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## Affect and Cognitive Control: The Influence of Naturalistic Mood on Interference Processing

Lorri A. Kais

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LOYOLA UNIVERSITY CHICAGO

AFFECT AND COGNITIVE CONTROL:

THE INFLUENCE OF NATURALISTIC MOOD ON INTERFERENCE PROCESSING

A DISSERTATION SUBMITTED TO

THE FACULTY OF THE GRADUATE SCHOOL

IN CANDIDACY FOR THE DEGREE OF

DOCTOR OF PHILOSOPHY

PROGRAM IN CLINICAL PSYCHOLOGY

BY

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## CHAPTER ONE

### STUDY OVERVIEW AND RATIONALE

Historically, cognitive functions have been studied within a vacuum void of affective input or grounded within discrete diagnostic classifications, such as depression or schizophrenia (Blanchette & Richards, 2010; Mitchell & Phillips, 2007). More recently, academic and clinical interests regarding the interaction between dimensional affective processes and cognition have increased (Blanchette & Richards, 2010; Blanchard-Fields, 2005; Mitchell & Phillips, 2007; Yiend, 2004), largely in response to the National Institute of Mental Health (NIMH) Research Domain Criteria Initiative (RDoC; Insel & Cuthbert, 2009). In order to more accurately characterize dimensions of function that underlie the range of normal to abnormal human behavior, the RDoC encourages applying dimensional approaches to transcend traditional categorical nosology regarding normal and abnormal psychological function and integrate information across multiple methods/levels of analysis (Insel & Cuthbert, 2009).

Within the broader domain of cognitive systems, RDoC identifies cognitive control as a dimension of human behavior necessitating further investigation. Cognitive control processes, which are also commonly referred to as executive functions (EFs), are the mechanisms through which humans use internal intentions to guide thought and behavior (Banich, 2009). Miller and Cohen (2001) describe cognitive control as higher-order processes that optimize and schedule lower-order processes. Further, cognitive



control processes are engaged when prepotent (predominant, more automatic) modes of responding are not sufficient to meet contextual demands experienced during daily life as well as in completion of experimental tasks. The ability to refrain from prepotent response patterns in favor of more regulated responding requires the engagement of inhibitory-related cognitive control processes.

For almost a century, modified versions of the classic Color-Word Stroop Test (CWST, Stroop, 1935; Klein, 1964; Delis et al., 2001) have been utilized to investigate cognitive function, including inhibitory-related cognitive control processes (Harnishfeger, 1995; MacLeod, 1991). The CWST requires cognitive control processes to engage during conflict situations to disregard irrelevant stimulus information and maintain task instructions (thus ignoring a prepotent response option) in order to make an appropriate response. A correct response in the context of conflicting stimulus input reflects the successful processing of interference. The CWST has been used to measure the range of normative EF abilities to deficits in pathological populations (Lufi, Cohen & Parnish-Plass, 1990; Wagner et al., 2006; West, 2004).

Despite common use of the CWST, little is known regarding the influence of naturally occurring state affect on the recruitment /allocation of cognitive control resources during interference processing. Affect is an individual's subjective experience of emotion (Diener, Suh, Lucas & Smith, 1999). Affect can be represented by state and trait components (Watson, Clark & Carey, 1988). While trait affect represents relatively stable patterns of emotional experience, state affect is thought to vary over time in relation to exogenous and endogenous factors (Watson, Clark & Carey, 1988). Within this context, state affect can also be referred to as an individual's mood or mood state, and reflects current emotional experience at time of

assessment. Although laboratory-based explorations of affect have largely utilized mood induction procedures to create greater positive or negative mood states within participants, individual differences in naturalistic state affect can be measured without induction (Blanchette & Richards, 2009; Mitchell & Phillips, 2007). While induced mood differentially influences components of cognitive control (Bartolic, Basso, Schefft, Glauser, & Titanic-Schefft, 1999; Blanchette & Richards, 2009; Mitchell & Phillips, 2007), understanding how everyday mood impacts EF is not as well characterized.

Most behavioral and neuroimaging studies of the CWST in “healthy” populations have not explored the potential role of natural state affect or previous psychopathology, yet evidence from several studies indicate that these factors may indeed play a role in behavioral and neurophysiological response (Heller, 1993; Liotti, Mayberg, McGinnis, Brannan, & Jerabek, 2002). In fact, the interplay between cognitive and affective processing is evident within the structural and functional organization of the cortex, such that frontocingulate cortical structures that support affective function also are engaged during cognitive tasks, and vice versa (Mohanty et al., 2007).

Electroencephalography (EEG) is a noninvasive method of monitoring electrical activity elicited by the brain. EEG measures excitatory and inhibitory postsynaptic potentials of highly synchronized cortical neurons (Luck, 2012). Recorded EEG activity may be time-locked to specific events in order to analyze event-related potentials (ERPs). ERPs are stereotyped electrophysiological responses that are recognizable as peaks and troughs within the EEG waveform. ERPs are observed in response to endogenous or exogenous stimuli and provide measurement of neural response within milliseconds of stimulus onset. The temporal specificity

of event-related potentials (ERPs) makes them an ideal vehicle for examining the sequential dynamics of affective state on cognitive control processes engaged during the CWST. Previous investigations have identified several ERP components relevant to the CWST; however, methodological differences across studies have contributed to mixed findings.

The purpose of this study is two-fold. First, this investigation aims to further clarify/elucidate the nature and time course of cognitive resources engaged during the CWST within a sample of healthy controls and participants with remitted depression. Healthy controls were identified as individuals with no current or lifetime history of psychiatric illness and individuals with a lifetime history of depression who do not currently meet diagnostic criteria for a major depressive episode were included in the remitted depressed group. Consistent with previous research, this study proposes that neural resources will be differentially recruited and allocated dependent on task-related demands (presence or absence of interference). Secondly, given the established interplay between affect, cognition and behavior, the present investigation also aims to investigate how naturally occurring positive mood will influence recruitment of cognitive resources and behavioral performance during interference processing.

Investigating dimensional variations in state affect, including naturally occurring positive mood, is important as affect has been found to influence abilities essential for day-to-day functioning. Greater understanding of the influence of naturally occurring state affect on EF has implications for cognitive interventions, as cognitive demands are likely influenced differently by high and low levels of positive and negative affect. The exploration of state affect and EF may help to answer questions such as, “how do individual factors influence the ability to overcome cognitive conflict/interference and effectively direct behavior and actions?” Naturally

occurring Positive affect is theorized to be a key individual difference factor that influences the neural correlates of cognitive function.

## CHAPTER TWO

### REVIEW OF THE RELEVANT LITERATURE

#### **Affect**

Variations in mood (affective state) and dispositional affectivity (affective trait) have been found to significantly influence everyday activities by modulating thoughts and actions (Blanchette & Richards, 2010). Foundational literature has established the dominance of two dimensions of valenced affective experience: Positive Affect (PA) and Negative Affect (NA) (Davidson, 1998; Heller, 1993; Russell & Carroll, 1999; Russell & Pratt, 1980; Watson & Clark, 1991; Watson & Tellegen, 1985). The present project conceptualizes PA and NA as discrete variables in accordance with the seminal works of Watson, Clark, and Tellegen (Clark & Watson, 1991; Watson & Tellegen, 1985; Watson & Clark, 1991; Watson, Clark & Tellegen, 1988); however, it should be acknowledged that the extant literature is charged with debate as to whether PA and NA represent two relatively orthogonal facets of affect, or are better conceptualized as bipolar extremes of a singular dimension (Barret & Russell, 1999; Carroll, Yik, Russell, Barret, 1999; Kendall et al., 2015; Russell, 1980). While recent research has explored additional affective dimensions of intensity, frequency, and arousal (Brown, Chorpita & Barlow, 1998; Diener, Larsen, Levine & Emmons, 1985; Heller, 1993; Larsen & Diener, 1987), the present investigation focuses primarily on PA

State affect/mood represents transient fluctuations in emotional experience. As such, methods assessing state affect focus on an individual's current experience of emotion across a limited span of time (e.g., several hours or a day). State PA reflects how much an individual feels enthusiastic, active and alert across a specified time constraint. Individuals experiencing high state PA often endorse affirmative mood states such as feeling pleased, engaged, and/or energetic. On the opposing extreme of the PA dimension, individuals experiencing low state PA often describe feelings of lethargy and apathy. Therefore low state PA is characterized by the absence of pleasantness, vigor, and interest. State NA reflects subjective feelings of distress and engagement in unpleasurable experience (Watson & Clark, 1997; Watson & Tellegen, 1985; Watson & Naragon-Gainey, 2010). An individual experiencing high state NA may describe multiple aversive states such as anger, fear, nervousness, disgust, and hatred. In contrast, low state NA is effectively represented in tranquility and peace.

### **Positive and Negative Affect: Distinct but Related Dimensional Psychological Constructs**

Building upon previous findings (Bradburn and Caplovitz, 1965), Bradburn (1969) proposed that psychological well-being is best conceptualized as a function of two independent dimensions, positive and negative affect. Citing evidence from several national samples, Bradburn (1969) found, that when measured separately, PA and NA varied independently. In other words, the amount of PA endorsed by an individual did not substantially correlate with the level of endorsed NA. Prior to Bradburn's seminal works, most researchers had conceptualized affect as a single hedonic dimension with PA and NA defined and measured as bipolar opposites. By allowing positive and negative affect to vary independently, subsequent research has found that PA and NA often correlate differently with other psychological and neurobiological

variables (Cherlin & Reeder, 1975; Harding, 1982; Warr, 1978; Heller, Nitschke & Miller, 1998; Mitchell & Phillips, 2007).

The two-dimensional structure of PA and NA has also been validated and applied within the empirical and clinical domains of psychopathology. Given significant overlap in symptomatology/diagnostic comorbidity between depressive and anxiety disorders, clarifying etiological distinctions and maintenance factors of illness became a significant focus of work in order to improve measurement sensitivity and specificity. The tripartite model of depression and anxiety theorizes that low positive affect (PA) is specific to depression and anxious arousal is specific to anxiety. The model also asserts that high NA is a common factor of both depression and anxiety (Clark & Watson, 1991; Watson, Clark & Carey, 1988; Watson, Clark & Tellegen, 1988). High NA has also more broadly been viewed as a potential general risk factor for psychopathology (Clark and Watson, 1991, 1994; Mineka et al. 1998) and low PA has been specifically related to the psychiatric construct of anhedonia (Blanchard, Mueser, & Bellack, 1998; Crawford & Henry, 2004; Kendall et al., 2015). Watson and colleagues (1995) directly tested the predictions of the tripartite model on student, adult, and psychiatric patient samples through the administration of the Mood and Anxiety Symptom Questionnaire (MASQ; Watson & Clark, 1991). Findings indicated superior diagnostic classification of depressive and anxiety disorders with incorporation of tripartite model (i.e., depression was differentiated via low levels of PA).

**Evidence from neuroscience.** Complementing conventional research that has postulated PA and NA as distinct but related affective dimensions, human neuroscience research has treated these primary affective dimensions in a similar manner with the goal of mapping them onto brain structures and functions. Dating to observations from lesion studies in the early 1970s (e.g.,

Gainotti, 1972), affect implementation in the brain has been conceptualized as lateralized, with structures supporting pleasant affect lateralized to left prefrontal cortex (PFC) and those supporting unpleasant affect lateralized to right PFC (Heller, Nitschke, & Miller, 1998). Results of electroencephalographic (EEG) studies have consistently evidenced increased cerebral activation in the left hemisphere relative to activation of the right hemisphere during positive mood states (Allen & Kline, 2004; Cacioppo, 2004; Davidson, Ekman, Saron, Senulis, & Friesen, 1990; Heller, 1993; Heller, Nitschke, Etienne, & Miller, 1997; Heller, Nitschke, & Miller, 1998) as well as relative greater activation in the right hemisphere compared to left during negative mood states (Davidson et al., 1990; Heller, Nitschke, & Miller, 1998; Lee et al., 2004). Blunted positive and negative affectivity have been associated with bilateral decreases in prefrontal cortical activity (Davidson, 1998). These findings further support the proposal that PA and NA should be treated as distinct but related dimensional psychological constructs.

In summary, a large body of work, ranging from self-report to neurophysiological methodologies, subdivides affect into two distinct yet related affective dimensions based upon valence (PA and NA). Additionally, variations in individual experience of PA and NA can be dimensionally assessed through self-report measures such as the Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988). Given the relative independence of these affective dimensions, a low rating of one dimension does not necessitate a high rating of the other dimension.

### **Affect and Cognitive Control**

Affective processing of both endogenous and exogenous information is fundamental to human behavior. As such, all decisions and actions occur in an affective context in which cognitive processes, such as cognitive control, are constantly modulated by affective state.



Commonalities across several theories of cognitive control suggest that cognitive control processes are engaged when: (a) resisting distracting/task-irrelevant input; (b) inhibiting familiar/stereotyped behaviors; (c) creating and maintaining attentional set; (d) prioritizing/sequencing behaviors; (e) shifting task-demands; (f) utilizing relevant information for decision making; (g) organizing/categorizing conceptually-related information about stimuli; and (h) processing novel input regarding task demands and situational context (Banich, 2009; Friedman & Miyake, 2004).

A growing body of work provides evidence for the role of affect in cognitive control processes. While many studies have examined differences in cognitive control processes across categorical classifications of abnormal affective function (i.e., various psychiatric illnesses), less is known regarding the dimensional influence of naturally occurring positive and negative mood states on cognitive control. The following section first examines psychological theories of affect and cognitive control and then reviews empirical investigations of cognitive processes in the context of affect. While only literature investigating the effects of affect/mood on cognition will be examined, it should be acknowledged that executive processes may play an important role in the maintenance and regulation of mood as well, as is likely true in the context of cognitive reappraisal (Hofmann, Schmeichel & Baddeley, 2012; Ochsner et al., 2004; Buhle et al., 2014).

### **Psychological Theories of Affect and Cognitive Control**

Several psychological theories posit unique implications when examining the effects of mood or state affect on cognition/cognitive control. While the majority of research supporting these theories has focused on induced-affect/mood, considerations may be drawn for natural state characteristics as well.

**Affect and cognitive load/resource depletion.** This framework of affect and cognitive control underscores that human processing capacity is limited (Kahneman, 1973) and that competition for neural resources will occur under conditions of dual demands (Broadbent, 1958; Treisman & Gelade, 1980; Underwood, 1977). As such, both positive and negative affective states would be expected to burden cognitive resources in comparison to neutral states due to the associated increased activation of widespread networks involved in emotion-related processing. For example, the presence of high NA has been associated with rumination about mood-relevant thoughts, which may be task-irrelevant (Mitchell & Phillips, 2007; Ottowitz, Dougherty & Savage, 2002). According to resource depletion theory, the engagement in task-irrelevant processing decreases available resources/capacity to engage in presented cognitive tasks. Therefore, according to this framework high levels of both PA and NA would have detrimental effects on cognitive control tasks, including inhibitory-related functions tapped during Stroop interference processing.

**Affect and information processing style.** The feelings-as-information framework set forth by Schwarz, Clore and colleagues (Schwarz, 1990; Schwarz & Bohner, 1996; Schwarz & Clore, 1996) initially postulated that an individual's present affect informs him or her about the "goodness" or "badness" of the environment, which then influences subsequent information processing style. PA has been associated with increased utilization of heuristic and creative processing styles in comparison to neutral moods (Bless, Bohner, Schwarz, & Strack, 1990; Bodenhausen et al., 1994; Bohner, Chaiken & Hunyadi, 1994). It is thought that given the absence of threats in the environment (as assumed when experiencing PA), an individual's motivation to alter his or her environment is attenuated and therefore opens resources for more creative processing styles. In contrast, experience of NA generally signals that the environment

is problematic and that action should be taken to alter current circumstances. Therefore, according to the feelings-as-information framework, NA results in an information processing style that is relatively systematic, effortful, and vigilant.

