

RUNNING HEAD: Associated mechanisms of self-regulation

Mechanisms of self-regulation: Associations between cognitive control and
emotion regulation

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Abstract

This study sought to expand on the current understanding of a relationship between two self-regulatory systems: cognitive control and emotion regulation. Cognitive control ability was measured through the error-related negativity (ERN) and post-error behavioral measures during a Stroop task. Emotion regulation ability was assessed by measures of cortisol levels and heart rate after a stress manipulation as well as a self-report measure of coping skills (the CISS). We predicted that individuals with better cognitive control abilities would show lower levels of cortisol and heart rates in response to the stress manipulation and higher scores on the CISS problem focused coping subscale. While we found that higher levels of cognitive control were correlated with lower levels of cortisol after the Stroop task, we did not find this expected correlation in response to the stress manipulation. There were also no significant correlations between our other measures of emotion regulation (heart rate and the CISS) and cognitive control ability. Implications of this finding as well as directions for future research are discussed.

Introduction

The ability to flexibly control one's behaviors is important to adaptive human functioning. Self-regulation allows us to learn from our mistakes and react accordingly. Recent research in cognitive neuroscience has shown that this type of self-regulation in the cognitive domain, cognitive control, relies on two separate but critical mechanisms: an evaluative and a regulatory component. Research in the field of emotion has also investigated a similar mechanism called emotion regulation, the process by which humans can control emotional responding to stimuli. Conceptual and neural overlaps of these two processes lead to the question of whether these two forms of self-regulation rely on the same or overlapping control mechanisms in the brain. While one recent study provides convincing evidence in favor of this idea, we aim to further explore and expand on these initial findings. A link between these two processes would add to a growing body of research on self-regulatory mechanisms and would provide more information into how both cognitive control and emotion regulation mechanisms function.

Cognitive control

Self-regulation in the cognitive domain has been extensively studied. Cognitive control allows us to exhibit flexible, corrective, goal directed behavior on cognitive tasks and react to our mistakes in an adaptive fashion. This mechanism is also essential for such high level cognition as language and learning. Cognitive control can be defined as “adaptive, goal-directed behavior [that] involves monitoring of ongoing actions and performance outcomes, and subsequent adjustments of behavior and learning” (Ridderinkhof, Ullsperger, Crone, & Nieuwenhuis, 2004: 443). In short, this system is responsible for the ability to monitor and correct for errors. Research has shown that

cognitive control consists of two processes: an evaluative component that involves monitoring one's performance and an executive/regulatory component that institutes increased control and implements appropriate changes in behavior (van Veen & Carter, 2006). Therefore, cognitive control consists of a system for optimizing information processing and behaviors needed for the task at hand that depends on the monitoring and evaluation of one's actions and performance outcomes (Ridderinkhof et al., 2004).

The first step of cognitive control (the evaluative step) involves error monitoring and detection. One way that this error monitoring can be measured is by using EEG technology. EEG, which stands for electroencephalogram, is a technique used to record electrical activity in cortical regions of the brain through electrodes placed on the scalp. Changes in frequencies and patterns of electrical activity can be related to brain activity and can help us to understand physiological measures of such complex processes as sleep, attention and emotion. Electrical activity in the brain as measured through the EEG is described by the amplitude and frequency of a given electrical signal. While some electrical activity in the brain is very rhythmic signaling states such as sleep and alertness, other brain activity is related to particular events in the environment. When an environmental stimulus occurs, such as a flash of light, certain patterns of electrical activity are observed. These electrical responses to stimuli are called event-related potentials, or ERP's (Stern, Ray, & Quigley, 2001). ERP's are time-locked events in that they show stable time course characteristics of electrical activity after a stimulus. The electrical voltages of ERP's are smaller than overall EEG recordings, but after averaging many trials one can see the regular pattern of electrical activity that follows a particular event (Luck, 2005; Stern et al., 2001).

One such ERP is the ERN, or error related negativity, that is evoked contemporaneously with errors. The ERN is a response-locked sharp, negative-going deflection of up to 10 μV that is associated with incorrect responses in choice reaction-time tasks (Gehring, Goss, Coles, Meyer, & Donchin, 1993). A wide range of tasks elicit the ERN, including the Eriksen Flankers task, the Sternberg Memory Scanning task, sentence verification tasks, and the Stroop task (Bernstein, Scheffers, & Coles, 1995). This illustrates the flexible nature of the ERN and shows that it is generated by erroneous responses across modalities and tasks. The ERN begins around the time of an incorrect response (shortly after muscle activity is detected in the responding limb) and peaks close to 100ms later. The amplitude of this negative-going deflection is largest at electrodes placed in the middle and front of the scalp. The ERN is absent in trials in which participants respond correctly, suggesting that the ERN can be seen as an electrophysiological measure of neural activity responsible for performance and error monitoring. This neural system seems to detect when responses do not match with the original intent (Bernstein et al., 1995; Gehring et al., 1993)

Research has further elucidated the properties of the ERN and shown that its magnitude is influenced by the features of a given task. In one of the earliest studies on the ERN, Gehring et al. (1993) demonstrated that the ERN is sensitive to the importance that an individual places on accuracy during a task. Through experimentally manipulating the importance of accuracy versus speed of response, the researchers found that ERN's were highest for participants in the accuracy condition while participants in the speed condition showed lower ERN amplitudes. In addition, the amplitude of the ERN is also sensitive to the degree of error. This can be seen in a study utilizing a four choice reaction

time task in which participants had to respond by pressing one of four buttons with one of two fingers on either hand. On trials where errors were committed with both the wrong finger and wrong hand, ERN's were larger than on trials where errors were made with only the wrong finger or the wrong hand (Bernstein et al., 1995). These findings are consistent with the idea of the ERN reflecting a neural system for performance monitoring and error detection relative to the goals of a given task.

Questions still remain however as to how this neural system detects errors and is activated. One such theory relies on dopamine. Phasic increases in dopamine carried from the mesencephalic dopamine system may signal when an event is better than predicted and phasic decreases signal when an event is worse than predicted. Therefore, the ERN may reflect changes in activity (specifically modulations in the activity of motor neurons in the pMFC) signaled by a reduction of dopamine due to the loss of a reward, indicating that events (errors) are worse than anticipated (Holroyd & Coles, 2002).

While EEG techniques demonstrate the timing of electrical brain activity in relation to error detection (the ERN), they cannot precisely locate the source of this activity within the brain. Converging evidence using other techniques suggests that the ERN is generated in the anterior cingulate cortex (ACC). By recording both magnetoencephalogram and EEG activity during reaction –time tasks, researchers found a magnetic equivalent of the ERN and through source analysis localized this activity to the ACC (Miltner, Lemke, Weiss, Holroyd, Scheffers, & Coles, 2003). In addition, fMRI studies have also yielded similar findings. fMRI studies of brain activity associated with incorrect responses while participants engaged in a go/no-go task revealed activation in the rostral ACC (Kiehl, Liddle, & Hopfinger, 2000). This would presumably locate the

ERN to this brain region as well. By correlating EEG measures of the ERN with fMRI activity during corresponding tasks, ERN activity was located to both the caudal and rostral ACC, with a stronger correlation being shown in the rostral ACC (Mathalon, Whitfield, & Ford, 2003). In addition, activity in the ACC is modulated by error relevance and the importance of accuracy. Higher levels of ACC activity were found if the importance of accuracy over speed was emphasized in a flanker task paradigm (Ullsperger & Cramon, 2004) just as ERN amplitude was found to vary with the importance placed on accuracy (Gehring et al., 1993). Taken together this evidence points towards the ACC as the generator of the ERN and as a brain region involved in performance monitoring and error detection.

The ACC has been implicated in more general aspects of performance monitoring and cognition as well. It is located on the medial portion of the frontal lobe and is situated at the interface between cortical and subcortical limbic regions. This location positions the ACC to serve both cognitive and emotional processing roles. This region appears to be involved in many different aspects of cognition and self-evaluation, such as motor control, response selection, processing demanding tasks, the processing of pain, as well as emotion processing (Banich, 2004).

