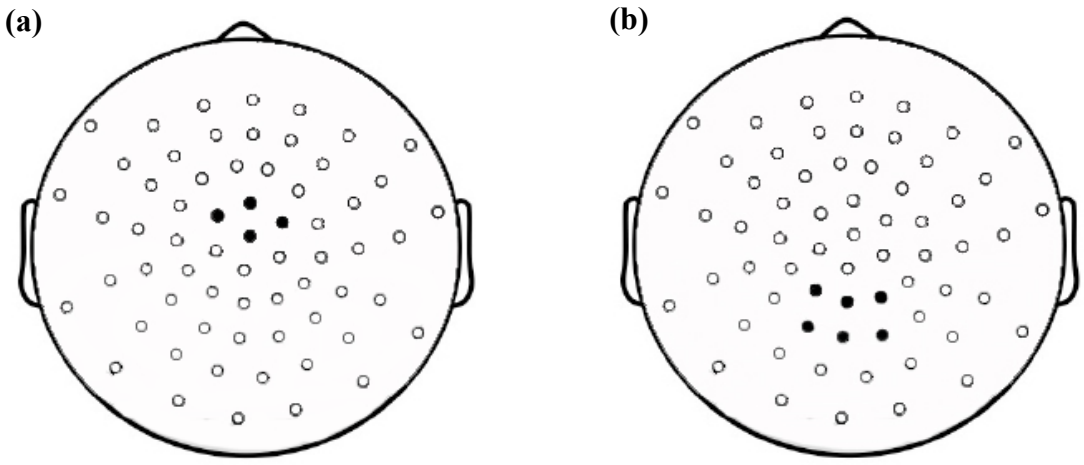
EEG data reduction. The following EEG data processing steps were implemented in Brain Electrical Source Analysis software (BESA Version 6.0; Berg & Scherg, 1994). EEG data were re-referenced to a common average reference and digitally filtered with a half-power amplifier bandpass at 0.01–100 Hz, with a cutoff attenuation of 12 dB/octave. Muscle (e.g., eye blink, eye movement) and other artifact were removed and/or corrected via visual inspection of the raw EEG signal and a trial-by-trial rejection criterion during averaging (see Silton et al., 2010 for specific methods).

Stimulus-locked averages were calculated to ascertain the frontocentral N200/P300 and parietal P300 components. The data were baseline-adjusted by subtracting the average amplitude for 200 milliseconds before stimulus onset. Waveform averages will be smoothed using a 101-weight, .1– 12 Hz (half-amplitude) digital filter. Electrode sites and scoring windows were selected based upon *a priori* scoring windows derived from other studies investigating these ERPs in similar contexts (N200/P300:

Enriquez-Geppert et al., 2010; Stockdale et al., 2015; parietal P300: Polich, 2007), as well as visual inspection of the data in the present study.

The N200/P300 complex was measured from 140 to 440 ms post stop-signal onset for correct "stop" trials of the Stop-Signal Task. A cluster of four frontocentral electrodes was identified for N200/P300 analyses (Figure 6a). On average, participants had 94 accepted correct "stop-happy" trials and 96 accepted correct "stop-sad" trials. The parietal P300 was measured from 230 to 530 ms post stimulus onset for correct "switch" trials of the Color-Word Stroop. A cluster of six parietal electrodes was identified for P300 analyses (Figure 6b). On average, participants had 55 accepted correct "switch" trials. Amplitude scores were obtained for the frontocentral N200/P300 and parietal P300 following methods used in a study by Silton and colleagues (2010).

Figure 6. Electrode localization for frontocentral N200/P300 and parietal P300 ERPs



(a) Frontocentral N200/P300 electrode cluster. (b) Parietal P300 electrode cluster.

CHAPTER FOUR

RESULTS

Descriptive Analyses

The means and standard deviations for study variables can be found in Table 1.

Table 1. Descriptive statistics for study variables

Self-Report Measures	***M***	***SD***
MASQ-AD	54.75	16.85
CRI Avoidance Coping Subscale	51.38	9.74
Coping Flexibility Scale	22.75	4.55
Behavioral Measures	***M***	***SD***
Stop-Signal Delay (ms)	470.73	51.95
CWS Switch Cost (ms)	88.01	53.91
EEG Measures	***M***	***SD***
N200 / P300 Amplitude (μV)	2.41	1.19
Parietal P300 Amplitude (μV)	2.66	1.41

Note. MASQ-AD = Anhedonic Depression Subscale of the Mood and Anxiety Symptom Questionnaire; CRI = Coping Responses Inventory; CWS = Color-Word Stroop; Switch Cost = reaction time for "switch" trials minus reaction time for "non-switch" trials.

Correlational Analyses

Simple bivariate correlational analyses were conducted to assess the relations among study variables. As can be seen in Table 2, neither Stop-Signal Task performance nor N200/P300 amplitude was significantly correlated with disengagement coping or depression symptoms. Furthermore, disengagement coping was not significantly correlated with depression symptoms. Coping flexibility was negatively correlated with

depression symptoms. There were no significant correlations between coping flexibility and Color-Word Stroop performance or parietal P300 amplitude (see Table 3).

Table 2. Correlations among study variables included in inhibitory control analyses

	Stop-Signal Delay	N200/P300 Amplitude	Disengagement Coping	Depression Symptoms
Stop-Signal Delay	--	-.13	-.11	-.07
N200/P300 Amplitude		--	.15	-.17
Disengagement Coping			--	.14
Depression Symptoms				--

Table 3. Correlations among study variables included in set-shifting analyses

	CWS Switch Cost	P300 Amplitude	Coping Flexibility	Depression Symptoms
CWS Switch Cost	--	.14	-.18	.05
P300 Amplitude		--	.08	.23
Coping Flexibility			--	-.52***
Depression Symptoms				--

Note. CWS = Color-Word Stroop
*** $p < .001$.

Mediation Analyses

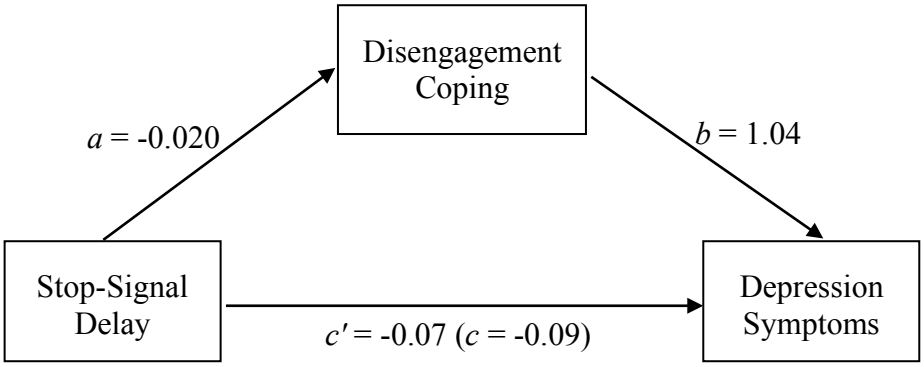
All mediation models were tested using the PROCESS macro (Hayes, 2012) for SPSS. As recommended for small samples (e.g., Preacher & Hayes, 2004; Preacher, Rucker, & Hayes, 2007), nonparametric bootstrapping analyses were used, generating a

95% bias-corrected confidence interval for the indirect effect of each model using 10,000 bootstrap samples. All sub-hypotheses (i.e., the "causal steps") of each mediation model were tested. However, contrary to the "causal steps approach" to analyzing mediated effects as outlined by Baron and Kenny (1986), Hayes (2009, 2013) proposed that *X* (the independent variable) can exert an indirect effect on *Y* (the dependent variable) through *M* (the mediator) in the absence of an association between *X* and *Y*. Thus, the indirect effect of the independent variable through the mediator was calculated for each model, regardless of analytical outcomes for the relevant sub-hypotheses.

