Monitoring the Mind:
The Relationship Between Individual Differences in Cognitive Control and Emotion Regulation

Alexandra Amen
Haverford College
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In collaboration with Jessica Dodd and Stephanie Fineman
Advisor: Rebecca Compton

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Abstract

Cognitive control and emotion regulation are two separate mental processes that are each crucial in terms of internal goal-fulfillment. Past research has mapped these skills to overlapping areas of the brain, suggesting that individuals may likely exhibit system functionalities that are positively correlated. In order to address such a possibility, the current study quantitatively measured individual participants' cognitive control abilities using EEG technology. It also assessed their ability to regulate emotion, using increases in cortisol and heart rate during a laboratory-induced stress manipulation and self-report questionnaires. Results showed no correlation between anxiety induced during the stress manipulation and measures of cognitive control. However, cortisol increase during the EEG task was negatively correlated with measures of cognitive control, indicating that participants who were less able to regulate stress in the EEG portion of the experiment were also less able to exert cognitive control during this task. Such findings may be taken as support for the idea that individual abilities to regulate cognition and emotion are related.

Background

Overview

The ability of humans to control our actions, thoughts, and emotions is a part of what has made us a successful and unique species. In order to promote personal well-being it is necessary to recognize when intentions and goals are being thwarted, and then to compensate accordingly. The fulfillment of goals and needs is generally considered to be one of the main motivators of conscious and unconscious behavior in psychology (Terborg & Miller, 1978). If we could not control behavior and emotion to the extent that they are in line with desired outcomes, we would often find ourselves in situations that would not lead to internal goal-fulfillment. Therefore, it is clearly adaptive to be able to monitor when things are not in a preferred state, and to then be able to change our responses in order to fix this.

It seems reasonable and likely that the brain exerts control over cognition and emotion through similar mechanisms. Cognitive control and emotion regulation both exist to accomplish the same general task – as described, the monitoring of mental processes so that they are in line with goal directed states. Though there are of course differences in how the brain deals with cognitive versus emotional events, there is some support for the idea that the machinery that detects an overall deviance from internal goal-state and sends out signals to initiate change is the same. Recent research has shown that suppressing emotion leads to a decrease in ability to exert cognitive control (Inzlicht & Gutsell, 2007). This finding can be interpreted as preliminary evidence for an overlapping mechanism between cognitive control and emotion regulation since when areas involved in emotion regulation are occupied with a task, cognitive control is

harmed. In the currently published literature the idea that a link could exist between an individual's cognitive control and ability to regulate emotion has never been tested, but based on these ideas, we hypothesize that the abilities of individuals to control behaviors and emotions should be related. Specifically, individuals who are better at exerting cognitive control should also be better at regulating their emotions.

Cognitive Control

Cognitive control is an overarching term for the process by which relevant information is selected, information processing is organized, and optimal (goal-directed) behavior is produced – thus it serves to make sure that thought and behavior are consistent with internal goals and plans. As defined by Ridderinkhof et al (2004), cognitive control is "adaptive goal-directed behavior [that] involves monitoring of ongoing actions and performance outcomes, and subsequent adjustments of behavior and learning" (pp. 443). Thoughts and behaviors must be constantly judged to make sure that they are facilitating goal achievement. This highly adaptive, flexible system is generally activated in order to inhibit responses that would otherwise be produced by environmental stimuli (Holroyd & Coles, 2002). For example, when on a diet, a person might decide not to eat a steak (a generally tempting food). The system is crucial to processes such as attention and learning - when situational factors cause the cognitive control system to be activated, the brain then 'learns' what response should be made the next time the situation arises, and what information should be attended (Ridderinkhof et al, 2004).

Cognitive control is often broken down into two distinct aspects. The first is the monitoring of conflict between actual and optimal states. The second is the meeting of the requirements of a current task, and making sure that information is processed in such a way as to elicit a desired response. In general, the first aspect involves recognizing a need for control, while the second aspect works to implement the necessary increase in control (MacDonald et al, 2000). It is widely accepted that the posterior medial frontal cortex, most especially the anterior cingulate cortex (ACC), is involved in the conflict-monitoring aspect. The ACC is thought to be involved in processes such as attentional control, controlling and planning of motor function, and detecting errors (Ridderinkhof et al, 2004). The fact that it is involved in error detection makes the ACC a clear candidate for the role of conflict-monitoring in cognitive control. The ACC may be generally divisible into a dorsal (dACC) region involved in cognition, and a rostral (rACC) region involved in emotion. It is the dACC, then, that is thought to be activated during cognitive control (Bush et al, 2000). This is, however, not clear-cut – there are some areas of overlap between cognition and emotion in the ACC (Mohanty et al, 2007).

After the ACC detects conflict or error, the prefrontal cortex (PFC) seems to be responsible for the exertion of control and production of a "correct" response. This too makes sense given that the PFC is generally implicated in planning, decision-making, and controlling behavior (van Veen & Carter, 2006). The most widely accepted theoretical mechanism for the system of cognitive control proposes that the ACC becomes active when conflicting information enters the brain. When the ACC detects conflict it activates the PFC, which then serves to resolve the conflict and keep behavior in line with what is ultimately desired as determined by internal goals (Kerns, 2006). Based on a review of

neurophysiological and neuroimaging studies of cognitive control, Miller and Cohen (2001) suggest that the PFC contains multiple specific patterns of activity that each represent an internal goal. When the PFC receives information that a goal is being thwarted, it activates the particular pattern of activity representing that goal, which sends signals to other brain structures. These structures then work to make sure that behavioral outputs are consistent with internal desires.

The role presented here for the ACC is supported by the fact that fMRI imaging shows an elevation in ACC activity following error response trials, and also during trials in which the response was correct but the information presented was incongruent (Kerns et al, 2004; MacDonald et al 2000). Specifically, in a Stroop color-naming task, participants who responded incorrectly (which would result in conflict in information processing, as the internal goal is to respond correctly) showed increased ACC activation. In addition, simply viewing the word "red" written in green (as seen in 'incongruent' trials) resulted in increased ACC activation, presumably because this incongruence caused conflict in the information processing system. In addition, neurophysiological research has indicated that the anterior cingulate contains areas of error-recognition. Niki & Wantabe (1979) used single-cell recording in rhesus monkeys to show an increase in activity of these areas following error response. The areas were also activated when the monkeys were denied an expected reward, and thus were experiencing a kind of conflict, or error, condition.

Neuroimaging data also supports the idea of a role for the PFC in cognitive control. As can be predicted from the proposed cognitive control model, conflict (as understood by the ACC) should signal a need for control, causing the PFC to select for

relevant as opposed to irrelevant information (for example, the color of the word as opposed to the word itself in a Stroop task), leading to desired behavior. In the case of the Stroop task, attending to the color of the word as opposed to the word itself requires greater attentional control, and therefore should lead to greater PFC activation. MacDonald et al (2000) showed that if subjects were told they would later be performing a Stroop color-naming task (and were therefore motivated to correctly name word color), during task preparation the left dorsolateral prefrontal cortex was more active when subjects named the color of the words than when they simply read the word. It also appears that activity in the PFC is increased in trials following error and incongruence conditions. In fact, the activation of the ACC on conflict trials seems to positively predict the activation of the PFC on following trials (Kern et al, 2004; van Veen & Carter, 2002).

Measuring Cognitive Control

Cognitive control can be measured in the laboratory through several different methods. As has been discussed, neuroimaging with fMRI technology is often used to show activation of brain regions in response to cognitive control conditions. However, another common method that allows for the study of cognitive control involves using electroencephalography (EEG) technology to observe the error-related negativity (ERN) waveform.

EEG methodology was first discovered in 1929 when it was found to be possible to measure electrical activity in the human brain by placing electrodes on the scalp (Berger, 1929). When brain areas become active action potentials are occurring in the

neurons of that area, and this produces a change in voltage. The scalp electrodes measure voltage in specific brain areas as compared to a baseline electrode. EEG can therefore be used to look at voltage changes during different human cognitive processes in different areas of the brain. Generally such processes are observed through events known as ERPs (event-related potentials), which are the patterns of brain activity seen as a result of both exposure to specific stimuli and response events. ERPs can include anything from the decrease in voltage observed in specific areas as a subject prepares to make a response to the increase in voltage seen in other areas when subjects look at faces as compared to non-face objects (Stern, 2007). The ERN is one specific type of ERP.

The ERN is defined as a sudden drop of about 10μV that occurs after the presentation of a stimulus that results in an incorrect response, or error (Gehring et al, 1990). This negative peak can be seen at about 80ms after response intention is decided. The areas through which electrodes tend to record an ERN response are located directly over the ACC, linking the ERN to activation of the ACC (Ridderinkhof et al, 2004). In fact, Mathalon et al (2003) demonstrated that during Go/NoGo tasks, incorrect responses were associated with simultaneous EEG-recorded ERN waveforms and fMRI recorded activation of the ACC. Based on evidence such as this, the ACC is believed to produce the ERN, providing even more support for the model of the ACC as a detector of conflict.

The amplitude of the ERN is larger when subjects are striving for accuracy (Gehring et al, 1990), and therefore fits with the idea that it is related to the brain's detection of conflict and re-establishment of goal-states. It also appears that as ERN amplitude increases after error in conditions in which accuracy is emphasized, reaction time to subsequent trials increases (Gehring et al, 1993). This is the sort of behavioral

modification that would be expected in cases of good cognitive control – after an incorrect response, the brain is motivated to make sure that the following tasks are completed correctly, and therefore responds more slowly and carefully. However, while a reaction time behavioral modification is often observed, other research has implied that the slowing of response is not always associated with error and ERN amplitude, and might simply be occurring as a result of momentary confusion or distress following error response (Hajcak et al, 2003b). Another more consistently observed behavioral modification in individuals with large ERN amplitudes is the increase in probability of a correct response following error – that is, after a subject responds incorrectly, cognitive control serves to make sure that response will be correct the next time. However, in terms of behavioral response data in general, it has been proposed that this is less a reflection of cognitive control ability and more a reflection of adaptive motor abilities (Ullsperger & Cramon, 2006). Therefore, although behavioral measures of error-related adjustment can be useful, the most reliable data that is obtained from ERN studies seems to be the amplitude of the ERN peak.

