Cognitive and Emotional Aspects of Error Responsiveness in Depressive College Students

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Abstract

The purpose of this study was to analyze the error responsiveness of depressive college students and to attempt to determine the contributions of emotional and cognitive processes to this response. This response was analyzed by measuring three event-related electrical signals originating in the Anterior Cingulate Cortex (ACC)—the response Error-Related Negativity (rERN), feedback ERN (fERN), and error Positivity (Pe)—as well as reaction time and accuracy following an error. A high depression group and an anxiety-matched control group were tested on an emotional and cognitive Stroop task. The expected increase in amplitude after errors was seen for the rERN and Pe but not for the fERN. While none of the error signals or behavioral compensation measures differed significantly between groups, there were surprising interactions found for the rERN and Pe suggesting that depression might be related to a larger Pe and a smaller rERN. This study provides many important points of comparison for other ERP and ACC studies in anxiety and other disorders.
Background
Overview and Rationale

In 2004, seventeen million adults experienced at least one Major Depressive Episode (MDE) during the past year and 14.8% of adults (36 million adults) had experienced a MDE at least once in their lifetime—making depression one of the most common psychiatric disorders and a leading cause of disability in America (SAMHSA, 2005; Kemp et al, 2005). These high prevalence rates, along with the associated economic and social costs, make depression a critical focus in psychological research.

By studying the neural correlates of depression and its associated cognitive deficits, researchers can develop more effective treatments and diagnostic tools, as well as a better understanding of depression’s relationship to other psychiatric disorders. The two leading cognitive theories of depression—Hopelessness theory and Beck’s Cognitive theory, both involve abnormal reactions to negative events, making this a core feature of the disorder (Gibb et al, 2004). The neurological basis of this hypersensitivity has yet to be determined and is critical in understanding and treating this disorder.

Depression involves deficits in both affective and cognitive control. Patients show heightened distress and emotional reactivity as well as problems with attention and strategic reasoning. One well-studied area of the brain that has consistently been implicated in both affective and cognitive control is the Anterior Cingulate Cortex (ACC), situated in the medial frontal lobe, dorsal and rostral to the corpus callosum. The ACC appears to have two subregions specifically involved in each of these two functions—activity in the rostral ACC (rACC) is related to emotion and distress and activity in the dorsal ACC (dACC) is related to cognition and attention (Bush et al, 2000). Because of their affective and cognitive deficits, it follows logically that depressed
patients would show abnormal activity in the rACC and dACC—and this has in fact been found using various brain imaging and electrical recording techniques (e.g. Drevets et al, 2001; Davidson et al, 2002; Pizzagalli et al, 2001; Elliott et al, 1997).

One of the most common ways of studying ACC functioning is through the measurement of electrical signals generated there. Of special interest are signals that occur in response to errors—the two focused on in this study are called the Error-Related Negativity (ERN) and the Error Positivity (Pe), and studies have suggested that these signals originate in the dACC and rACC, respectively. While the ERN has been found to be amplified in several anxiety disorders, the ERN findings in depression have been contradictory and have not included analysis of the Pe. By measuring these two error signals and by using emotional and neutral stimuli (neither of which has been done in an ERP study of depression), this study attempted to better understand depressed subjects’ responses to errors, and the differential contributions of emotional and cognitive processes to this response. Studying the error responses of depressed subjects in this more comprehensive way will help to quantify how this response differs from that in other disorders, particularly anxiety disorders.

The ACC

Connections and functions

The ACC is part of the limbic system and, like the system as a whole, is involved in affect regulation and motivational processes. It has connections with motor systems, the brain stem, the hypothalamus, and other regions of the frontal cortex (Devinsky et al, 1995; Luu et al, 2003). ACC activity has been studied using both functional imaging and
electrical recording techniques, and in many different subject groups. In general, the ACC is thought to be involved in evaluating and regulating behavior (Kiehl et al., 2000; MacDonald et al., 2000). ACC activity has been associated with response selection, motivational processes, goal-directed behavior, selective attention, error responsiveness, and language generation (Kiehl et al., 2000). More specifically, increased ACC activity is seen in situations where there is a need to change behavior (Gehring & Taylor, 2004). Situations calling for behavior change are generally negative and include erroneous responses, the loss of a reward, being excluded from a social group, and experiencing physical pain (Gehring & Taylor, 2004; Eisenberger et al., 2003; Tucker et al., 2003).