One unified theory of ACC function is that it is involved in performance monitoring in relation to the failure *or* the reduced probability of obtaining an anticipated reward (Ridderinkhof et al., 2004). This has come from research suggesting activity in the ACC during other aspects of performance monitoring in addition to error detection. One such line of research is in the role of the ACC in conflict monitoring. When the information processing system is presented with conflicting information, such as a

mismatch between a word spelling one color but printed in a different color ink as during the Stroop task, this could signal a reduced probability of obtaining a reward due to suboptimal information processing efficiency and a lack of attention to relevant stimuli. Therefore, the ACC increases its activity during correct high conflict trials as well as during trials leading to erroneous responses consistent with its role in performance monitoring (van Veen & Carter, 2006).

fMRI studies have confirmed increased activation of the ACC consistent with the conflict monitoring hypothesis. High conflict trials leading to correct responses as well as erroneous trials both produce activation of the ACC. This suggests that error detection may be one form of conflict monitoring given the fact that errors are more likely to occur during high conflict trials (Kerns, Cohen, MacDonald III, Cho, Stenger, & Carter, 2004). Therefore the ACC may monitor performance through the detection of response competition in the cognitive system. Activation of the ACC in high conflict correct and incorrect trials has been confirmed by other studies as well (Carter, Braver, Barch, Botvinick, Noll, & Cohen, 1998; Mathalon et al., 2003), although errors and high conflict correct responses may recruit slightly different regions of the ACC (Mathalon et al., 2003). In addition, ACC activity has been observed in relation to other aspects of performance monitoring that indicate the reduced probability or loss of a reward. These include activity in response to unexpected negative feedback after a task indicating that one did commit an error as well as during tasks involving decision uncertainty where a set of responses are all equally likely to be correct (Ridderinkhof et al., 2004; Botvinick, Cohen, & Carter, 2004). Together these studies point towards a role of the ACC in general performance monitoring.

While performance monitoring is necessary to determine when one has made a mistake or gone astray, behavioral adjustment must then be implemented in order to respond adaptively to the given situation and learn from one's past behavior. Performance monitoring in and of itself is not effective unless it is followed by compensatory behavior (Garavan, Ross, Murphy, Roche, & Stein, 2002). Performance monitoring signals the need for increased levels of control, leading to adaptive behavioral changes. These behavioral changes represent the regulatory/executive component of the cognitive control process. Behavioral changes such as slowing down after one has made an error as well as improved accuracy following an error are adaptive behavioral adjustments following error detection. Behavioral adjustments following high conflict trials may include faster response times owing to increased attentional control and focus on the relevant information in the task at hand (van Veen & Carter, 2006; Kerns et al., 2004). These examples of behavioral changes are all indicative of increased and more effective levels of cognitive control and more efficient information processing.

These enhanced levels of control are associated with enhanced performance monitoring and ACC activity. Gehring and colleagues' early study of the ERN demonstrated that after committing errors participants showed more compensatory behaviors. Larger ERN's were associated with adaptive behavioral changes, such as post error slowing on the next trial as well as a higher probability of a correct response after an error (Gehring et al., 1993). This suggests that ACC activity is associated with behavioral changes. Further studies have also shown that trials with higher amounts of behavioral adjustment are associated with greater amounts of ACC activity on the preceding high conflict and erroneous trials. In particular, greater ACC activity on trials

involving errors in the Stroop task was correlated with greater behavioral adjustments on subsequent trials (Kerns et al., 2004). These changes reflect more efficient information processing and higher levels of attentional control in the cognitive system.

This second step of cognitive control involving increased control and adaptive changes in behavior seems to be implemented by the prefrontal cortex (PFC), and more specifically the lateral prefrontal cortex (LPFC). This is consistent with other PFC functions. The PFC is important for working memory, attentional control, representations of goal directed behavior, and in executive functions more generally. In addition, the PFC seems to have extensive connections with the limbic system (Banich, 2004). fMRI studies have demonstrated that high levels of behavioral adjustments after errors and high-conflict trials are associated with increased activation of the LPFC (Ridderinkhof et al., 2004; Kerns et al., 2004; Garavan et al., 2002). Tasks that require high levels of control have also been shown to recruit activation in the LPFC (Ullsperger & von Cramon, 2004). Hemispheric LPFC activity may be distinguishable by task as well, with activity in the left LPFC correlated with the compensatory behaviors following erroneous responses and the right LPFC being involved in response inhibition during high conflict tasks (Garavan et al., 2002).

Therefore, the ACC and the LPFC are both important to cognitive control since both performance monitoring and behavioral adjustment are necessary for adaptive responses to one's actions. While these two regions may be tied to different steps in the cognitive control process, evidence also exists that these two regions work together as an integrated system to allow for adaptive responses to behavior. For example, fMRI results of trials tied to erroneous responses revealed activity in both the rostral ACC and the left

LPFC (Kiehl et al., 2000) while activity in the caudal ACC as well as the dorsal LPFC were observed on high conflict trials (Mathalon et al., 2003). In addition, both LPFC and ACC activity are involved in behavioral responses after high conflict trials (Garavan et al., 2002). These studies demonstrate that activity in both regions is necessary for effective cognitive control. Furthermore, fMRI studies have demonstrated that ACC activity on high conflict and erroneous trials is significantly correlated with LPFC activity on the following trial beyond what could be explained by random fluctuations in brain activity (Kerns et al., 2004). Therefore this research underlines the fact that these brain regions are part of an integrated mechanism and that high levels of functioning in both the ACC and LPFC are essential to the cognitive control system.

Emotion regulation

While self-regulation has been widely researched in the cognitive domain, questions remain as to whether cognitive control might be tied to other forms of self-regulation, namely emotion regulation. Emotion regulation has been defined differently in the literature, however one definition that seems to capture the complexity of this ability is as follow: “the process by which we influence which emotions we have, when we have them, and how we experience and express them” (Gross, 2002; p. 282). As a follow-up to this broad definition, Ochsner and Gross (2005) have offered a more specific definition of the regulation of emotion; “attending to or interpreting emotion-eliciting situations in ways that limit emotional responding” (p. 243), which leads to a neutralization of the negative emotion (Gross, 2002). While emotion regulation may apply to both positive as well as negative emotional experiences, we will concentrate on negative emotions since these are more likely types of emotions that one would need to

down-regulate for adaptive functioning. Emotional dysregulation can take a huge toll on one's physical, mental, and social well-being and can also lead to more serious mental health disorders such as major depression (Gross & Munoz, 1995). Therefore, emotion regulation can be thought of as an adaptive response to emotion eliciting stimuli that one encounters.

Researchers are beginning to elucidate the many ways in which humans may achieve this form of self-regulation. There are various strategies one may use to down-regulate a certain emotion. These include situation selection, or the avoidance of certain emotion eliciting situations, situation modification where one modifies a given situation to lessen the emotional impact, and attentional control (the amount of attention that one pays to a given emotional stimulus). Distraction is another approach one can take to limit the amount of attention paid to a certain stimulus. Cognitive change involves attributing a particular meaning to a given situation or stimulus that will lessen emotional responding or change the emotion itself. For example, reappraisal involves reinterpreting the meaning of a given emotional stimulus in order to limit emotional responding (Gross, 2002; Ochsner & Gross, 2005). Regulation of emotion through cognitive strategies, and in particular reappraisal strategies, is associated with the experience of less negative emotion (Gross, 2002).

Both conceptual and anatomical similarities between cognitive control and emotion regulation point to a potential relation between these two forms of self-regulation. On a conceptual level, both cognitive control and emotion regulation can be seen as adaptive responses to environmental stimuli. One must first recognize that an emotional response to a given stimulus is not adaptive or does not match one's goals.

While emotions are often essential for life's demands, they can also be ill-matched to a particular situation or environment. For example, if one is afraid of heights but has an office on the top floor of a tall building, this fear response is not conducive to one's goals of succeeding at work everyday. Therefore, one must first recognize that an emotional state or emotion-producing situation will cause us more harm than good and conflict with our goals. However, one must then do something to down-regulate these emotions in order to adaptively respond to a given stimulus or environment (Gross, 2002).

Researchers have also described emotion processing and regulation in a manner similar to the steps involved in cognitive control. For example, Phan and colleagues (2004) state that humans must both perceive and react accordingly to emotions, similar to the two steps described in cognitive control. As these researchers describe, emotional responses can be seen as reflexive in nature and attempts at regulating these emotions may overlap with the cognitive control of behavior and rely on similar brain regions to do so (Phan, Fitzgerald, Nathan, Moore, Ude, & Tancer, 2004). In addition, both cognitive control and emotion regulation seem to rely on mechanisms that are involved in both attentional control in relation to goals as well as resolving conflicts between interfering emotional stimuli and task relevant stimuli (Bantick, Wise, Ploghaus, Clare, Smith, & Tracey, 2002; Bishop, Duncan, Brett, & Lawrence, 2004; Ochsner et al., 2004).

Evidence from various neurological dysfunction and neuroimaging supports this theoretical connection between cognitive control and emotion regulation systems. Studies of patients with brain lesions in the PFC have suggested that this region may be involved in emotion regulation given the fact that these patients showed abnormalities in the ability to adaptively regulate their emotions and showed more mental disturbances as a

result. For example, individuals with abnormal circuitry in the PFC are at risk for increased aggressive impulses and violence (Davidson, Putnam, & Larson, 2000).