Model 1: Disengagement coping utilization as a mediator of the relation between response inhibition (task performance) and depression symptoms. In this model, a simple mediation analysis conducted using ordinary least squares path analysis estimated the total and direct effects of performance on a task of response inhibition (as measured by the length of the stop-signal delay) on depression symptoms through self-reported utilization of disengagement coping strategies. Figure 7 presents the statistical model. There was no significant association between Stop-Signal Task performance and self-reported depression symptoms (c = -0.09, p = .44) or utilization of disengagement coping (a = -0.02, p = .10). There was also no significant association between level of disengagement coping utilization and depression symptoms (b = 1.04, p = .48). Additionally, there was no evidence that task performance was directly associated with depression symptoms after accounting for the effect of disengagement coping (c' = -0.07, p = .57). A 95% bias-corrected bootstrap confidence interval for the indirect effect (ab = -.02) based on 10,000 bootstrap samples included zero (-0.13 to 0.02). Thus, task

performance on a task of inhibitory control did not indirectly influence depression symptoms through its effect on level of disengagement coping utilization.

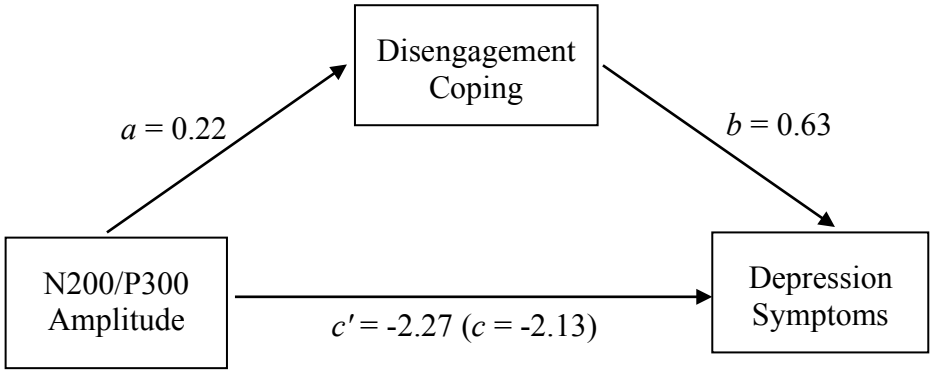
Figure 7. Statistical diagram: Utilization of disengagement coping strategies mediating the relation between performance on a task of inhibitory control and depression symptoms



Model 2: Utilization of disengagement coping strategies as a mediator of the relation between neural correlates of inhibitory control (N200/P300) and depression symptoms. The simple mediation analysis for this model estimated the total and direct effects of frontocentral N200/P300 amplitude during "stop" trials of the Stop-Signal Task on depression symptoms, as well as the indirect effect of N200/P300 amplitude on depression symptoms through self-reported utilization of disengagement coping strategies. As can be seen in Figure 8, there was no significant association between N200/P300 amplitude and self-reported depression symptoms (c = -2.13, p = .32) or utilization of disengagement coping (a = 0.22, p = .34). There was no significant association between level of disengagement coping utilization and depression symptoms (b = 0.63, p = .70). Additionally, the direct effect of N200/P300 amplitude on depression

symptoms was not significant (c' = -2.27, p = .30). A 95% bias-corrected bootstrap confidence interval for the indirect effect (ab = 0.14) based on 10,000 bootstrap samples included zero (-0.41 to 1.93). Therefore, contrary to hypotheses, neural correlates of inhibitory control did not indirectly influence depression symptoms through its effect on level of disengagement coping utilization.

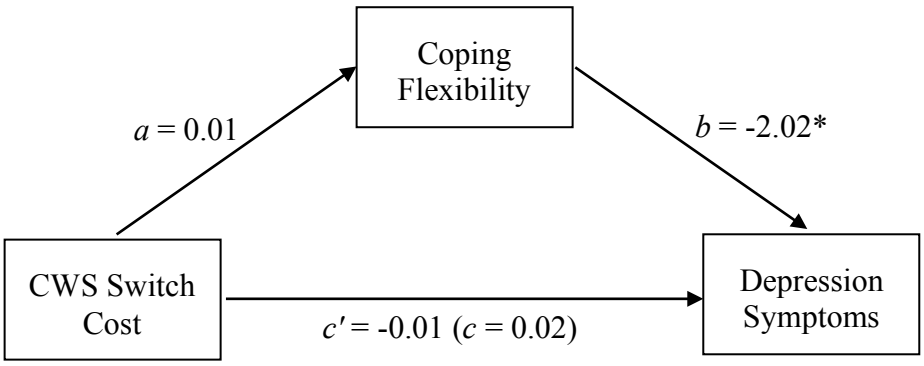
Figure 8. Statistical diagram: Utilization of disengagement coping strategies mediating the relation between frontocentral N200/P300 amplitude and depression symptoms



Model 3: Coping flexibility as a mediator of the relation between set-shifting task performance and depression symptoms. This mediation analysis estimated the total and direct effects of performance on a task of set-shifting ability on depression symptoms, as well as the indirect effect of set-shifting performance on depression symptoms through coping flexibility. Task performance was measured by reaction time of correct responses on "switch" trials versus "non-switch" trials of the Color-Word Stroop. As can be seen in Figure 9, there was no significant association between set-shifting and self-reported depression symptoms (c = 0.02, p = .73) or coping flexibility (a = -0.01, p = .24). The association between coping flexibility and depression symptoms

was significant (b = -2.02, p <.001). Additionally, there was no evidence that set-shifting performance on the Color-Word Stroop was significantly associated with depression symptoms after accounting for coping flexibility (c' = -0.01, p = .73). A 95% bias-corrected bootstrap confidence interval for the indirect effect (ab = 0.03) based on 10,000 bootstrap samples included zero (-0.01 to 0.10). Contrary to hypotheses, set-shifting ability did not indirectly influence depression symptoms through its effect on coping flexibility.