Others have also categorized PA as inducing more global/holistic information processing attributes, with negative affect prompting a more localized/focused analysis of information (Bolte, Goschke & Kuhl, 2003; Fredrickson & Branigan, 2005). Similar to the feelings-as-information framework discussed above, the broaden-and-build theory set forth by Fredrickson (1998; 2001) contends that positive emotions broaden attentional scope and expands transient thought-action repertoires. By increasing cognitive access to various potential thoughts and actions, positive emotion ultimately increases the likelihood of engaging in thoughts and actions beyond basic functional requirements. Examples of thoughts and behaviors related to positive emotion include play, exploration, savoring, and complex integration (Fredrickson, 2001). The theory also highlights the adaptive benefits of select negative emotions (e.g., anger and anxiety) momentarily limiting available thought-action repertoires to increase likelihood of engaging in context-appropriate action (Fredrickson & Branigan, 2005). A number of empirical studies support the broaden-and-build account of mood on attentional focus. For example, Biss, Hasher and Thomas (2010) found that individuals in a positive mood were more likely than others to utilize previously irrelevant information (distractor stimuli on a previous task) to facilitate performance on a subsequent implicit task. Results from this study support a broader, more global focus of processing under conditions of high PA as positive mood was associated with implicit use of distraction.

Rowe, Hirsh and Anderson (2007) investigated the effects of positive, neutral, and sad mood induction on visuospatial (Eriksen Flanker task) and conceptual (remote associates task,

RAT) attentional domains. No significant differences were found on performances for either task between sad and neutral inductions, however positive mood induction was related to increased access to semantic associations on the RAT and impaired visual selective attention on the Flanker task. Together, these results were interpreted to suggest that high PA attenuates inhibitory control to increase breadth of conceptual and visuospatial attention.

Storbeck and Clore (2005) examined whether induced positive or negative moods would influence the formation of false memories in two related studies. Utilizing the Deese-Roediger-McDermott paradigm, which lures individuals to produce false memories, Storbeck and Clore (2005) found that negative mood resulted in attenuated false memory effects compared to positive mood and non-manipulated mood groups. To further examine whether this effect took place at the point of encoding or retrieval, a second study required participants to list words they identified as related to the target list of words but were not actually included on the list. Results indicated no interaction between lure identification type (lure listed as a part of the target list or lure identified as a related word) and mood induction. However, a main effect for mood induction found that individuals within the negative mood group were less likely to identify lures in general, which supported differences in mood-related processing style at the stage of encoding rather than retrieval. Overall, these findings suggest negative mood is related to a more refined, limited-scope of processing when encoding information in comparison to positive mood.

*Affect-as-cognitive-feedback account.* Emerging in response to empirical evidence contradicting the fixed effects of PA and NA on cognition, affect-as-cognitive feedback theory suggests that the relation between affect and cognitive processing is malleable (Huntsinger 2014). This viewpoint asserts that the impact of mood on perceptual processing/attentional scope depends upon accessibility of global or local processing orientations, with positive mood

generally amplifying whichever processing style is contextually dominant. This theory also suggests that when global and local orientations are equally accessible, differences in mood will not be related to differences in attentional scope.

Several studies provide supporting evidence for this theory. Huntsinger, Clore and Bar-Anan (2010) examined the relation between positive mood and processing focus (global or local) across two experiments with each utilizing different mood induction, perspective priming, and assessment methodologies to increase generalizability. Results from both studies indicated that positive mood amplified processing type dependent on which focus was primed. In other words, positive mood was not wholly related to global processing, but rather facilitated adaptation of context-specific processing in comparison to negative mood. These findings were further substantiated in more recent work by Huntsinger (2012), which examined the effects of focal priming on performance during a flanker task. Flanker tasks assess an individual's perceptual attention and inhibitory control. The task requires participants to focus on a target stimulus while inhibiting attention to distracter stimuli flanking it. Results indicated that positive mood was related to adaptation of whichever perceptual orientation was primed. Further, when global and local orientations were primed equally, affect was not found to influence scope of attention.

Given the above theoretical frameworks, it is evident that the pertinent question is not *whether* mood state influences cognition/cognitive control, but rather *how* mood state influences these higher order processes. Models rooted in concepts of limited capacity and resource depletion, suggest that affective input is deleterious to cognitive control processes as it occupies related and limited resources. In contrast, recent advances in theories of information processing and mood suggest an increasingly malleable role between cognitive approach and affective state. More specifically, high positive affect increases adaptability to whichever processing style is

most accessible given task context. As the vast majority of existing processing style literature has focused on laboratory induced-mood, the relation between naturally occurring mood states and information processing style warrants further investigation.

### **The Effect of Mood State on Cognitive Control**

While a substantial amount of literature has examined the influence of pathological levels of negative affect (e.g., unipolar and bipolar depressive disorders) on cognitive control, less is understood regarding sub-threshold dimensional effects of negative mood. Although several studies have shown that NA influences cognitive control processes in the context of affective stimuli, null findings from a number of studies exploring NA and cognitive control (Blanchette & Richards, 2010; Mitchell & Phillips, 2007) suggest that NA may have a more limited role in cognitive control processes than PA. In contrast, PA has been related to increased cognitive flexibility, creativity, and globally focused processes. High PA has also been related to impairments in working memory and inhibition of irrelevant information. Relevant empirical investigations are discussed below.

Martin and Kerns (2010) investigated the effect of positive mood on working memory and inhibition. In two studies, participants underwent positive or neutral mood induction via video clips and then completed a task of working memory storage capacity (Running Memory Span) and prepotent response inhibition (CW-Stroop or Flanker). Results indicated that positive mood induction resulted in poorer performance on the test of working memory in comparison to neutral mood induction for both studies. No effect for positive mood induction compared to neutral was found in either study of inhibitory-related function, suggesting that positive mood differentially affects cognitive control functions by impairing working memory but having no effect on inhibitory-related function. However, it should be noted that these inhibitory-related

findings contrast the findings of Rowe, Hirsh and Anderson (2007), who found that positive mood was related to impaired performance on a flanker task.

The differential effect of positive mood on specific EFs was also examined by Phillips, Bull, Adams and Fraser (2002). Subsequent to neutral or happy mood induction, thirty-six participants completed four conditions of the CW-Stroop task. Compared to neutral mood induction, positive mood was found to impair performance on the switching condition of the Stroop test. In a second study, sixty participants underwent neutral or positive mood induction and then completed three verbal fluency tasks. Positive mood was related to improved performance on a test of verbal fluency requiring the naming of as many novel uses for a cup as possible. Significant differences for mood condition were not observed for fluency tasks based on initial word letter or alternating between semantic category and phonemic criteria. Overall, results from these studies suggest that positive mood is related to increased fluency requiring creative responses, but resulted in poorer performance on a task of cognitive flexibility that did not require creative response suggesting that PA facilitative effects on cognitive flexibility may depend on level of creativity/engagement in novel processes required to complete demands.

Chepenik, Cornew and Farah (2007) induced and prolonged sad and neutral mood in thirty-three healthy volunteers across two laboratory sessions. Following mood induction participants completed seven cognitive tasks. Tasks assessed working memory (Object Two-Back, Digit Span), inhibitory-related function (CW-Stroop, Go/No-Go), and attention/perception/memory for valenced materials (Attention Probe, Free Recall and Recognition Memory, Facial Emotional Recognition). Sad mood was found to affect memory for emotional words and facial emotion recognition, but not performance on other tasks. Sad mood was found to bias memory toward negatively valenced words relative to neutral mood

conditions, however overall percentage of word recognition was not affected by mood. Sad mood was also found to impair facial emotion recognition compared to neutral mood and this effect was generalized across recognition of neutral, happy and sad faces. Results of this study indicated that sad mood influences cognitive processes that involve an emotional stimulus, but not cognitive tasks that do not explicitly involve emotional stimuli.

In another study, 14 student volunteers completed a CWST paradigm across three blocks of positive, neutral, and negative mood induction (Yuan et al., 2011). Behavioral results did not differ between mood induction groups regarding response latency or accuracy. The Stroop-related N450 ERP component, which is related to conflict effects emanating from anterior cingulate cortex and discussed in more detail later, was more pronounced for individuals in the positive mood induction group but not the negative mood group in comparison to the neutral mood state. Results of this study suggest increased neural processing was required for the positive mood induction group to achieve similar behavioral results. Results of this study also indicate that investigations utilizing only behavioral methodologies may not be adequate to capture the nuanced effects of mood on cognitive control processes.

Laboratory-induced mood has been related to specific effects in cognitive control processes engaged during the CW-Stroop task. However, to date no investigations have explored the dimensional affect of naturally occurring PA on Stroop interference and related neural response.

### **Color-Word Stroop Task (CWST)**

Recent efforts towards providing an integrative and comprehensive account of cognitive control have consistently identified inhibitory-related function as a core component of EF (Banich, 2009; Friedman & Miyake, 2004). Inhibitory-related control processes include the



ability to suppress irrelevant or interfering information at the sequential levels of processing and response output. Demands related to everyday life necessitate an ability to control impulses such as inhibiting prepotent responses or delaying gratification.

Modified versions of the classic CWST (Stroop, 1935; Klein, 1964) have been frequently utilized to investigate inhibitory-related function. A typical CWST paradigm involves several task modules, eliciting a number of neural and behavioral response patterns from participants. Module variations may include: (a) *word-reading*, reading color-words (e.g., “BLUE” or “RED”) or non-color words (e.g., “MATH” or “BUN”) in black/grey or color ink; (b) *color-naming*, naming ink of color swatches or symbol series/non words (e.g., “XXXX” in red); (c) *congruent-word color-naming*, naming matched ink color of word, such as the word “RED” in red ink; (d) *incongruent-word color-naming*, naming mismatched ink color of word, such as the word “RED” in blue ink; and (e) *switching trials*, which instruct participants to switch between task demands (word-reading or color-naming) in the context of incongruent and congruent stimuli throughout the condition.

Additionally, color-naming modules may be presented in “blocked” form during which the entire presentation of stimuli is of the same congruency (e.g., all congruent or all incongruent), or “mixed” during which the presentation of stimuli alternates between congruencies (e.g., both congruent and incongruent types are present within same module). One of the most robust findings of the CWST is commonly referred to as the *Stroop interference effect*, described in detail below.

### **Stroop Interference**

Classic CWST paradigms direct participants to focus on one component of a stimulus, typically ink color, while ignoring other facets of the stimulus, including the written word.

Computational and mathematical models assert that independent pathways exist for processing color and word information (Cohen & Servan-Schreiber, 1992; Miller & Cohen, 2001).

Therefore, interference is observed when the task-irrelevant dimension conflicts with the relevant dimension resulting in different input for each pathway.

As the CWST elicits conflict between more automated response (word-reading) and more controlled action (color-naming), it requires the engagement of two related inhibitory control processes in order to produce a correct response. First, interference control reflects the ability to selectively inhibit the processing of irrelevant information (Nigg, 2000). In the context of the typical CWST, the irrelevant information is the written word. The other inhibitory-related function is prepotent response inhibition, which is the ability to modulate dominant or automatic responses (Friedman & Miyake, 2004). A prepotent response is a reflexive response due to habituation or the presence of immediate positive/negative reinforcement. In the case of the CWST, word-reading represents the more habituated/automated response which must be inhibited in choice of the more controlled action of color-naming.

Within the context of the CWST, interference is observed as an increase in response latency under incongruent task conditions in comparison to neutral or congruent conditions (hereafter referred to as Stroop interference – reaction time). In some studies, interference has also been observed as an increase in relative number of errors under incongruent task conditions in comparison to neutral or congruent conditions (hereafter referred to as Stroop interference – accuracy). Participants with typical levels of interference control generally have high accuracy scores and shorter response times for the congruent trials with somewhat lower accuracy scores and increased response latency for incongruent trials. Participants with low interference control generally demonstrate a larger difference in accuracy scores and response latencies between

congruent and incongruent conditions compared to participants with high interference control abilities.

Several theories have emerged in efforts to explain the observed behavioral and neural implications of Stroop interference. Theories of early processing, limited response capacity, and parallel processing are described below.

**Perceptual encoding.** The perceptual-encoding perspective provides an early processing theory of Stroop interference. In comparison to theories placing emphasis on processing after encoding, theories emphasizing encoding failed to garner significant theoretical popularity or empirical support (MacLeod, 1991). Broadly, this perspective suggests that the perceptual encoding speed for information regarding ink-color (e.g., blue) is attenuated due to conflicting information from an incongruent color word (e.g., “RED”) in comparison to a neutral word (e.g., “STEP”). Support of this theory comes from the work of Hock and Egeth (1970). They altered the CWST to require binary verbal classification of colors (“yes” or “no”) to remove the semantic relation between responses and stimuli. Interference for incongruent stimuli was still observed. Hock and Egeth (1970) interpreted these results as suggesting that interference occurred at the stage of implicit color identification/coding rather than at the stage of overt response competition. Others have criticized this assumption, indicating that interference at the level of response is still present despite binary demands (Dalrymple-Alford & Azkoul, 1972; Dyer, 1973).

**Relative speed of processing.** This theory posits that Stroop interference occurs due to the differential speed of processing of relevant (ink color) and irrelevant (word) stimulus information, that ultimately converges upon a singular response channel that only has room for one input (Morton & Chambers, 1973). As this theory posits that only one piece of information

(color or word) may be accepted into the response channel at a time, priority of response is determined by the *speed* of processing attributed to the respective information pathway (color or word). This results in a sort of “attentional bottleneck” to be the first input to reach the response stage. Typically words have been found to be read faster than colors are named (Cattell, 1886; Fraisse, 1969), therefore this theory suggests that Stroop interference occurs when irrelevant stimuli information (word) reaches the response output stage before slower relevant information (ink color) resulting in conflict in selecting the correct color response (MacLeod, 1991). Evidence against a basic speed-of-processing account comes from studies in which the color and word components of the Stroop stimulus were presented with varying stimulus onset asynchronies (SOAs; Dyer, 1971; Glaser & Glaser, 1982; Sugg & McDonald, 1994). If interference in the CW-Stroop task is primarily due to word meaning being processed more quickly than ink color, classic interference should be eliminated if color information is presented sufficiently before a word resulting in reverse Stroop interference/facilitation. To date, this pattern of results is not typical within SOA empirical efforts suggesting that a speed of processing account does not sufficiently explain Stroop interference (MacLead 1991; Maclead 2015).

**Continuum of automaticity.** Similar to the speed of processing theory, an automaticity theory of Stroop interference is defined by the parallel processing of multiple stimuli characteristics that ultimately bottleneck at the stage of response, as these theories suggest only one stream of processing may be inputted into the response selection stage at any given moment. This theory differs slightly from the speed of processing theory by emphasizing degree of information automaticity and related recruitment of attentional/processing resources (LaBerge & Samuels, 1974; Posner & Synder, 1975). While purely automatic processes are defined as

operating outside the realm of volitional attention, other processes can develop strong automaticity through learning/practice. Processes with high levels of automaticity are believed to interfere with less automatic processes (MacLeod & Dunbar, 1988). Kahneman and Chajczyk (1983) categorize Stroop interference as an example of partially automatic forces (word reading) at odds with less automatic forces (color naming). According to this theory, Stroop interference occurs because the level of automaticity is stronger for word reading than color naming, thus naming ink color recruits more heavily from attentional resources than reading, ultimately increasing response latency when ink color and written word are incongruent. Within this context, word reading is often viewed as obligatory (given its large level of automaticity), while color naming remains optional requiring top-down attentional control to engage more effortful processing.

**Parallel distributed processing model.** Expanding upon the work of Logan (1980), Cohen, Dunbar and McClelland (1990) put forth one of the most comprehensive models of Stroop interference to date. The Parallel Distributed Processing Model includes many of the strengths established in speed of processing and automaticity theories while exchanging the limited serial stage approach for a more inclusive system loop. Emphasis within this model is placed on pathway strength and not relative speed. Level of automaticity is determined as a function of the strength of each pathway and pathways are strengthened through practice. Pathways necessitating fewer processing resources are stronger at lower levels of engagement compared to those less automated pathways. The volitional direction of cognitive resources can increase pathway strength. Therefore, the likelihood and degree of interference occurring is determined by the relative strengths and information processing capacities of each engaged pathway (MacLeod, 1991). In the context of the CWST, the engaged pathways would be color

and word information. This theory refutes the limited-capacity of a singular response channel (assumed in both speed of processing and automaticity theories) and instead suggests that there can be as many response channels as there are responses. Whichever response channel is most strongly activated will ultimately produce the response.