More recent fMRI studies also suggest that brain regions involved in cognitive control (the pre frontal cortex and the ACC) are activated during emotional self-regulation. Beauregard, Lévesque and Bourgouin (2001) demonstrated that voluntary inhibition of sexual arousal in response to erotic film excerpts through distancing oneself from the stimuli led to increased activation in the dorsolateral PFC (BA 10) and the right ACC (BA 32). Reappraisal strategies also seem to lead to similar patterns of brain activation. The lowering of subjective negative affect in response to photographs through the use of reappraisal strategies was associated with activation in the left LPFC. In addition, higher right ACC activity was also correlated with more effective reappraisal (Ochsner, Bunge, Gross, & Gabrieli, 2002). A similar study further replicated these findings. Participants who used reappraisal to regulate their emotional response to negative pictures showed activation in both the dorsal and ventral LPFC as well as in the dorsal ACC. In addition, increased activity in the dorsal ACC and the dorsal LPFC was negatively correlated with intensity of negative emotion, indicating more effective emotion regulation (Phan, Fitzgerald, Nathan, Moore, Uhde, & Tancer, 2005).

Together these studies suggest that activity in both the ACC and the LPFC is associated with emotion regulation and that higher levels of activity indicate more effective regulation. These results have been replicated in other studies using different types of emotion regulation strategies in response to various types of emotion, such as the use of distraction to modulate the emotional experience during painful stimuli (Bantick et al., 2002). While there is evidence that somewhat different patterns of activation are

present depending on the emotion regulation strategy one uses, overall evidence supports the notion that the LPFC and the ACC are both critical to adaptive emotion regulatory ability (Ochsner et al., 2004). Even if individuals successfully modulate their emotional response to negative photographs but are given no specific strategy to employ, similar brain regions, specifically the LPFC, are still activated (Kim & Hamann, 2007). These studies point to the flexible nature of emotion regulation with regards the type of emotion being regulated as well the type of strategy that is employed to achieve this regulation.

Research indicates that emotion regulation is also accompanied by attenuation of activity in regions important in emotional processing (namely the limbic structures) and suggests a neural mechanism through which this regulation is achieved. This decrease in activity in limbic regions such as the amygdala would lead to less subjective experience of emotion since these regions are critical to emotional processing and evaluation and therefore the experience of emotional salience. Decreased activity of limbic regions such as the amygdala, hypothalamus, and medial orbital frontal cortex are associated with activation in the ACC and LPFC (Phan et al., 2005; Ochsner et al., 2004; Beauregard et al., 2001; Kim & Hamann, 2007). More specifically, levels of ventral LPFC activity were negatively correlated with amygdala and medial orbital frontal cortex activity, suggesting that LPFC may modulate this activity more directly through connecting neural circuits (Ochsner et al., 2002).

Stress regulation

While there are many instances where emotion regulation is necessary for adaptive behavior, the ability to effectively cope with stressful situations is very important to human functioning. High levels of distress and anxiety can result in

psychopathology as well as reduced physical health, making the regulation of this type of emotion extremely important in daily life. Stress can be defined as a process that results from “a mismatch between the demands of a given situation and the individual’s perceived ability to deal with those demands” (Matthews, Deary, Whiteman, 2003: 242). Stressors are external events or situations that one appraises as taxing or impossible to deal with. These stressors then elicit stress symptoms, such as feelings of anxiety or general distress. However, one may try to down-regulate these feelings of distress and limit emotional responding to stressors through coping, a form of emotion regulation in stressful situations (Matthews, Deary, & Whiteman, 2003). Therefore, stress regulation may be seen as one form of emotion regulation where one engages in coping/regulatory strategies to limit a negative emotional response to the stressor.

Neuroimaging studies also show that emotion regulation attenuates feelings of anxiety in response to stressors and that the same brain regions implicated in forms of emotion regulation described above are also activated during stress regulation. Research has shown that the use of a cognitive regulation strategy (detaching oneself from an emotional stimuli) in response to a particular type of stressor, namely the anticipation of a painful shock to the hand, was associated with activity in the right anterior lateral PFC. This form of stress regulation also attenuated feelings of subjective anxiety in participants (Kalisch et al., 2004). In addition, Bishop and colleagues (2004) demonstrated that individuals who scored higher on measures of state anxiety levels also showed lower general levels of activation in the rostral ACC. This finding implies that ACC activation is necessary for the successful down-regulation of anxiety and that individual differences in activation levels are inversely associated with anxiety levels. Therefore these findings

indicate that the efficacy of stress regulation as well as emotion regulation correspond to levels of activation in the ACC and LPFC. Successful stress regulation, as well as emotion regulation more generally, seems to rely on the activation of the same neural mechanisms.

A Link Between Control Systems

Based on these conceptual and neural parallels between two different forms of self-regulation (emotion regulation and cognitive control), one would expect that cognitive control and emotion regulation rely on similar control systems in the brain. An analysis of the research in both of these areas of self-regulation points to many similarities between brain regions activated during cognitive control as well as emotion regulation. If there is a functional overlap between the neural mechanisms involved in both forms of self-regulation, then one would expect to see that more effective cognitive control is associated with more effective emotion regulation due to more efficient processing and activity in the ACC and LPFC.

While most prior studies regarding the neural correlates of emotion regulation have noted this plausible connection, this link has not been thoroughly researched. As Ochsner and Gross have recently suggested, “the consistent involvement of control-appraisal system dynamics in various forms of regulation suggests a common functional architecture” and points to the striking similarities between these two forms of self-regulation (Ochsner & Gross, 2005; p.246). This connection has also been suggested by other researchers who have demonstrated activation in the ACC and LPFC during emotion regulation (Ochsner et al., 2002; Phan et al., 2005). However, direct correlational studies linking measures of both forms of self-regulation are lacking in the literature

making it difficult to draw a more direct connection between cognitive control and emotion regulation.

One study however does point to a significant correlation between cognitive control and emotion regulation. Compton, Robinson, Quandt, Fineman, Carp, and Ode (in press) demonstrated that individuals with more effective and efficient cognitive control systems (as measured by higher ERN's and changes in performance speed and accuracy following errors on a Stroop task) also showed more effective stress regulation measured by the amount of subjective anxiety individuals experienced in response to daily stressors. Both error monitoring ability as well as behavioral changes in response to errors independently predicted the degree to which levels of anxiety increased in response to stressors, further suggesting that high levels of functioning in both the ACC as well as the LPFC are critical to the ability to down-regulate non-adaptive emotions such as anxiety. Therefore, this study demonstrates that individuals who show more effective levels of cognitive control also experience less subjective feelings of anxiety in response to daily stressors.

While this study does imply that cognitive control and emotion regulation draw on similar neural processes and overlapping abilities, there are several limitations to this study that hinder a conclusive result. First, this study relied on self-report measures of stressors and anxiety. Information about participants' responses to daily stressors and their resulting levels of anxiety were collected via online self-report measures over a two-week period. This leads to a potential confounding variable in that different individuals may interpret the meaning of anxiety differently and thus these self-ratings may not reflect the actually amount of subjective anxiety experienced. A second limitation to this

study is that the type of stressor experienced was not controlled. In particular this study looked at five different types of stressors (for example “a lot of responsibilities” or “health problems or fatigue”). This adds variability to the study since participants may have experienced stressors that were not on this list. Therefore, while this study provides preliminary evidence that cognitive control and emotion regulation rely on the same fundamental mechanism, more research is needed to further address the existence as well as the foundations of this link.

The Current Study

In the current study, we investigated individual differences in cognitive control and emotion regulation in response to a stressor using more controlled measures of emotion regulation. In this way we intended to expand upon the knowledge regarding these two separate systems and help to establish a more nuanced view of the potential overlap between these two control systems. First of all, we extended previous findings by controlling the type of stressor participants experienced through the induction of anxiety in the lab through a stress manipulation. In this way all participants experienced the same environmental stressor, which minimizes one potential confounding variable. Second, instead of relying on self-report measures of anxiety, we assessed the effectiveness of emotion regulation in more objective ways.

In particular, this study addressed the association between these two control systems in a different manner than was previously used to examine this question. We investigated the relationship between cognitive control and physiological measures of emotion regulation, a relationship that has not yet been examined in the literature. Physiological responses to a stressor or other environmental stimuli are one aspect of an

emotional response and provide a more direct unbiased measure of emotional responding. In addition, a relationship between cognitive control and emotion regulation may be more directly seen through physiological rather than self-report measures since the connection between these two processes is evident in overlapping neural correlates.

Psychological stress is associated with particular physiological changes in the body that we will measure. When an individual appraises and evaluates an external event as overwhelming one's resources at hand, a chain of events is initiated. This begins in the cerebral cortex, including the prefrontal cortex, where incoming information concerning all aspects of a given situation including sensory information, interpretation of the environmental situation, and one's past experience are synthesized and evaluated. If the environmental stimulus is interpreted as a threat, then the next step in the process is initiated. This step relies on pathways between the frontal cortex and the amygdala, a structure well known for its importance in emotional processing. This connection to the amygdala is an important component in the formulation of an emotional response to a psychological stressor, such as anxiety. However, it is also critical to the endocrine response to anxiety and stress, namely the release of the hormone cortisol (Lovallo & Thomas, 2000).