Figure 9. Statistical diagram: Coping flexibility mediating the relation between set-shifting task performance and depression symptoms

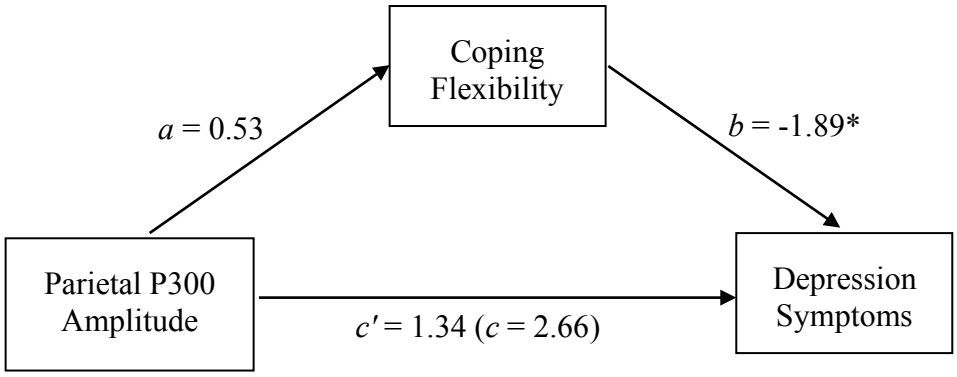


Note: * = p < .001

Model 4: Coping flexibility as a mediator of the relation between neural correlates of set-shifting (parietal P300) and depression symptoms. The simple mediation analysis for this model estimated the total and direct effects of parietal P300 amplitude during "switch" trials of the Color-Word Stroop on depression symptoms, as well as the indirect effect of parietal P300 amplitude on depression symptoms through coping flexibility. As can be seen in Figure 10, there was no significant association

between P300 amplitude and self-reported depression symptoms (c = 2.66, p = .17) or coping flexibility (a = 0.53, p = .24). There was again a significant association between coping flexibility and depression symptoms (b = -1.89, p = .007). Additionally, the direct effect of N200/P300 amplitude on depression symptoms was not significant (c' = 1.34, p = .41). A 95% bias-corrected bootstrap confidence interval for the indirect effect (ab = -1.00) based on 10,000 bootstrap samples included zero (-2.79 to 0.87). Contrary to hypotheses, neural activity (parietal P300) as an indicator of set-shifting ability did not indirectly influence depression symptoms through its effect on coping flexibility.

Figure 10. Statistical diagram: Coping flexibility mediating the relation between parietal P300 amplitude and depression symptoms



Note: * = p < .01

Post-Hoc Analyses

Given that each of the variables investigated in the current study are multifaceted, there are several potential explanations for the lack of significant associations between the identified variables. Alternative, theory-driven conceptualizations/measurements of depression symptoms, coping strategy, and neural correlates of inhibitory control and set-shifting ability were evaluated.

Anhedonic Depression subscale. Although depression symptomatology as an outcome variable is often treated as unidimensional clinical construct, depression is undoubtedly multifaceted and heterogeneous (Kendall et al., 2015; Monroe and Anderson, 2015). Recent studies have shown anhedonic depression symptoms to consist of two primary factors: negative affect and positive affect (Kendall et al., 2015; Prenoveau et al., 2011; Naragon-Gainey, Gallagher, & Brown, 2013). The Anhedonic Depression subscale of the Mood and Anxiety Symptoms Questionnaire (MASQ-AD; Watson & Clark, 1991) used in the present study to assess depression symptoms consists of two subscales: an 8-item Negative Affect Scale (MASQ-AD 8; e.g., "Felt withdrawn from other people") and a 14-item Positive Affect Scale (MASQ-AD 14; e.g., "Felt like I had a lot of energy). These factors have been supported in a college student sample and individuals at risk for depression (Bredemeier et al., 2010; Casillas & Clark, 2000; Kendall et al., 2015; Nitschke, Heller, Imig, McDonald, & Miller, 2001).

Watson and Clark (1998) theorized that, whereas high negative affect is not specific to one disorder and may be considered a general risk for psychopathology (Clark and Watson, 1991, 1994; Mineka et al. 1998; Watson and Clark, 1998), low positive affect is specific to depression (Watson and Clark, 1998). Recent research has demonstrated that individuals with depression are characterized by low positive affect and high negative affect (Kendall et al., 2015). Furthermore, low positive affect has been associated with unremitting depression and greater risk for future depressive episodes (Pine, Cohen, Cohen, & Brook, 1999; Wilcox & Anthony, 2004). Low positive affect has been shown to be predictive of depression symptoms even after accounting for high trait negative affect, which suggests a distinct contribution of low positive affect to depression

symptoms over time (Loh, Schutte, & Thorsteinsson, 2014; Verstraeten, Vasey, & Raes, 2009). Thus, low positive affect may represent an ongoing vulnerability to depression. Post-hoc exploratory analyses examined the association between negative affect and positive affect with aspects of coping and inhibitory control.

Coping. The current study hypothesized that deficits in inhibitory control would be associated with greater reliance on disengagement coping strategies and higher levels of depression symptoms. However, disengagement coping was not significantly related to (behavioral or neural indicators of) inhibitory control or depression symptoms. These findings may be explained by the circumscribed way in which coping strategy was operationalized in the study. For example, deficits in executive function abilities such as inhibitory control and set-shifting may be more likely to predict less utilization of engagement coping strategies, rather than increased reliance on disengagement coping strategies. Post-hoc exploratory analyses examined engagement coping (in addition to disengagement coping) and the sub-domains assessed by the Coping Responses Inventory (i.e., cognitive approach, behavioral approach, cognitive avoidance, behavioral avoidance), and their relative associations with negative affect and positive affect, as well as the behavioral and neural correlates of inhibitory control.

Neural correlates. The present study investigated the event-related potentials (i.e., frontocentral N200/P300 amplitude and parietal P300 amplitude) as neural correlates of executive function abilities (i.e., inhibitory control and set-shifting). Another indicator of cognitive processing efficiency often examined using EEG methodology is ERP peak latency; this measurement emphasizes the time course of neural activity, rather than signal strength (i.e., recruitment of cognitive resources). Prolonged peak latencies

typically represent slowed cognitive processing. Research has shown that individuals with "melancholic" or anhedonic depression frequently have longer frontocentral N200/P300 (Urretavizcaya et al., 2003) and parietal P300 peak latencies (Schlegel, Nieber, Herrmann, & Bakauski, 1991; Vandoolaeghe, van Hunsel, Nuyten, & Maes, 1998). Post-hoc exploratory analyses examined associations between frontocentral N200/P300 and parietal P300 peak latencies with negative affect, positive affect, and coping strategy.

Results. The means and standard deviations for variables included in the post-hoc analyses can be found in Table 3.