### **Neural Correlates of Stroop Interference**

Empirical evidence from a number of electrophysiological and functional imaging studies indicate involvement of several regions of the brain during interference control, including the anterior cingulate cortex (ACC), the dorsolateral prefrontal cortex (dlPFC), posterior parietal cortex, and inferior parietal cortex (Egner, Delano & Hirsch, 2007; MacLeod and MacDonald 2000; Peterson et al. 2002; Siltan et al., 2010; Van Veen & Carter, 2005). ERP studies provide the temporal sensitivity necessary to identify the rapid succession of events related to interference processing. While several ERP components have been associated with Stroop interference, variable findings suggest that the time course of neural processes engaged during Stroop interference remains an open question.

ERPs indicate how the brain responds to a specific event. ERP waveforms are typically described in terms of latency, amplitude, and scalp distribution and are generally calculated using the averages of numerous trials. Latency refers to the temporal onset of the ERP in relation to the event it is “locked” to (e.g., stimulus onset or response). Amplitude reflects the difference in voltage between a given electrode site (or collapsed grouping of sites) and the assigned reference point. Amplitudes can be positive inflections or negative deflections within the EEG waveform. Standard nomenclature for ERPs often reflect both amplitude and latency characteristics, typically beginning with a “P” for positive inflections and “N” for negative deflections followed by numbers to indicate general latency. ERP amplitudes are typically

measured/reported in microvolts ( $\mu\text{V}$ ). Common methods to calculate amplitudes include instantaneous (amplitude at a single latency), peak (the most positive or most negative point within a specified time window), and mean amplitude (averaged voltage within a specified time window) (Luck, 2012; Sur & Sinha, 2009; Woodman, 2010). Associations between neural correlates (ERP latencies and amplitudes) and neural processes must consider how contextual factors influence these variables (Luck, 2012; Sur & Sinha, 2009). Previous literature investigating the time course of the CWST has analyzed various ERPs including the N200, P300, N450 and a late slow wave component discussed below in order of temporal onset.

**N200.** The N200 is a negative deflection that peaks approximately 200 to 250 ms following the stimulus presentation. The N200 encompasses a family of neural responses that differ based upon eliciting context, such as stimulus modality (Ceponien, Rinne & Naatanen, 2002; Donchin, Ritter & McCallum, 1978; Sur & Sinha, 2009). Subcomponents within the N200 wave include N200a/Mismatch negativity (N200a/MMN), N200b, and N200c. MMN is elicited subsequent to an infrequent change in repetitive stimuli in any sensory modality, but has been most frequently studied utilizing auditory paradigms and has a frontocentral distribution (Naatanen, Paavilainen, Rinne & Alho, 2007; Pazo-Alvarez, Cadaveira & Amenedo, 2003). The N200b, also located frontocentrally, occurs slightly later than the N200a following a task-relevant change in the stimulus characteristic (Pritchard, Shappell & Brandt, 1991; Folstein & Petten, 2007). The N200b has been related to the detection of perceptual novelty, response conflict, error monitoring, and inhibiting a prepotent response system (Folstein & Petten, 2007). The N200c presents as posterior maximal for visual stimuli and is elicited when the classification of disparate stimuli is required, as in identifying targets from non-targets (Pritchard, Shappell & Brandt, 1991; Folstein & Petten, 2007). The N200 wave has been observed in CWST paradigms

(Holmes and Pizzagalli, 2008; Siltan et al, 2010) as well as in other visual interference and conflict monitoring tasks (Folstein & Petten, 2007; Kopp, Rist & Mattler, 1996; Yeung, Botvinick & Cohen, 2004). Relevant to the CWST, the N200b has differentiated between congruencies, with greater amplitudes for incongruent than congruent conditions.

**P300 (P3).** The P3 is often considered an index of cognitive control and has been implicated in several CWST studies (e.g., Badzakova-Trajkov, Barnett, Waldie, & Kirk 2009; Duncan-Johnson & Kopell, 1981; Ilan & Polich, 1999). The P300 is a positive inflection peaking approximately 300 ms following stimulus onset and was first reported over 50 years ago (Sutton et al., 1965). The P300 has been broadly regarded as a marker of task-relevant stimulus processing/stimulus evaluation and is further subdivided into the P3a and P3b subcomponents (Polich, 2007). The P3b is regarded as the “traditional” P300 and is observed to have maximum amplitude in parietal regions and it is typically elicited in tasks with two types of stimuli of unequal probability, with larger P3b amplitude observed for the infrequent stimuli type (Polich, 2007).

In contrast, the P3a evidences a fronto-central distribution and is related to distracter information, such as the written word in the context color-naming demands (Polich, 2007). The N200b is consistently observed in combination with the P3a, a subcomponent of the P300. Several studies have found that the P3a amplitude elicited by incongruent stimuli is smaller than that elicited by congruent stimuli (Houston et al., 2004; Ilan & Polich, 1999). Badzakova-Trajkov, Barnett, Waldie, and Kirk (2009) found no discernible difference between congruent and incongruent conditions at P3, but both elicited larger P3 than neutral conditions at central and parietal sites. Other studies have also found no difference between congruent and



incongruent conditions regarding P3b amplitude (Hanslmayr et al., 2008; Liotti et al., 2000; Markela-Lerenc et al., 2004).

**N450.** The N450 is one of the most commonly identified ERP components associated with the CWST. Distinguishable as a negative-going deflection 300-500ms following stimulus onset with fronto-central topography, The N450 ERP waveform typically peaks around 450ms following the presentation of a stimulus with high levels of conflict (Liotti et al., 2000; van Veen & Carter, 2002; West & Alain, 1999). Results regarding the N450 are robust, with studies consistently reporting that incongruent conditions elicit increased amplitude relative to congruent or neutral conditions (West, 2003; West, Jakubek, Wymbs, Perry & Moore, 2005). ERP source localization efforts have consistently found that dipoles placed within the ACC account for the most variance in the topography of the N450 (Liotti, Woldorff, Perez & Mayberg, 2000; Szucs, Soltesz & White, 2009; Hanslmayr et al., 2008). Further, evidence accrued from functional magnetic resonance imaging studies (fMRI) have implicated increased ACC activity in response to incongruent compared to congruent stimuli, consistent with a role in conflict processing (MacDonald et al., 2000). While some studies have argued that the N450 denotes the resolution of conflicting information (Liotti et al., 2000), others regard it as marking the detection of conflict (Hanslmayr et al., 2008). Seeking to clarify this discrepancy, Szucs and Soltesz (2012) utilized electro-myography (EMG) in conjunction with ERP to determine whether the N450 is related to stimulus or response conflict processing. Despite manipulating level of response conflict on a trial-by-trial basis (as confirmed by EMG recording), N450 effect remained stable suggesting that N450 is best conceptualized as a marker of stimulus conflict rather than response conflict.

**Late activity (Conflict SP).** Several studies have identified a conflict-sensitive slow potential (sometimes referred to as the Conflict SP) characterized by a sustained parietal positivity and lateral frontal negativity beginning around 500ms post stimulus onset. The Conflict SP has been found to be more positive following incongruent than congruent trials over temporo-parietal sites (Liotti, Woldorff, Perez III, and Mayberg (2000) and more negative in the same comparison at fronto-central sites (Hanslmayr et al, 2008). Liotti and colleagues (2000) referred to this later waveform as the Late Positive Complex (LPC) and suggested that this later activity may be related to additional processing of word meaning. Hanslmayr and colleagues (2008) offered a different perspective, referring to the component as the Late Negativity (LN); they suggested that the sustained activity likely reflected additional recruitment/engagement of cognitive control mechanisms necessary to select proper response in the context of interference. This interpretation is similar to one offered by West and colleagues (2005) who found that Conflict SP was correlated with overall RT and accuracy, thus suggesting that it may be more reflective of response selection. Additional research is needed to better characterize the time course, topography and functional implications of this late ERP complex that has been repeatedly observed in the context of the CWST.

Overall, the N450 remains the most reliable ERP marker of congruency effects in the CWST. While not as consistently addressed in CWST literature as N450, N200 has been related to the detection of response conflict and prepotent response inhibition in visual interference and conflict monitoring tasks. Findings remain mixed regarding processes indexed by P3 and localization of later sustained slow wave activity in the context of Stroop interference.. As prior research has not addressed the influence of naturally occurring PA on the recruitment/allocation of neural resources during interference processing, the present study will investigate the

influence of PA on the two most established ERP components related to interference processing, N450 and N200.

### **Research Overview: Aims & Hypotheses**

The present study investigated the relationship between naturally occurring positive mood state (PA) and interference processing during the Color-Word Stroop Task. This study proposed that neural resources are differentially recruited and allocated dependent on task-related demands (presence or absence of interference) and that implementation of these resources are influenced by levels of positive affect.

#### **Aim 1**

This study investigated the behavioral and neural mechanisms of interference processing during a modified CWST. Previous investigations utilizing ERPs have resulted in mixed findings; therefore the first aim of the present study was to establish the time course and pattern of activation of the most reliable ERP components related to interference processing (N200 and N450) for different stimulus presentations (blocked and mixed) within this specific version of the CW-Stroop task.

**Hypothesis 1a (reaction time).** A behavioral cost of interference is reflected in increased response latency (reaction time; RT) for incongruent compared to congruent conditions for both blocked and mixed presentations.

**Hypothesis 1b (accuracy).** A behavioral cost of interference is reflected in decreased accuracy (increased number of errors) for incongruent compared to congruent conditions for both blocked and mixed presentations.

**Hypothesis 2a (N200 amplitude)** Presence of interference elicits increased negativity at fronto-central electrode sites occurring approximately 200 – 300 ms post stimulus-onset (N200)

in incongruent compared to congruent conditions. This has been suggested to reflect the detection of response conflict and prepotent response inhibition.

**Hypothesis 2b (N200 latency)** Presence of interference results in delayed peak latency of a negative component occurring approximately 200 – 300ms post stimulus-onset (N200) in incongruent compared to congruent trials.

**Hypothesis 3a (N450 Amplitude).** Presence of interference elicits increased negativity at fronto-central electrode sites occurring approximately 350-500ms post stimulus-onset (N450) in incongruent compared to congruent conditions. This has been suggested to reflect the detection of interference and recruitment of central executive processing resources rather than the classical semantic incongruity effect (Hanslmayr et al., 2008).

**Hypothesis 3b (N450 Latency).** Presence of interference results in delayed peak latency of a negative component occurring approximately 350-500ms post stimulus-onset (N450) in incongruent compared to congruent trials.

## **Aim 2**

This study examined the associations between self-reported positive naturalistic state affect (PA) and performance on a modified CWST. Behavioral (RT and accuracy) and ERP (N200 and N450) indices of performance are analyzed. High Positive Affect (PA) is predicted to have a deleterious effect on CW-Stroop interference processing. As individuals with remitted depression were included, analyses also accounted for variance associated with diagnostic history.

**Hypothesis 1a (Reaction Time).** Based on previous mood induction findings, Stroop interference – RT is higher for individuals with Higher PA and lower for individuals with Lower PA for both blocked and mixed presentations.

**Hypothesis 1b (Accuracy).** Based on previous mood induction findings, increased Stroop interference – accuracy is associated with Higher PA for both blocked and mixed presentations.

**Hypothesis 2a (N200 Amplitude)** Higher levels of PA is related to increased N200 amplitude on incongruent trials as individuals with high PA will require increased neural resources to detect response conflict and inhibit irrelevant information for both blocked and mixed presentations.

**Hypothesis 2b (N200 Latency)** Higher levels of PA is related to increased N200 latency as individuals with high PA will have slower response time and slower recruitment of neural resources necessary to complete trials with interference (incongruent stimuli) for both blocked and mixed presentations.

**Hypothesis 3a (N450 Amplitude).** Higher levels of PA is related to increased N450 amplitude on incongruent trials as individuals with high PA will require increased neural resources to inhibit irrelevant information in order to process conflicting information for both blocked and mixed presentations.

**Hypothesis 3b (N450 Latency).** Higher levels of PA is related to increased N450 latency since individuals with high PA will have slower response time and slower recruitment of neural resources necessary to complete trials with interference (incongruent stimuli).

## CHAPTER THREE

### METHODS

#### **Study Overview**

The present research study was a component of a larger research study. After participants were deemed eligible for inclusion in the broader research study, they were invited for three laboratory visits during which they completed informed consent, a semi-structured clinical interview, self-report measures, and behavioral tasks while electroencephalography (EEG) data were recorded. The university Institutional Review Board approved all recruiting and experimental methods.

#### **Participants**

Participants in this study were native English-speaking undergraduate students from an urban, Midwestern university. Participants were recruited from a participant-pool based registry maintained by a university research laboratory.

Participants were excluded from the study if they endorsed any of the following exclusion criteria, which are known to influence EEG recording: 1) colorblindness, 2) neurological medical condition, 3) a sensory impairment (i.e., colorblindness) that would influence 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.

History and current status of depression was determined through diagnostic interview (Mini-International Neuropsychiatric Interview, MINI; Sheehan et al., 1998) that was based on criteria determined by the Diagnostic and Statistical Manual, 4<sup>th</sup> edition revised (DSM-IV-TR; American Psychiatric Association, 2000). Participants included in the present study either met DSM-IV-TR criteria for a lifetime history of depression but did not meet criteria for current depression (remitted depressed group) or never met DSM-IV-TR criteria for depression (control group). Individuals who met current diagnostic criteria for depression were excluded from the present study.

Participants ( $N = 49$ ) were paid \$15 per hour for their participation. Two participants were eliminated from further analysis for the Color-Word Stroop Task due to poor task performance (i.e., <50% accuracy on the mixed incongruent condition); eight participants were excluded from analyses due to low-quality EEG signal (e.g., number of rejected trials during cleaning, electrical noise).

The final sample included 39 participants with behavioral and EEG data on the Color-Word Stroop (37 with PANAS data). Of these 39 participants, 20 participants did not have a lifetime history of depression and were categorized as controls (age  $M = 19.75$ ,  $SD = 1.80$ , range = 18-24; 55% female; 62.7% White, 27.3% Asian American, 10% Hispanic/Latino). 19 participants presented with a history of depression without meeting criteria for current depression and were categorized as remitted depressed (age  $M = 19.53$ ,  $SD = 1.10$ , range = 18-22; 79% female; 68% White, 32% Asian American, 0% Hispanic/Latino).

## **Procedure**

**Demographic information.** Participants reported information regarding age, gender, handedness, and ethnicity collected online via survey software utilizing a tablet device in the research laboratory.

**Diagnostic evaluation/screening.** Participants completed a diagnostic interview using the MINI International Neuropsychiatric Interview (MINI; Sheehan, et al., 1998). In the present study, the MINI was used to assess for current presentation and history of major depressive episodes. Interviewers were master-level students who were trained and supervised by Rebecca Siltan, Ph.D. Accuracy of diagnosis was reviewed in consensus meetings in order to ensure consensus regarding diagnosis.

**Depression severity.** To evaluate current depression severity, the nine-item Patient Health Questionnaire (PHQ-9; Kroenke et al., 2001) was administered. PHQ-9 items are scored from “0” (not at all) to “3” (nearly every day) and are based on the depression criteria from the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition.

**State affect.** Participants completed the Positive and Negative Affect Scale (PANAS; Watson, Clark, & Tellegen, 1988) before completion of behavioral/EEG measures. The PANAS is a 20-item scale that measures the distinct constructs of positive and negative affect. Participants answer on a five-point Likert scale how accurately words describe their current mood. For example, negative affect words include "afraid, nervous, and guilty" and positive affect words include "active, enthusiastic, and interested." The PANAS yields Positive Affect (PA) and Negative Affect (NA) subscales. In the present study, the internal consistencies of the



PA ( $\alpha = .86$ ) and NA scales ( $\alpha = .88$ ) were adequate and commensurate with past studies (i.e., Crawford & Henry, 2004; Mehrabian, 1998; Roesch, 1998).

**Behavioral tasks and EEG measurement.** Participants completed two cognitive tasks while electroencephalography (EEG) data was recorded. Only behavioral and EEG data from the modified Color-Word Stroop task were included in the present study. Participants were seated 100 cm from a 21-inch CRT monitor in a quiet, noise-shielded room. The stimuli were presented and responses recorded using E-Prime 2.0 (Schneider, Eschman, & Zuccolotto, 2002).