The amygdala connects to the hypothalamus via multisynaptic pathways that lead to the paraventricular nucleus (PVN) of the hypothalamus. This is the beginning of the hypothalamic-pituitary-adrenal axis (the HPA axis), which is responsible for the production of the hormone cortisol under normal conditions as well as stressful ones. Specialized neurons called corticotropin releasing factor (CRF) neurons release CRF, which is then carried to the pituitary gland, where ACTH is formed. ACTH then causes

the adrenal cortex to secrete cortisol. Cortisol is needed under normal states to maintain growth and development as well as metabolic functions. Cortisol levels vary throughout the day and serve to regulate metabolic processes in the body. Under stress, however, the production of cortisol is enhanced (Lovallo & Thomas, 2000).

This secretion of cortisol is associated with negative affect and situations resulting in failure and loss of reward. Cortisol secretion was found to rise in relation to the perceived aversiveness of a given situation and can therefore be tied to the levels of distress associated with a situation (Lovallo, 1997). This was also observed in a naturalistic study where participants were told to report stressors in their lives and their accompanying emotional state six times a day and to provide cortisol samples. Cortisol levels increased in response to stressors and were associated with negative affect (Smyth, Ockenfels, Porter, Kirschbaum, Hellhammer, & Stone, 1998). Heart rate is another physiological measure that is affected by activation of the HPA axis, with CRF neurons activating the sympathetic nervous system and increasing heart rate. In response to a laboratory stressor, individuals with high heart rates also showed high cortisol levels, suggesting that these two physiological responses are tied and that individual differences in physiological stress reactivity are reliable (Sgoutas-Emch, Cacioppo et al., 1994).

Given this information, levels of cortisol secretion (as well as heart rate) can be seen as a physiological component of emotion regulation in response to a stressor. This relation between cortisol and stress regulation is two-fold. First of all, on a conceptual level, higher levels of stress regulation will lead to lower levels of cortisol as well as a reduction of anxiety and related emotions. Based on studies cited above, if a given situation is not associated with negative affect, the situation will not be appraised as

stressful and the cascade of events involved in the production of cortisol will not be initiated. There is evidence supporting a strong link between depression, a form of emotion dysregulation, and a dysregulated HPA axis (Brown, Varghese, & McEwen, 2004). This implies that cortisol responses and HPA axis activity are associated with levels of stress regulation.

However, the relationship between stress regulation and cortisol can be more strongly seen on an anatomical level. Activity in the ACC and LPFC during emotion regulation has also been linked to a concurrent reduction and attenuation of activity in the amygdala. This was observed in conjunction with the strategy of reappraisal in response to negative images as well as during inhibition of sexual arousal (Beauregard et al., 2001; Ochsner et al., 2002; Ochsner et al., 2004; Phan et al., 2005). More specifically, lower levels of negative affect (associated with higher levels of activation in the ACC and LPFC) were significantly correlated with decreases in amygdala activation (Ochsner et al., 2004). However, a more interesting finding coming from two separate studies is that activity in the ventral LPFC is negatively correlated with activity in the amygdala (Ochsner et al., 2002; Phan et al., 2005). This demonstrates that activity in the LPFC may directly inhibit or modulate amygdala activation. As described above, amygdala activation is critical to the production of cortisol in response to stressors. Therefore, these studies suggest a direct mechanism by which efficient activation of the ACC and the LPFC (in particular the ventral LPFC) may lower the physiological response to stress as well as reduce the subjective experience of anxiety.

This also points to the role of the anatomical connections between the PFC and the ACC with regards to the limbic system and in particular to the amygdala. The frontal

cortex and the ACC both have anatomical connections to the amygdala as well as the hypothalamus, both structures involved in cortisol production (Beauregard et al., 2001; Feldman, Conforti, & Weidenfeld, 1995). The ACC is also considered by some as part of the limbic system and related to emotional processing. It is well connected to the amygdala, pariaqueductal gray, nucleus accumbens, hypothalamus, and the orbitofrontal cortex, and by way of these connections can influence endocrine systems connected to stress, such as cortisol (Bush, Luu, & Posner, 2000). This therefore provides additional support for this mechanism through which the LPFC and the ACC could modulate cortisol release in response to stress.

In addition to examining a physiological measure of emotion regulation, this study also utilized a self-report measure of efficacy of emotion regulation in response to stress. This allowed us to look at another measure of emotion regulation in addition to physiological changes to further explore the relationship suggested by Compton et al. (in press). While the emotion regulation literature has identified potential strategies one uses in order to down-regulate one's emotions, the coping literature has done the same for stress regulation. Coping strategies can be broken down into three main types: problem focused coping (directly confronting the problem at hand), emotion focused coping (focusing on the emotions resulting from a stressful situation), and avoidance coping (ignoring the stressor) (Cosway, Endler, Sadler, & Deary, 2000).

Problem focused coping seems to be the most effective of the three and can be related to effective stress regulation. It has been negatively correlated with outcome measures such as depression, anxiety, and overall psychological distress (Cosway et al., 2000), demonstrating its effectiveness in stress regulation. In addition, problem focused

coping may overlap with more general forms of emotion regulation in that problem focused coping may be one form of situation modification, where an individual directly focuses on modifying the situation, or stressor, to regulate their anxiety (Gross, 2002). Individuals do seem to use one type of coping strategy over another consistently across situations, suggesting that these strategies possess trait-like qualities and differences between individuals (Endler & Parker, 1994). The extent to which individuals use these three different types of coping strategies can be measured by the Coping Inventory for Stressful Situations (CISS) (Endler & Parker, 1990a, 1990b, 1994, 1999) and therefore provides information on individual differences in effective emotion regulation in response to stressors.

Hypotheses

Conceptual parallels between the steps involved in cognitive control and emotion regulation as well as the overlapping brain regions (the ACC and the LPFC) responsible for these two forms of self-regulation point to a link between the two regulatory systems. Therefore, we predicted that more effective levels of cognitive control (and more effective functioning of the ACC and LPFC) would be correlated with more effective emotion regulation and less anxiety in response to a laboratory-manipulated stressor. Our particular hypotheses were as follows:

- 1) Individuals displaying higher levels of cognitive control (as measured by ERN amplitudes, correct responses after errors on a Stroop task, and a post-error decrease in performance speed on the Stroop task) would show lower cortisol levels in response to a stressor

- 2) Individuals with higher levels of cognitive control would have lower heart rates in response to the stressor
- 3) Individuals with higher levels of cognitive control would score higher on the CISS problem-focused coping measure, indicative of more effective stress regulation

Methods

Participants

37 Haverford College undergraduate students (19 females) participated in this study in exchange for payment. Participants were recruited via online postings and e-mail. Participants who completed a prescreening questionnaire for the study were entered in a lottery to win \$50. Participants eligible for the study received \$30 for their participation. Prescreening excluded people with a learning disability, neurological history, uncorrected visual defects, or the regular use of substances or medication that affect the central nervous system. Participants were also asked to refrain from smoking and drinking caffeinated beverages, milk or coffee one hour prior to coming into the lab.

Design

This study used a correlational design to compare individual differences in cognitive control (as measured by ERN amplitude, accuracy after errors on the stroop task, and reaction times after errors on the Stroop task) to individual differences in emotion regulation (as measured by cortisol levels, heart rate, and the CISS questionnaire).

Procedure

Cognitive Task

To measure error monitoring ability and correct responses after committing errors, we followed the same methods used by Compton et al. (in press). This task consisted of a 6-choice version of the Stroop color-identification task. On each trial, participants indicated the color of the target word (red, orange, yellow, green, blue, or purple) with the first three fingers of each hand on a keyboard. The task started with a practice block of 24 trials during which accuracy feedback was given to ensure that participants understood the task. For the main experiment, 10 blocks of 66 trials each were used (660 trials total) without accuracy feedback. At the beginning of each trial a blank screen was presented for 500 ms, followed by the presentation of the target word for 150 ms and then another blank screen that remained until a keypress or 2 ms elapses. There were short breaks during each trial block, during which participants were reminded of the directions for the task. In each block there were 30 incongruent trials where the word color and spelling did not match (e.g., the word “red” spelled in green ink), 6 congruent trials where the word spelling and color did match, and 30 neutral trials where non-color words were displayed in various colors. All stimuli were presented against a black background.