Table 4. Descriptive statistics for variables included in post-hoc analyses

Self-Report Measures	***α***	***M***	***SD***
MASQ-AD 22-Item Subscale	.95	54.75	16.85
MASQ-AD 8-Item Subscale	.92	19.92	6.95
MASQ-AD 14-Item Subscale	.96	46.36	11.90
CRI Approach Coping Subscale	.89	63.74	12.49
CRI Avoidance Coping Subscale	.80	51.38	9.74
CRI Cognitive Approach Coping Subscale	.85	32.08	7.02
CRI Behavioral Approach Coping Subscale	.82	31.66	6.88
CRI Cognitive Avoidance Coping Subscale	.75	26.86	5.97
CRI Behavioral Avoidance Coping Subscale	.72	24.56	5.73
Coping Flexibility Scale	.77	17.52	4.42
Behavioral Measures		***M***	***SD***
SST Stop-Signal Delay (ms)		470.73	51.95
EEG Measures		***M***	***SD***
N200 / P300 Mean Amplitude (μV)		2.41	1.19
N200 / P300 Peak Latency (ms)		319.41	41.65
Parietal P300 Mean Amplitude (μV)		2.66	1.41
Parietal P300 Peak Latency (ms)		354.18	47.96

Note. MASQ-AD = Anhedonic Depression Subscale of the Mood and Anxiety Symptom Questionnaire; CRI = Coping Response Inventory; SST = Stop-Signal Task; CWS = Color-Word Stroop; Switch Cost = response reaction time on "switch" trials minus response reaction time on "non-switch" trials.

Associations between coping and affect. Correlation analyses revealed that disengagement coping was not significantly associated with negative affect or positive affect, while engagement coping was negatively associated with negative affect and positively associated with positive affect (Table 4). When coping strategy was parsed into cognitive and behavioral components, correlation analyses showed that both *cognitive* and *behavioral engagement* strategies were negatively associated with negative affect and positively associated with positive affect. As can be seen in Table 4, *cognitive disengagement* coping was positively correlated with negative affect but not significantly correlated with positive affect. *Behavioral disengagement* coping was not significantly related to either negative affect or positive affect. Coping flexibility was negatively correlated negative affect and positively correlated with positive affect.

Associations with behavioral task performance. Simple bivariate correlation analyses demonstrated that performance on a task of inhibitory control during implicit emotion regulation was not significantly associated with any aspect of coping strategy (i.e., disengagement, engagement, cognitive disengagement, behavioral disengagement cognitive engagement, or behavioral engagement), negative affect, or positive affect (Table 4). Similarly, performance on a set-shifting task (difference in reaction time for correct responses in "switch" minus "non-switch" trials) was not significantly associated with coping flexibility, negative affect, or positive affect (Table 5).

Associations with neural correlates of inhibitory control and set-shifting. Post-hoc bivariate correlation analyses showed that frontocentral N200/P300 amplitude was not significantly associated with any aspect of coping strategy (i.e., disengagement, engagement, cognitive disengagement, behavioral disengagement, cognitive engagement,

or behavioral engagement), negative affect, or positive affect (Table 4). However, frontocentral N200/P300 *peak latency* was positively correlated with cognitive engagement coping and positive affect; it was not significantly associated with other coping variables or negative affect. Similarly, parietal P300 amplitude was not significantly associated with coping flexibility, negative affect, or positive affect. Parietal P300 *peak latency* was negatively associated with negative affect and positively associated with positive affect; it was not significantly correlated with coping flexibility (Table 5).

Table 5. Correlations among coping strategies, affect, and behavioral and neural correlates of inhibitory control

	SST	N2/P3 MA	N2/P3 PL	Dis Cope	Eng Cope	Cog Dis	Beh Dis	Cog Eng	Beh Eng	NA	PA
Stop-Signal Delay	--	.23	-.34*	-.24	.07	-.07	-.11	-.03	-.13	-.044	.073
N200/P300 MA		--	-.34*	.02	.19	.16	-.18	-.17	-.18	-.12	.08
N200/P300 PL			--	.18	.31*	.04	.26	.37*	.20	-.02	.34
Disengagement Coping				--	.43**	.84***	.84***	.51***	.28	.13	-.041
Engagement Coping					--	.06	.66***	.90***	.91***	-.42**	.47**
Cognitive Disengagement						--	.40**	.21	-.08	.33*	-.26
Behavioral Disengagement							--	.65***	.55***	-.12	.19
Cognitive Engagement								--	.65***	-.32*	.39**
Behavioral Engagement									--	-.44**	.46**
Negative Affect										--	-.94**
Positive Affect											--

Note. SST = Stop-Signal Task Performance; N2/P3 = N200/P300; MA = Mean Amplitude; PL = Peak Latency; Dis Cope = Disengagement Coping; Eng Cope = Engagement Coping; Cog Dis = Cognitive Disengagement; Beh Dis = Behavioral Disengagement; Cog Eng = Cognitive Engagement; Beh Eng = Behavioral Engagement; NA = Negative Affect; PA = Positive Affect

* $p < .05$. ** $p < .01$. *** $p < .001$.

Table 6. Correlations among coping flexibility, affect, and neural correlates of set-shifting ability

	P300 Amplitude	P300 Peak Latency	Coping Flexibility	Negative Affect	Positive Affect
P300 Amplitude	--	-.16	.08	.17	-.22
P300 Peak Latency		--	.16	-.36*	.34*
Coping Flexibility			--	-.49***	.47***
Negative Affect				--	-.96***
Positive Affect					--

* $p < .05$. ** $p < .01$. *** $p < .001$.

CHAPTER FIVE

DISCUSSION

Overview

Within the clinical psychology literature, the ability to cope effectively with stressful or negative life events has consistently been associated with mental health outcomes (e.g., Aldwin & Revenson, 1987; Carver & Connor-Smith, 2010). However, the cognitive and neurophysiological correlates of the ability to cope are not well understood. A considerable amount of research has demonstrated evidence of executive dysfunction and abnormal neural activity in depression (e.g., Langenecker et al., 2007; Levin et al., 2007; Rogers et al., 2004; Silton et al., 2011).

The present study tested the hypothesis that executive functions (i.e., inhibitory control and set-shifting abilities) play a critical role in coping skills (i.e., strategy and flexibility), which in turn are associated with depression symptoms. To measure executive function, participants were administered computerized cognitive tasks while scalp electroencephalography (EEG) was measured. Coping and depression symptoms were assessed with self-report questionnaires. Overall, relations among maladaptive coping and increased depression symptoms were observed; however, findings did not support the primary hypothesis that coping mediates the relation between executive dysfunction and depression symptoms.

Executive functioning, coping, and depression symptoms are multifaceted constructs, and the primary study analyses used monolithic variables to represent these constructs. Theory-driven post-hoc analyses were conducted to further evaluate the constituents of these variables. Results showed that cognitive disengagement coping was positively correlated with negative affect, and engagement coping strategies were negatively associated with negative affect and positively associated with positive affect. Furthermore, frontocentral N200/P300 peak latency during a task of inhibitory control was positively correlated with cognitive engagement coping and positive affect, and parietal P300 peak latency during a set-shifting task was positively correlated with positive affect.