In order to motivate a change in behavior, the ACC appears to be responsible for linking negative events with emotional distress—signaling that something has gone wrong and that there is a need for change (Gehring & Taylor, 2004; Luu et al., 2000). Support for this idea comes from the fact that lesions to the ACC remove the affective distress associated with chronic pain and the finding that stimulating the ACC causes negative emotions (Tucker et al., 2003).

There are many interesting similarities between physical pain and errors that provide insight into the ACC’s functions. Pain and errors elicit very similar ACC activity and observable behavior, supporting the fact that similar neural pathways are involved in processing both events (Tucker et al., 2003; Davis et al., 1997). Both painful stimuli and errors can be detrimental to an individual’s fitness and survival. Therefore, it is adaptive to find these events distressing, and for this distress to lead to avoidance of the pain or error-related stimuli in the future (Gehring et al., 1993). In other words, ACC activity is necessary for a motivational state that leads to adaptive behavior under stress (Tucker et
The ACC appears to influence post-error behavioral adjustment through its connection with the frontal lobe, and more specifically the prefrontal cortex (PFC) (Botvinick et al, 2004). Therefore, emotional distress seems to affect future attentional and cognitive processes through this connection of the ACC to the PFC.

While this behavior monitoring and regulation in response to negative events is adaptive, it would be maladaptive for these events to be so distressing that future compensatory and avoidance behavior is hindered. This is the proposed situation in depressed subjects. That is, in depression, the distress response from the ACC may be overwhelming and not used constructively to adjust future behavior. Depressed patients show a lowered pain tolerance and increased pain-signaling behavior, and so if error processing involves this same pathway then one would also expect a hyperactive response to errors in these individuals (Tucker et al, 2003; Davis et al, 1997). Therefore, poor post-error adjustment in depressed patients, along with ruminations on errors and negative events, might be due to less activity the ACC which leads to less PFC activity or ineffective connectivity between the ACC and the PFC (Elliott et al, 1997).

**The rACC and the dACC**

The ACC is divided into two functionally distinct subregions with distinct anatomical connections, the rACC and dACC. The rACC, sometimes referred to as the Anterior Cingulate affective division (ACad), the subgenual or the ventral ACC, includes Brodman’s rostral areas 24a-c and 32 and ventral areas 25 and 33 (Bush et al, 2000). This region has connections with limbic and paralimbic structures, including the amygdala, nucleus accumbens, orbitofrontal cortex (OFC), periaqueductal gray, and autonomic brainstem motor nuclei (Pizzagalli et al, 2001). These connections, especially
with the amygdala, frontal cortex and motor areas, support its role in both affective
distress and motivating changes in future behavior. The dACC, also referred to as the
pregenual, caudal, or posterior ACC, includes Brodman’s areas 24b’-c’ and 32’ as well as
cingulate motor areas. The dACC has projections to the spinal cord, the parietal cortex,
and the red nucleus which is involved in movement (Devinsky et al, 1995; Pizzagalli et
al, 2001).

Imaging methods have associated rACC and dACC activity with distinct
functions (Bush et al, 1999; 2000). In general, the dACC appears to be involved in
attentional, cognitive and motor functions, with little to no effect on emotional processes
(Yeung et al, 2004; Fellows, 2005). More specifically, the dACC is involved in
visuospatial and memory functions as well as cognitively demanding information
processing, response selection under high conflict conditions, and adjusting allocation of
attention (Kiehl et al, 2000; Carter et al, 1998; Fellows et al, 2005). This region also
plays a role in pre-motor functions and tracking task parameters such as feedback valence
and conflicting task demands (Devinsky et al, 1995; Tucker et al, 2003).