EEG Recording and Signal Processing

Electrodes were applied using an elastic cap (Quick-Caps) fitted with sintered Ag/AgCl electrodes. We recorded data continuously from 8 sites on the scalp: Fz, FCz, Cz, Pz, F3/F4, and C3/C4. Since error-related ERP's are normally observed on the fronto-central midline sites (Fz, FCz, Cz, and Pz) we focused on these sites. In order to amplify signals, a NuAmps amplifier (controlled by Neuroscan software) with a sampling rate of 1000 Hz and a bandpass of 0.1-40 Hz (-3dB) was used. We referenced data on-

line to the right mastoid and digitally re-referenced data off-line to the average of the left and right mastoid. Electrodes above and below the left eye and at the outer canthus of each eye monitored eye movements. These recordings were then used to compute bipolar horizontal and vertical EOG channels off-line. We addressed artifacts off-line in three steps. First, after visually inspecting the EEG data portions large non-blink artifacts were manually excluded. Next, using Neuroscan software's regression-based algorithm for ocular artifact reduction the effects of blinks were reduced. Finally, any remaining artifacts in the EEG record were identified by using a ± 150 uv threshold and these epochs were excluded. Following baseline correction, we carried out response-locked signal averaging separately for correct and incorrect trials, with an epoch window of -200 to 600 ms surrounding the response.

Stress manipulation

In order to measure stress regulation in the lab, we induced stress under controlled circumstances. We followed the procedures used by Sgoutas-Emch et al. (1994) and Bolini et al. (2004), which have both been shown to elicit individual differences in physiological responding. This stressor consisted of a public speaking task as well as a mental arithmetic task. First, participants performed the public speaking task based on the procedures used by Sgoutas-Emch et al. (1994). Participants were asked to imagine that they were in a department store shopping and a security guard falsely accused them of shop-lifting. They were given 3 minutes to prepare a 2 minute speech that included telling their side of the story, telling the manager of the store why the security guard was wrong and why he might have falsely accused the participant of shop lifting, stating how the participant can prove that they are innocent, specifying what penalties the security

guard should face for the mistake, and summarize their points in the end. We told participants that their speech would be video-recorded and compared to other participants in the study. While there was a video camera present, participants' speeches were not actually recorded.

This was then followed by the mental arithmetic task based on that used by Bollini et al., 2004. Participants were presented with a series of numbers (from 1 to 15) on a computer screen. Each number was presented for half a second, followed by a + sign which was presented for 20 ms. The task consisted of adding the number that the participant saw on the screen with the number they saw just before it (while ignoring the answers they gave for the added numbers) and saying the number out loud to the experimenter. In addition, loud, aversive noises were presented via headphones to make the task more challenging. Noises included tires screeching, white noise, a horn, a vacuum, and nails on a chalkboard. The task included three blocks of 60 numbers with a practice block of 10 numbers. During the practice block, participants' answers were corrected by the experimenter and there were no noises presented. At the start of the task participants were told that their answers were being recorded by the experimenter and that they would be compared with other participants in the study on their accuracy (although again this was not the case)..

Both these types of lab stressors have been shown to induce anxiety and exaggerated physiological responses to stress, including high cortisol responses. This is due to the fact that stressors containing a social-evaluative threat (such as comparison with other participants) as well as uncontrollability (the random noise blasts) threaten central goals of an individual, such as social self-preservation, and are considered to be

stressful by most individuals. The combination of both of these stressors has been shown to elicit the largest cortisol response among participants in lab studies and to provide a larger range in which to analyze individual differences in physiological stress regulation (Dickerson & Kemeny, 2004).

Physiological measurements

We measured cortisol levels in participants three times during the lab session: when participants first arrived, after participants completed the cognitive task, and 20 minutes after the onset of the stress manipulation since this is when cortisol peaks (Dickerson & Kemeny, 2004). This took into account any change in baseline levels of cortisol levels induced by the cognitive task and the EEG measurement. Cortisol levels were collected from participants through saliva samples. Participants were asked to drool through a straw into a small test tube. These samples were immediately refrigerated and later analyzed for cortisol levels via an enzyme immuno assay (EIA) procedure. We used a corticosterone EIA kit commercially available from DS labs in Arlington, TX. To perform this assay we first centrifuged all the samples and then incubated them with the antibody. After performing the assay we read the cortisol concentrations on a plate reader at 600nm. We performed the assay on duplicate samples for each individual at each time period. A heart rate measurement was also taken via a wristband device at the same three points as the cortisol samples.

Self-report measures

After completing the stress manipulation, participants filled out a self-report questionnaire. Participants were first asked to rate their stress level at that point in time on a 10-point likert scale with 0 being no stress and 10 being very stressed. This was

followed by a subjective measure of anxiety level, the State-Trait Anxiety Inventory, which asked participants to rate on a scale of 1-4 which statements best described their feelings during the testing session (Spielberger, 1968). Items included statements such as “I feel calm”, “I feel strained”, or “I am tense”. In addition to this, participants completed the Coping Inventory for Stressful Situations (CISS) (Endler & Parker, 1990a, 1990b, 1994, 1999). This inventory consists of 48 items where individuals rate how much they engage in a certain activity using 5-point Likert scales. Sample items include “schedule my time better”, “blame myself for procrastinating”, and “try to be with other people”. This inventory measures the extent to which individuals utilize the three types of coping strategies (problem focused, emotion focused, and avoidance coping) and has proven to be a reliable measure of these coping styles. Each coping strategy is measured by 16 items and shows strong construct validity and external validity (Endler & Parker, 1994). The validity of this construct has been confirmed in other studies (Cosway et al., 2000).

Results

ERP Data

Grand-average response-locked waveforms are presented in Figure 1 for the FCz site for error versus correct trials. The ERN peak in each participant’s waveform was defined as the most negative point within the interval 0 to 100 ms surrounding the button press response during the Stroop task.

A 2X4 repeated measures ANOVA on peak amplitude with site location (Fz, FCz, Cz, and Pz) and trial accuracy (correct vs. incorrect) as factors was used to ensure the presence of an ERN in the group as a whole using Greenhouse-Geisser adjustments. As expected there was a main effect of trial accuracy, with a greater negativity following

errors ($M=-6.70$ μV , $SE=.75$) than following correct responses ($M=-1.69$ μV , $SE=.52$; $F(1, 35)= 48.15$, $p<.001$). There was also a significant interaction between site and trial accuracy ($F(3,105)=4.33$, $p<.05$), reflecting greater error vs. correct differentiation at the FCz site than at the other three sites (Fz, Cz, and Pz). Means and standard errors are listed in Table 1. Due to this significant interaction further analyses focus only on the FCz site, where the ERN was most pronounced.

Behavioral data

The group as a whole did significantly slow down after errors ($M=742.64$ ms, $SD=158.53$) as opposed to after correct responses ($M=657.06$ ms, $SD=110.42$); $t(36)=5.10$, $p<.001$. But, participants tended to be less accurate on trials following errors ($M=86\%$, $SD=.10$) than on trials following correct answers ($M=92\%$, $SD=.04$); $t(36)=-4.48$, $p<.001$.

Measures of cognitive control

To examine individual differences in cognitive control, we used three different measures. First, we constructed an ERN difference score using the FCz site (the amplitude after a correct response minus the amplitude after an incorrect response). A higher ERN difference score is therefore indicative of better ERN differentiation of errors and more effective cognitive control. We also calculated a reaction time (RT) difference score (RT on trials following correct responses minus RT on trials following errors) where a lower score indicated a greater slowing after errors, and an accuracy difference score (accuracy after errors minus accuracy after correct trials) where a higher score indicated a higher level of accuracy following errors. Individuals with higher ERNs displayed higher levels of accuracy following errors ($r=.42$, $p<.05$), but there was no

significant correlation between ERN amplitude and the RT difference score. Overall accuracy rates were positively correlated with the accuracy and ERN difference scores ($r=.48, p<.01$ and $r=.49, p<.01$, respectively), suggesting that both this behavioral measure of accuracy following errors as well as the electrophysiological measure of cognitive control (the ERN) predict successful performance on a cognitive task and cognitive control ability. There was no significant correlation between the RT difference score and overall accuracy.

Cortisol data

For the purposes of our analysis, we chose to use the average of the duplicate cortisol concentration readings we obtained for each participant since there was a high significant correlation between the duplicate readings ($r=.78, p<.01$). Contrary to our predictions, the results of a one-way ANOVA showed that overall salivary cortisol concentration levels in the group did not significantly differ between the three different times samples were taken (at the beginning of the lab session at time 1 (T1) ($M=1.25$ ug/dL, $SE=.12$), post cognitive task at time 2 (T2) ($M=1.29$ ug/dL, $SE=.14$), and after the stress manipulation at time 3 (T3) ($M=1.30$ ug/dL, $SE=.17$). Levels of cortisol concentration were not significantly correlated with the time of day the subject came into the lab. Cortisol levels were, however, highly correlated with one another (time 1 and 2: $r=.70, p<.001$; time 1 and 3: $r=.56, p<.001$; and time 2 and 3: $r=.71, p<.001$). This provides additional validation of our cortisol assay in that individuals with high levels of cortisol at T1 also showed high levels of cortisol at T2 and T3, a stable individual difference that would be expected.