Associations Between Coping Abilities and Subjective Reports of Affect

Initial analyses showed no significant association between disengagement coping and depression symptoms. However, further investigation of the constituents of each variable revealed that while *cognitive* disengagement was positively associated with negative affect, *behavioral* disengagement was not associated with either negative affect or positive affect. Additionally, cognitive and behavioral *engagement* coping strategies were negatively associated with negative affect and positively associated with positive affect, and *cognitive disengagement* was only positively associated with negative affect. These findings indicate that utilization of *behavioral disengagement* coping strategies had no influence on affect; however, participants who reported utilizing more *cognitive disengagement* coping reported more negative affect. Consistent with the literature, this suggests that cognitive disengagement strategies may have a more insidious effect on emotional functioning, as the attempted avoidance, denial, and suppression of stressful

thoughts can promote a paradoxical increase in rumination and intrusive thoughts about the stressor (Najmi & Wegner, 2008).

Alternatively, participants who reported utilizing fewer *engagement* coping (both cognitive and behavioral) strategies endorsed more depression symptoms, consisting of higher levels of negative affect and lower levels of positive affect. This suggests that greater utilization of engagement coping strategies may result in lower levels of negative affect and higher levels of positive affect, or vice versa, individuals with higher positive affect and lower negative affect may be more likely to utilize engagement coping strategies. Moreover, as expected, individuals who reported less coping flexibility endorsed more depression symptoms, more negative affect, and less positive affect. These findings are consistent with the considerable literature demonstrating the critical role of adaptive coping in resilience against depression symptoms (e.g., Connor-Smith & Compas, 2002; Herman-Stahl & Peterson, 1995; Jaser, Champion, Dharamsi, Riesing, & Compas, 2011). That is, greater coping flexibility and utilization of engagement coping strategies is associated with better subjective well-being.

Descriptive analyses showed that participants were more likely to endorse engagement coping than disengagement coping strategies, and *behavioral* disengagement strategies were reported least often. Thus, the low level of behavioral disengagement coping behaviors across participants may have diminished the association between disengagement coping and depression symptoms. This may be a function of a high-achieving undergraduate sample, perhaps comprised of individuals who do not frequently resort to behavioral disengagement strategies.

Behavioral Indicators of Inhibitory Control and Set-Shifting Abilities

Results from the present study demonstrated that behavioral performances on tasks of inhibitory control and set-shifting were not significantly associated with any aspects of coping, depression symptoms, or reported affect. Additionally, mediation analyses showed no significant indirect effects of these measures on depression symptoms through coping abilities. The lack of significant association between behavioral measures of inhibitory control and set-shifting with other variables may be due in part to the high-functioning, somewhat self-selected participant sample, comprised of undergraduate students who not only agreed to participate in the study, but ultimately completed the project's three laboratory visits. That is, behavioral measures of executive functions may not be sensitive enough to detect the nuanced individual variability in executive function abilities, particularly in high-functioning samples (Chan, Shum, Toulopoulou, & Chen, 2008).

Consistent with this finding, neuroimaging studies have frequently demonstrated that individuals with depression are able to achieve behavioral performance scores equivalent to control subjects on tasks of executive functions. That is, they are able to compensate for cognitive weaknesses and perform at the same level as others, but the task requires more cognitive effort and recruitment of additional cognitive resources. This cognitive exertion is then illustrated by the consequential differences in neural activation in frontal and various other brain regions of individuals with depression as compared to non-depressed controls (e.g., Harvey et al., 2005; Holmes et al., 2005; Langenecker et al., 2007; Silton et al., 2011; Wagner et al., 2006).

Associations Between Frontocentral N200/P300 Peak Latency, Engagement Coping, and Positive Affect

Frontocentral N200/P300 amplitude evoked during a task of inhibitory control and implicit emotion processing was not significantly associated with coping, depression symptoms, or trait affect. There was also no significant indirect effect of N200/P300 amplitude on depression symptoms through disengagement coping. However, N200/P300 *peak latency* was positively correlated with engagement coping and positive affect. Prolonged N200/P300 peak latency was associated with greater utilization of engagement coping strategies and more positive affect. These findings are contrary to hypotheses based upon previous research demonstrating prolonged frontocentral N200 and P300 peak latencies in individuals with depression (Kemp et al., 2008; Urretavizcaya et al., 2003). However, ERP components represent task-specific functions, and previous investigations of ERP components in depression have examined the N200 and P300 during an auditory oddball task, rather than a task that measures inhibitory control in the context of implicit emotional distractors.

Given the implicit emotion processing aspect of the Stop-Signal Task used in the present study, the associations between N200/P300 peak latency and engagement coping and positive affect were probed further by investigating N200/P300 peak latency distinctly for happy and sad faces, in order to determine if the associations were driven by an implicit attentional bias to valenced facial expressions. Analyses showed that the association between N200/P300 peak latency and positive affect was only significant when participants were asked to inhibit their response while implicitly attending to happy faces. Individuals who reported lower levels of positive affect exhibited more efficient

inhibitory control processes when shown happy faces than those who reported higher levels of positive affect. This indicates that their attention was not implicitly captured by happy emotional expressions (resulting in less difficulty inhibiting a response). Similarly, the association between N200/P300 peak latency and engagement coping was only significant when participants were instructed to inhibit their response while implicitly attending to happy faces. That is, individuals whose attention was not implicitly captured by happy emotional expressions reported lower levels of engagement coping. Alternatively, individuals whose attention was captured by happy emotional expressions reported more positive affect and higher levels of engagement coping.

These findings are consistent with previous research on affect and cognition demonstrating that individuals with lower levels of positive affect do not exhibit the positive attentional biases that characterize happy individuals; rather, they may filter out positive stimuli (e.g., Harding, Hudson, & Mezulis, 2014; Joormann & Gotlib, 2007). Furthermore, these individuals are less likely to utilize engagement coping strategies. There was no significant association of engagement coping or positive affect with N200/P300 peak latency when participants were asked to inhibit their response while implicitly attending to sad faces. This suggests that positive affect did not correspond with an implicit attentional bias to negative emotional stimuli.

The present findings have important implications for coping research, as they demonstrate associations between affect, early differences in the implicit processing of emotional information, and coping strategy. The majority of research on coping has focused on the construct as predictive of other outcome variables, and some studies have focused on coping patterns within the context of mental health diagnoses (e.g.,

depression, anxiety) or medical conditions; however, less is known about the association of positive emotions on coping behaviors. A literature on this topic has only recently begun to emerge, indicating that individuals who are resilient to stress and depression may use positive emotions more effectively during emotion regulation processes, in order to support healthy coping strategies (Folkman & Moskowitz, 2000; Garland et al., 2010; Tugade & Fredrickson, 2004; Ong et al, 2006). The current project allowed us to examine the very early time course of implicit emotion processing, and this is one of the first known studies to explore the neural correlates of response to valenced stimuli within the context of coping. Findings from the present research support the theory that positive and negative affect represent two distinct systems, rather than opposite ends of the same dimension (Watson & Clark, 1992). Future research should consider the influence of positive and negative affect on coping abilities.