Recent research has shown that the ERN may occur as a "response ERN" in conditions where subjects are immediately aware that a mistake has been made, or as a "feedback ERN" in conditions where subjects are not made aware that they made a mistake until a later point in time. In the "feedback ERN" condition, once subjects are alerted to the error, the ERN peak occurs (Ridderinkhof et al, 2004). This shows that the ERN is not time-specific; it is simply a result of the processing of error occurrence (conflict, as processed by the ACC). In order for the ERN to occur, and thus for the ACC to have processed this conflict, it makes sense that it would be necessary for error to be

recognized by the brain. The ERN also does not appear to be dependent on the input or output modality of error trials (Miltner, Braun, & Coles, 1997), indicating that the mechanism is highly flexible, as would be expected of a cognitive control system.

There are different proposals as to how exactly the ACC creates an ERN event. One such proposal relates the mesencephalic dopamine system (MDS) with the generation of the ERN by the ACC. The mesencephalic dopamine system is a group of dopamine neurons that are important in reward, conditioning, and motivation (Montague et al, 1996). These neurons synapse in different areas of the frontal cortex, including the ACC. The theory suggests that the MDS is capable of transmitting information on how a task is going through the release of dopamine – specifically, it alerts the brain if outcomes are either better or worse than expected. More dopamine (reward) is released in the pathway when things are going well, and there is a decrease in dopamine when things are going poorly (Montague et al, 1996). In this way, the MDS appears to function as part of the conflict-monitoring aspect of cognitive control. Holroyd and Coles (2002) suggest that the ACC is given input from multiple different motor controllers, which each are linked to a specific behavioral response. However, especially in new situations, the ACC does not necessarily know which motor controller is best for the job. In conditions where outcomes are worse than expected (i.e. errors are made), this causes a decrease in activation of the MDS. Less dopamine transferred to the ACC then leads to a disinhibition of the ACC neurons (activation of the ACC), later causing the drop in voltage observed in the ERN waveform. The ACC uses such signals to select for the correct motor controller for a particular task. This theory suggests that the ACC immediately recognizes error (via the MDS) and initiates a correct response.

Such a proposal seems contrary to previously discussed roles for the ACC and PFC, in that earlier the ACC was discussed as the recognizer of conflict, and the PFC was given credit for the production of a correct response. Holroyd and Coles, however, suggest that the ACC is responsible for producing the correct response based on the information it receives from the MDS. In both cases the ACC is seen as processing conflict, but in the first theory it is the PFC that initiates behavioral change, while in the second the ACC can initiate change on its own. These two theories may be reconciled if the meaning of "behavioral change" is considered to differ between the two theories. Possibly Holroyd and Coles' theory explains automatic motor responses that often occur following error - for example, when subjects press the wrong key in a computer task and immediately correct themselves by pressing the right one. This type of automatic behavioral response to error may not require the type of attentional control exerted by the PFC, but might instead be a result of the ACC activating the correct motor controller. The ACC could simultaneously be alerting the PFC, which would then exert control and make sure of a correct response in the next trial as is consistent with the originally discussed theory.

In summary, using EEG technology to look at ERN peaks is a useful way to study the ability of an individual's brain to exert cognitive control. The ERN is probably a result of the disinhibition of neurons in the ACC in response to detection of the fact that behavior is not consistent with internal goals. The greater the amplitude of the ERN peak the greater the disinhibition of the neurons, and therefore the more effective the ACC will be at communicating to connecting brain areas, including motor areas and the PFC. If the ACC is more effective in alerting the PFC, the PFC will be able to exert a higher level of

control. Therefore, it seems that measuring the amplitude of ERN peaks in laboratory subjects (as well as some of the behavioral changes correlated with ERN amplitude) should give a valid idea of how effective their individual cognitive control system is.

Emotion Regulation

In the field of psychology, emotions are viewed are highly adaptive experiences which lead to tendencies to act in ways that facilitate survival and reproduction. Emotions provide us with information about the current relationship between ourselves and the environment, and then serve as motivation to make any necessary changes. They arise when a person attends to an environmental situation and evaluates it in terms of consistency with internal goals (Frijda, 1986). For example, fear will cause people to remove themselves from a situation – and a situation which elicits fear is probably one from which it is adaptive to be removed (consider getting off the street when you see a car headed towards you). At the same time, it can also be adaptive to regulate emotions. Sometimes an impulsive emotional response is actually not consistent with optimal survival and reproduction. When angry with a romantic partner, you may want to yell, but will restrain yourself from expressing your emotions in this way because it is not in the best interest of the relationship. Emotion regulation refers to the process by which the adaptivity of an emotion is compared to the environmental situation, which then influences how one experiences and expresses that emotion – "An ongoing process of the individual's emotion patterns in relation to moment-by-moment contextual demands" (Cole & Michel, 1994, pp.74) or "The process by which we influence which emotions we

have, when we have them, and how we experience and express them" (Gross, 2002, pp. 282).

Emotion regulation is clearly an important part of everyday life. Clinical psychiatric cases often involve people who have trouble regulating emotion (Cole & Michel, 1994). The ability of individuals to regulate their own emotions affects how they are perceived by others, and is therefore a specific facet of personality. People who are assessed as having more adaptive emotion regulation strategies based on self-report measures score higher on questionnaires designed to assess general well-being (Mauss, 2007). Just as cognitive control involves acting in accordance with internal goals, the ability to experience emotion in accordance with internal goals should help lead to goal-fulfillment, thus leading to increased well-being. For example, if you were to experience anxiety while walking down a darkened street at night this emotion would cause you to pay more attention, which is adaptive in terms of keeping yourself safe (an obvious internal goal). However, experiencing high levels of anxiety before an exam might cause you to perform poorly, and therefore in this situation, it would be adaptive to exert more emotion regulation and experience less anxiety.

Emotion regulation can be conscious (as in deciding not to yell at your romantic partner) or unconscious (as in expressing more pleasure than you feel when you receive an undesired gift). However, both conscious and unconscious regulation seem to involve the same basic mechanisms. Though unconscious emotion regulation is difficult to measure in the laboratory, individuals who exhibit avoidant attachment styles (i.e. avoid close relationships) are thought to inhibit emotional impulses, which would be the equivalent of unconscious repression of emotion. When asked to recall past

relationships, avoidant subjects show less negative emotion but greater physiological arousal than non-avoidant controls. This is the same trend seen in subjects asked to consciously repress emotional responses to negative images as compared to control subjects asked simply to look at the images (Mauss, 2007).

Differences in mechanisms of emotion regulation appear therefore to depend on the specific individual as opposed to the type of emotion regulation, and are thought to be predictive of certain aspects of personality. Emotion regulation strategies can be divided into two broad categories. The first is antecent-focused regulation, which involves a strategy that changes the response process before it has been fully activated. The second is response-focused regulation, which involves a strategy that is applied after the activation of the emotional response, while the emotion is already in existence. One example of an antecent-focused strategy is reappraisal, which involves re-evaluating a situation that would typically elicit an emotion so that it is non-emotional. An example of a response-focused strategy is suppression, which has to do with inhibiting the expression of current emotion (Gross, 2002).

A discussion by Gross (2002) of earlier studies looked at the benefit of using reappraisal versus suppression strategies, as seen by the difference in effect on the experience of emotion, emotion expression, and memory of the event. These studies generally showed an emotion-eliciting film clip and then asked participants to either think about the film in non-emotional terms (reappraisal) or to hide the emotion they were experiencing (suppression). Overall, reappraisal was associated with less emotion experience, less emotion expression, and accurate memory recall. Suppression, on the other hand, was associated with less emotion expression, but emotion experience was not

changed and memory recall was harmed. Additionally, it appears that people who use reappraisal strategies in general experience less negative and more positive emotion, while suppressors actually experience less positive and more negative emotion (Gross & John, 2003). Such evidence suggests that antecedent-focused emotion regulation strategies like reappraisal are more functionally adaptive than response-focused strategies like suppression.

Functional MRI studies of emotion regulation have been able to link this process to certain brain areas – in fact, the same brain areas implicated in cognitive control. Specifically, studies have mapped brain activity during reappraisal to the lateral prefrontal cortex, the medial prefrontal cortex, the medial orbitofrontal cortex (MOFC), and the amygdala (Ochsner & Gross, 2005). When subjects are told to use reappraisal strategies, levels of emotional distress in response to negative images, and thus the effectiveness of the individual's reappraisal strategy, can be measured by studying eyeblinks. More effective reappraisal strategies are correlated with increased lateral PFC (LPFC) activation, increased ACC activation, and decreased activation in the MOFC and amygdala (Ochsner et al, 2002). Ochsner et al suggest that emotion regulation involves three distinct processes. First, the LPFC serves to create a reappraisal strategy and to keep the strategy in mind throughout the stimulus presentation. Second, the ACC works to observe conflict between the neutralization of emotion through reappraisal and emotion that may still being produced, thus alerting the LPFC to the need for continued reappraisal. Here again, as in cognitive control, the ACC is involved in conflict-monitoring, presumably through the 'emotion' areas of this brain structure (the rACC,

specifically). Third, the medial PFC works to keep track of the new relationship between internal state and environmental stimuli, monitoring any need for change.

These three control processes together will then affect the activation of emotion-processing centers, specifically the amygdala and the MOFC. Patients with lesions to the brain affecting frontal cortex connections to the amygdala are able to process and understand emotional events, but have no affective response to them at all (Rolls, 1992). Amygdaloid connections, therefore, are crucial to the actual experience of emotion. The MOFC also plays a crucial role in our understanding of emotion. It appears that while the amygdala responds to the intensity of an emotional experience, the MOFC responds to the level of unpleasantness. A multitude of neurons connect these two structures, which then allow for the entire emotional experience to be had (Anderson et al, 2003). Both structures may be involved in emotion regulation because both intensity and unpleasantness lead to the need for system activation – that is, typically, highly negative emotions are the ones most in need of regulation.

Ochsner et al (2002) propose that the dorsolateral PFC (DLPFC) could use its anatomical connections to the orbitofrontal cortex to influence both MOFC and amygdala activity. It has also been shown that neurons in the ACC synapse with neurons in the amygdala (Cassel & Wright, 1986), and that inhibition of amygdala activity is disrupted by lesions to the medial prefrontal cortex (Morgan & LeDoux, 1995). This would support, then, a model where activation of an adaptive emotion regulation system, involving the PFC and the ACC, leads to less activation of the amygdala and the MOFC, and thus less emotion experience. Such a model is intriguingly similar to that discussed

for the implementation of cognitive control, where activation of the ACC and PFC causes an adaptive change in behavior.