Heart rate data

A measurement of heart rate was taken at the same three points in time as the salivary cortisol samples. A one-way ANOVA on heart rate revealed a significant effect of time ($F(2,66)=20.37, p<.001$). However, a post hoc pairwise comparisons test (the least significant differences test) showed that heart rate in the overall group significantly decreased ($p<.001$) from the first time point ($M=81.44, SE=3.00$) to the second time point ($M=71.94, SE=2.32$). There was no significant difference between time 2 and time 3 ($M=71.71, SE=2.25$) heart rate measures, again contrary to our predictions. The three different heart rate measures were significantly correlated with one another (T1 and T2: $r=.77, p<.001$; T2 and T3: $r=.79, p<.001$; T1 and T3: $r=.79, p<.001$).

Self-report measures

35 participants filled out the CISS online questionnaire after the stress manipulation. Overall, participants' scores on the problem focused coping subscale ranged from 2.13 to 4.69 ($M=3.65, SD=.72$). The CISS emotion subscale consisted of a mean of 2.66 ($SD=.75$) with a range in scores from 1.13 to 4.00. The CISS avoidance subscale showed a mean of 2.46 ($SD=.61$) and a range of 1.13 to 3.63. 34 participants completed our stress question ($M=5.44, SD=2.20$; range 0-9) and all 37 participants completed the STAI questionnaire ($M=2.53, SD=.45$; range 1.70-3.35). There was a significant positive correlation between the CISS emotion focused coping subscale and the STAI and stress question ($r=.45, p<.01$ and $r=.47, p<.01$, respectively). Individuals with higher scores on the emotion focused coping subscale of the CISS also reported more anxiety and stress during the stress manipulation. There was also a significant positive correlation between the CISS avoidance scale score and the stress question

($r=.54, p<.01$). Together these correlations provide validation for the CISS as a measure of stress regulation.

Relationship between cognitive control and emotion regulation

Cortisol and cognitive control

In order to test our first hypothesis that individuals with higher levels of cognitive control would show lower salivary cortisol levels in response to a stressor, we correlated our three measures of cognitive control (ERN amplitude, accuracy following errors, and post-error reaction time on the Stroop task) with a cortisol difference score: the change in cortisol levels between T1 and T2, T2 and T3, and T1 and T3.

There was a significant negative correlation between ERN amplitude and the change in cortisol levels between T1 and T2 ($r=-.44, p<.01$). Participants who had higher ERN amplitudes also showed smaller increases in cortisol levels between T1 (the baseline measure) and T2 (after the Stroop task). There was also a significant negative correlation between overall accuracy on the Stroop task and the cortisol difference score between T1 and T2 ($r=-.40, p<.05$). While these two effects were not completely independent of one another, the relationship between ERN amplitudes and changes in cortisol in response to the Stroop task still remained at trend level after a partial correlation controlling for overall accuracy (partial $r=-.32, p=.064$). Contrary to our hypotheses, however, there was no significant relationship between ERN amplitude and the change in cortisol levels after the stress manipulation (T3) compared to both baseline levels (T1) and after the Stroop task (T2) ($r=-.095, p=.58; r=.28, p=.105$, respectfully).

A relationship between the change in cortisol levels during the Stroop task and the behavioral measure of cognitive control (the accuracy difference score) also showed a

trend towards significance ($r=-.28, p=.092$) in that individuals who were more accurate following errors on the Stroop task also tended to have smaller cortisol increases during the Stroop task (T1 to T2). There were no significant correlations between the other cortisol difference scores and the accuracy difference score or the reaction time difference score.

Heart rate and cognitive control

There was no significant correlation between the three different heart rate difference scores (constructed in the same way as the cortisol difference scores) and the ERN amplitude, contrary to our second hypothesis.

Self-report measures and cognitive control

To test our third hypothesis that individuals with more effective cognitive control would score higher on the CISS measure of problem focused coping, we correlated our three measures of cognitive control with the CISS subscale scores. There were no significant relationships between any of the CISS subscales and the ERN. This was also the case for the STAI question as well as the stress question. There were also no significant correlations between the accuracy difference score and the CISS or STAI score. There was a significant correlation between the reaction time difference score and self-reported stress levels ($r=-.35, p<.05$). However, this was in the opposite direction than that predicted since individuals who slowed down more after errors reported higher subjective levels of stress.

Measures of emotion regulation

Correlations between the three heart rate and cortisol change scores were not significant. There were no significant correlations between the self-report measures and

cortisol expect for one negative correlation between the change in cortisol from T1 to T2 and the participant's subjective experience of stress at the end of the stress manipulation ($r=-.37, p<.05$). This finding is puzzling as well since participants who reported higher subjective levels of stress showed a smaller increase in cortisol levels between time 1 and time 2.

Discussion

The main goal of this study was to assess individual differences in cognitive control and emotion regulation. We predicted that individuals with more effective cognitive control levels (as measured by the amplitude of their ERN and their behavioral response after making an error on the Stroop task) would show better emotion regulation in response to a stressor. In particular, we predicted that individuals demonstrating higher levels of cognitive control would have lower cortisol levels and a lower heart rate in response to the stress manipulation and a higher score on the CISS problem focused coping subscale.

Relationship between cognitive control and cortisol levels

Our main finding in this experiment does show that cognitive control is correlated with a physiological measure of emotion regulation. There was a significant relationship between cognitive control and changes in cortisol levels during the Stroop task in the direction we predicted. Individuals who had higher ERN amplitudes also had smaller increases in cortisol levels in response to the Stroop task. Since overall accuracy on the Stroop task was also significantly correlated with individual changes in cortisol levels, we needed to rule out the possibility that overall accuracy on the Stroop task was mediating the relationship between the change in cortisol levels and cognitive control.

More specifically, it was possible that individuals with higher levels of cognitive control performed better on the Stroop task and therefore found the task less stressful. However, this relationship between the ERN and cortisol levels remained at trend level when we statistically controlled for overall accuracy. While there was no significant relationship between the behavioral measure of accuracy after errors and cortisol levels, there was a strong trend in that individuals who were more accurate following errors on the Stroop task (indicative of higher levels of cognitive control) also exhibited smaller increases in cortisol in response to the Stroop task. The lack of a relationship between the reaction time and cortisol change scores can be explained by the fact that we did not find any significant relationships between the reaction time score and ERN amplitude or overall task accuracy. This further validates the parallel findings of Compton et al. (in press) that reaction time is not an accurate measure of cognitive control.

These results are consistent with our hypothesis that there is a relationship between cognitive control and emotion regulation. We expected to find this relationship since similar regions of the brain (both the ACC and the LPFC) have been implicated in both cognitive control and emotion regulation. There are also conceptual parallels between the two methods of self-regulation, which leads to a logical connection between the cognitive control and emotion regulation. These results are also in line with previous research findings. Compton et al. (in press) found that individuals with higher ERN amplitudes and higher levels of accuracy following errors on a Stroop task reported less anxiety in response to daily life stressors. This effect was independent of mean daily stress scores, suggesting that this relationship was not mediated by overall success in meeting the demands of life. Therefore, our study further validates the existence of an

overlap between neural systems supporting both forms of self-regulation. Our results also demonstrate that cognitive control is connected to a physiological measure of emotion regulation, specifically cortisol levels, in addition to self-report measures of anxiety. Since this specific connection has not yet been examined in the literature, our finding adds another dimension to the link between cognitive control and emotion regulation.

However, this finding was not present with regards to the stress manipulation as we originally predicted. Therefore the Stroop task somehow tapped the association between cognitive control and emotion regulation in a way that the stress manipulation did not. While we did not initially predict to see a relationship between cognitive control and emotion regulation in response to the Stroop task, previous research has shown that the Stroop task is a type of stressor that can induce significant cortisol increases since it contains a social-evaluative threat (i.e., the participant feels like their competence is in question if they do not perform well) (Dickerson & Kemeny, 2004). In addition, results of a recent study suggest that high ERN amplitudes are tied to a larger decrease in salivary cortisol levels in response to a Flanker Task (a cognitive task similar to the Stroop task) (Tops, Boksem, Wester, Lorist, and Meijman, 2006). A larger decrease in cortisol levels could also be viewed as indicative of better emotion regulation ability. Therefore, these studies demonstrate that the Stroop task is a type of cognitive stressor and it reflects individual differences in emotion regulation ability.