Associations Between Parietal P300 Peak Latency and Affect

The current study found that parietal P300 amplitude during "switch" trials of the Color-Word Stroop were not associated with coping flexibility, depression symptoms, negative affect, or positive affect. There was also no significant indirect effect between parietal P300 on depression symptoms through coping flexibility. However, parietal P300 peak latency was negatively correlated with negative affect and positively correlated with positive affect. In other words, longer P300 peak latency (representing less efficient cognitive processing during a set-shifting task) was associated with lower negative affect and higher positive affect. This result is contrary to hypotheses based upon previous findings within the EEG depression literature, which cite prolonged P300 peak latency in depression (Schlegel et al., 1991; Vandoolaeghe et al., 1998). However, as previously

mentioned, these studies primarily investigated the P300 during auditory odd-ball tasks. Nevertheless, the association between longer parietal P300 latency, lower negative affect, and higher positive affect can be explained by affect-cognition theories.

Set-shifting is a complex cognitive task, requiring shifting attention and mental sets to changing environmental demands (Wager et al., 2006; Ward, Roberts, & Phillips, 2001). The Color-Word Stroop (Delis et al., 2001) in particular requires participants to switch from a prepotent response (i.e., word reading) to the inhibition of the prepotent response (i.e., color naming instead of word reading). Successful task completion requires flexible, context-dependent goal-setting and execution (e.g., Miller & Cohen, 2001; Wagner et al., 2006), and resolution of interference from competing task sets (i.e., instructions from previous trials; Allport, Styles, & Hsieh, 1994). To this end, positive affect has been shown to reduce perseveration (e.g., rumination) and promote cognitive flexibility, but at the cost of increased distractibility (Dreisbach & Goschke, 2004).

Research has demonstrated that positive emotions – whether related to trait affectivity or more momentary affective states – serve to enhance cognitive flexibility (Isen & Daubman, 1984; Isen, Niedenthal, & Cantor, 1992), reduce rigidity in problem solving (Green & Noice, 1988; Isen, 2008), and increase creativity (Isen, Daubman, & Nowicki, 1987). However, this increase in flexibility reflects a fundamental change in selective attention (Gasper & Clore, 2002; Fenske & Eastwood, 2003; Huntsinger, 2013). Some theorists propose that, in contrast to the "tunnel vision" of depression and negative affect (Derryberry & Reed, 1988; Easterbrook, 1959), positive affect may serve to broaden the scope of attention (i.e., the "broaden-and-build" theory of positive emotions; Fredrickson, 2001; Rowe, Hirsh, & Anderson, 2007). Others posit that positive affect acts

as a "go" signal that facilitates the use of relevant cognitive resources elicited by the task at hand, while negative affect acts as a "stop" signal, inhibiting the effective use of necessary cognitive processes (Clore & Huntsinger, 2009; Huntsinger, 2013).

Thus, positive affect appears to reduce perseverative behavior and enable individuals to access the broad attentional scope necessary to efficiently and flexibly switch between cognitive sets (Dreisbach & Goschke, 2004). However, it has been proposed that perseveration (i.e., narrow focus) and distractibility (i.e., broad focus) fall on opposite ends of the attentional spectrum (Goschke, 2003; Miller & Cohen, 2001). In other words, increased attentional breadth and greater cognitive flexibility indicate a decreased capacity to inhibit processing of task irrelevant information (i.e., distractibility; Dreisbach & Goschke, 2004). Consistent with this theory, the prolonged parietal P300 peak latency observed during "switch" trials of the Color-Word Stroop in the present study may represent a neural indicator of distractibility, as individuals with more positive affect may require additional time to process the additional information via their broadened attentional scope (activated to facilitate performance with set-shifting). Similarly, the "narrow" scope of negative affect in response to the set-shifting task may have paradoxically facilitated the processing of task-relevant information more efficiently, resulting in a shortened P300 peak latency.

Related, some researchers have postulated that positive mood states actually "impair" executive functions, in that both positive and negative mood states may lead to task-irrelevant thought processing (Oaksford et al., 1996). Phillips and colleagues found that individuals in a happy mood exhibited slower performance on the "switch" trials of the Stroop task, without corresponding slowness on any other condition of the task

(Phillips, Bull, Adams, & Fraser, 2001). Through this lens, individuals reporting more positive affect (and less negative affect) may need to exert more cognitive control to suppress task-irrelevant positive thoughts, which could be associated with the observed prolonged parietal P300 latency in the present study.

Implications and Future Directions

The findings from the present study have important implications for future clinical psychology research, as well as intervention strategies for the treatment or prevention of depression symptoms. Results demonstrated that greater coping flexibility and utilization of engagement coping strategies were associated with more positive affect and less negative affect, underscoring the role of coping abilities as protective factors, buffers, or indicators of resilience against the development of depression symptoms. However, further research is necessary to identify the foundational mechanisms involved in coping. Specifically, what are the cognitive abilities that facilitate the utilization of engagement versus disengagement strategies? And what causes dysfunction in these processes? Alternatively, what enhances these processes?

The present study found that individuals who did not implicitly attend to happy facial expressions reported utilizing fewer engagement coping strategies and reported lower levels of positive affect. However, the directional nature of the associations between these constructs remains unclear. For example, it may be the case that when there is a disruption in attentional processes (e.g., the implicit attentional bias to happy or positive stimuli is interrupted), an individual may not be able to identify sufficient (internal or external) resources to cope with a particular stressor, decreasing the amount of engagement coping strategies utilized, and subsequently leading to a decrease in

positive affect. Alternatively, low levels of positive affect may impair the typical attentional bias to positive stimuli, leading to less engagement coping utilization. These associations have important implications for therapeutic intervention or prevention strategies. Specifically, findings provide support for reduced attention to positive information in individuals with low positive affect, highlighting the potential benefit of attention training strategies of cognitive bias modification (CBM) programs, also known as attention bias modification (ABM). CBM has been defined as the "direct manipulation of target cognitive bias, by extended exposure to task contingencies that favor predetermined patterns of processing selectivity" (MacLeod, 2012). CBM programs address the selective biases in information processing that are considered to play an important role in the development, maintenance, and exacerbation of psychopathology. ABM teaches individuals to avoid negative stimuli (usually pictures or words) by directing their attention, without their knowledge, to neutral or positive stimuli. Increased attention to positive information may result in increased engagement coping and/or levels of positive affect.

However, the present study also implicates higher levels of positive affect in decreasing cognitive efficiency during a task of set-shifting (without congruent effects on behavioral performance). This emphasizes the importance of research examining the role of affect in the regulation of cognitive control. Low positive affect has been shown to play a significant role in the development, maintenance, and exacerbation of depression symptoms (Watson & Clark, 1992; Kendall et al., 2015; Loh et al., 2014; Werner-Seidler et al., 2013); yet, research into the associated mechanisms has only begun to emerge. Additionally, findings related to the association between positive affect and cognition

have been mixed, with some studies demonstrating cognitive processing deficits in individuals with low positive affect (e.g., Compton, Wirtz, Pajoumand, Claus, & Heller, 2004), while others suggest that high positive affect has detrimental effects on cognitive functions (Oaksford et al., 1996; Phillips et al., 2001). It is possible that both high and low positive affect have negative effects on cognitive processing; more research is necessary to identify optimal levels of positive affect, as well as mechanisms through which affect influences cognition and/or vice versa. Future research should examine executive functions, neural activity, coping, and affect using longitudinal methods, which would allow for investigations of directional associations to clarify the best targets for intervention strategies. Research should also consider variables that might influence the relation between cognitive processes (and brain circuitry) and coping behaviors.