**Apparatus and physiological recording.** EEG data were recorded from each participant using a Biosemi Active2 EEG system. Custom-designed Falk Minow caps with 64 equidistant Ag/AgCl active electrodes were used for data collection. Common Mode Sense (CMS) and Driven Right Leg (DRL) sensors were placed near the vertex and formed a feedback loop serving the job of typical “ground” electrodes. Two electrodes were placed on the mastoid bones. Two electrodes were placed on the outer canthus of each eye to monitor horizontal eye movements (HEOG). Two additional electrodes were placed on the inferior edge of the orbit of each eye to monitor vertical eye movements (VEOG). After placement of the electrode cap, electrode positions were digitized. Data were recorded with a band pass filter of 0–104 Hz, and sampled at a rate of 512 Hz

**Stroop interference.** To assess interference processing, participants completed an electronic Color-Word Stroop task, based upon the Delis-Kaplan Executive Function System (D-KEFS) Color-Word Interference Task (Delis, Kaplan, & Kramer, 2001). Interference occurs when conflicting stimuli are presented simultaneously. In the case of the Color-Word Stroop task, conflicting stimuli are presented such that the name of one color (e.g., the word “RED”) is printed in the ink of another color (e.g., blue). These task trials are referred to as incongruent

trials. Interference does not occur when stimuli characteristics are in agreement (e.g., the word “RED” is printed in red ink). Trials in which the word name and ink color match are referred to as congruent trials. To obtain an index of accuracy interference, accuracy on incongruent trials was subtracted from accuracy on congruent trials. To obtain an index of reaction time interference, reaction time on congruent trials was subtracted from reaction time on incongruent trials.

**Task design.** Stimulus presentation times were based upon previous Stroop tasks optimized for EEG recording (Compton et al., 2011; Lansbergen & Kenemans, 2008; Siltan et al., 2010). Each trial began with a fixation cross in the center of the screen, the presentation of which varied between 500-1100 ms to minimize potential habituation to the stimuli presentation format. The stimulus was then be presented for 150 ms, followed by a blank screen, during which participants had 1500 ms to respond before the onset of the next trial. On each trial, participants indicated their response (blue, green, red, or yellow) via a button press. The four response options were mapped onto a response box with color-coded keys.

Participants completed the following task blocks: (a) word-reading congruent (b) word-reading incongruent, (c) color-naming congruent block, (d) color-naming-incongruent block, (d) color-naming mixed congruent and incongruent block, and (e) task-switching. Participants were provided 90-second breaks in between each test block. The color-naming blocked and mixed conditions for congruent and incongruent stimuli were analyzed for the present study; these blocks are further detailed below.

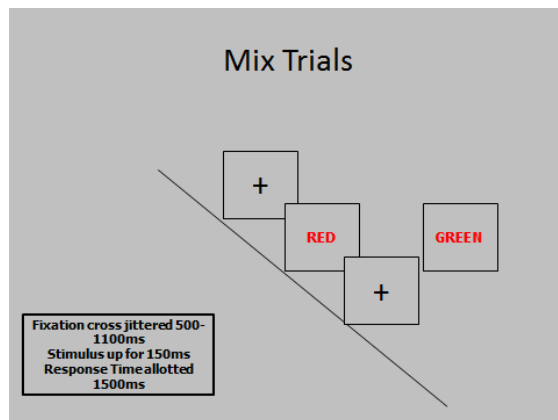
Prior to beginning the Color-Word Stroop task, participants completed color identification practice (16 trials) with written feedback (“correct” or “incorrect”) displayed on the screen after each trial. Sixteen practice trials with accuracy feedback preceded each of the

task blocks; twenty-four practice trials preceded the switching trials. Feedback was not provided throughout the testing blocks of trials.

**Blocked color-naming condition.** Participants completed a congruent and incongruent condition of color-identification of words. The congruent condition only presented congruent stimuli (e.g., the word “RED” printed in red ink), and the incongruent condition only presented incongruent stimuli (e.g., the word “RED” printed in blue ink). Each condition was presented in two consecutive sets of 48 trials with a 10 second break in between (96 total congruent/incongruent total trials were presented).

**Mixed color-naming condition.** Participants completed one mixed presentation of color-identification (see Figure 1). For the mixed condition, the incongruent and congruent stimuli were randomly intermixed throughout each presentation. This condition was divided into four consecutive sets of 48 trials, with a ten second break in between each set, totaling 96 congruent and 96 incongruent trials.

Figure 1. Schematic of Color-Word Stroop Mixed Color-Naming Block



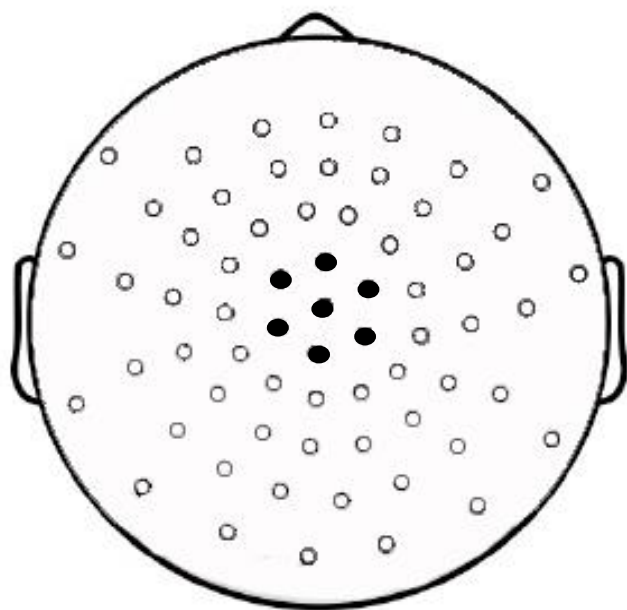
**Note.** Color-Word Stroop mixed color-naming blocks included 96 congruent and 96 incongruent intermixed trials that were randomized within each block. The block was presented in sets of 48 trials, with a ten second break between each trial.

**EEG data reduction.** EEG data processing was performed in Brain Electrical Source Analysis software (BESA, Version 6.0). Following the adaptive artifact correction method (Ille, Berg, Scherg, 2002) ocular artifacts were corrected using a spatial PCA filter. Muscle and other artifact were removed through automated and visual inspection of raw data. Participants with fewer than 30% rejected trials across conditions were included in ERP analyses. Baseline adjustment was computed using the averaged amplitude of 100 ms pre-stimulus onset. Data were referenced using an average reference and were not digitally filtered for analyses.

Stimulus-locked averages were calculated to ascertain mean amplitudes and peak latencies for central N200 and N450 components across conditions. On average, participants had 77 accepted correct “blocked congruent” trials, 75 accepted correct “blocked incongruent” trials, 73 “mixed congruent” trials, and 66 “mixed incongruent” trials.

Electrode selection and temporal windows were informed by visual inspection of data as well as *a priori* judgments based on findings from previous studies investigating these ERP components in similar contexts (i.e., Siltan et al., 2010). The N200 component was measured from 225 to 325 ms post stimulus onset for correct trials of the CW-Stroop Task. The N450 was measured from 350 to 500 ms post stimulus onset for correct trials of the CW-Stroop Task. A cluster of seven central electrodes was identified for N200 and N450 analyses (Figure 2). Amplitude and peak latency data for stimulus-locked averages were calculated for the aforementioned N200 and N450 time windows.

Figure 2. Electrode Localization for N200 and N450 ERP Analyses.



## CHAPTER FOUR

### RESULTS

#### Descriptive Analyses

Skewness analyses were conducted for all variables using guidelines established by Tabachnick and Fidell (2001). Conservative alpha levels (.001) were employed to evaluate the significance of skewness, in which z-score values greater than 2.00 were considered significantly skewed. Since no variables were skewed, data were analyzed without transformation. The means and standard deviations for study variables are presented in Table 1. Two-tailed t-tests were calculated to assess whether the healthy control and remitted depressed groups differed on affect and depression severity. Results did not indicate significant group differences. ERP waveforms are presented in Figure 3 and Figure 4.

Table 1. Descriptive Statistics for Study Variables

| Self-Report Measure | Diagnostic Group |           |                    |           |
|---------------------|------------------|-----------|--------------------|-----------|
|                     | Healthy Controls |           | Remitted Depressed |           |
|                     | <i>M</i>         | <i>SD</i> | <i>M</i>           | <i>SD</i> |
| PANAS - PA          | 29.00            | 5.98      | 27.22              | 6.54      |
| PANAS - NA          | 15.33            | 5.86      | 15.56              | 5.94      |
| PHQ-9               | 4.76             | 4.27      | 6.81               | 4.40      |

| <b>Congruent Trials – Blocked Condition</b>   | <i>M</i> | <i>SD</i> | <i>M</i> | <i>SD</i> |
|---|----------|-----------|----------|-----------|
| <b>Accuracy</b>                               | 0.95     | 0.04      | 0.95     | 0.02      |
| <b>Response Time (ms)</b>                     | 395.92   | 100.22    | 383.32   | 99.10     |
| <b>N200 Amplitude</b>                         | 0.58     | 1.99      | -0.27    | 1.37      |
| <b>N200 Latency (ms)</b>                      | 264.16   | 17.24     | 273.87   | 21.82     |
| <b>N450 Amplitude</b>                         | 2.84     | 2.34      | 1.89     | 1.89      |
| <b>N450 Latency (ms)</b>                      | 403.16   | 32.50     | 411.96   | 36.14     |
| <b>Incongruent Trials – Blocked Condition</b> | <i>M</i> | <i>SD</i> | <i>M</i> | <i>SD</i> |
| <b>Accuracy</b>                               | 0.92     | 0.06      | 0.91     | 0.04      |
| <b>Response Time</b>                          | 542.77   | 125.76    | 523.71   | 131.45    |
| <b>N200 Amplitude</b>                         | 0.73     | 1.94      | -0.37    | 1.35      |
| <b>N200 Latency</b>                           | 268.99   | 14.72     | 280.32   | 20.16     |
| <b>N450 Amplitude</b>                         | 2.28     | 2.00      | 0.94     | 1.91      |
| <b>N450 Latency</b>                           | 415.63   | 36.74     | 424.98   | 32.09     |
| <b>Congruent Trials - Mixed Condition</b>     | <i>M</i> | <i>SD</i> | <i>M</i> | <i>SD</i> |
| <b>Accuracy</b>                               | 0.96     | 0.04      | .96      | 0.03      |
| <b>Response Time</b>                          | 434.43   | 88.18     | 434.60   | 122.95    |
| <b>N200 Amplitude</b>                         | 0.45     | 2.27      | -0.87    | 1.59      |
| <b>N200 Latency</b>                           | 266.75   | 18.34     | 274.62   | 18.98     |
| <b>N450 Amplitude</b>                         | 2.18     | 2.00      | .88      | 1.86      |
| <b>N450 Latency</b>                           | 413.77   | 39.86     | 402.12   | 33.15     |
| <b>Incongruent Trials - Mixed Condition</b>   | <i>M</i> | <i>SD</i> | <i>M</i> | <i>SD</i> |

|  |          |           |          |           |
|--|----------|-----------|----------|-----------|
| <b>Accuracy</b>                                | 0.90     | 0.07      | 0.87     | 0.06      |
| <b>Response Time</b>                           | 575.75   | 126.40    | 571.09   | 131.95    |
| <b>N200 Amplitude</b>                          | 0.46     | 2.24      | -0.42    | 1.41      |
| <b>N200 Latency</b>                            | 271.44   | 16.28     | 278.77   | 18.42     |
| <b>N450 Amplitude</b>                          | 1.97     | 2.25      | 1.04     | 2.23      |
| <b>N450 Latency</b>                            | 402.93   | 33.32     | 424.99   | 38.18     |
| <b>Stroop Interference – Blocked Condition</b> | <i>M</i> | <i>SD</i> | <i>M</i> | <i>SD</i> |
| <b>Accuracy (Con Acc – Inc Acc)</b>            | 0.03     | 0.04      | 0.03     | 0.04      |
| <b>Reaction Time (Inc RT – Con RT)</b>         | 146.85   | 62.14     | 139.39   | 85.26     |
| <b>Stroop Interference – Mixed Condition</b>   | <i>M</i> | <i>SD</i> | <i>M</i> | <i>SD</i> |
| <b>Accuracy (Con Acc – Inc Acc)</b>            | 0.06     | 0.06      | 0.09     | 0.07      |
| <b>Reaction Time (Inc RT – Con RT)</b>         | 141.32   | 65.77     | 136.49   | 60.66     |

Figure 3. ERP Waveforms at Cz for Blocked Conditions

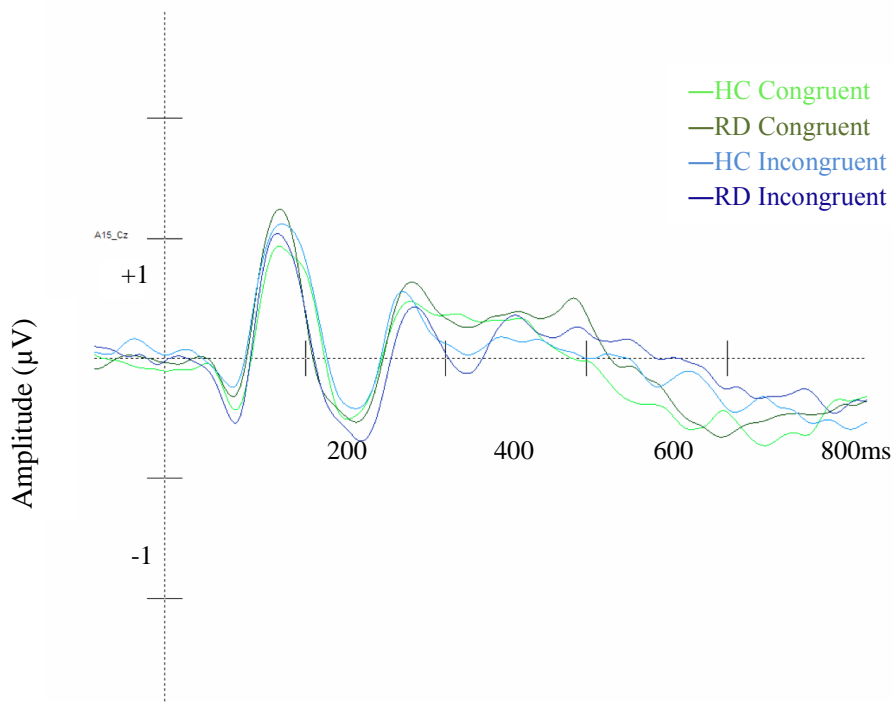
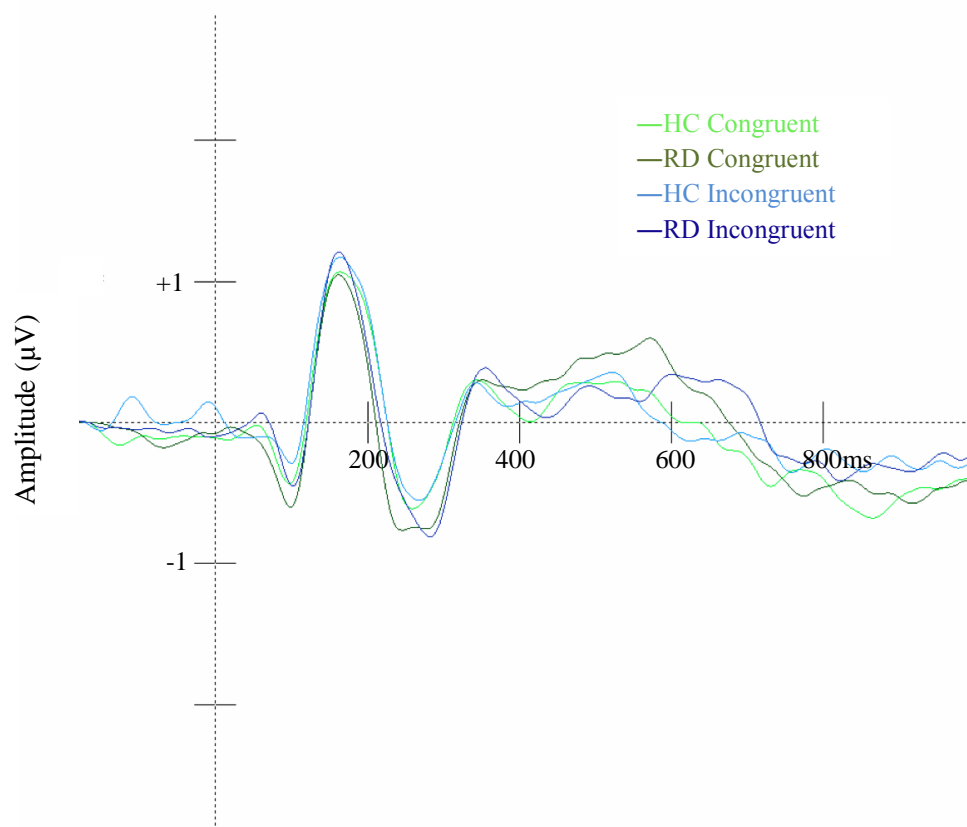




Figure 4. ERP Waveform at Cz for Mixed Conditions



### Correlational Analyses

Correlational analyses were conducted for each diagnostic group (healthy control or remitted depressed) to assess the relations among relevant study variables. Findings are presented in Table 2 and Table 3. Significant correlations involving primary study constructs are characterized below.