Regulating Emotional Responses to Stress

Stress is a shared experience of all living organisms. A stressor (the stimulus causing the stress experience) may be situational, biological, or psychological. The dictionary definition of stress describes it as a "mental or bodily tension that results from factors that tend to alter the existing equilibrium" (Merriam-Webster Medical Dictionary, 2007). As this definition implies, it is the recognition of a discrepancy between the way things are and the way the organism would like them to be. A stress response leads to greater focus and attention on the stressor, which is adaptive in low levels. However, high levels of stress and chronic stress are not adaptive – they are significantly associated with decreased well-being (Sjogren, 2006). Stress regulation, therefore, can be seen as a necessary subtype of emotion regulation. Like emotion regulation, stress regulation is a predictor for certain facets of personality. For example, stereotypically "inhibited" children show greater stress responses to novel stimuli than do stereotypically "bold" children (Kagan, Reznick, & Snidman, 1987). Here, we will be thinking of stressors as generally leading to anxiety, which is subdued through previously discussed emotion regulation mechanisms.

Past research has provided a convincing argument for stress regulation as a subtype of emotion regulation. As stated, stressors lead to anxiety, which is generally a negative emotion and is therefore likely to be one that is highly regulated. The amygdala shows a significant decrease in activity when individuals are told to regulate responses to

threat-related stimuli through reappraisal (Phan, 2005). Such reappraisal also, as expected, leads to increased DLPFC and ACC activation. ACC activation and amygdala deactivation are inversely correlated with negative emotion, defined as fear-invoked anxiety (Eippert et al, 2007). It appears too that injury to any of these areas causes a change in the body's response to stressors. Lesioning the amygdala of rhesus monkeys led to the disappearance of anxiety-produced behavior when they were separated from cohabitants and introduced to human presence (Kalin & Shelton, 2003). The amygdala must be crucial for the production of anxiety since lesioning it appears to eliminate the emotion altogether. Other studies have looked at the effect of post-traumatic stress disorder (PTSD) on stress regulation. PTSD is a condition that arises when individuals are subjected to a horribly stressful event, which then impairs their ability to cope with stress in the future. Subjects with PTSD show significantly less activation of the ACC when presented with unpleasant or threatening stimuli than control subjects (Bremner et al, 2004). It appears that the loss of ability to normally regulate stress in PTSD is correlated with the impairment of the role of the ACC in the emotion regulation process.

Just as individuals use different strategies to regulate emotion, there are different strategies for coping with stress. The modern theories of stress-coping strategies come from Folkman & Lazarus (1988), who proposed eight different ways of coping with stress - confrontive coping, distancing, self-control, seeking social-support, accepting responsibility, escape-avoidance, planful problem-solving, and positive appraisal. These eight strategies can be broken down into two main categories, generally called problem-focused coping (i.e. positive appraisal), and emotion-focused coping (i.e. escape-avoidance, Cosway et al, 2005). Problem-focused coping involves changing the

relationship between the person and the environment, while emotion-focused coping involves changing an emotional response to the stressor/environment (Endler & Parker, 1994). Avoidance coping is one other main stress coping strategy that has been suggested in recent years. Avoidance coping involves (as the name suggests) avoiding the stressor, and can be broken down into person-oriented tasks (seeking out other people) or behavior-oriented tasks (distracting oneself by performing some other behavior, Endler & Parker, 1990).

As discussed, using the cognitive strategy of reappraisal to regulate emotion appears to be more adaptive than other strategies. In the same vein, some coping strategies seem to give better results than others. Problem-focused coping is theorized to be most adaptive in situations in which individuals have high levels of perceived control, and thus are able to change the person-environment relationship. Emotion-focused coping is theorized to be more adaptive in situations where individuals have low levels of perceived control (Avero et al, 2003). However, emotion-focused coping is only adaptive as a short-term coping strategy. When used for long-term coping, it has actually been shown to be correlated with an increase in anxiety (Endler & Parker, 1990). Avoidance coping is also, not surprisingly, correlated with anxiety and distress (Endler & Parker, 1994). When academic performance (measured by exam grades) was studied in relation to coping strategies, it was found that problem-focused coping was correlated with an increase in performance as compared to the other two strategies (Endler, Kantor, & Parker, 1994). Therefore, in terms of long-term success in coping with stressful situations, it is clearly adaptive to use problem-focused coping strategies.

If emotion regulation – and therefore stress regulation – involves an underlying mechanism similar to that of cognitive control, it would be logical to predict that the levels of functionality of these processes are correlated within an individual. The use of adaptive emotion regulation strategies and stress coping styles should be related to the use of adaptive cognitive control strategies. That is, it seems likely that individuals who are more successful at implementing cognitive control are also more likely to use problem-focused coping strategies.

Physiological Measures of Stress

In order to study the success of individuals' stress-coping styles, it is necessary to determine the "amount" of stress that they feel in response to different stimuli. Perception of stress is often evaluated through self-report forms, but given the subjective nature of self-report methods, different types of questionnaires have sometimes been shown to produce different results (Cohen et al, 1983). Because of this, researchers often turn to biological measures of stress. Stress-related anxiety leads to several well-understood physiological changes in the body, which can be observed and used as dependent measures of stress level.

Physiological arousal associated with the stress response includes such changes as an increase in heart rate and blood pressure, inhibition of the digestive system, release of nutrients used by muscles, and an increase in focus and attention. All of these changes allow the organism to respond to the stressor in an effective way. Biological mechanisms underlying the body's response to stress are fairly well understood. Stress response begins, as do most subjective experiences, in the frontal cortex, where information about

the environment is evaluated and processed. This is the step during which the PFC might initiate stress regulation – for example, during problem-focused coping, it is the step where control would be exerted and the perceived person/environment relationship would be changed, leading to an alteration of the stress experience. Information processed by the frontal cortex is then sent to the amygdala, as has been previously discussed. The emotional experience of stress is processed by the amygdala. Cognitive feedback mechanisms then allow the amygdala to send information about the processed emotion back to the frontal cortex areas (Amaral et al, 1992). In terms of stress regulation, this may be how the ACC is able to detect conflict between the desired neutralization of emotion via the PFC and the continued production of anxiety. As discussed, in conditions of conflict the ACC will alert the PFC, which will then work harder to reduce anxiety-provoking information that is sent to the amygdala (Ochsner et al, 2002).

The amygdala also sends information to brain regions that lead to behavioral and nervous system responses to emotion – here, anxiety. It connects via the pariaqueductal grey to the spinal cord ventral horn, which then initiates motor movement as determined by the emotion experience. It also projects via other brain structures to the vagus nerve, which is responsible in part for parasympathetic nervous system activation, and the intermediolateral column of the spinal cord, which is responsible in part for sympathetic nervous system activation (LeDoux, 1995). The parasympathetic division of the autonomic nervous system exists to help the body save energy when the environment allows for a relaxed state while the sympathetic division gives rise to the “fight or flight” response, associated with stress-induced physiological changes such as heart rate and blood pressure increase. Presumably, in response to anxiety or fear the amygdala would

cause the intermediolateral column to initiate a sympathetic nervous system response and the vagus nerve to suppress parasympathetic function (Hardy & Pollard, 2006). This means that anxiety can lead to activation of the sympathetic and suppression of the parasympathetic nervous systems via the amygdala, which explains in part the effects of stress on the nervous system. Sympathetic nervous system activity, therefore, can be used to evaluate the amount of anxiety provoked by a stressful stimulus. A subject experiencing high stress-related anxiety would have greater amygdala activations and therefore larger increases in heart rate and blood pressure than a subject experiencing less anxiety.

The body's stress response can also be examined in terms of hormone release. Amygdala activation initiates activity of the hypothalamic-pituitary-adrenocortical (HPA) axis though its connections with the paraventricular nucleus (PVN) of the hypothalamus. The neuroendocrine system is known to be dependant on the amygdala – for example, rats with damage to the amygdala do not show any increase in neuroendocrine activation in response to stress, as measured by corticosterone levels (Wolterink et al, 2001). Activation of the PVN causes the release of CRF (corticotropin releasing factor) and vasopressin. CRF molecules then move to the anterior pituitary, known to have a high concentration of CRF receptors. When these receptors bind to CRF molecules, CRF together with vasopressin presence causes the anterior pituitary to release ACTH (andrenocorticotropin) into the bloodstream. ACTH moves down to the adrenal cortex, which responds to ACTH presence by releasing cortisol - the hormone most often associated with stress in humans – into the bloodstream (Stansbury & Gunnar, 1994).

Thus, cortisol level in the blood is another measure that can be used to study individual stress response.

When cortisol is released, the majority of it binds to receptors - however, it is the unbound cortisol that is biologically active. Cortisol is always being produced in low levels by the HPA axis, as it is a necessary part of tissue regulation. This baseline cortisol is crucial for general health and function. The term 'glucocorticoid' refers to a general class of molecules containing cortisol (present in humans) and corticosterone (present in rodents). It has been shown that rats that are not able to produce corticosterone exhibit many motor deficits as well as a disruption in learning and memory processes (Russell & Wilhelmi, 1954). During the stress response, the major role of cortisol is actually to regulate other stress-related processes. That is, stress causes a change in other biological mechanisms such as immune response or heart rate monitoring that could lead to damage if not mediated by cortisol (Lovallo & Thomas, 2000). In sudden high quantities, as would be seen during stress response, cortisol also leads to heightened energy, concentration, and memory function, and lower sensitivity to pain (Stansbury & Gunnar, 1994). All of these are functions associated with the "fight or flight" response, and are adaptive in that they allow for the body and brain to attend to and take care of the stressor.

The adrenocortical system contains multiple internal mechanisms to shut itself off after stress-induced activation. When cortisol is present in the bloodstream it sends signals back to the anterior pituitary, the PVN, and the hippocampus, telling each of these structures to stop the stress response. The hippocampus works to inhibit PVN-induced release of CRF, the PVN inhibits the release of CRF on its own, and the anterior pituitary

downregulates its release of ACTH (Lovallo & Thomas, 2000). This system of internal regulation is necessary because while temporary activation of the neuroendocrine system may be adaptive in response to stress, long-term activation is not. The presence of chronically high levels of cortisol is equally as maladaptive as no cortisol at all. Cushing's disease is a condition characterized by chronic excess cortisol, and its sufferers show physiological deterioration of the bones, muscles, and some areas of the brain. They are also prone to mood swings and depression, and exhibit impaired learning and memory (Lovallo & Thomas, 2000). Clearly it is necessary to have some cortisol present in the body at all times, and clearly an increase in cortisol is adaptive in responding to stressful situations – however, if the body is unable to downregulate the stress response quickly, this leads to serious side-effects. Because glucocorticoid levels are directly increased in response to stressful stimuli and are downregulated quickly in the absence of stressors, these hormones are very useful in the study of stress.