Present findings also have important implications for continued translational research investigating the cognitive and neural correlates of coping. For example, the exploration of coping should encompass the various aspects and dimensions of the construct. This strategy allows for a more ecologically valid assessment of coping and may also reveal sample-specific coping patterns. For example, when faced with stressful situations or negative life events, individuals likely utilize both engagement and disengagement coping strategies, perhaps even simultaneously; coping strategies also change over time and in accordance with the demands of stressful situations (Folkman & Moskowitz, 2004; Kato, 2012). Additionally, as demonstrated by the present study sample, different populations may demonstrate preferred/typical coping strategies (e.g., Monteiro, Balogun, & Oratile, 2014; Ptacek, Smith, & Zanas, 2006). This provides support for strategies that allow for a comprehensive assessment of coping repertoires.

Similarly, research investigations should address the heterogeneous nature of depression symptoms. Depression is a complex disorder identified by symptoms that fall along various dimensions, including mood/affect, behavior, and cognitive variables. As mentioned above, findings from the present study highlight the particular importance of positive affect when exploring coping abilities and neural correlates of inhibitory control and set-shifting. Although the interaction between negative affect and cognition has long been an active focus of research, research on the role of positive affect in cognition is in its infancy.

Finally, though the present study provided limited support for the initial mediation models, further research is warranted. For example, executive dysfunction may influence coping abilities through other constructs, such as rumination or decreased self-esteem. Coping variables may also be associated with different neurophysiological markers than those proposed in the present study. For example, research has shown resting EEG asymmetries – specifically reduced left frontal and increased right frontal activity – in individuals with depression (Davidson et al., 2002; Heller & Nitschke, 1997; Stewart et al., 2011). This is consistent with the "valence hypothesis" and circumplex model of hemispheric brain function, suggesting that positive affect is differentially lateralized to the left hemisphere and negative affect is differentially lateralized to the right hemisphere (Davidson & Henriques, 2000; Heller, 1990; Heller and Nitschke, 1997; Tomarken, Davidson, Wheeler, & Doss, 1992). This is also consistent with the "motivational hypothesis" of cerebral asymmetry, which suggests that the left hemisphere differentially specializes in approach motivation, whereas the right hemisphere differentially specializes in avoidance motivation (Harmon-Jones, Gable, & Peterson, 2010; Sutton &

Davidson, 1997). Thus, future research should explore the associations between resting EEG asymmetry and coping strategies; that is, increased activation in the left hemisphere may indicate a tendency toward engagement coping strategies, while increased activation in the right hemisphere may indicate a tendency toward disengagement coping strategies.

Limitations

Several limitations of the study should be noted. The final participant samples were relatively small, limiting the power to detect indirect effects for the proposed mediation models. Specifically, given an estimated three variables (i.e., the independent variable (IV) the mediator variable, and the interaction), a power analysis using G*Power version 3.1 (Faul, Erdfelder, Lang, & Buchner, 2007), with power (1 - β) set at 0.80 and α set at .05, indicated that a total sample size of $N = 107$ participants would be necessary to detect a medium effect size ($f^2 = 0.15$; Cohen, 1988) and $N = 48$ would be necessary to detect a large effect size ($f^2 = 0.35$) in the proposed study. Thus, the sample sizes of 40-51 in the current study likely limited the ability to detect even large indirect effects in the proposed mediation models.

Another limitation involves the measurement of coping strategy and flexibility using self-report questionnaires. There are many benefits of utilizing this method to assess coping behaviors. For example, it allows for a more comprehensive evaluation of the different dimensions of coping, while also providing the flexibility to assess situation-specific coping thoughts and behaviors. Furthermore, questionnaires are convenient and simple to administer. However, there are pitfalls to this assessment technique as well. One of the most prominent criticisms of relying on self-report measures to assess coping

behaviors is the problem of retrospective report, specifically the accuracy of recall about specific thoughts and behaviors in response to a situation that occurred in the past (Coyne & Gottlieb, 1996; Folkman & Moskowitz, 2004; Stone et al., 1998). For example, the Coping Responses Inventory (Moos, 1993) instructs participants to "think of the most significant problem [they] have faced in the past month" and indicate how often they have used various coping strategies to deal with that problem. This method places a considerable amount of trust on participants' memories and level of insight.

Previous studies have shown that retrospective reports of coping can be unreliable. For example, Stone and colleagues (1998) found that when comparing retrospective self-reports to data collected using ecological momentary assessment (EMA) methodology, one-third of individuals failed to retrospectively report strategies that they had reported on EMA, and another third of the sample retrospectively reported strategies that they did not report on EMA (Stone et al., 1998). Additionally, as with any measurement that relies upon subjective memory, recall of personal events and behaviors may be subject to distortion due to efforts to create a coherent and/or meaningful narrative of events (Ptacek et al., 1994; Stone et al., 1998).

However, a strength of the present study also lies in the methodology. Previous research investigating executive functions (EF) and coping in psychopathology has relied primarily on self-report measures of EF, which have the same weaknesses as those noted above for self-report inventories of coping (Toplak, West, & Stanovich, 2013). The present study utilized lab-based, computerized measures of inhibitory control and set-shifting, as well as scalp EEG techniques to explore candidate neural correlates. This multi-method approach allows for the investigation of behavioral measures of EF (e.g.,

response accuracy, reaction time) in addition to more nuanced measures of EF abilities, including neural indicators of cognitive effort (i.e., ERP peak amplitude) or cognitive processing efficiency (on the order of milliseconds; i.e., ERP peak latency).

Conclusions

In conclusion, the present study provides limited evidence for the hypothesis that executive dysfunction and associated frontocingulate brain abnormalities are associated with depression symptoms through their effects on coping abilities. However, results suggest that coping strategy and flexibility are associated with positive affect and negative affect, emphasizing the role of coping skills as protective factors against the development of depression symptoms. Furthermore, though neither coping nor affect variables were associated with the amount of cognitive effort required during executive function tasks, affect was observed to influence cognitive efficiency during a task of set-shifting. Furthermore, a decrease in implicit positive attentional bias was associated with lower levels of positive affect and lower levels of reported utilization of engagement coping strategies. Thus, while the present study is limited with regard to demonstrating causal associations between behavioral and neural indicators of executive functions, coping, and depression symptoms, findings highlight important associations between affect, patterns of cognitive processing, and coping variables.

APPENDIX A

SELF-REPORT MEASURES

Coping Flexibility Scale
(Kato, 2012)

When we feel stress, we try to cope using various actions and thoughts. The following items describe stress-coping situations. Please indicate how these situations apply to you by choosing one of the following for each situation: "very applicable," "applicable," "somewhat applicable," and "not applicable."