#### Affect and Behavioral/ERP Correlations

Higher levels of self-reported positive affect (PA) were related to faster onset of N200 latency during mixed congruent trials for the healthy control group ( $r = -.52$ ), but later onset N200 for the same condition for the remitted depressed group ( $r = .47$ ). Similarly, higher levels of self-reported negative affect (NA) were related to attenuated N200 amplitude for mixed

congruent trials for the healthy control group, but larger N200 amplitude during the same condition for the remitted depressed group.

### **Blocked Condition Behavioral and ERP Correlations**

For the healthy control group, correlations between behavioral performance and neural correlates showed an association between Stroop interference reaction time (Stroop interference-RT, incongruent trials reaction time – congruent trials reaction time) and N450 latency for incongruent blocked trials ( $r = -.52$ ); higher levels of Stroop interference conflict were related to faster onset of N450 latency for incongruent blocked trials. Stroop interference-RT was related to N450 amplitude, such that higher levels of interference correlated with increased N450 amplitude during incongruent blocked trials.

Stroop interference accuracy (congruent accuracy – incongruent accuracy) for blocked trials directly correlated with Stroop interference accuracy for mixed trials for both the healthy control group ( $r = .74$ ) and the remitted depressed group ( $r = .52$ ). Increased Stroop interference accuracy during the blocked presentation of conditions was also related to increased N200 amplitude during blocked incongruent ( $r = -.49$ ) and congruent conditions ( $r = -.46$ ) for healthy controls. Increased Stroop interference accuracy during the blocked condition correlated with delayed onset of N200 ( $r = -.51$ ) and more negative N450 amplitude ( $r = -.56$ ) during blocked incongruent trials for the remitted depressed group.

### **Mixed Condition Behavioral and ERP Correlations**

Lastly, for healthy controls, Stroop interference accuracy during the mixed condition was related to more negative N200 amplitudes for mixed congruent ( $r = -.56$ ) and incongruent ( $r = -.55$ ) conditions.

Table 2. Pearson Correlations for the Healthy Control Group

|                           | 1    | 2    | 3    | 4     | 5    | 6     | 7     |
|---------------------------|------|------|------|-------|------|-------|-------|
| 1. PA                     | --   |      |      |       |      |       |       |
| 2. NA                     | -.14 | --   |      |       |      |       |       |
| 3. PHQ-9                  | .07  | .22  | --   |       |      |       |       |
| 4. Block Interference-RT  | .31  | -.16 | .04  | --    |      |       |       |
| 5. Mix Interference-RT    | -.21 | -.14 | .12  | -.03  | --   |       |       |
| 6. Block Interference-Acc | -.02 | -.15 | -.09 | .06   | -.18 | --    |       |
| 7. Mix Interference-Acc   | -.05 | -.21 | .05  | .05   | .10  | .74*  | --    |
| 8. N200 BC Amplitude      | -.07 | .42  | .02  | .08   | .11  | -.46* | -.57* |
| 9. N200 BC Latency        | -.25 | .23  | .30  | -.27  | .23  | .39   | .52*  |
| 10. N200 BI Amplitude     | -.19 | .56* | .12  | .13   | -.06 | -.49* | -.49* |
| 11. N200 BI Latency       | .37  | .22  | -.03 | .04   | .07  | .27   | .31   |
| 12. N200 MC Amplitude     | -.13 | .57* | .16  | .15   | -.14 | -.41  | -.56* |
| 13. N200 MC Latency       | .52* | .31  | -.36 | -.13  | .22  | .01   | .23   |
| 14. N200 MI Amplitude     | -.15 | .46  | .09  | .22   | -.10 | -.53* | -.55* |
| 15. N200 MI Latency       | -.09 | .00  | -.16 | .14   | -.02 | .32   | .42   |
| 16. N450 BC Amplitude     | .16  | .06  | -.22 | .15   | -.03 | -.19  | -.11  |
| 17. N450 BC Latency       | -.18 | -.16 | -.14 | -.20  | .28  | -.03  | -.14  |
| 18. N450 BI Amplitude     | .05  | .13  | -.10 | .10   | .09  | -.29  | -.16  |
| 19. N450 BI Latency       | -.23 | .20  | .14  | -.52* | -.08 | .30   | .09   |
| 20. N450 MC Amplitude     | .14  | .25  | -.06 | .10   | -.16 | -.18  | -.27  |
| 21. N450 MC Latency       | -.41 | .34  | -.19 | -.36  | .18  | .33   | .08   |
| 22. N450 MI Amplitude     | -.01 | .19  | .03  | .24   | .12  | -.34  | -.35  |
| 23. N450 MI Latency       | -.37 | .06  | -.22 | -.51* | .03  | .31   | .30   |

Notes. PA = positive affect; NA = negative affect; RT = reaction time; Acc = accuracy; BC = blocked congruent; BI = blocked incongruent; MC = mixed congruent; MI = mixed incongruent; \* =  $p < 0.05$

Table 3. Pearson Correlations for the Remitted Depressed Group

|                           | 1     | 2     | 3     | 4     | 5    | 6     | 7    |
|---------------------------|-------|-------|-------|-------|------|-------|------|
| 1. PA                     | --    |       |       |       |      |       |      |
| 2. NA                     | .33   | --    |       |       |      |       |      |
| 3. PHQ-9                  | .15   | .72*  | --    |       |      |       |      |
| 4. Block Interference-RT  | -.25  | -.30  | -.07  | --    |      |       |      |
| 5. Mix Interference-RT    | -.35  | -.20  | .02   | .44   | --   |       |      |
| 6. Block Interference-Acc | -.29  | -.09  | .03   | .17   | -.21 | --    |      |
| 7. Mix Interference-Acc   | -.27  | -.30  | -.24  | .26   | .46* | .51*  | --   |
| 8. N200 BC Amplitude      | .07   | -.61* | -.63* | -.39  | -.34 | .06   | .13  |
| 9. N200 BC Latency        | .40   | .18   | -.49  | -.09  | -.06 | .09   | .31  |
| 10. N200 BI Amplitude     | .27   | -.10  | -.42  | -.25  | -.21 | -.40  | -.10 |
| 11. N200 BI Latency       | .37   | .06   | -.25  | .01   | -.40 | .51*  | .23  |
| 12. N200 MC Amplitude     | -.28  | -.54* | -.73* | -.15  | -.10 | -.01  | .18  |
| 13. N200 MC Latency       | .47*  | .04   | -.26  | .02   | .01  | .03   | .22  |
| 14. N200 MI Amplitude     | -.28  | -.32  | -.36  | -.23  | -.13 | -.17  | -.09 |
| 15. N200 MI Latency       | .46   | .00   | -.35  | .00   | -.06 | .18   | .26  |
| 16. N450 BC Amplitude     | -.15  | -.19  | .40   | -.28  | -.12 | -.01  | -.17 |
| 17. N450 BC Latency       | -.02  | -.18  | -.27  | -.13  | -.24 | -.29  | -.30 |
| 18. N450 BI Amplitude     | .05   | .04   | .18   | -.50* | -.08 | -.56* | -.43 |
| 19. N450 BI Latency       | .13   | .14   | .40   | .07   | .08  | .00   | .19  |
| 20. N450 MC Amplitude     | -.52* | -.29  | -.21  | -.20  | -.11 | .03   | -.16 |
| 21. N450 MC Latency       | .10   | .25   | .00   | .04   | -.10 | -.40  | -.27 |
| 22. N450 MI Amplitude     | -.44  | -.22  | .03   | -.39  | -.03 | -.14  | -.19 |
| 23. N450 MI Latency       | .39   | .22   | .21   | -.20  | -.13 | .00   | -.07 |

*Notes.* PA = positive affect; NA = negative affect; RT = reaction time; Acc = accuracy; BC = blocked congruent; BI = blocked incongruent; MC = mixed congruent; MI = mixed incongruent; \* =  $p < 0.05$

## Hypothesis Testing

### Aim 1 (Stroop Interference)

A series of repeated measures ANOVA analyses were conducted to evaluate Aim 1 regarding behavioral and neural correlates of Stroop Interference effects for healthy controls and individuals with remitted depression. Behavioral data (i.e., response time or response accuracy) and event related potential (ERP) components (i.e., N200/N450 ERP amplitudes and latencies) were within-subject variables using a 2x2 design of condition (blocked or mixed) and congruency (congruent or incongruent). For all analyses, diagnostic group (healthy controls or remitted depressed) was entered as a between-subject factor. Wilks' lambda statistics are reported for all multivariate analyses unless analyses rejected the null hypotheses for homogeneity of variance/covariance across diagnostic groups, Pillai's Trace was conducted to account for violation in statistical assumptions.

A power analysis with a significance level of  $p < .05$  (Aiken & West, 1991; Cohen, 1992) was conducted using G\*Power version 3.1 (Faul, Erdfelder, Lang, Buchner, 2007) to estimate the sample size needed to detect within subject differences between conditions and congruency and between-subject differences for lifetime history of depression. A review of literature regarding remitted depression and cognition indicated power for a medium effect size (Hasselbalch, Knorr & Vedel Kessing, 2011), but how this specifically translates to different components of cognitive control is unclear. The power analysis indicated that a sample of  $N = 44$  participants would be needed to detect a large effect size. A sample of  $N = 220$  would be need to detect a medium effect size. As such the following results are discussed in the context of limited power.

**Hypothesis 1a (reaction time).** Hypothesis 1a predicted that a behavioral cost of interference would be observed via increased reaction time (RT) for incongruent compared to congruent trials. A repeated measures ANOVA was conducted to compare the main effects of block condition, congruency, and diagnostic group and the interaction effects between these variables on RT. As hypothesized, a main effect was found for congruency,  $F(1, 37) = 266.90, p < .01, \eta_p^2 = .89$ , with larger RT observed on incongruent trials ( $M = 553.33, SE = 19.18$ ) compared to congruent trials ( $M = 412.32, SE = 16.02$ ) confirming the impact of interference processing on RT (see Figure 5). A main effect was also found for block condition (see Figure 6),  $F(1, 37) = 17.60, p < .01, \eta_p^2 = .32$ , in that RT was longer for mixed trials ( $M = 503.97, SE = 18.27$ ) in comparison to blocked trials ( $M = 461.68, SE = 17.44$ ). Diagnostic group and interaction factors between variables were not significant ( $p$ 's  $> .05$ ).

Figure 5. Main Effect of Congruency on Reaction Time

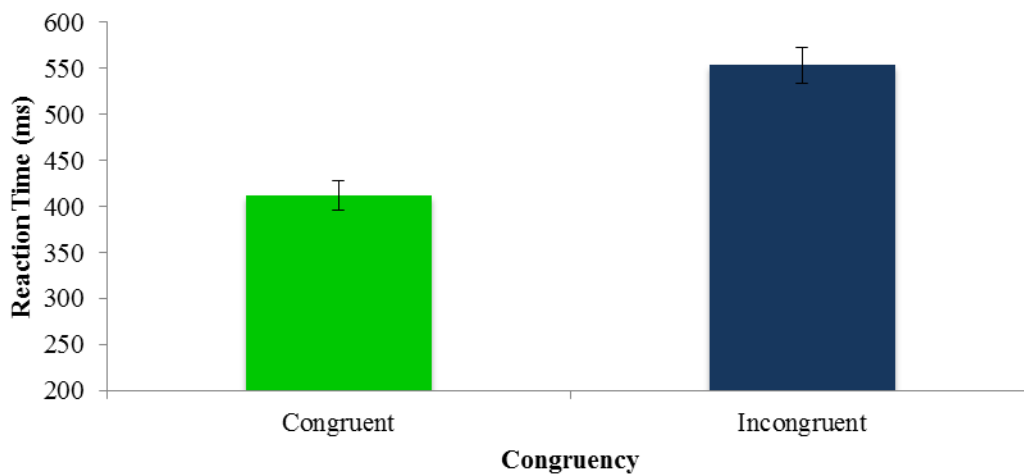
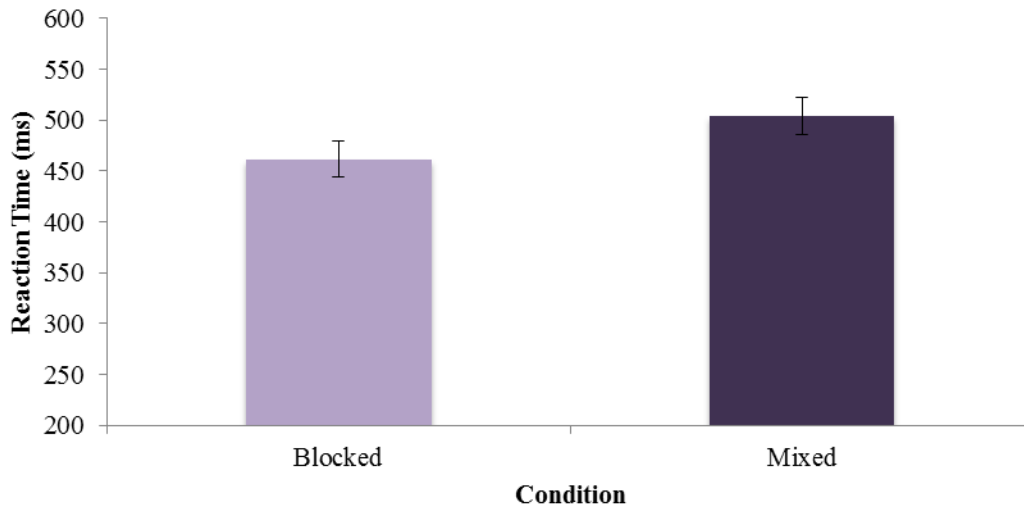


Figure 6. Main Effect of Block Condition on Reaction Time



**Hypothesis 1b (accuracy).** Hypothesis 1b predicted that a behavioral cost of interference would be observed in decreased accuracy for incongruent compared to congruent trials. A repeated measures ANOVA was conducted to compare the main effects of block condition, congruency, and diagnostic group and the interaction effects between these variables on accuracy. As hypothesized, a main effect was found for congruency,  $F(1, 37) = 51.97, p < .01, \eta_p^2 = .58$ , with decreased accuracy observed on incongruent trials ( $M = .90, SE = .01$ ) compared to congruent trials ( $M = .95, SE = .01$ ) confirming the impact of interference processing on task accuracy (see Figure 7). Similar to findings regarding RT, a main effect was also found for block condition (see Figure 8),  $F(1, 37) = 4.64, p < .05, \eta_p^2 = .11$ . Accuracy was lower on mixed trials ( $M = .92, SE = .01$ ) in comparison to blocked trials ( $M = .93, SE = .01$ ).