Another class of hormones that have been studied in relation to the stress response are catecholamines. Specifically, dopamine and norepinephrine are released from the sympathetic nerves in response to acetylcholine during stress response, while ACTH causes norepinephrine and epinephrine to be released from the adrenal medulla. All of the catecholamines have individual effects, but both dopamine and norepinephrine can be converted to epinephrine (adrenaline) via enzymes present in the blood stream (Lovallo & Thomas, 2000). The exact functions of the catecholamines in the stress response are not fully understood, but it is clear that they act on the sympathetic nervous system to produce aspects of the "fight-or-flight" response. Epinephrine, for example, is necessary for the stress-induced increase in heart rate and blood pressure (Bao et al, 2007). Stress-

produced catecholamines also appear to act on areas of the brain such as the medial prefrontal cortex (where the ACC is located), and from there may cause HPA axis activation to continue (Lovallo & Thomas, 2000).

Based on what we know about the body's response to stress, physiological measures such as glucocorticoids, catecholamines, heart rate, and blood pressure can be used to subjectively study the "amount" of stress-provoked anxiety an organism is undergoing in response to a particular stressor. Heart rate, blood pressure, cortisol, and epinephrine have all been shown to significantly increase in humans in response to laboratory stressors (McCann et al, 1993). Equally important, self-reports of distress and lack of control in response to negative stimuli are correlated with increases in cortisol (Vedhara et al, 2003) and changes in the cardiovascular system (Sgoutas-Emch et al, 1994). However, stressful situations that create the need for increased attention and focus but are perceived as controllable and energizing as opposed to distressing are correlated with significant catecholamine increase and changes in the cardiovascular system (Lundberg & Frankenhaeuser, 1980). Therefore, clearly the best type of physiological measure to use in measuring stress is dependent on the specific type of stressor. However, catecholamines are only present in the blood and urine, while cortisol is able to move through the partoid gland into the saliva and thus can be measured using saliva samples, making it much more accessible.

If cortisol is to be used as a laboratory measure of response to stress, it is necessary to understand specifically what situations lead to significant increases in cortisol. Stressful situations that cause negative affect and are perceived to be uncontrollable elicit significant changes in cortisol (Vedhara et al, 2003). Indeed,

uncontrollability is thought to be a major factor in increasing cortisol response. When confronted with an uncontrollable stressor nothing can be done behaviorally to change the situation, which means that the stressor poses a highly significant goal threat. Sapolsky et al (1993) showed that primates show greater cortisol increases in response to uncontrollable versus controllable stressors.

In addition to uncontrollability, social-evaluative threat is another factor that can be used to increase cortisol response. Maintaining a positive social image is a nearly universal internal goal, and situations involving social evaluative threat create the potential for the self to be judged negatively by others. Internal traits such as competence and intelligence are thought to be the most widely susceptible to this effect (Dickerson & Kemeny, 2004). Overwhelming evidence shows that laboratory stressors such as public speaking tasks that pose a social-evaluative threat cause increases in physiological stress measures, specifically cortisol and heart rate/blood pressure (Al'Absi et al, 1997; Sgoutas-Emch et al, 1994).

Overall, when the brain perceives a situation to be stressful and causes the amygdala to produce anxiety, several physical changes take place. One type of change occurs in the autonomic nervous system, where an increase in factors such as heart rate and blood pressure can be observed. These responses are easily measured, and can be used as indicators of individuals' varying stress levels. Another type of change involves the activation of the neuroendocrine system, which leads to elevated release of glucocorticoids and catecholamines. Stress hormones, therefore, may also be used as a measure of stress level. Because it is present in the saliva, cortisol is the easiest of these to access. Negatively stressful stimuli that introduce uncontrollability and social-

evaluative threat lead to a substantial increase in cortisol. Therefore, a subject's ability to regulate negative emotion involved in stress may be studied in the laboratory by subjecting him or her to such a stimulus and observing changes in cortisol level, as well as autonomic nervous system activity. Individual differences in activation of the neuroendocrine system and autonomic nervous system would be indicative of individual differences in the experience of stress. They can therefore be used as one way to determine a subject's ability to cope with stress, and thus to regulate emotion in general. Again, if (as we expect) emotion regulation and cognitive control mechanisms are related, these physiological measures of emotion regulation should correlate with individual adaptivity of cognitive control systems.

Current Study

The present literature on cognitive control and emotion regulation gives strong reason to believe that these two mental processes are carried out using a similar underlying mechanism. From a general standpoint, the control of cognition and emotion exist to accomplish similar tasks – the recognition of internal or external events that are not consistent with an internal goal state, and the subsequent change of these events (Cole & Michel, 1994; Ridderinkhof et al, 2004). For cognitive control, this might mean recognizing that you have answered a question incorrectly, and changing your response strategies so that you are correct the next time. For emotion regulation, this might mean working to decrease anxiety before taking a difficult exam. Either way, these processes help you to keep on track and accomplish your goals – to do well on the exam, for example. From a biological standpoint, cognitive control and emotion regulation appear

at the highest level to use the same brain structures, specifically the PFC and the ACC. Though their roles differ slightly between cognitive and emotional control, the ACC is crucial for the detection of goal conflict, and the PFC is necessary in recognizing situational events and exerting control (Kerns, 2006; Ochsner et al, 2002).

Both cognitive control and emotion regulation have been well studied in isolation, but the current literature is clearly lacking in examination of how these processes might be associated with one another. Now that the physiological events behind both functions are more clearly understood, we should be able to hypothesize about their connections with and effects on other neural mechanisms. Because of the previously discussed similarities between cognitive control and emotion regulation, it seems logical that these two systems are related. In the currently published literature, researchers report deficits in both cognitive control and emotion regulation when studying individuals with disorders ranging from insomnia to ADHD to Borderline Personality Disorder (Cortoos et al, 2006; Stein, 2008; Walcott & Landau, 2004). If these two systems can directly be correlated this would help us to understand one aspect of general individual personality and behavior differences, as well as in part what may be happening in various mental health conditions. However, while past studies have looked individually at between subject variability in cognitive control and emotion regulation abilities, they have not attempted to directly demonstrate a relationship in system functionality by examining within subject similarities in the processes.

As discussed, Inzlicht & Gutsell (2007) did provide preliminary evidence for an association between the mechanisms of the two systems, but still did not question how their functionalities might be related. It seems probable that if the same parts of the brain

are involved in both systems, the ability of an individual to engage in these processes (the functionalities of the systems) should be positively correlated. In the first and only study to test this hypothesis, Compton et al (in press) found that subjects who reported less anxiety to daily stressors in a two-week survey were also better at error monitoring and error correction as shown using ERN response data. In this experiment, we will attempt to take these findings further by showing that individuals who exhibit signs of greater emotion regulation in laboratory situations will also show higher levels of cognitive control.

Two possibly confounding factors in the Compton et al study were the subjective nature of self-report data, as well as possible differences in day-to-day stressors encountered by individuals. Using physiological measures to study emotion regulation in a controlled, laboratory setting would eliminate both of these. Therefore, if such a study could correlate emotion regulation results with cognitive control ability, this may be seen as furthering the argument for overlapping regulatory mechanisms by demonstrating that when exposed to the same stressful stimulus, individuals who respond with less heightened physical arousal also show better cognitive control through EEG measures. In a more general sense, it may also be seen as evidence for the idea that the capability of a brain region to support one process is positively correlated with its capability to support another. In addition, based on the literature, we would expect coping styles to correlate with individual cognitive control and emotion regulation as measured here, which may have interesting implications in terms of possibly bettering cognitive control through working to improve methods of coping with emotion. Therefore, participants' general coping styles as reported using a stress-coping questionnaire should also be examined.

In this experiment, we will assume that using EEG technology to study ERN responses is an accurate representation of individual cognitive control ability. Given that ERN amplitude is considered to be a reflection of ACC activity in response to conflict (Gehring et al, 1991) this seems reasonable. We will also assume that stress regulation is a type of emotion regulation, and that physiological factors associated with the body's activation of stress response are indicative of the amount of anxiety being experienced. Specifically, large increases in cortisol and heart rate in response to laboratory stressors suggest a lack of stress regulation (Dickerson & Kemeny, 2004; William et al, 2007). We expect to show that these physiological measures correlate with subjective ratings of distress in response to the stress manipulation. Because in a laboratory setting the internal goals of participants presumably include successfully accomplishing the presented tasks, and anxiety would make successful completion more difficult, we will assume that smaller stress responses are indicative of a more adaptive emotion regulation system in general. As a final assumption, we will take problem-focused stress-copers to be more successful at emotion regulation than those who use emotion-focused or avoidance strategies, since problem-focused coping appears to lead to long-term successful accomplishment of goals (Endler, Kantor, & Parker, 1994). Based on these ideas and assumptions, we make the following predictions:

H1) Subjects who show greater ERN amplitudes, increased reaction time to stimuli following incorrect responses, and increased correct responses following incorrect responses (greater cognitive control) during the EEG task will also show smaller increases in salivary cortisol and heart rate and less reported anxiety in response to a laboratory stressor (greater emotion regulation).

H2) Subjects who show greater cognitive control will be more likely to use problem-focused coping strategies in response to stressors.

Methods

Participants

Participants were 18 male and 19 female undergraduate students at Haverford College who voluntarily responded to a request for psychology subjects. They were compensated $30 for their involvement. Participants were prescreened to ensure that they had normal vision, no neurological history, and were not taking any medications that would affect cortisol levels in the bloodstream or sympathetic nervous system activity.

EEG Data Recording

Electrophysiological ERP recordings were taken during the cognitive task using a Quik-caps system with Ag/AgCl electrodes and a NuAmps amplifier. Electrical activity was measured at a frequency of 1000Hz from the Fz, FCz, Cz, and Pz sites, using a bandpass of 0.1-40Hz. Data was collected using the right mastoid (A_2) as the reference site, thus comparing the right mastoid to the ground site and the active sites to the ground site. Eye movements were monitored from the left temple, the right temple, above the left eye, and below the left eye using leads X_1, X_2, X_3, and X_4 respectively.