The rACC, on the other hand, is involved in autonomic functions and emotional
processing (Tucker et al, 2003; Yeung et al, 2004; Bush et al, 2000; Devinsky et al,
1995). Besides regulating autonomic and endocrine functions, the rACC is involved in
goal-directed behavior, assessing motivational content, assigning emotional valence to
stimuli, conditioned emotional learning, and vocalizations expressing internal states
(Devinsky et al, 1995; Tucker et al, 2003; Devinsky et al., 1995). Healthy subjects show
increased blood flow to the rACC in response to induced sadness (along with less activity
in the corresponding areas of the dorsal frontal lobe) (Luu et al, 2000; Tucker et al,
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2003). Also, healthy subjects with rACC lesions show abnormal autonomic responses to emotionally provocative stimuli, supporting the theory that this region is necessary for affective control (Drevets et al, 2001).

**Error-Related Electrical Signals from the ACC**

The Electroencephalogram (EEG) is a method of measuring ACC activity that is useful in studying error response due its high temporal resolution. By studying EEG activity during or after specific events (event-related potentials (ERPs)) researchers can get a clearer picture of how ACC activity relates to specific events, and these findings can be a useful complement to brain imaging studies. The Error-Related Negativity (ERN) and the error positivity (Pe) are two well-researched signals originating in the ACC. Both of these signals are event-related peaks in ongoing electrical activity that are measured non-invasively from scalp electrodes. These signals reflect summed neuronal activity near the skull, and thus have less spatial resolution than many brain imaging techniques. The ERN and the Pe have been localized to the ACC, and in more recent studies to subregions within it, using advanced EEG methods or EEG along with concurrent brain imaging techniques (van Veen & Carter, 2002; Luu et al, 2000; Yeung et al, 2004; Botvinick et al., 1999; Braver et al., 2001; Carter et al., 1998; Kiehl et al., 2000; Tucker et al, 2003; Falkenstein et al, 2000).

The ERN occurs after erroneous or high conflict responses on various cognitive tasks, and is thought to reflect conflict monitoring between possible responses (van Veen & Carter, 2002). This signal appears to be a peak in ongoing activity of the dACC, and imaging studies support the role of this region in conflict monitoring (van Veen & Carter, 2002; Luu et al, 2000; 2001; Yeung et al, 2004; Botvinick et al., 1999; Braver et al.,
2001; Carter et al., 1998; Kiehl et al., 2000; Tucker et al., 2003). The types of trials that this region responds to are those with more possible responses, causing more cognitive interference and usually requiring the inhibition of a pre-potent response (Tucker et al., 2003; Carter et al., 1998). Error feedback is not necessary to elicit activity in the dACC, and activity here does not rely on conscious awareness of the error, suggesting that this region is involved in more automatic or subconscious conflict monitoring (Holroyd et al., 2004; Nieuwenhuis et al., 2001). The onset of the ERN is simultaneous with the subject’s response on cognitive tasks and has a negative peak at approximately 50-100 milliseconds (ms) later (Tucker et al., 2003). This signal is referred to as the response ERN (rERN), to distinguish it from a similar, less-studied signal seen after negative feedback (the feedback ERN, or fERN).

The fERN is a negative peak seen 300-500 ms after feedback indicating an error. It is postulated to have a stronger affective component than the rERN, since it appears to originate in both the rACC and dACC (Tucker et al., 2003). The fERN is thought to reflect more conscious error processing, and to relate more to external signals of error as opposed to internally-driven self monitoring.

The final error signal of interest is the Pe, a positive peak which occurs approximately 200-250 ms later than the rERN and is seen only after error trials and not high conflict trials (van Veen & Carter, 2002). This signal originates in the rACC and is thought to reflect affective processing of errors (van Veen & Carter, 2002; Falkenstein et al, 2000; Kiehl et al, 2000). Activity here has been shown to be greater for perceived than unperceived errors, suggesting that this region is involved in more conscious error processing than the dACC (Nieuwenhuis et al, 2001). The rERN and Pe have been found
to vary differentially, suggesting that they reflect different underlying cognitive processes (Falkenstein et al, 2000).

There have been no studies measuring all three of these signals simultaneously, especially in depressed subjects. By comparing signals which originate in different subregions of the ACC, it will theoretically be possible to see if error reactivity differs in these two subregions in the high and low depression groups.