Figure 7. Main Effect of Congruency on Accuracy

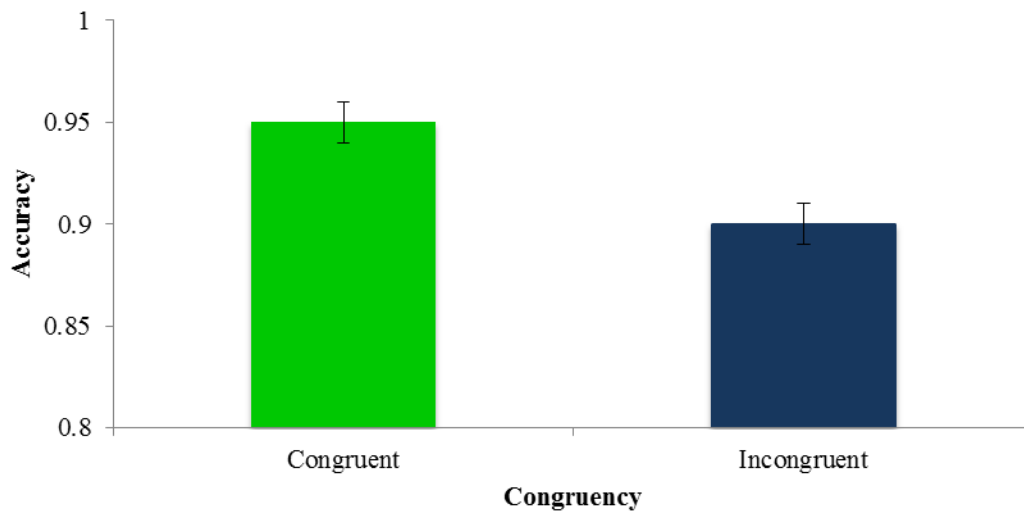
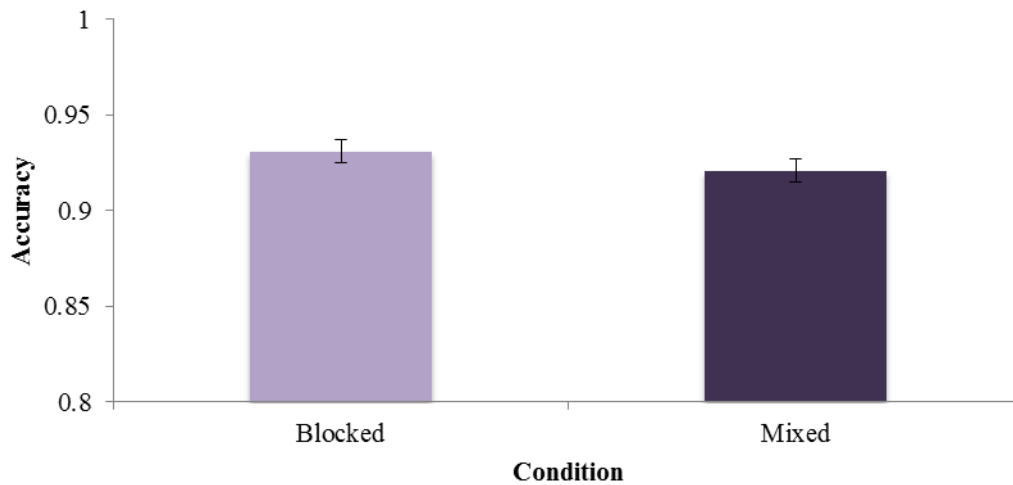


Figure 8. Main Effect of Block Condition on Accuracy

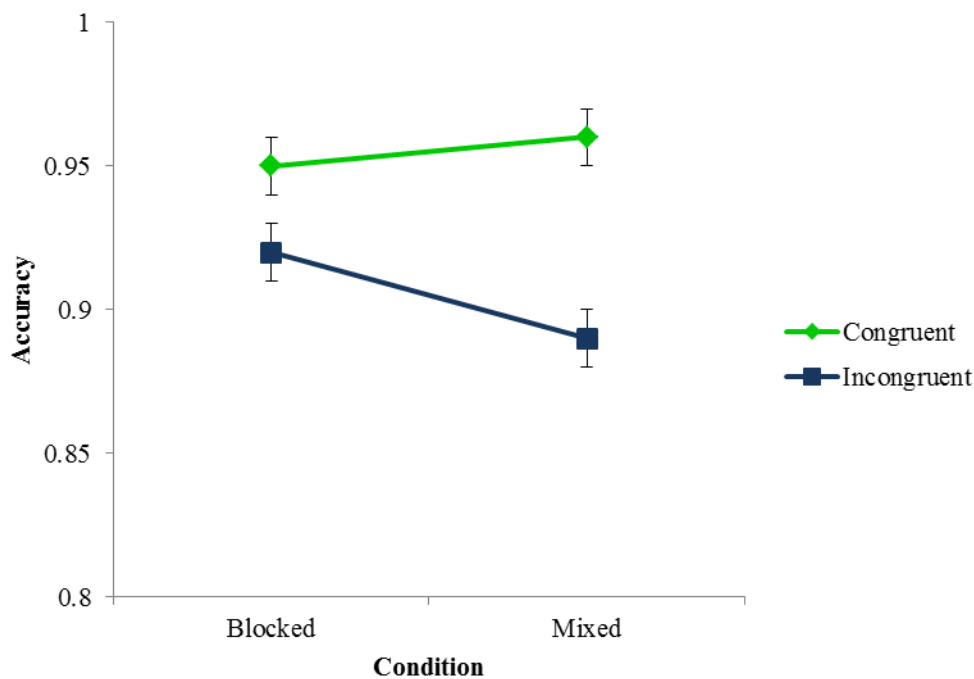


Additionally, a significant interaction for block condition by congruency was observed,  $F(1, 37) = 30.66, p < .01, \eta_p^2 = .45$ . Holm's sequential Bonferroni post-hoc analyses revealed simple main effects for congruency during the blocked condition,  $F(1, 38) = 26.09, p < .001, \eta_p^2 = .41$ , and mixed condition,  $F(1, 38) = 52.74, p < .01, \eta_p^2 = .58$ . Post-hoc analyses also confirmed simple main effects for block condition on congruent trials,  $F(1, 38) = 7.30, p < .05$ ,



$\eta_p^2 = .16$ , and mixed block condition,  $F(1, 38) = 18.63, p < .01, \eta_p^2 = .33$ . As shown in Figure 9, participants were most accurate on mixed congruent trials ( $M = .96, SE = .01$ ) and blocked congruent trials ( $M = .95, SE = .01$ ) and had more difficulty on blocked incongruent trials ( $M = .92, SE = .01$ ) and mixed incongruent trials ( $M = .89, SE = .01$ ). Diagnostic group and other interaction factors between variables were not significant ( $p$ 's  $> .05$ ).

Figure 9. Interaction of Block Condition x Congruency on Accuracy

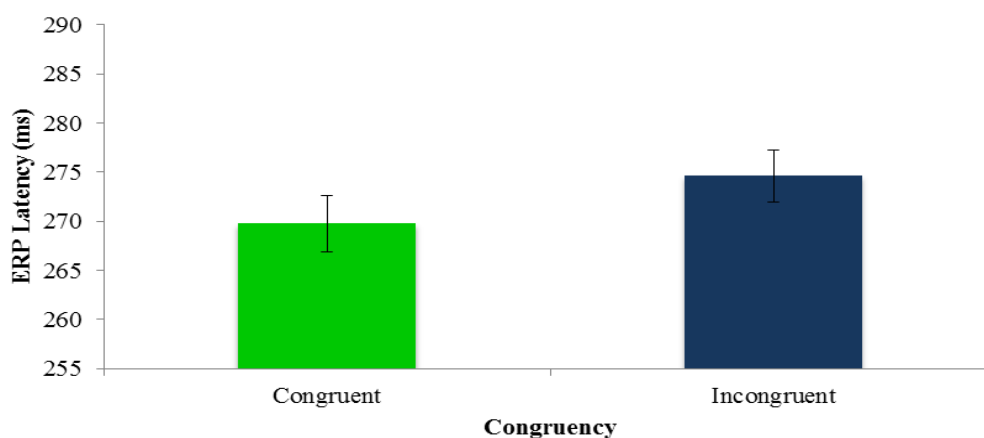


**Hypothesis 2a (N200 ERP amplitude).** Hypothesis 2a predicted that a neural correlate of interference processing would be observed in increased amplitude at central electrode sites occurring 225 to 325 ms post stimulus onset (N200 ERP component) for incongruent compared to congruent conditions. A repeated measures ANOVA was conducted to compare the main effects of block condition, congruency, and diagnostic group and the interaction effects between

these variables on N200 amplitude. No significant main effects or interaction effects were observed ( $p$ 's  $>.05$ ).

**Hypothesis 2b (N200 ERP latency).** Hypothesis 2b predicted that a neural correlate of interference processing would be observed in delayed onset (peak latency) of the N200 component for incongruent compared to congruent conditions. A repeated measures ANOVA was conducted to compare the main effects of block condition, congruency, and diagnostic group and the interaction effects between these variables on N200 latency. A significant main effect was found for congruency,  $F(1, 36) = 10.09, p < .01, \eta_p^2 = .22$ . As hypothesized and shown in Figure 10, onset of N200 was delayed for incongruent conditions ( $M = 274.63, SE = 2.65$ ) compared to congruent conditions ( $M = 269.79, SD = 2.88$ ). No additional significant main effects or interaction effects were observed ( $p$ 's  $>.05$ ).

Figure 10. Main Effect of Congruency on N200 Latency



**Hypothesis 3a (N450 ERP amplitude).** Hypothesis 3a predicted that a neural correlate of interference processing would be observed in increased negativity at central electrode sites occurring 350 to 500 ms post stimulus onset (N450) for incongruent compared to congruent conditions. A repeated measures ANOVA was conducted to compare the main effects of block

condition, congruency, and diagnostic group and the interaction effects between these variables on N450 amplitude. A main effect was observed for congruency (see Figure 11),  $F(1, 36) = 5.94, p < .05, \eta_p^2 = .14$ . As hypothesized, incongruent conditions resulted in more negative amplitude at N450 ( $M = 1.59, SE = .32$ ) than congruent conditions ( $M = 1.99, SE = .30$ ). A main effect was also observed for block condition (see Figure 12),  $F(1, 36) = 4.15, p < .05, \eta_p^2 = .10$ , in that N4 amplitude was larger for mixed conditions ( $M = 1.58, SE = .32$ ) in comparison to blocked conditions ( $M = 2.00, SE = .32$ ).

Figure 11. Main Effect of Congruency on N450 Amplitude

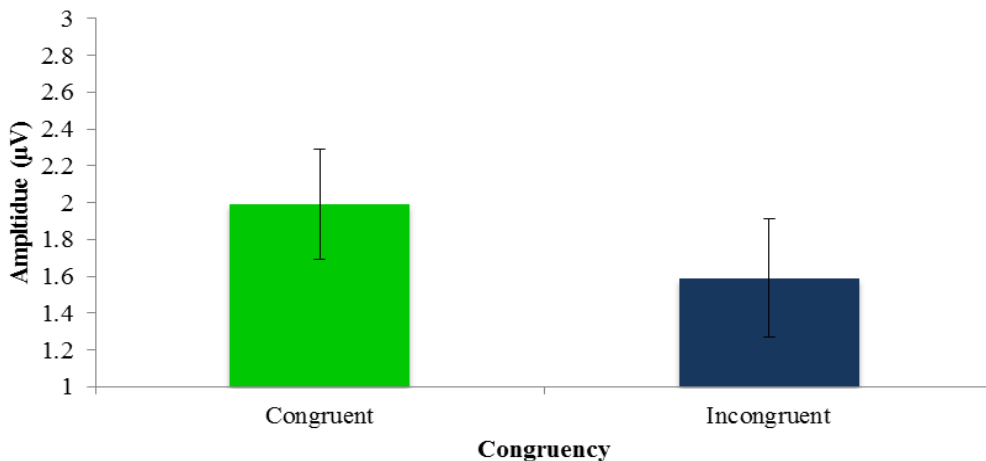
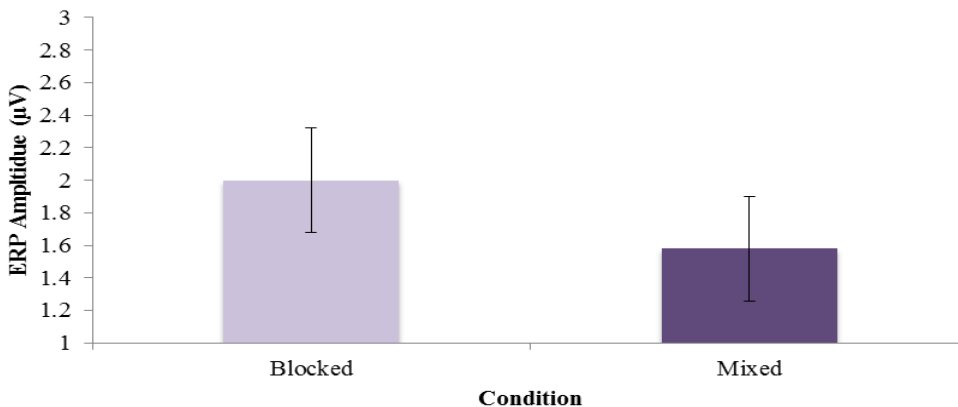
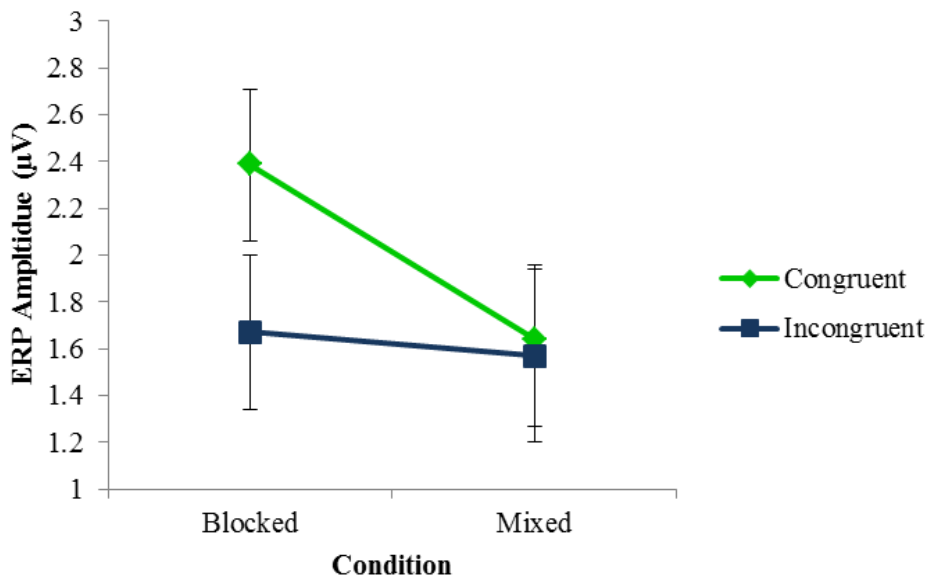


Figure 12. Main Effect of Block Condition on N450 Amplitude



Additionally, a significant interaction for block condition by congruency was observed,  $F(1, 36) = 5.76, p < .05, \eta_p^2 = .14$ . Holm's sequential Bonferroni post-hoc analyses revealed a simple main effect for congruency on blocked trials,  $F(1, 37) = 12.79, p < .01, \eta_p^2 = .26$ , and a simple main effect for block condition on congruency,  $F(1, 37) = 8.96, p < .01, \eta_p^2 = .20$ . As displayed in Figure 13, N450 amplitude was larger for blocked incongruent trials ( $M = 1.67, SE = .33$ ) compared to blocked congruent trials ( $M = 2.39, SE = .35$ ). Additionally, N450 amplitude was larger for mixed congruent trials ( $M = 1.64, SE = .32$ ) than blocked congruent trials ( $M = 2.39, SE = .35$ ). Diagnostic group and remaining interaction factors between variables were not significant ( $p$ 's  $> .05$ ).

Figure 13. Interaction of Block Condition x Congruency on N450 Amplitude



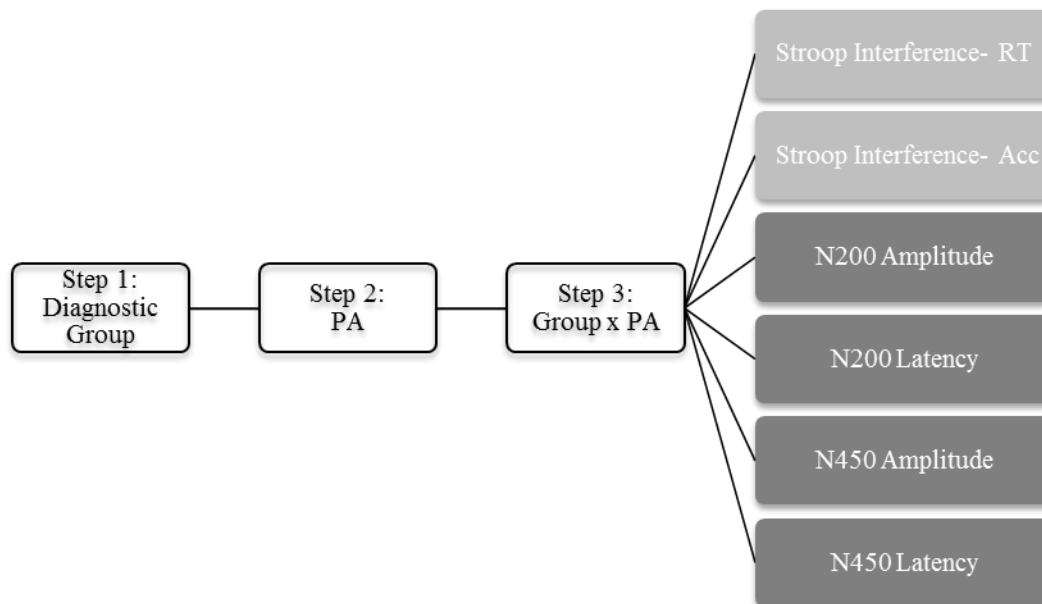
**Hypothesis 3b (N450 latency).** Hypothesis 3b stated that a neural correlate of interference processing would be observed in delayed onset (peak latency) of the N450 component for incongruent compared to congruent conditions. A repeated measures ANOVA was conducted to compare the main effects of block condition, congruency, and diagnostic group

and the interaction effects between these variables on N450 latency. Contrary to the proposed hypothesis, no significant main effects or interaction effects were observed ( $p$ 's  $>.05$ ).