After raw ERP collection, data were re-referenced to the average of the 2 mastoid sites (A_1 and A_2) thereby creating a symmetric reference point. Manual removal of gross artifacts was performed, as well as regression-based blink reduction algorithms (using Scan software) to remove the effects of blinks on scalp electrode sites. An epoch of

-200ms to 600ms was created around each response (correct and incorrect) with 0ms being the time of response. Correct and incorrect trial epochs were analyzed separately, and responses across the epochs within each subject's data were averaged to create the ERP waveform. The ERN was defined as being the most negative point on the waveform between response (0ms) and 100ms post-response.

Stroop Task

In order to measure cognitive control we used a Stroop color-identification task, which is known to elicit error response (Gehring et al, 1993). The specific Stroop task here was modeled after that used by Compton et al (in press), as it produced a significant post-error ERN and behavioral changes. During each trial the participant indicated the color of the word presented. There were 6 possibilities - red, orange, yellow, green, blue, and purple. A practice set of 24 trials was first given to ensure that the subject understood how to perform the task. The task then included 10 blocks of trials, each containing 66 separate trials. Within each block, 30 trials were incongruent (i.e. the word "blue" written in purple), 30 were neutral (a non-color word written in one of the 6 colors), and 6 were congruent (i.e. the word "blue" written in blue). These trials occurred in random order. Words were presented for 150ms, followed by a blank screen that remained until response by key press or a maximum 2s elapsed. A blank screen appeared for 600ms following the keypress response. For each trial, accuracy and reaction time were measured and recorded.

Stress Manipulation

A synthesis of laboratory stress and cortisol response research indicates that combinations of public speaking and cognitive tasks elicit the greatest changes in cortisol level (Dickerson & Kemeny, 2004). We followed procedures that have previously been shown to be successful in causing an increase in cortisol and sympathetic nervous system response (Bollini et al, 2004; Sgoutas-Emch et al, 1994). Following a script (Appendix A), participants were asked to imagine that they had been falsely accused of shoplifting. They were told to prepare a 2 minute speech which would a) present their side of the story, b) explain what the security guard did wrong and why they were falsely accused, c) explain how they could prove they were not guilty, d) describe consequences for the security guard based on the mistake, and e) summarize their points. Participants were given 3 minutes to prepare and 2 minutes to give the speech. They were told that they were being videotaped and compared to other participants in the study (introducing social-evaluative threat) though in fact the camera was not recording them (Sgoutas-Emch et al, 1994).

Next, participants were asked to complete a computerized mental arithmetic/working memory task. This task involved the presentation of numbers, one at a time, on a computer screen. Again following a script (Appendix B) participants were asked to report out loud the sum of the number they were currently seeing plus the number they had just seen. Numbers included digits 1-15. The task began with a practice block of 10 numbers, followed by 3 trial blocks each including 60 numbers. Numbers were presented on the screen for 500ms following by a plus (+) sign between presentations. During the three trial blocks, subjects wore headphones that played loud,

randomized, distracting noise blasts via computer software in conjunction with the + sign (introducing an uncontrollable aspect). Subjects were aware that the purpose of these noises was to make the task more difficult. Number presentation in the trials blocks was randomized, and no record was kept of subject accuracy (Bollini et al, 2004).

Self-Report Measures

The Coping Inventory for Stressful Situations (CISS) was developed by Endler and Parker (1994) as a way to measure self-reported stress-coping style – problem-focused, emotion-focused, or avoidant. It is a 48-item questionnaire that lists different possible activities in which people may engage in response to stress. Subjects are asked to rate on a scale of 1-5 how much they engage in each activity after encountering a stressful situation. Problem-focused coping is evaluated by high ratings to responses such as "Focus on the problem and see how I can solve it," emotion-focused coping is evaluated by high ratings to responses such as "Blame myself for having gotten into this situation," and avoidance oriented coping is evaluated by high ratings to responses such as "Think about the good times I've had." The CISS has been shown to indeed be predictive of how people respond to specific stressful situations (Cosway et al, 2000). Participants were asked to fill out the CISS questionnaire as a way to evaluate their general coping strategies.

Following the stress manipulation, participants were asked rate how "anxious" they felt on a scale of 1-10 (the Subjective Stress Scale, or SSS). In addition, they were asked to fill out the State section of the State-Trait Anxiety Inventory (STAI, Spielberger, 1983). This questionnaire involves 2 different scales, each consisting of 20 items, and

subjects are asked to rate on a scale of 1-4 how much they agree with each item. The S-Anxiety scale measures "state", or how subjects feel "right now, at this moment". Items on the S-Anxiety scale include statements such as "I feel at ease" or "I feel upset". The T-Anxiety scale measures "trait", or how subjects feel "generally". Items on the T-Anxiety scale include statements such as "I am a steady person" or "I lack self-confidence". Appropriately, the scale has been shown to have low stability for the S-Anxiety scale, and high stability for the T-Anxiety scale (Jacobs et al, 1988). Observing scores on the SSS and state anxiety allowed us to quantify the amount of stress that subjects felt in response to the stress manipulation.

Procedure

Participants arrived and were immediately asked to give a saliva sample by spitting through a straw into a 1mL micro-centrifuge tube from which we measured baseline levels of cortisol (T_1). They were also given a heart rate-monitoring wrist-watch from which we determined baseline heart rate. Participants were then fitted with the EEG cap and asked to perform the Stroop color-identification task while the cap electrodes measured voltage changes in the brain.

Following the EEG task, participants were asked to give another saliva sample as the cap was being removed, and heart rate was again measured (T_2). This allowed us to observe whether the EEG task caused an increase in HPA axis or sympathetic nervous system activation. After removal of the EEG cap, the stress manipulation began. It lasted approximately 15 minutes total. Heart rate was measured at the end of the stress manipulation (T_3). Following the stress manipulation, subjects were asked to rate their

anxiety level and to fill out the STAI and CISS. After the questionnaires were completed, subjects were asked to give a final saliva sample (T_3) since around 20 minutes post-stressor onset seems to be when the most cortisol is present in the saliva (Dickerson & Kemeny, 2004).

Saliva samples taken at T_1 (baseline), T_2 (post-EEG task), and T_3 (post-stress manipulation) were frozen and later analyzed for cortisol presence using a commercially available enzyme immunoassay kit (DS labs, Arlington, TX). Directly before the assay, samples were allowed to thaw at room temperature and were centrifuged to eliminate particles in the saliva. Samples were incubated with the provided cortisol antibody, and cortisol levels were read at 600nm under a spectrophotometer. Duplicate samples were run for each subject at each time point. Cortisol levels were log transformed for analyses.

Results

Stroop Task

ERN Analysis

A 4x2 repeated measures ANOVA was performed in order to analyze the size of the ERP waveform peak amplitudes in the specified time period (0-100ms surrounding trial response). Peak amplitudes were evaluated at 4 sites known to shown the ERN (Fz, FCz, Cz, and Pz) as a function of accuracy (correct versus incorrect). A main effect of accuracy was found ($F(1,35)=48.15$, $p<.01$), such that the peak amplitude was significantly larger following error trials (M=-6.67μV, SEM=0.75μV) than following correct trials (M=-1.69μV, SEM=0.52μV), averaged across electrode sites (see Figure 1). This indicates the presence of an ERN, consistent with our hypothesis that the Stroop task

would elicit an ERN response. There was also a significant interaction effect between accuracy and site ($F(3,105)=4.33$, $p<.01$), such that the greatest difference between peak size following error versus correct trials occurred at site FCz (see Table 1). Within each subject, difference scores for ERN peak amplitude (FCz(error)-FCz(correct)) were calculated.

Behavioral Changes

Subjects' overall accuracy ($M=0.91$, $SEM=0.01$) and reaction times ($M=669$, $SEM=19$) during the Stroop task were analyzed. Subjects showed significant changes in accuracy following error responses during the EEG task ($t(36)=-4.48$, $p<.01$) such that subjects were less accurate in trials following an error response ($M=0.86$, $SD=0.10$) than following a correct response ($M=0.92$, $SD=0.04$). There were also significant changes in reaction time following error responses ($t(36)=5.10$, $p<.01$), such that subjects responded

Table 1

Means and standard errors for the ERP waveform peak amplitude at each site in the designated epoch, following both error and correct responses

	Correct		**Error**	
Site	**Mean (μV)**	**SEM (μV)**	**Mean (μV)**	**SEM (μV)**
Fz	-1.32	0.54	-6.17	0.90
FCz	-1.20	0.64	-7.27	0.86
Cz	-1.15	0.57	-6.50	0.81
Pz	-3.09	0.66	-6.85	0.78

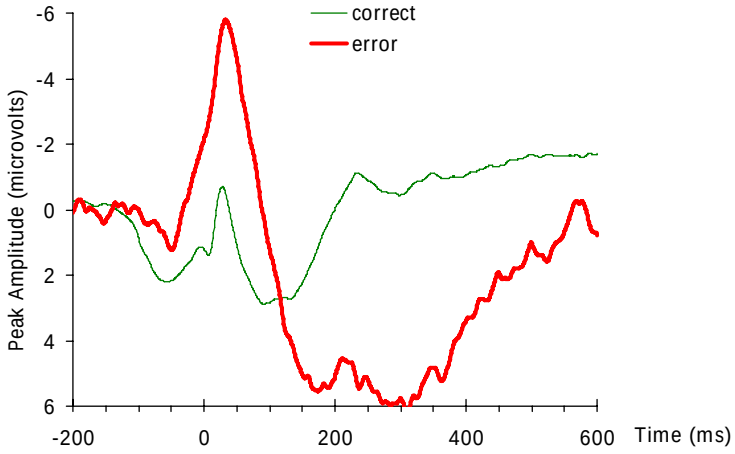

Figure 1. ERP waveforms at site FCz after correct and error trials. The ERN is clearly visible at around 80ms post response.

more quickly following correct responses (*M*=657, *SD*=110) than following error responses (*M*=743, *SD*=159). Both of these overall behavioral results are consistent with the findings of prior studies (Compton et al, in press; Hajcak et al, 2003b).

The ERN difference score was significantly correlated with subjects' overall accuracy (r =0.49, p<.01). In addition, changes in accuracy following error (calculated as accuracy following correct responses subtracted from accuracy following error responses) were significantly correlated with the ERN difference score (r =0.17, p<.05) and overall accuracy (r=0.48, p<.01). This suggests that subjects with larger ERN peak amplitudes performed better overall and showed greater improvement following error trials than subjects with small ERN peak amplitudes. However, the ERN difference score was not significantly correlated with reaction time.