One explanation for the lack of a significant relationship between cognitive control and cortisol reactivity to the stress manipulation is that cognitive control is more closely linked to stress regulation in response to cognitive stressors, such as the Stroop task, rather than to other types of stressors. This is unlikely, however, given that we used

a cognitive stressor in our stress manipulation (namely the mental arithmetic task) and we did not see an association with respect to this manipulation. In addition, previous research by Compton et al. (in press) demonstrated a relationship between cognitive control ability and subjective levels of anxiety in response to many different types of stressors, such as health problems and not having enough time to meet obligations.

Another possibility is that there was some human error in our cortisol assay, but this is highly unlikely since the correlation between the duplicate readings of salivary cortisol levels for each participant were very high. In addition, the correlation between the three different cortisol measurements at time 1, time 2, and time 3 further validates our assay since participants who had higher cortisol levels at time 1 also had higher cortisol levels at time 2 and time 3. This would be expected given stable biological differences between individuals in cortisol production and HPA axis functioning.

A more plausible explanation of the current results is that while both the Stroop task and the stress manipulation did not produce overall group increases in cortisol levels, the Stroop task did produce individual variation in cortisol reactivity that could be explained by individual differences in anxiety and emotion regulation. However, this was not true for the stress manipulation because this manipulation was not effective in producing increases in anxiety. Therefore, any individual variation that was present in cortisol levels during the stress manipulation was most likely due to random variation that cannot be explained by anxiety levels and individual differences in functioning of the ACC and the LPFC. If fluctuations in cortisol levels were not tied to increases in anxiety, then we would not expect to see any relationship between cognitive control and cortisol levels since this physiological measure would not be measuring emotion regulation but

merely random individual differences in cortisol production. This would explain why individual changes in cortisol levels in response to the Stroop task were correlated with cognitive control ability while cortisol levels in response to the stress manipulation were not.

Our finding that cortisol levels did not significantly change in the group as a whole throughout the study leads us to question the validity of our stress manipulation since previous researchers who have used the same stress manipulations have found large increases in cortisol across participants (Sgoutas-Emch et al., 1994; Bollini et al., 2004). Since the college at which this study was conducted is small and intimate, however, perhaps participants did not feel anxious during our manipulation since the experimenters were not complete strangers. Other studies that have used these same manipulations were conducted at large universities. The setting of the study itself, therefore, may have affected the level of stress the manipulation produced. If participants did not feel very anxious during the stress manipulation, then this would not have led to a significant increase in cortisol levels in the group as a whole during the stress manipulation or to individual differences in cortisol reactivity that were related to emotion regulation ability.

Even though participants did generally report a high subjective level of stress during the manipulation, this response may not have reflected their actual levels of anxiety and hence cortisol production. Different individuals may have interpreted the word “stress” differently and may have misinterpreted the question and replied in a more general manner instead of thinking about their particular emotions during the stress manipulation. If this were true then the subjective measure of stress may have been tapping a more stable personality characteristic instead of a state of mind during the

testing session. Participants could have reported high levels of general anxiety even if they were not very stressed during the manipulation. Therefore this measure would not be expected to mirror physiological changes in response to a laboratory stressor. This could explain why there was a discrepancy between high subjective reports of stress among participants and no significant increase in cortisol levels in the group during the stress manipulation.

Relationship between cognitive control and heart rate

We did not find any significant relationships between our measures of heart rate and levels of cognitive control. The group as a whole even significantly decreased in the measure of heart rate from time 1 to time 2. There were many problems with the heart rate measure in this study that made it an invalid measure of emotion regulation. First, many participants came to the lab after previously working out or running up the four flights of stairs to where our lab is located. This would explain the overall significant elevation in heart rate at time 1. In addition, the wrist-band device we used to measure heart rate did not always work (there were several subjects for which we could not gather heart rate measurements). While the correlations between the time 1, time 2, and time 3 measurements were high reflecting stable individual differences in heart rate, this measure was not related to our other measures of cognitive or emotion regulation and did not contribute to our investigation of cognitive control and emotion regulation.

Relationship between cognitive control and self-report measures

Neither measure of cognitive control (the ERN amplitude or behavioral data) was significantly correlated with any of the CISS subscale scores. This finding does not fit with our hypothesis. It also does not fit with previous research or correlations performed

in our analysis, which both demonstrate that the coping styles measured by the CISS are related to anxiety levels. Previous studies have shown that individuals who engage in problem focused coping strategies are less distressed, anxious, and depressed (Cosway et al., 2000). In addition, individuals who scored higher on the emotion focused coping subscale of the CISS in the present study also reported higher subjective levels of stress and anxiety according to our stress question and the STAI. This further supports the use of the CISS as a measure of stress regulation and would suggest that individuals scoring higher on the problem focused coping subscale are better at emotion regulation and would subsequently show higher levels of cognitive control. However, this measure did not seem to tap the relationship between emotion regulation and cognitive control in the same way that our measurements of cortisol levels did.

Given that previous research has shown that self-report measures of anxiety do reflect emotion regulation and cognitive control ability (Compton et al., in press), this finding suggests that the CISS does not measure emotion regulation ability in the same way that other self-report measures of anxiety do. While the general use of problem focused coping may be negatively related to distress and anxiety, perhaps other variables, such as personality differences, are mediating this relationship instead of emotion regulation ability. Research has shown that individuals scoring high in emotion focused coping also score high on scales measuring the personality factor of Neuroticism (Cosway et al., 2000). Therefore, individuals who engage in emotion focused coping strategies may also have personalities that pre-dispose them to higher levels of distress and anxiety, independent of their emotion regulation abilities.

In addition, since the CISS measures the use of certain coping strategies in daily life and is not specific to certain situations, this measure may not be useful in determining individual differences in emotion regulation. Even though problem focused coping seems to be the most effective general strategy in reducing anxiety, perhaps one's ability to down-regulate anxiety is specific to the situation and requires different coping skills at different times. In previous studies, self-reported anxiety and emotion regulation ability were measured during specific situations in response to the use of specific emotion regulation strategies (e.g., Kalisch et al., 2004). Therefore, while the CISS might measure different general coping styles and their relation to overall anxiety and distress, this measure may be too broad and might not tap into subtle individual differences in emotion regulation ability and ACC/LPFC functioning. This would mean that the general use of problem focused coping strategies is not necessarily indicative of more superior emotion regulation and cognitive control ability.

Self-report measures also have their flaws in that they are subjective and are subject to many biases. For example, participants in the current study may not have accurately answered the questions in the CISS because they wanted to appear more competent or because certain responses seemed like the "right" answer. Participants may also not have been aware of which strategies they use more often and their responses may have not been an accurate measure of how they cope with stress.

Directions for future research

The results of this study suggest important directions for future research. First, these results need to be replicated. This would allow further exploration of the relationship between cognitive control and emotion regulation on a physiological level. In

addition, since our stress manipulation was not effective (perhaps due to the fact that it was run at a small college), this study should be repeated at a large university to see if there are individual differences in cortisol reactivity due to a laboratory stress manipulation that are in fact related to levels of cognitive control. If our initial findings do hold for future studies, then this will further add to the current knowledge of the overlapping neural networks responsible for both cognitive control and emotion regulation (ACC and the LPFC) and how they function.

In addition to merely repeating the current study, future research could also examine how cognitive control relates to different measures of emotion regulation. These could involve other physiological measures, such as heart rate or blood pressure, that would help to develop a more nuanced view of the how ACC and LPFC activity contributes to physiological changes in the body. Since stress and anxiety are associated with many negative psychological as well as health consequences, understanding these mechanisms are very important. Therefore this study can help to direct and motivate future research in this area.

This study also demonstrates the need for developing a better understanding of how to measure emotion regulation ability. According to Gross (2002), emotion regulation is the process through which we regulate which emotions we experience, when we experience them, and how we experience them. However, the understanding of how we achieve this form of self-regulation is not yet well understood. While many different types of emotion regulatory strategies have been proposed (such as attentional control, distraction, problem focused coping, and cognitive change), research has not converged onto which of these many regulation strategies is the most effective and under what

circumstances it is effective. Based on the current study a physiological measure of anxiety (i.e., cortisol levels) seems to be a good indicator of emotion regulation while the same does not hold true for the CISS. Therefore future studies could aim to define what “effective emotion regulation” is and how to best measure it. While the current results as well as those of Compton et al. (in press) support the idea of a link between cognitive control and emotion regulation, a more thorough understanding of emotion regulation would help to further explore this connection.

Conclusion

This study does replicate previous findings demonstrating an association between emotion regulation and cognitive control ability. We found a significant relationship between high levels of cognitive control and low levels of cortisol reactivity, but only in response to a Stroop task. We did not find this relationship to be present in response to our stress manipulation. In addition, we did not find any correlation between our self-report measure of emotion regulation (the CISS) and cognitive control. Methods of self-regulation and the flexibility to control one’s behaviors are very adaptive and important to human functioning. Without this ability, humans would not be able to learn from their mistakes or react to errors. Therefore, this relationship between cognitive control and emotion regulation needs to be further researched in order to continue to elucidate the connections between ACC and LPFC functioning and these two forms of self-regulation.