### **Aim 2 (Positive Affect)**

A series of hierarchical regressions were conducted to examine Aim 2 regarding the effect of positive affect (PA) on performance during incongruent trials during blocked and mixed conditions of the Color-Word Stroop Task. For all regression models, Group (Healthy Controls = 0, Remitted Depressed = 1) was entered in the first step, Affect (PA) was entered in the second step, and the interaction of Group x Affect was entered in the third step. Outcome variables were behavioral and ERP data (see Figure 14). All interaction tests, including post-hoc tests when applicable, were conducted using the approach for depicting interaction effects described in Aiken and West (1991). Significant findings are reviewed below and regression results for all models can be found in Table 4 and 5.

Figure 14. Aim 2 Regression Analyses



*Note.* Diagnostic group, PA, Group x PA are entered sequentially as independent variables. Behavioral outcomes are indicated in light grey and ERP outcomes in dark grey boxes. Hierarchical linear regression analyses were conducted for blocked and mixed conditions of incongruent trials.

A power analysis with a significance level of  $p < .05$  (Aiken & West, 1991; Cohen, 1992) was conducted using G\*Power version 3.1 (Faul, Erdfelder, Lang, Buchner, 2007) to determine whether the sample size was appropriate to detect hypothesized effects. A review of literature regarding induced mood state and cognition indicated power for a medium effect size (Rowe et al., 2007; Gray 2001). Given that the effects of natural mood state on cognition is unknown, analyses were conducted for both medium and large effect sizes. The power analysis indicated that a total sample of  $N = 36$  participants would be needed to detect a large effect size and a total sample of  $N = 77$  would be need to detect a medium effect size. As such the following results are sufficiently powered to detect a large effect size and underpowered to detect a medium or smaller effect.

**Stroop interference – reaction time.** Regression analyses for incongruent trials in the blocked and mixed conditions showed that group, PA, and Group x PA were not significant predictors of Stroop Interference – Reaction Time.

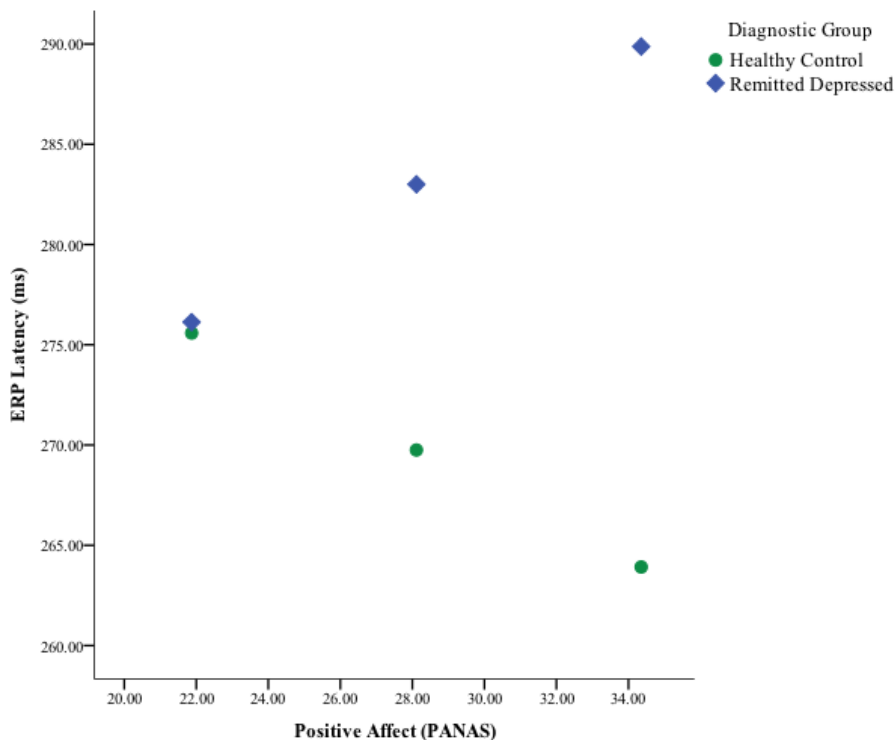
**Stroop interference – accuracy.** Regression analyses for incongruent trials in the blocked and mixed condition did not elicit significant models for Stroop Interference - Accuracy.

**N200 amplitude.** Results from the first step of the regression analyses showed that diagnostic history significantly contributed to the regression model,  $F(1,34) = 4.93, p < .05$ ) and accounted for 10% of the variance in N200 amplitude for incongruent trials in the blocked condition. Findings showed that the remitted depression group demonstrated more negative N200 amplitude ( $\beta = -.36, p < .05$ ) than the healthy control group on blocked incongruent trials.

PA and Group x PA did not contribute significantly to the regression model,  $F(2,33) = 2.40, p > .05$ ;  $F(3,32) = 2.16, p < .05$ , respectively. Regression analyses for the same trials in the mixed condition did not elicit significant models.

**N200 latency.** Results from the first step of the regression analyses showed that diagnostic history significantly contributed to the regression model,  $F(1,34) = 5.10, p < .05$  and accounted for 13% of the variance in N200 latency for incongruent trials in the blocked condition. Findings showed that the remitted depression group exhibited later onset of N200 ( $\beta = .36, p < .05$ ) than the healthy control group on blocked incongruent trials. PA did not significantly contribute to the regression model,  $F(2,33) = 2.55, p > .05$ . Group x PA significantly predicted N200 latency,  $F(3,32) = 3.55, p < .05$ , and accounted for an additional 12% of the variance in N200 latency during the mixed congruent condition. As shown in Figure 15, findings indicated that at higher levels of PA those in the remitted depressed group demonstrated later onset N200 and those in the healthy control demonstrated earlier onset N200 on blocked incongruent trials ( $\beta = 1.61, p < .05$ ). Regression analyses for the same trials in the mixed condition did not elicit significant models for N200 Latency.

Figure 15. N200 ERP Latency Predicted by Group x PA



**N450 amplitude.** Results showed that diagnostic history contributed significantly to the regression model,  $F(1,34) = 5.60, p < .05$  and accounted for 14% of the variance in N450 latency for incongruent trials in the blocked condition. Findings showed that the remitted depression group demonstrated more negative N450 amplitude ( $\beta = -.38, p < .05$ ) than the healthy control group on blocked incongruent trials. PA and Group x PA did not contribute significantly to the regression model,  $F(2,33) = 2.78, p > .05$  and  $F(3,32) = 1.79, p > .05$ , respectively. Regression analyses for the mixed condition did not elicit significant models.

**N450 latency.** Regression analyses for incongruent trials in the blocked and mixed condition not elicit significant models for N450 latency.



Table 4. Regression Summary Statistics for Blocked Incongruent Trials

**A) Reaction Time**

| Variable                     | Model 1  |             |         | Model 2  |             |         | Model 3  |             |         |
|------------------------------|----------|-------------|---------|----------|-------------|---------|----------|-------------|---------|
|                              | <i>B</i> | <i>SE B</i> | $\beta$ | <i>B</i> | <i>SE B</i> | $\beta$ | <i>B</i> | <i>SE B</i> | $\beta$ |
| Diagnostic Group             | -4.21    | 25.39       | -.03    | -4.81    | 26.03       | -.03    | 182.16   | 119.74      | 1.23    |
| PA                           | --       | --          | --      | -.33     | 2.12        | -.03    | 3.33     | 3.06        | .27     |
| Group * PA                   | --       | --          | --      | --       | --          | --      | -6.63    | 4.15        | -1.28   |
| $R^2$ Change                 |          | .00         |         |          | .00         |         |          | .07         |         |
| <i>F</i> for change in $R^2$ |          | .03         |         |          | .03         |         |          | 2.55        |         |

**B) Accuracy**

| Variable                     | Model 1  |             |         | Model 2  |             |         | Model 3  |             |         |
|------------------------------|----------|-------------|---------|----------|-------------|---------|----------|-------------|---------|
|                              | <i>B</i> | <i>SE B</i> | $\beta$ | <i>B</i> | <i>SE B</i> | $\beta$ | <i>B</i> | <i>SE B</i> | $\beta$ |
| Diagnostic Group             | .02      | .01         | .26     | .02      | .01         | .24     | .04      | .06         | .59     |
| PA                           | --       | --          | --      | .00      | .00         | -.11    | .00      | .00         | -.03    |
| Group * PA                   | --       | --          | --      | --       | --          | --      | .00      | .00         | -.36    |
| $R^2$ Change                 |          | .07         |         |          | .01         |         |          | .01         |         |
| <i>F</i> for change in $R^2$ |          | 2.36        |         |          | .41         |         |          | .20         |         |

**C) N200 Amplitude**

| Variable                     | Model 1  |             |         | Model 2  |             |         | Model 3  |             |         |
|------------------------------|----------|-------------|---------|----------|-------------|---------|----------|-------------|---------|
|                              | <i>B</i> | <i>SE B</i> | $\beta$ | <i>B</i> | <i>SE B</i> | $\beta$ | <i>B</i> | <i>SE B</i> | $\beta$ |
| Diagnostic Group             | -1.27    | .57         | -.36*   | -1.27    | .59         | -.36*   | -4.67    | 2.74        | -1.31   |
| PA                           | --       | --          | --      | .00      | .05         | .00     | -.07     | .07         | -.22    |
| Group * PA                   | --       | --          | --      | --       | --          | --      | .12      | .10         | .97     |
| $R^2$ Change                 |          | .13         |         |          | .00         |         |          | .04         |         |
| <i>F</i> for change in $R^2$ |          | 4.93*       |         |          | .00         |         |          | 1.61        |         |

**D) N200 Latency**

| Variable                | Model 1  |             |         | Model 2  |             |         | Model 3  |             |         |
|-------------------------|----------|-------------|---------|----------|-------------|---------|----------|-------------|---------|
|                         | <i>B</i> | <i>SE B</i> | $\beta$ | <i>B</i> | <i>SE B</i> | $\beta$ | <i>B</i> | <i>SE B</i> | $\beta$ |
| Diagnostic Group        | 13.11    | 5.80        | .36*    | 13.41    | 5.94        | .37*    | -43.97   | 26.42       | -1.21   |
| PA                      | --       | --          | --      | .17      | .48         | .06     | -.93     | .68         | -.32    |
| Group * PA              | --       | --          | --      | --       | --          | --      | 2.04     | .92         | 1.61*   |
| $R^2$ Change            |          | .13         |         |          | .00         |         |          | .12         |         |
| $F$ for change in $R^2$ |          | 5.10*       |         |          | .13         |         |          | 4.94*       |         |

**E) N450 Amplitude**

| Variable                | Model 1  |             |         | Model 2  |             |         | Model 3  |             |         |
|-------------------------|----------|-------------|---------|----------|-------------|---------|----------|-------------|---------|
|                         | <i>B</i> | <i>SE B</i> | $\beta$ | <i>B</i> | <i>SE B</i> | $\beta$ | <i>B</i> | <i>SE B</i> | $\beta$ |
| Diagnostic Group        | -2.57    | .66         | -.38*   | -1.55    | .68         | -.37*   | -1.48    | 3.25        | -.36    |
| PA                      | --       | --          | --      | .01      | .06         | .04     | .02      | .08         | .05     |
| Group * PA              | --       | --          | --      | --       | --          | --      | .00      | .11         | -.02    |
| $R^2$ Change            |          | .14         |         |          | .00         |         |          | .00         |         |
| $F$ for change in $R^2$ |          | 5.60*       |         |          | .07         |         |          | .00         |         |

**F) N450 Latency**

| Variable                | Model 1  |             |         | Model 2  |             |         | Model 3  |             |         |
|-------------------------|----------|-------------|---------|----------|-------------|---------|----------|-------------|---------|
|                         | <i>B</i> | <i>SE B</i> | $\beta$ | <i>B</i> | <i>SE B</i> | $\beta$ | <i>B</i> | <i>SE B</i> | $\beta$ |
| Diagnostic Group        | 6.88     | 11.67       | .10     | 6.37     | 11.96       | .09     | -50.91   | 56.21       | -.74    |
| PA                      | --       | --          | --      | -.29     | .97         | -.05    | -1.39    | 1.44        | -.25    |
| Group * PA              | --       | --          | --      | --       | --          | --      | 2.03     | 1.95        | .85     |
| $R^2$ Change            |          | .01         |         |          | .00         |         |          | .03         |         |
| $F$ for change in $R^2$ |          | .35         |         |          | .09         |         |          | 1.09        |         |

\*  $p < .05$

Table 5. Regression Summary Statistics for Mixed Incongruent Trials  
**A) Reaction Time**

| Variable                | Model 1  |             |         | Model 2  |             |         | Model 3  |             |         |
|-------------------------|----------|-------------|---------|----------|-------------|---------|----------|-------------|---------|
|                         | <i>B</i> | <i>SE B</i> | $\beta$ | <i>B</i> | <i>SE B</i> | $\beta$ | <i>B</i> | <i>SE B</i> | $\beta$ |
| Diagnostic Group        | -7.21    | 21.81       | -.06    | -12.40   | 21.49       | -.10    | 11.73    | 102.59      | .09     |
| PA                      | --       | --          | --      | -2.02    | 1.75        | -.28    | -2.45    | 2.62        | -.24    |
| Group * PA              | --       | --          | --      | --       | --          | --      | -.86     | 3.56        | -.19    |
| $R^2$ Change            |          | .00         |         |          | .08         |         |          | .00         |         |
| $F$ for change in $R^2$ |          | .11         |         |          | 2.80        |         |          | .06         |         |

**B) Accuracy**

| Variable                | Model 1  |             |         | Model 2  |             |         | Model 3  |             |         |
|-------------------------|----------|-------------|---------|----------|-------------|---------|----------|-------------|---------|
|                         | <i>B</i> | <i>SE B</i> | $\beta$ | <i>B</i> | <i>SE B</i> | $\beta$ | <i>B</i> | <i>SE B</i> | $\beta$ |
| Diagnostic Group        | .03      | .02         | .21     | .02      | .02         | .18     | .09      | .10         | .73     |
| PA                      | --       | --          | --      | .00      | .00         | -.18    | .00      | .00         | -.05    |
| Group * PA              | --       | --          | --      | --       | --          | --      | .00      | .00         | -.56    |
| $R^2$ Change            |          | .04         |         |          | .03         |         |          | .01         |         |
| $F$ for change in $R^2$ |          | 1.52        |         |          | 1.06        |         |          | .48         |         |

**C) N200 Amplitude**

| Variable                | Model 1  |             |         | Model 2  |             |         | Model 3  |             |         |
|-------------------------|----------|-------------|---------|----------|-------------|---------|----------|-------------|---------|
|                         | <i>B</i> | <i>SE B</i> | $\beta$ | <i>B</i> | <i>SE B</i> | $\beta$ | <i>B</i> | <i>SE B</i> | $\beta$ |
| Diagnostic Group        | -1.00    | .65         | -.26    | -1.11    | .65         | -.28    | -.99     | 3.11        | -.25    |
| PA                      | --       | --          | --      | -.06     | .05         | -.19    | -.06     | .08         | -.18    |
| Group * PA              | --       | --          | --      | --       | --          | --      | .00      | .11         | -.03    |
| $R^2$ Change            |          | .07         |         |          | .04         |         |          | .00         |         |
| $F$ for change in $R^2$ |          | 2.40        |         |          | 1.30        |         |          | .00         |         |