Summary

Analysis of the ERP data indicated the clear presence of an ERN peak following error response (most notable at site FCz), and a decrease in accuracy and slowing of reaction time following error. It also suggested a positive relationship between the size of the ERN, overall accuracy, and improvement following error, which is in line with our assumption that these factors are indicative of cognitive control ability. In this study, reaction time did not appear to be related to cognitive control.

Cortisol

Validity of Assay

Duplicates for all samples were found to be highly significantly correlated with each other (r=0.78, p<.01). Cortisol levels in each pair of duplicate samples were averaged to create the final values used in statistical analysis. Within subjects, cortisol samples at T_1 were significantly correlated with samples at T_2 (r=0.70, p<.01) and at T_3 (r=0.56, p<.01), and cortisol samples at T_2 were significantly correlated with those at T_3 (r=0.71, p<.01). Because duplicate samples were similar and cortisol levels across time points for each subject were in the same general range, this argues for reliable data provided by the enzyme immunoassay kit (DS labs, Arlington, TX).

Overall Changes in Cortisol

Time of day was not significantly correlated with cortisol levels across subjects at T_1, T_2, or T_3, which demonstrates that cortisol was not affected by variance in scheduled experiment times. Because of this, time of day was not controlled for when looking at

cortisol change. A 1-way ANOVA was used to analyze differences between cortisol level averages at T_1, T_2, and T_3 across subjects. While mean cortisol levels appear to rise over the three time points, there were no significant changes. This indicates that overall, contrary to our hypothesis, the stress manipulation did not cause subjects' stress levels to increase in such a way that cortisol levels increased as well (see Table 2).

Cortisol and the ERN

Within each subject, difference scores for cortisol levels (T_2-T_1, T_3-T_1, and T_3-T_2) were calculated in order to compare increases in ERN amplitude following error (cognitive control) to changes in cortisol throughout the experiment. Cortisol levels at T_2-T_1 were significantly correlated with cortisol levels at T_3-T_1 (r=0.56, p<.01) but not at T_3-T_2, indicating that subjects' anxiety during the EEG Stroop task was not predictive of their anxiety during the stress manipulation. Contrary to our hypothesis, the ERN difference score was not significantly correlated with either T_3-T_1 or T_3-T_2, but was significantly negatively correlated with T_2-T_1 (r=-0.44, p<.01), showing that subjects with

Table 2

Cortisol level means and standard error of the mean over time points 1, 2, and 3

Time	Mean (μg/dL)	SEM (μg/dL)
1	1.248	0.123
2	1.287	0.142
3	1.302	0.165

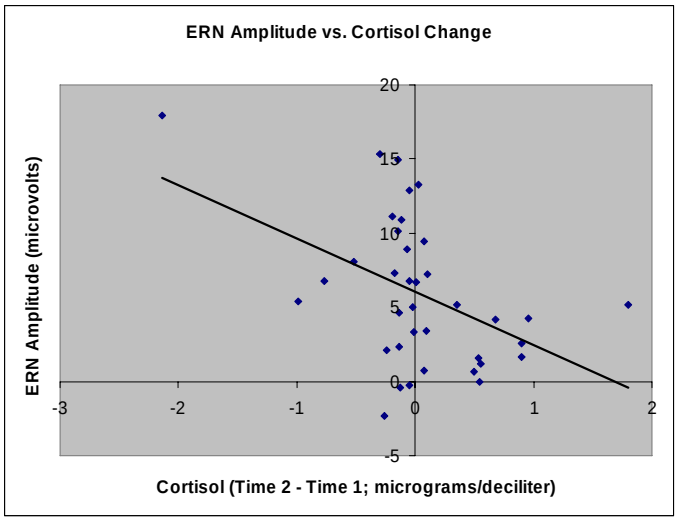

Figure 2. ERN difference score (site FCz) versus Cortisol level (T_2-T_1)

larger ERN peaks experienced smaller increases in cortisol between time 1 and time 2 (see Figure 2). In looking at the plot, the data appear to exist in a negative, linear relationship whether including or excluding the major outlying points. Subjects' overall accuracy was also correlated with cortisol levels at T_2-T_1 (r=-0.40, p<.01). In addition, changes in accuracy following errors trended towards a significant negative correlation with cortisol level at T_2-T_1 (r=-0.28, p=.09). In general, this suggests that subjects who experienced smaller increases in cortisol during the EEG Stroop task also exhibited larger ERN peak amplitudes while performing the task, were more accurate following error, and were more accurate in their responses overall.

As was shown earlier, subjects with larger ERN amplitudes tended to have higher accuracy rates, and high accuracy here was correlated with small cortisol increase. It seemed possible, therefore, that the correlation between small increases in cortisol and large ERN amplitudes occurred simply as an artifact of accuracy – that is, ERN amplitude was related to accuracy, and accuracy might have affected anxiety (and thus

cortisol increase), causing ERN amplitude and anxiety to appear to be correlated. To address this, we performed a partial correlation between the ERN differences scores and cortisol at T_2-T_1 while controlling for accuracy. ERN difference scores still trended towards a significant negative correlation with cortisol levels at T_2-T_1 (r=-0.32, p=.06), suggesting that this relationship cannot be explained simply as an effect of accuracy. In fact, when ERN difference scores were controlled for there was no significant correlation between cortisol change and overall accuracy, suggesting that the correlation between cortisol change and accuracy can be explained by differences in ERN amplitude. These results support the general main idea of our hypothesis – that smaller increases in cortisol are associated with larger ERN peaks.

Summary

Neither the stress manipulation nor the EEG Stroop task caused an overall increase in cortisol, and the stress manipulation was not successful in demonstrating a correlation between individual differences in cortisol increase and measures of cognitive control. However, individual differences in cortisol increase were observed during the EEG task such that small increases in cortisol were correlated with large ERN peaks, even when controlling for accuracy.

Self-Report Measures

Overall Coping Styles and Anxiety

New variables were created for each subject giving their average individual score on the CISS for problem-focused coping (M=3.66, SD=0.72), emotion-focused coping

(*M*=2.66, *SD*=0.75), and avoidance coping (*M*=2.46, *SD*=0.61) styles. A main effect of coping style was found ($F(2,68)=31.99$, $p<.01$), and a post hoc pairwise comparison using a Least Significant Difference test indicated that subjects were significantly more likely to engage in problem-focused coping than in either emotion-focused or avoidant coping styles ($p<.01$). There was no significant difference between emotion-focused or avoidant coping styles. The average state anxiety score of each subject was also computed. Subjects overall reported the stress manipulation as causing a moderate level of anxiety, measured by the state aspect of the STAI (*M*=2.53, *SD*=0.45) and the SSS (*M*=5.44, *SD*=2.21).

Self-Report Intervariable Correlations

Coping style, state anxiety, and score on the SSS were significantly correlated amongst each other such that state anxiety was significantly correlated with the SSS ($r=0.25$, $p<.05$) and the CISS emotion-focused coping score ($r=0.45$, $p<.01$), and score on the SSS was significantly correlated with both the CISS emotion-focused coping score ($r=0.47$, $p<.01$) and the CISS avoidance coping score ($r=0.54$, $p<.01$). This was in line with the idea that higher emotion-focused and avoidance coping scores should relate to less ability to regulate anxiety, though contrary to the assumption that problem-focused coping should result in better stress regulation outcomes, high problem-focused coping scores were not correlated with less reported anxiety.

Correlations Between ERP data and Cortisol

None of the self-report measures were significantly correlated with ERN difference scores, but the SSS was significantly negatively correlated with cortisol difference scores at time T_2-T_1 (r=-0.37, p<.05). This shows that higher perceived anxiety following the stress manipulation was associated with lower physiological stress during the EEG Stroop task. Scores on the SSS were also significantly negatively correlated with reaction time difference scores (r=-0.35, p<.05) indicating that subjects who slowed down following error trials reported more stress following the stress manipulation.

Summary

The self-report measures suggested that emotion-focused and avoidant coping styles were related to more perceived stress during the stress manipulation, but (contrary to our hypothesis) were not correlated with biological changes or cognitive control measures. However, high levels of reported anxiety following the stress manipulation were linked to smaller changes in cortisol and also to the tendency to slow down following error trials during the EEG Stroop task.

Heart Rate

Overall Changes in Heart Rate

A 1-way ANOVA analysis of heart rate showed a significant overall effect of time (1, 2, and 3) on heart rate (F(2,66)=20.37, p<.01). A post hoc pairwise comparison using a Least Significant Differences test indicated a significant difference (p<.01) such

that heart rate was significantly higher at T_1 (*M*=81, *SEM*=3) than at T_2 (*M*=72, *SEM*=2) or T_3 (*M*=72, *SEM*=2), which was contrary to our hypothesis. Heart rate at T_2 did not differ significantly from heart rate at T_3.

Individual Differences in Heart Rate

Within subjects, heart rate at T_1 was significantly correlated with heart rate at T_2 (r=0.77, p<.01) and T_3 (r=0.79, p<.01), and heart rate at T_2 was significantly correlated with heart rate at T_3 (r =0.79, p<.01). This indicates that across the three time points, subjects' heart rates behaved similarly. However, heart rate measures did not support our hypothesis in that no significant correlations were found in comparing difference scores for heart rate (T_2-T_1, T_3-T_1, and T_3-T_2) to the ERN and cortisol difference scores and self-report measures. There were also no significant correlations between heart rate difference scores and the behavioral ERP data (overall accuracy and reaction time).

Summary

Heart rate, as measured here, did not appear to be an effective measure of anxiety. Though it was consistent within subjects, it did not increase in response to either the stress manipulation or the EEG Stroop task, and was not correlated with any of the other stress variables.

Discussion

Overview

The general aim of this study was to investigate the relationship between individual abilities in cognitive control (as measured by the ERN and behavioral changes observed during an EEG task) and emotion regulation (as measured by increases in cortisol and heart rate and self-report scales during a stressful situation, and stress coping styles). The stress manipulation was not successful in inducing changes in cortisol or producing self-report measures such that individual differences were associated with cognitive control measures. Stress coping styles were also not found to be correlated with cognitive control variables. However, the EEG Stroop task induced individual changes in cortisol such that smaller increases in cortisol were associated with larger ERN peaks during that same task. These results generally support the idea that individuals who have stronger cognitive control abilities are also better able to regulate emotion.