1. When a stressful situation has not improved, I try to think of other ways to cope with it.
2. I only use certain ways to cope with stress.
3. When stressed, I use several ways to cope and make the situation better.
4. When I haven't coped with a stressful situation well, I use other ways to cope with that situation.
5. If a stressful situation has not improved, I use other ways to cope with that situation.
6. I am aware of how successful or unsuccessful my attempts to cope with stress have been.
7. I fail to notice when I have been unable to cope with stress.
8. If I feel that I have failed to cope with stress, I change the way in which I deal with stress.
9. After coping with stress, I think about how well my ways of coping with stress worked or did not work.
10. If I have failed to cope with stress, I think of other ways to cope.

Coping Responses Inventory (CRI)

Please think of the most significant problem you have faced in the past month and indicate how often you have used each of the coping strategies below to deal with that problem.

	Not at all	Once or Twice	Sometimes	Fairly Often
1. Think of different ways to deal with the problem.	1	2	3	4
2. Tell yourself things to make yourself feel better.	1	2	3	4
3. Talk with your spouse or other relative about the problem.	1	2	3	4
4. Make a plan of action and follow it.	1	2	3	4
5. Try to forget the whole thing.	1	2	3	4
6. Feel that time will make a difference: that the only thing to do is wait.	1	2	3	4
7. Try to help others deal with a similar problem.	1	2	3	4
8. Take it out on other people when you feel angry or depressed.	1	2	3	4
9. Try to step back from the situation and be more objective.	1	2	3	4
10. Remind yourself how much worse things could be.	1	2	3	4
11. Talk with a friend about the problem.	1	2	3	4
12. Know what has to be done and try hard to make things work.	1	2	3	4
13. Try not to think about the problem.	1	2	3	4
14. Realise that you have no control over the problem.	1	2	3	4
15. Get involved in new activities.	1	2	3	4
16. Take a chance and do something risky.	1	2	3	4
17. Go over in your mind what you would say or do.	1	2	3	4
18. Try to see the good side of the situation.	1	2	3	4
19. Talk with a professional person (eg. doctor, lawyer, clergy).	1	2	3	4
20. Decide what you want and try hard to get it.	1	2	3	4
21. Daydream or imagine a better time or place, than the one you are in.	1	2	3	4
22. Think that the outcome will be decided by fate.	1	2	3	4
23. Try to make new friends.	1	2	3	4

24.	Keep away from people in general.	1	2	3	4
25.	Try to anticipate how things will turn out.	1	2	3	4
26.	Think about how you are much better off than other people with similar problems.	1	2	3	4
27.	Seek help from persons or groups with the same type of problem.	1	2	3	4
28.	Try at least two different ways to solve the problem.	1	2	3	4
29.	Try to put off thinking about the situation, even though you know you will have to at some point.	1	2	3	4
30.	Accept it; nothing can be done.	1	2	3	4
31.	Read more often as a source of enjoyment.	1	2	3	4
32.	Yell or shout to let off steam.	1	2	3	4
33.	Try to find some personal meaning in the situation.	1	2	3	4
34.	Try to tell yourself that things will get better.	1	2	3	4
35.	Try to find out more about the situation.	1	2	3	4
36.	Try to learn to do more things on your own.	1	2	3	4
37.	Wish the problem will go away or somehow be over with.	1	2	3	4
38.	Expect the worst possible outcome.	1	2	3	4
39.	Spend more time in recreational activities.	1	2	3	4
40.	Cry to let your feelings out.	1	2	3	4
41.	Try to anticipate the new demands that will be placed on you.	1	2	3	4
42.	Think about how this event could change your life in a positive way.	1	2	3	4
43.	Pray for guidance and/or strength.	1	2	3	4
44.	Take things a day at a time, one step at a time.	1	2	3	4
45.	Try to deny how serious the problem really is.	1	2	3	4
46.	Lose hope that things will ever be the same.	1	2	3	4
47.	Turn to work or other activities to help you manage things.	1	2	3	4
48.	Do something that you don't think will work, but at least you are doing something.	1	2	3	4

MASQ

Below is a list of feelings, sensations, problems, and experiences that people sometimes have. Read each item and mark the appropriate choice in the space next to that item. Use the choice that best describes how much you feel or experienced things this way in general. Use this scale when answering:

1	2	3	4	5
Not at all	A little bit	Moderately	Quite a bit	Extremely

1.	Startled easily	1	2	3	4	5
2.	Felt faint	1	2	3	4	5
3.	Felt really bored	1	2	3	4	5
4.	Felt numbness or tingling in my body	1	2	3	4	5
5.	Felt withdrawn from other people	1	2	3	4	5
6.	Felt like nothing was very enjoyable	1	2	3	4	5
7.	Felt like it took extra effort to get started	1	2	3	4	5
8.	Felt like there wasn't anything interesting or fun to do	1	2	3	4	5
9.	Had pain in my chest	1	2	3	4	5
10.	Had hot or cold spells	1	2	3	4	5
11.	Felt dizzy or lightheaded	1	2	3	4	5
12.	Felt unattractive	1	2	3	4	5
13.	Was short of breath	1	2	3	4	5
14.	Hands were shaky	1	2	3	4	5
15.	Felt like I was choking	1	2	3	4	5
16.	Felt really slowed down	1	2	3	4	5
17.	Had a very dry mouth	1	2	3	4	5
18.	Muscles twitched or trembled	1	2	3	4	5
19.	Was afraid I was going to die	1	2	3	4	5
20.	Heart was racing or pounding	1	2	3	4	5
21.	Was trembling or shaking	1	2	3	4	5
22.	Had to urinate frequently	1	2	3	4	5
23.	Had trouble swallowing	1	2	3	4	5
24.	Hands were cold or sweaty	1	2	3	4	5
25.	Thought about death or suicide	1	2	3	4	5

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VITA

Catherine Lee was born and raised in Shaker Heights, Ohio. She completed her undergraduate studies at Cornell University, where she earned a Bachelor of Arts in psychology in 2008. She completed her graduate studies in clinical psychology at Loyola University Chicago, where she earned a Master of Arts in 2013 and a Doctor of Philosophy in 2016. Prior to attending Loyola, she worked as a research assistant in the Department of Psychiatry and Psychology at the Cleveland Clinic.

At Loyola, Dr. Lee's initial research interests primarily involved psychopathology. Her master's thesis investigated the effects of self-esteem, perceived social support, and coping style on depression symptoms during the transition to college. Through her clinical experiences, she developed a particular interest in neuropsychological assessment and spent the remainder of her graduate training specializing in adult neuropsychology. This ultimately influenced her research interests and contributed to the concept for her doctoral dissertation, which examined the cognitive and neural correlates of coping and resilience in depression. She completed her clinical internship with a neuropsychology emphasis at the Minneapolis Veterans Affairs (VA) Health Care System and is currently completing a two-year postdoctoral residency in clinical neuropsychology at NorthShore University HealthSystem in Evanston. She hopes to become a board-certified clinical neuropsychologist and plans to continue participating actively in scientific and clinical research.