**D) N200 Latency**

| Variable                     | Model 1  |             |         | Model 2  |             |         | Model 3  |             |         |
|------------------------------|----------|-------------|---------|----------|-------------|---------|----------|-------------|---------|
|                              | <i>B</i> | <i>SE B</i> | $\beta$ | <i>B</i> | <i>SE B</i> | $\beta$ | <i>B</i> | <i>SE B</i> | $\beta$ |
| Diagnostic Group             | 9.05     | 5.73        | .26     | 10.09    | 5.75        | .29     | -32.72   | 26.36       | -.95    |
| PA                           | --       | --          | --      | .58      | .47         | .21     | -.25     | .67         | -.09    |
| Group * PA                   | --       | --          | --      | --       | --          | --      | 1.52     | .91         | 1.26    |
| $R^2$ Change                 |          | .07         |         |          | .04         |         |          | .07         |         |
| <i>F</i> for change in $R^2$ |          | 2.50        |         |          | 1.55        |         |          | 2.76        |         |

**E) N450 Amplitude**

| Variable                     | Model 1  |             |         | Model 2  |             |         | Model 3  |             |         |
|------------------------------|----------|-------------|---------|----------|-------------|---------|----------|-------------|---------|
|                              | <i>B</i> | <i>SE B</i> | $\beta$ | <i>B</i> | <i>SE B</i> | $\beta$ | <i>B</i> | <i>SE B</i> | $\beta$ |
| Diagnostic Group             | -1.06    | .76         | -.23    | -1.21    | .76         | -.26    | 3.00     | 3.57        | .66     |
| PA                           | --       | --          | --      | -.08     | .06         | -.23    | .00      | .09         | .00     |
| Group * PA                   | --       | --          | --      | --       | --          | --      | -.15     | .12         | -.94    |
| $R^2$ Change                 |          | .05         |         |          | .05         |         |          | .04         |         |
| <i>F</i> for change in $R^2$ |          | 1.92        |         |          | 1.84        |         |          | 1.46        |         |

**F) N450 Latency**

| Variable                     | Model 1  |             |         | Model 2  |             |         | Model 3  |             |         |
|------------------------------|----------|-------------|---------|----------|-------------|---------|----------|-------------|---------|
|                              | <i>B</i> | <i>SE B</i> | $\beta$ | <i>B</i> | <i>SE B</i> | $\beta$ | <i>B</i> | <i>SE B</i> | $\beta$ |
| Diagnostic Group             | 19.52    | 12.04       | .27     | 20.13    | 12.33       | .28     | -102.13  | 54.60       | -1.40   |
| PA                           | --       | --          | --      | .35      | 1.00        | .06     | -2.02    | 1.40        | -.34    |
| Group * PA                   | --       | --          | --      | --       | --          | --      | 4.34     | 1.89        | 1.71*   |
| $R^2$ Change                 |          | .07         |         |          | .00         |         |          | .13         |         |
| <i>F</i> for change in $R^2$ |          | 2.63        |         |          | .12         |         |          | 5.25*       |         |

\*  $p < .05$

## CHAPTER FIVE

### DISCUSSION

#### **Overview**

As deficits in cognitive and emotional functioning are well documented across many neurological disorders (e.g., depression, Alzheimer’s disease, and epilepsy), advancing our understanding of the interaction between cognitive and emotional processes in both normal and abnormal functioning is critical to further elucidating etiological and maintenance factors of emotional and cognitive dysfunction (Pessoa, 2008). In addition to implications for pathological conditions, understanding the nuanced impact of state affect on cognitive processes, such as cognitive control functions, is central to discerning optimal levels of mood in the completion of everyday activities as well. While a robust body of literature details the curvilinear nature of “optimal” anxiety/arousal (i.e., Zone of Optimal Functioning; Hanin, 1980), the “optimal” levels of positive or negative affect for different cognitive demands are not as well defined. In fact, empirical findings and theoretical models regarding the impact of positive and negative affect on cognitive control processes are riddled with inconsistent and at times contradictory messages. Therefore, the purpose of the present study was to replicate and expand upon previous research by examining behavioral and neural correlates of interference processing during the CWST and to evaluate the impact of varying levels of positive affect on performance.

As expected from previous findings (e.g., MacLeod, 1991; Harnishfeger, 1995; West, 2004; Sifton et al., 2011), incongruent trials on the CWST in the present study resulted in increased difficulty (i.e., increased reaction time and error rate) and increased recruitment of neural resources (e.g., increased ERP amplitude) compared to congruent trials. In addition to similarities in observed outcomes across blocked and mixed paradigms of the CWST, differences in interference processing across conditions were significant and provide insight into discrepant findings in the literature.

While little is known regarding the influences of naturally occurring positive affect and previous psychopathology on the recruitment/allocation of cognitive control resources, evidence from several studies indicate that these factors may indeed play a role in behavioral and neurophysiological response (Hur et al., 2015; Heller, 1993; Liotti et al., 2002). In fact, the interplay between cognitive and affective processing is evident within the structural and functional organization of the cortex, such that frontocingulate cortical structures that support affective function also are engaged during cognitive tasks, and vice versa (Mohanty et al., 2007; Blanchette and Richards, 2009). Findings from the limited studies that have investigated the influence of PA on interference processing during the CWST are inconsistent, in that high levels of PA were found to either have a deleterious effect (Yuan et al., 2011; Phillips, Bull, Adams and Fraser 2002) or did not influence performance (Martin & Kerns, 2010). Of note, these investigations did not report nor account for lifetime history of depression. In the present study, PA was found to moderate the relationship between diagnostic group and onset of N200 during blocked incongruent trials. At high levels of PA, N200 peak latency was delayed in the remitted depressed group and had an earlier onset in the healthy control group. These findings expand upon previous literature that has identified pervasive cognitive and emotional processing

differences in individuals with remitted depression (Paelecke-Habermann, Pohl, & Lepow, 2005) and suggest that the differential effects of emotional state upon cognitive control is dependent upon history of psychopathology.

### **Interference Processing**

Interference processing occurs when the presentation of task-irrelevant information conflicts with task-relevant information. Therefore, in order to successfully complete a task in the context of interference, an individual must disregard irrelevant data in favor of task-relevant information. In the CWST, interference processing is pronounced as task-irrelevant information is characterized as an automatic/habituated process that is in direct conflict with the prioritization of task-relevant information that requires effortful processing (MacLeod, 1991; Kahneman and Chajczyk, 1983). While findings in the extant literature illustrate a consistent pattern of behavioral outcomes (i.e., increased reaction time and decreased accuracy for incongruent trials), data detailing neural correlates of interference processing are inconsistent in regards to relevant ERP components and corresponding neural generators (Harnishfeger, 1995; MacLeod, 1991; Lufi, Cohen & Parnish-Plass, 1990; Wagner et al., 2006; West, 2004; Silton et al., 2011).

### **Behavioral and Neural Correlates of Interference Processing**

Several behavioral and neural correlates of interference processing were observed in both blocked and mixed CWST conditions and underscore the robust effects of interference processing that transcends factors such as diagnostic group and task presentation. Replicating and building upon previous research (MacLeod, 1991; Silton et al., 2011), both healthy control and remitted depressed groups showed behavioral costs of interference processing in increased reaction time and decreased accuracy for incongruent compared to congruent trials in blocked

and mixed conditions. Additionally, increased Stroop interference-accuracy for blocked trials was related to increased Stroop interference- accuracy for mixed trials for both groups.

While differences in N200 amplitude for incongruent compared to congruent trials have been observed in other studies (Holmes and Pizzagalli, 2008; Siltan et al., 2010), N200 amplitude was not significantly influenced by congruency in the present study. However, incongruent trials resulted in delayed onset of N200 peak latency compared to congruent trials across both blocked or mixed presentations. Taken together, these findings in combination with relevant literature (Holmes and Pizzagalli, 2008; Siltan et al., 2011) further establish N200 as a marker of interference processing. In the context of interference processing, N200 has been associated with the detection of conflict and source localization analyses have identified regions within the anterior cingulate cortex (ACC) as a potential generator (West, Kropf, Bowry & Doll, 2004; Holmes & Pizzagalli, 2008; van Veen & Carter, 2002).

Shortly after N200, N450 was observed in the present study, which is similarly thought to reflect ACC function. While there is currently debate within the literature as to whether N450 is reflective of conflict detection (Hanslmayr et al., 2008; Szucs and Soltesz, 2012) or response resolution (Liotti et al., 2000), it is generally agreed upon that increased N450 amplitude is associated with increased engagement of cognitive control abilities in relation to task demands. Consistent with observed behavioral differences between blocked and mixed conditions in the present study, N450 amplitude was attenuated in the blocked condition for congruent trials. These findings suggest that additional cognitive resources are recruited in order to successfully meet the higher task demands of a mixed presentation (e.g., switching between congruent and incongruent stimuli) compared to a blocked presentation. In the present study, N450 amplitude differentiated congruency conditions only in the blocked presentation. Since N450 amplitude did



not differ for congruent and incongruent trials in the mixed block presentation, this finding suggests that increased N450 amplitude reflects a broader recruitment of cognitive control resources rather than those specific to interference processing. Despite observed behavioral and N450 differences between blocked and mixed conditions, no significant differences in N200 were observed which further establishes N200 as an indicator of conflict detection.

### **Lifetime History of Depression and PA Influence CWST Performance**

In order to investigate the influence of affect on cognitive function with consideration for long-term implications of previous psychopathology, individuals with a lifetime history of depression as well as healthy controls participated in the present study. While there is evidence for residual low levels of PA, often characterized as anhedonia, in individuals with remitted depression (Wichers, Geschwind, van Os & Peeters, 2010; DelDonno et al., 2017), other findings have found no difference in dimensional ratings of mood/depressive symptom severity in remitted depression (Vanderhasselt, De Raedt, Dillon, Dutra, Brooks & Pizzagalli, 2012; Bylsma, Salomon, Clift, Morris & Rottenberg, 2014). In the present study, there were no significant differences between current depression severity as measured by the PHQ-9 or levels of PA or NA between healthy control and remitted depressed groups. Diagnostic group was a significant predictor of several ERP outcomes during the blocked condition of the CWST. Specifically, more negative N200 and N450 amplitude were observed for the remitted depression group compared to the healthy control group on blocked incongruent trials. This expands upon previous research (Vanderhassel et al., 2012) detailing the residual impact of remitted depression on cognition and reflects the greater recruitment of cognitive resources in order to complete task demands. Additionally, the remitted depression group exhibited later onset of N200 than the healthy control group on blocked incongruent trials. While self-reported PA alone did not

significantly predict behavioral or neural outcome measures, PA was found to moderate the relationship between diagnostic group and N200 latency, in that higher levels of PA were related to later onset N200 for the remitted depressed group and earlier N200 onset for the healthy control group on blocked incongruent trials. This finding calls attention to the differential affect PA has on cognitive control processes in the context of history of psychopathology and may further account for some of the differences found in existing research as both dimensional and categorical classifications of emotional functioning are not frequently examined in the same study. Lastly, diagnostic history and level of PA were not significant predictors of behavioral or ERP outcomes for incongruent trials during the mixed condition or behavioral outcomes in either condition. The discrepancy between conditions likely speaks to the difference in task demands and nuances of cognitive control processes.

### **Limitations and Future Directions**

The present study had several limitations that are discussed below. The final participant sample was relatively small, limiting the power to detect small to moderate effects across analyses. The participant sample was also predominantly Caucasian with limited diversity across other ethnicity or racial groups. Although an area of strength in terms of understanding affective and cognitive control processes in early adulthood, the limited age range of the present sample also limits generalizability to younger or older populations.

While this study expands upon existing CWST findings with the incorporation of congruent/incongruent trials presented in blocked and mixed conditions, the absence of a neutral condition limits comparisons to the absolute differences between congruency conditions. In addition to accounting for possible facilitative effects in congruent task demands, the inclusion of

a neutral condition would provide an additional comparison condition to better elucidate what cognitive processes are sensitive to high/low levels of PA and remitted depression.

Currently, emotional experience and diagnostic history of mood disorder are infrequently assessed in cognitive neuroscience research unless emotional constructs are central to study aims. Looking to the future, the interplay of affective and cognitive processes should be further investigated to include other cognitive control functions and negative affect. Also, given strong empirical evidence for the affective role of intensity, frequency, and arousal variables (Brown, Chorpita & Barlow, 1998; Diener, Larsen, Levine & Emmons, 1985; Heller, 1993), consideration of these constructs is warranted.

### **Conclusions**

In sum, the results of this study expand upon existing literature regarding recruitment of neural processes during different conditions of a CWST while also offering novel insight regarding the influence of low/high PA in the context of remitted depression. While similar patterns were observed for blocked and mixed conditions (i.e., behavioral measures of Stroop interference as well as N200 and N450 ERP components), significant effects were also observed for condition on behavioral and ERP outcomes such as attenuated N450 amplitude for congruent blocked trials and increased response latency for mixed trials. Differences between blocked and mixed conditions underscore that while Stroop interference is a robust effect involving conflict detection and response resolution, the strength of this effect is sensitive to task design and variations in task design have likely contributed to conflicting findings in CWST literature. Further, similarities between N450 amplitude for mixed congruent and mixed incongruent trials indicates that N450 may not be a direct correlate of conflict detection, but rather broader recruitment of cognitive control resources. Findings from the present study also provide further

evidence for residual effects of remitted depression on cognitive control processes and indicate that high levels of PA moderate the relationship between lifetime history of depression and neural processing during the CWST. Higher levels of PA were related to later onset N200 for the remitted depressed group and earlier N200 onset for the healthy control group on blocked incongruent trials. This suggests that high PA may have a facilitative effect in the recruitment of neural resources for individuals without a history of depression, but that the same effect is not observed in individuals with a lifetime history of depression. These findings further establish the interplay between affective and cognitive processes and posit that this relationship is malleable with consideration for individual history of psychopathology.

APPENDIX A  
SELF-REPORT MEASURE OF STATE AFFECT

## PANAS

This scale consists of a number of words and phrases that describe different feelings and emotions. Read each item and then mark the appropriate answer in the space next to the word. Indicate to what extent you feeling this way right now, that is, at the present moment. Use these numbers to record your answers.

| 1<br>very slightly<br>or not at all | 2<br>a little | 3<br>moderately    | 4<br>quite a bit | 5<br>extremely    |
|-------------------------------------|---------------|--------------------|------------------|-------------------|
| _____ cheerful                      |               | _____ sad          |                  | _____ active      |
| _____ guilty                        |               | _____ enthusiastic |                  | _____ attentive   |
| _____ afraid                        |               | _____ joyful       |                  | _____ downhearted |
| _____ nervous                       |               | _____ distressed   |                  | _____ happy       |
| _____ excited                       |               | _____ determined   |                  | _____ strong      |
| _____ hostile                       |               | _____ proud        |                  | _____ alert       |
| _____ jittery                       |               | _____ interested   |                  | _____ irritable   |
| _____ upset                         |               | _____ delighted    |                  | _____ ashamed     |
| _____ inspired                      |               | _____ blue         |                  | _____ scared      |

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## VITA

Lorri Kais was born on February 14, 1988 in Milwaukee, Wisconsin to Paul and Marilyn Kais. She graduated Cum Laude in Psychology from the University of Wisconsin, Milwaukee in May 2010. She completed postbaccalaureate research examining neuropsychological functioning in children with Neurofibromatosis-1, Williams syndrome, and posterior fossa tumor resection at the University of Wisconsin, Milwaukee and the Medical College of Wisconsin. In December 2015, she graduated with a Masters of Arts in Clinical Psychology at Loyola University Chicago. As a graduate student, Ms. Kais was involved in research led by Dr. Rebecca L. Sifton, which investigated the neural correlates of affective and cognitive processes utilizing electroencephalography. She completed her graduate and doctoral research under the mentorship of Dr. Sifton.

Ms. Kais' clinical work involved neuropsychological assessment, consultation, and intervention with children, adolescents, young adults, and their families, with an emphasis on medical and neurodevelopmental populations. Ms. Kais completed her clinical psychology internship at the University of Minnesota Medical School. She has accepted a postdoctoral fellowship in pediatric neuropsychology at The University of Colorado/ Colorado Children's Hospital and plans to pursue a career involving clinical care, research, and mentorship of future trainees.