Cognitive Control

As expected, we found highly significant ERN responses following incorrect trials in the EEG Stroop task, and also behavioral changes such that error responses caused subjects overall to be less accurate and to slow down. Such overall behavioral changes have been observed in previous studies as well (Compton et al, in press; Hajcak et al, 2003b**)**. ERN size was correlated with accuracy overall and (as expected) with an increase in accuracy following error, showing that large ERN peaks are related both to more accurate responses in general and to the ability to self-correct following error. This

agrees with current results in the literature (Ullsperger & Cramon, 2006), and supports the previously stated idea that ERN size and self-correction following error are both measures of cognitive control. We had hypothesized that slowing down following error could also be used as a measure of good cognitive control, but the fact that this variable was not correlated with either ERN size or accuracy improvement implies that reaction time may not be a valid measure of cognitive control. Perhaps (as suggested previously) people slow down following error trials because they are flustered or confused, and not because they are more concerned with accuracy (Hajcak et al, 2003b).

Emotion Regulation

The stress manipulation was not successful in generally increasing subjects' cortisol levels, and thus no significant overall changes in cortisol were observed between times 2 and 3 during the experiment. In this way, we failed to replicate the findings of the previous studies off of which our methods were based (Bollini et al, 2004; Sgoutas-Emch et al, 1994). We also did not observe the expected correlation between individual differences in cortisol change during the stress manipulation and cognitive control variables, which was contrary to our main hypothesis – that individual differences in cognitive control and emotion regulation should be related. However, because the stress manipulation failed to increase cortisol levels overall, it is reasonable to argue that individual differences in cortisol increase may still have been correlated with cognitive control measures had the stress manipulation been successful in inducing anxiety. Therefore, though these results do not support our main hypothesis, they do not oppose it either.

Interestingly, a link between cognitive control and emotion regulation measures can be seen in looking at data from the EEG Stroop task alone. Stroop tasks (like the one in the EEG section of our experiment) have been used in the past as stress manipulations on their own, since error-threat during tasks like these can be perceived as stressful (Rector & Roger, 1997). In this way the EEG task may have served as a sort of stress manipulation itself. There was no significant increase overall in cortisol during the EEG task. However, subjects who responded to error trials with larger ERN peaks showed smaller increases in cortisol levels during the EEG task. In addition to the correlation between cortisol increase and ERN size, smaller increases in cortisol were associated with greater overall accuracy and self-correction following error.

Though neither the stress manipulation nor the EEG Stroop task significantly elevated cortisol levels overall, looking at mean cortisol levels across the three time points shows that the EEG Stroop task caused a greater cortisol increase than the stress manipulation. This may be why we were not able to find a correlation between cognitive control variables and individual differences in cortisol change during the stress manipulation, but did observe that subjects who found the EEG task to be stressful (thereby showing less emotion regulation) also exhibited less cognitive control (as shown by smaller ERN peaks). These results are arguably in line with our original hypothesis – that individuals who are better at regulating emotion are also better at exerting cognitive control. Another recent study has reported the same correlation between small increases in cortisol and large ERN peaks during an EEG task, indicating that our results would be replicable (Tops et al, 2006).

Because we already saw that good performance was associated with greater ERN peak size, this raises the possibility that small increases in cortisol were associated with large ERNs as a result of the fact that subjects who experienced less anxiety performed better. However, when accuracy was controlled for, ERN amplitude still trended strongly towards a significant negative correlation with increases in cortisol. This shows that the relationship was not an artifact of accuracy, and the fact that the relationship between cortisol and accuracy disappeared when ERN size was controlled for indicates that stress and accuracy were associated only because of a shared association with the ERN. There are many examples in the current literature of subjects with generally high stress reactivity performing less well on laboratory tasks (Rector & Roger, 1997). Potentially these results can be explained in part through individual differences in ERN size, in that high stress reactivity (poor emotion regulation) is associated with poor cognitive control (a smaller ERN), which in turn results in less accurate responses.

One unfortunate aspect of observing such results during the EEG task as opposed to the designed stress manipulation is the presence of possible confounding factors. An issue that should be addressed here is the possibility that the ERN is sensitive to affective state such that emotion might itself cause a change in ERN size (Luu et al, 2003). That is, less emotion regulation ability could actually be impairing cognitive control. The observed correlation between cortisol increase and ERN size could be indicative of the fact that an increase in anxiety caused a decrease in ERN amplitude, as opposed to demonstrating a relationship between the functionality of subjects' general emotion regulation and cognitive control skills.

Previous EEG studies have investigated the idea that affect might cause a change in ERN size. Cavanagh & Allen (2007) showed that the ERN was diminished in subjects who reported negative (fear and shame) affective states, potentially reflecting task disengagement as caused by these emotions. However, in the study by Cavanagh & Allen, reported anxiety following the stressful tasks was not associated with a reduced ERN, possibly because feelings of anxiety do not lead to task disengagement. In fact, ERN size in state-anxiety (phobic) subjects has been shown to be equivalent to ERN size in a non-anxious control group (Hajcak et al, 2003a). This indicates that the experience of induced anxiety does not affect ERN size. If ERN amplitude does not change in response to induced anxiety, then ERN size during the EEG task can be assumed to reflect general cognitive control ability for all subjects, regardless of individual differences in state anxiety. Cortisol increase during the EEG task may be seen as indicative of stress regulation ability, and therefore the correlation between cortisol increase and ERN amplitude is still best explained by a correlation between emotion (stress) regulation and cognitive control system functionalities.

Considering self-report measures of perceived anxiety during the stress manipulation, just as cortisol levels during the stress manipulation did not increase in accordance with our hypothesis, these did not behave as predicted either. State anxiety and the SSS were significantly correlated with each other, indicating that they were measuring related variables. However, high anxiety reported on the SSS (but not the STAI) was correlated with smaller increases in cortisol during the EEG task (though not during the stress manipulation) and also with the tendency to slow down following error trials. If high SSS scores are taken to be indicative of less effective emotion regulation,

this again is contrary to the idea of using slowing down following error as a measure of cognitive control. Though state anxiety and SSS scores were closely associated, the larger range on the scale of the SSS (1-10) may have caused subjects to be more extreme their responses, while the smaller range (1-4) and multiple questions in the STAI section may have resulted in less extreme data. Perhaps state anxiety was not correlated with cortisol increase during the EEG task because of differences in the STAI and SSS scale ranges. Neither measure was associated with ERN size or accuracy improvement following error.

We had hypothesized that low scores on the STAI and SSS would be correlated with good cognitive control and assumed they would be associated with low increases in cortisol during the stress manipulation, but our results do not support these ideas. Given that the stress manipulation did not significantly elevate cortisol, and therefore presumably did not generally induce anxiety, it is reasonable that correlations with cognitive control variables did not occur. However, though it is understandable that STAI and SSS scores did not correlate with cognitive control measures, it is surprising that high reported anxiety following the stress manipulation was associated with less stress (smaller increases in cortisol) during the EEG task, since this implies that subjects who responded with less anxiety to the EEG task experienced more anxiety during the stress manipulation. This is somewhat counterintuitive since we might have assumed that overall, subjects who were better at regulating stress during the EEG task would also be better at regulating stress during the stress manipulation (showing a consistent ability to regulate emotion).

Possibly the correlation between SSS score and EEG cortisol increase occurred as an effect of contrast. Cortisol increases between times 1 and 2 were not correlated with cortisol increases between times 2 and 3. Because of this, it appears that finding the EEG task stressful was not related to anxiety experienced during the stress manipulation. Perhaps subjects who experienced greater anxiety during the EEG task (i.e. greater cortisol increases) felt that, compared to their previous experience, the stress manipulation was not stressful, and therefore responded with a lower rating on the SSS. Subjects who experienced less anxiety during the EEG task, then, may have been more inclined to report that the stress manipulation was stressful because they were not influenced by a previous increase in anxiety during the EEG task.

While coping styles as measured by the CISS did appear to relate to anxiety during the stress manipulation, they were not associated with cognitive control. The CISS questionnaire indicated that subjects were more likely overall to engage in problem-focused than emotion-focused or avoidant coping. We had predicted that individuals who engaged in emotion-focused or avoidant coping strategies would be less able to regulate stress, and our results support this idea. Subjects who reported using more emotion-focused and avoidant coping also reported more perceived anxiety as measured by state anxiety and the SSS. However, we also thought that problem-focused coping would lead to better stress regulation. Instead, our results suggest that while emotion-focused and avoidant coping may be harmful, engaging in problem-focused coping does not affect how stressful an individual perceives a situation to be – it does not harm or help. Also contrary to our hypothesis, we did not find a relationship between CISS

scores and cognitive control variables. From these results it does not appear that stress coping strategies as reported by subjects relate to cognitive control.

The fact that stress coping styles were not associated with ERN size or behavioral data was surprising given the study by Compton et al (in press) showing that ERN response data was associated with reported anxiety to daily stressors. However, Compton et al were looking at how subjects actually handled stress (i.e. how anxious they became), whereas the current study looked at how subjects would theoretically react to stressors. Enough literature exists surrounding stress coping styles that it seems fairly clear that problem-focused coping should result in better stress regulation outcomes (Endler, Kantor, & Parker, 1994). Perhaps when completing the CISS here subjects tended to report engaging in behaviors that they know they should do, or wish they did, as opposed to what they actually do. Often the options for problem-focused coping were clearly preferable or more positive (i.e. “I think about the event and learn from my mistakes”) compared to options for emotion-focused or avoidant coping (i.e. “I focus on my general inadequacies”). If subjects rated themselves as engaging in more problem-focused coping than is actually the case, this would also explain why problem-focused coping was not associated with better emotion regulation (i.e. lower scores on the SSS and STAI, or smaller cortisol increases during the EEG task).

Contrary to our hypothesis, analysis of heart rate showed significantly higher results at the first time point than at the other two, and changes in heart rate were not associated with any of the cognitive control variables or self-report measures. Elevated heart rates at the first time point can be explained by the fact that most subjects walked up five flights of stairs on their way to the lab, and some came straight from gym

workouts. In addition, because the stress manipulation failed to induce changes in cortisol that were associated with cognitive control, it is logical that it did not induce such changes in heart rate either. Since subjects did experience increases in cortisol following the EEG task that were associated with cognitive control, it might have been expected that larger decreases in elevated heart rate from time 1 to time 2 (implying more relaxation and thus less anxiety during the task) would be associated with greater ERN size. However, heart rate begins to drop more quickly than cortisol level (Sgoutas-Emch et al, 1994), and T_2 measures of both cortisol and heart rate measures were not taken until some time after the EEG task. Therefore, it is not surprising that changes in heart rate over the first two measurements were not correlated with cognitive control. In general, because of potentially confounding factors relating to variations in physical activity prior to the experiment and because of flaws in the timing of the measurement, this variable was generally discounted in terms of its implications towards emotion regulation success.