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Figure 1.

Grand-average waveform at the FCz site for error versus correct trials. Time 0 represents the button press during the Stroop task.

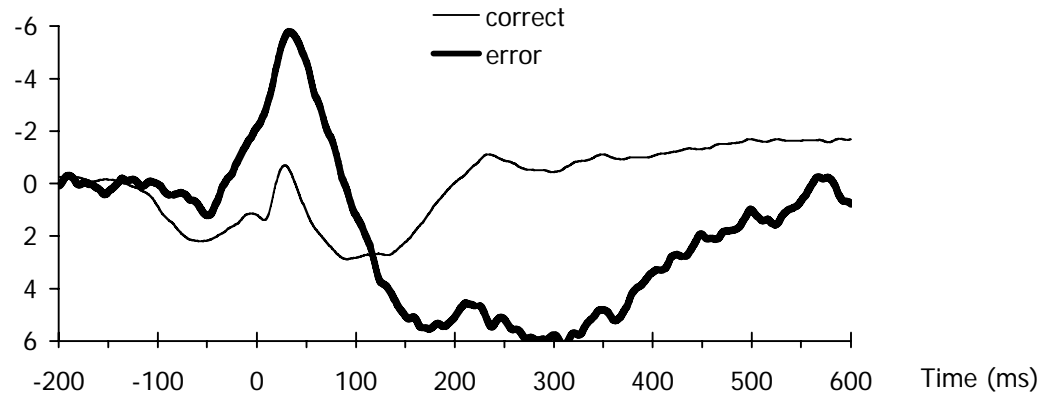


Table 1.

Table showing the mean amplitudes and standard errors of the ERP data after error versus correct trials. The mean amplitudes after error trials are more negative, demonstrating the presence of an ERN in the group as a whole. This table shows a site by accuracy interaction in that there was better differentiation of errors versus accuracy at the FCz site.

Mean amplitudes (in uv) and standard errors of ERPs showing a site X accuracy interaction

Site	Error	SE	Correct	SE
FCz	-7.27	.87	-1.20	.63
Fz	-6.17	.90	-1.32	.54
Cz	-6.50	.81	-1.15	.57
Pz	-6.85	.78	-3.09	.66

Appendix

The self-report questionnaire used in this study is available at:

http://www.haverford.edu/psych/rcompton/JASthesis_quest.htm

Screening questionnaire for potential participants:

The purpose of this questionnaire is to identify eligible participants for research studies that will be conducted in coming weeks and months in Professor Rebecca Compton's lab in the Psychology Department at Haverford. If you complete the questionnaire below, which should take only about 5 minutes, we'll enter your name into a lottery drawing for a \$50 cash prize. As long as you complete this questionnaire, your name will be entered into the lottery, even if your responses on this questionnaire make you ineligible for participating in any studies later on.

If your responses indicate that you are eligible for one of our ongoing studies, we will contact you within a few weeks to tell you more about what the study involves, and to see if you might be interested in participating. If you complete this questionnaire, it does not mean that you are committing yourself to doing anything else. If we contact you in the future about opportunities to participate in a study, you can decide at that time if you want to do it. Your responses to the questionnaire are confidential; only Prof. Compton will have access to them.

If you have any questions about this questionnaire, please contact Prof. Compton at 610-896-1309 or rcompton@haverford.edu. If you have questions or concerns about your rights as a research participant, you may also contact Prof. Rob Scarrow (610-896-1218, rscarrow@haverford.edu). Prof. Scarrow is chair of the Haverford College Institutional Review Board, which oversees the protection of research participants.

- 1) Male
Female
- 2) What is your graduation year?
2008 2009 2010 2011 Other
- 3) Which phrase best describes your handedness when writing or drawing?
 Strongly left-handed
 Weakly left-handed
 Strongly right-handed
 Weakly right-handed
 Ambidextrous (using both hands equally)

4) Please indicate whether any of the following statements apply to you. We have grouped these statements together to protect your privacy. If you check “yes” at the bottom of the list, no one will be able to tell the statement(s) to which you are responding.

- I have abnormal vision that is not corrected by glasses or contact lenses (e.g., color blindness, glaucoma, detached retina, etc.)
- I have a history of neurological problems, such as epilepsy (seizures), head injury, stroke, brain tumor, multiple sclerosis, etc.
- I regularly take medication that is known to affect the central nervous system. (Such medications could include anti-depressants, anti-anxiety medications, anti-psychotic drugs, drugs for epilepsy or other neurological disorders, etc.)
- I regularly consume non-medical substances that are known to affect the central nervous system (e.g., performance-enhancing drugs or substances such as marijuana, cocaine, heroin, ecstasy, etc.; do not include occasional use of alcohol, caffeine, or cigarettes).

Yes, at least one of the above statements describes me.

No, none of the above statements describes me.

I am not sure whether any of the statements above describes me.

In the box below, you may explain your answer to the above question if you wish, but it is not necessary to do so. _____

(5) Have you ever participated in a study at Haverford that involved recording your EEG (brainwaves)?

Yes No Not Sure

If your answer is yes (or not sure), do you remember when you participated and what the study involved? Your answer to this question does not necessarily affect your eligibility to participate in studies this year, because we usually have several different studies ongoing. However, your answer could help us to avoid running a given person in the same study by mistake. _____

(6) Would you be interested in receiving e-mails about opportunities to participate in our lab studies in the future? Our studies are run throughout the year. They typically involve cash payment for hour-long testing sessions in which you get to see your own brainwaves! Your answer to the question below does not affect whether you will be entered into the lottery for filling out this questionnaire (you will be entered into the lottery either way).

___ Yes, I'd like to hear about opportunities to participate in paid studies throughout the year.

___ No, please do not contact me about opportunities to participate in studies.

Please submit your name and e-mail address below. We will use this contact information to get in touch with you in the future if you are eligible to participate in studies in our lab.

Name

Haverford e-mail address

PLEASE NOTE: Your responses will not be submitted to our database until you click on the "SUBMIT" button below. By clicking on the SUBMIT button, you are granting your consent for your responses to be included in our database.

Recruitment e-mail:

Hi!

Want to earn \$30 for 1 hour of participation in a psychology study? Your response on the screening questionnaire indicated that you are eligible for our study.

The study is part of my senior thesis project, which I am conducting in Professor Compton's lab. The purpose of the study is to examine individual differences in how the brain responds to certain kinds of visual images. The study involves measuring "brainwaves", or electrical activity generated by the brain. We are also interested in how certain brainwave patterns are related to individual differences in responsiveness to challenging situations.

If you participate in the study, you will have a stretchy fabric cap placed over your head. Embedded within the cap are numerous sensors that detect the electrical signals generated by your brain. Wires from the cap are connected to a recording device, which then feeds into a computer. This allows us to obtain a read-out of your brain's electrical activity (your EEG, or electroencephalogram). During the study, your EEG would be recorded while you view a series of visual images and press buttons in response.

"Brainwave" recording is non-invasive, meaning that it simply records what your brain is doing, and does not affect your brain. You should be aware that in order to enhance the signals recorded from the cap, we will need to apply some gel between the cap sensors and your head. This gel washes out easily with shampoo. (But when scheduling an appointment, remember that you will probably want to shower right after the experiment!) Also, sensors will be placed around your eyes so that we can monitor your eye movements. Neither the stretchy elastic cap nor the eye sensors are painful! I have

worn the cap many times, and it doesn't hurt! It is just a little weird. It's actually really neat to watch your own brainwaves!

In addition to the brainwave part of the experiment, we are interested in measuring how people respond differently to challenging situations. As part of the study, you would be asked to make up and deliver a short speech (3 min) in front of a video camera, and you would also be asked to do a short but challenging arithmetic task in your head. You don't need to do anything in advance to prepare for this; complete instructions will be given during the study itself. While some people find these challenging tasks to be a little bit stressful, other people find them to be fun!

The entire study takes about an hour, and you would receive the \$30 payment as soon as the experiment ends.

We are running subjects in Professor Compton's lab (Sharpless 506) during this semester, beginning immediately and continuing for the next few weeks. When can you participate? Please e-mail us any times you are available for one hour during the next week or two to participate, including morning, afternoon, evening, & weekends. We'll be sure to find a time that suits your schedule.

Quick recap:

1. Painless experiment where you get to see your brainwaves!
2. There will be some gel in your hair after the experiment, so you probably want to schedule an appointment where you can shower afterwards!
3. Best part: earn \$30 cash. On the spot. For 1 hour! Wow!

Let us know when you're available, and hope to see you soon!

Please send responses and/or questions to [student's name] at Compton.Lab@gmail.com