The fact that the multiple measures of stress regulation in this study were not generally intercorrelated brings up the question of whether or not these variables were truly measuring the same processes. What is again interesting to note here is the fact that cortisol changes during the EEG task were not correlated with changes during the stress manipulation (and were in fact negatively correlated with the SSS), which implies that subjects responded differently to different potentially stressful situations. When these correlations were discussed before, it was assumed that the stress manipulation was not perceived as stressful by any of the subjects, and thus did not cause cortisol increases or self-reported anxiety scores that would be positively associated with each other or cortisol increase during the EEG task. However, it seems that a significant problem in

studying emotion regulation is the fact that individuals respond to emotion-inducing situations differently, and this may or may not be a reflection of their emotion-regulation ability. For example, one subject may have had multiple positive past experiences involving public speaking, and another may have had multiple negative experiences. Because of this, the second person may have experienced a stronger anxiety reaction to the idea of a speech task than the first, causing them to need more emotion regulation ability in order to exhibit comparable cortisol increases or reported stress scores. On the other hand, it is impossible to determine causality, since it could be that a highly negative emotional response to a certain stimulus is actually a result of poor emotion regulation in the past (leading to poor outcomes, leading to an expectation of poor outcomes). In order to obtain a broader, more accurate view of an individual's emotion regulation ability, it might be necessary to use methods of assessing emotion regulation in multiple circumstances, and in response to induction of multiple emotions.

Taking these ideas of individual differences in relation to the current study, it is possible that cortisol increase in response to the EEG task, cortisol increase in response to the stress manipulation, and SSS and STAI scores were not correlated because of the differing stressful tasks in the experiment. Cortisol increase following the EEG task was presumably caused by a stressful factor in that part of the experiment (the Stroop task, for example). Cortisol increase during the stress manipulation was most likely to be influenced by the public speaking portion of the manipulation because the speech occurred first, at a time designed to later show maximum cortisol elevation in the third saliva sample (Dickerson & Kemmeny, 2004). However, subjects filled out the self-report measures directly following the mental arithmetic task, and therefore their

perceived anxiety on the math task was probably more likely to affect their SSS and STAI responses. Each task may have affected each individual subject differently - that is, one subject may have felt more anxious during the EEG task, another during the speech task, and another during the mental arithmetic task. If this were the case, it would explain why overall these separate measures were not associated, even though they were all technically measuring susceptibility to stress.

Overall, neither of our hypotheses were supported in the ways in which we predicted, since the stress manipulation failed demonstrate a relationship between individual differences in cortisol increase and cognitive control, and stress coping styles were not associated with cognitive control measures either. However, as discussed, the fact that subjects who showed smaller cortisol increases during the EEG task also had larger ERN peaks is an interesting result in relation to our hypothesis. The EEG Stroop task can be viewed as a type of stress manipulation, and in this stress manipulation we saw the expected trend – subjects who were more successful at regulating stress were also more able to exert cognitive control (as measured by ERN amplitude). These results support our general prediction that cognitive control and emotion regulation system functionalities are related.

Limitations and Future Directions

The most obvious limitations to this study arise from the fact that the stress manipulation did not work as predicted. Although the manipulation followed procedures previously shown to increase physiological measures of stress (Bollini et al, 2004; Sgoutas-Emch et al, 1994), there may have been some environmental factor that rendered

it less stressful. Possibly previous experiments using such procedures involved an experimenter who was, or appeared to be, an authority figure in relation to the subjects. This may have added an additional stressful component. In our case, experimenters were probably seen by subjects as equals, causing them to feel less anxious. It could also be a reflection of the fact that subjects were slightly anxious in general about coming into the lab, and so were more relaxed overall by the end of the experiment (during the stress manipulation) than at the beginning (during the EEG task). Subjects may additionally have inferred the point of the stress manipulation, which could have made performing well feel less necessary (thus decreasing anxiety), while during the EEG task data were clearly being recorded and performance monitored.

Considering limitations due to the subject pool, the recruitment message informed subjects that they would be giving a speech and performing a mental arithmetic task, so potentially individuals who volunteered for this study were less likely to become anxious during such procedures than the norm. In addition, the EEG task may have been perceived as more stressful by the subjects who were less successful at regulating anxiety than the stress manipulation because as college students, subjects were used to giving speeches and performing math tasks. However, the EEG recording and Stroop task were unfamiliar, and novel stimuli are often perceived as stressful (Kloet et al, 1999). More generally, a larger sample size would also have been preferable, although this would serve mostly to push trends to the point of significance.

Evidence for a link between emotion regulation as measured by physiological reactivity to stress and cognitive control as measured by ERN size can be seen here, but clearly research needs to be taken further. In order to control for the possibility that

stress level during the Stroop task affected the ERN, a study addressing our initial hypothesis – in which cortisol increases during a designed stress manipulation would correlate with ERN size – should be performed. The presence of social evaluation appears to be the threat that causes the greatest increase in subjects' cortisol levels (Dickerson & Kemmeny, 2004). The current study attempted to induce this threat through the presence of a video camera, but overall the stress manipulation was not effective. Perhaps a future study could make the idea of social evaluative threat more salient by creating a stress manipulation in which all participants come in at once, and deliver speeches in front of each other and the experimenters. Subjects could then come back for a second session during which EEG data recording could occur.

Future research should also attempt to further examine the relationship between stress coping styles, anxiety in response to daily stressors, and cognitive control as measured by the ERN. Compton et al (in press) asked subjects to fill out a daily questionnaire for two weeks in order to measure anxiety response to daily stressors. A future study might have subjects complete a similar two-week questionnaire in which they would report both anxiety to stressors and items on the CISS indicating how they coped with each specific stress experience. If reported stress coping strategies were related to a specific instance of stress, subjects might be less likely to idealize their response. Such a study could then compare reported anxiety to stress coping style (presumably finding problem-focused coping strategies to be more effective), and then compare each of these to the ERN – and possibly as well to physiological changes during a stress manipulation.

Additionally, as was briefly discussed, in order to convincingly demonstrate a link between individual emotion regulation and cognitive control abilities cognitive control should be linked to subjects' abilities to regulate various emotions, not just anxiety. In the future, subjects could be asked to look at upsetting or disturbing images, and emotion regulation could then be evaluated either through self-report measures of negative affect (Eisenberg, 2000) or by observing eyeblinks during image presentation (Oschner et al, 2002). Individual emotion regulation success could then be observed in relation to measures of cognitive control, such as the ERN and behavioral changes during an EEG task. We would expect to see that negative affect would be negatively correlated with cognitive control measures.

Overall, the current study should be replicated taking more care to make the stress manipulation effective and in the administration of the CISS. Further research will also be necessary in order to persuasively argue that general emotion regulation ability is predictive of the functionality of the cognitive control system. However, our results do add some interesting ideas to the current literature. We provide more evidence for the idea that cognitive control as measured by ERN size is related to accuracy improvement following error. More importantly, in correlating cortisol changes during the EEG task with ERN amplitude, this is the first paper to suggest that such results are indicative of a relationship between subjects' cognitive control and emotion regulation abilities. Our physiological data suggests that greater ACC activation (producing larger ERN peaks, Mathalon et al, 2003) is associated with smaller increases in cortisol, which theoretically can be mapped as well to increased ACC activation (Ochsner et al, 2002). We can say with some certainty that, as shown by our results, the brain's ability to exert emotion

regulation may be positively associated with its ability to exert cognitive control, which should help in further understand conditions involving the impairment of these processes.

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Appendix A

Speech Task Script

For the 1st challenge task, we are going to ask you to make up and deliver a short speech (2 min in length) in front of a video camera and in front of us. Do you have any questions or concerns?

Okay, let's begin. Please sit down in this chair. We are going to be sitting on the other side of the room facing you and controlling the video camera.

So, here is a piece of paper and pen for you to briefly jot down your thoughts while preparing your speech. You will have 3 minutes to prepare a speech about the prompt. We are also giving you a piece of paper listing information that should be included in your speech. The paper you use to jot things down on cannot be used at all during your speech- it is just for you to brainstorm. However, the list of the components required for your speech will be posted in the room for you to remind yourself what you should talk about. Remember you only have 3 minutes to prepare and think about your speech, you cannot use the notes you jot down during your speech, and your speech is limited to 2 minutes.

Here is the prompt: Imagine that you were in a department store shopping when a security guard falsely accused you of shoplifting. You will prepare a 2-min speech to

(a) tell your side of the story

(b) tell the manager what the security guard did wrong and why the security guard may have suspected you of shoplifting

(c) say how you can prove you did not steal the item

(d) specify what should happen to the security guard for the mistake

(e) summarize your points.

Please give intelligent and well thought-out answers because your speech will be recorded and compared with the speeches of others. You will have 3 minutes to prepare your speech and 2 minutes to present your speech.

Experimenter Notes:

-if the subject stops speaking, immediately instruct the subject to keep speaking and to continue speaking till the end of the 2 minutes

Appendix B

Mental Arithmetic Task Script

For the 2nd challenge task, we are going to ask you to perform a challenging math task in front of us. Do you have any questions or concerns?

Okay, let's begin. Please sit down in this chair. We are going to be sitting next to you and recording how many answers you get right and wrong. So, here are the instructions for this task:

> I would like you to complete an arithmetic task that is quite simple. At the same time that you perform the task, you will hear a variety of noises through the headphones. Most people find these noises loud and unpleasant. These noises are supposed to make the task more challenging. In this arithmetic test, you will be asked to add simple numbers one after the other. Your job will be to add the number you see with the number you saw just before it, ignoring the answer you give for the added numbers.
>
> (SEE EXAMPLE SHEET- show to subject & explain)
>
> This is one example, but you will have one practice set where I tell you if your answers are right or wrong.
>
> The numbers on the screen will be presented very quickly. It is important that you try to give an answer after each number and that you accurate in your answers. Your accuracy rate will be compared with others. I will be recording your answers, so please speak loudly enough that I can hear you. I will not be correcting you after each answer. There will be 3 blocks of trials and after each block there will be a short break where I will give you additional instructions. It is important that you complete the task.