**The ACC and the Stroop Task**

The Stroop task is a cognitive interference task that has consistently been shown to activate the ACC (Botvinick et al, 2004; Whalen et al, 1998; MacDonald et al, 2000). The Stroop task requires that subjects inhibit certain interfering information quickly and successfully in order to make accurate responses about the stimuli. Many different variations of the Stroop task have been designed for different clinical populations, in order to tap their specific difficulties in attentional control (George et al, 1997). However, when it became clear that the ACC was divided into two functionally distinct subregions Bush, Whalen and colleagues devised two versions of the Stroop task to activate each of these regions in healthy controls. The tasks they devised were the counting Stroop (cStroop) to activate the dACC and the emotional counting Stroop (ecStroop) to activate the rACC (Bush et al, 1998; Whalen et al, 1998).

Both the cStroop and the ecStroop require the subject to report the number of words that appear on the computer screen. Since the tasks were designed to be used in conjunction with MRI imaging, subjects respond by pressing a button instead of by verbally responding like in the classic Stroop task. As opposed to the classic Stroop task, where the subject must say the color of the word, the subject’s goal is to respond based
on how many times a word appears on the screen. The difference between the two tasks is the type of word that appears.

For the cStroop, the word is either a number or a neutral word (see Appendix B). The high-conflict (or interference) trials are those with number words, since they provide direct interference with the correct response. For example, the word “two” would appear three times on the screen, and the correct response is three. Bush et al (1998) found that there was significantly more rACC activity during interference trials of this task than neutral trials. In the ecStroop, the words are either emotional words or neutral words. In this task, emotional salience provides the interference for the high-conflict trials. Whalen et al (1998) found that there was significantly more rACC activity during the interference trials than the neutral trials in initial trial blocks of this task. Therefore, the selective ACC activation by interference trials on these two tasks supports the distinctive functions of the rACC and dACC. However, these tasks have not been used with depressed subjects or in conjunction with ERP or post-error behavioral data. By using these two tasks, the current study aims to investigate the effects of this subregion activation on error signals originating in these areas of the ACC, and whether this activation has different effects on depressed and control groups. It is expected that this activation will have different effects on the two groups because of past findings showing abnormal activity in ACC in depressed subjects.
The ACC in Depression

The dACC and depression

Depressed patients have been shown to have less activity in the dACC than controls in studies using Positron Emission Tomography (PET), functional Magnetic Resonance Imaging (fMRI), and single photon emission computed tomography (Davidson et al, 2002; Drevets et al, 1998; Elliott et al, 1997). Recovery from acute depressive symptoms is associated with an increase in dACC activity, suggesting that activity is state-dependent rather than trait-dependent (Davidson et al, 2002). This reduced activity is almost always seen along with decreased activation in the PFC, and has been associated with certain cognitive deficits seen in this disorder, including adjusting behavior (Gehring and Taylor, 2004; Davidson et al, 2002; Drevets et al, 1998; George et al, 1997; Elliott et al, 1997). More specifically, patients with damage to their dACC are worse at adjusting their behavior, especially after negative feedback (Gehring & Taylor, 2004).

In addition to showing lower dACC activity, it appears that depressed patients do not show increased activity here when controls do—in situations where behavioral adjustment is needed, both after negative feedback and increases in task difficulty. Therefore, there seems to be a certain amount of dACC activity that is needed to respond effectively to changes in a task and to errors, and depressed patients do not show this requisite amount of activity. Davidson et al (2002) hypothesize that this hypoactivation of the dACC is related more generally to the impaired modulation of attention or the impaired response selection seen in depressed patients. This hypoactivation could
provide the neurological explanation for depressed patients’ tendency to dwell on negative events and feelings and not actively try to improve them (Elliott et al, 1997).

**The rACC and depression**

As opposed to the dACC, depressed subjects show greater activity in the rACC than controls (Davidson et al, 2002; Pizzagalli et al, 2001; Mayberg et al, 1997). Within these depressed subjects, differences have been found between treatment responders and non-responders. Depressed patients who respond to drug treatment and sleep deprivation have been shown to have hyperactive rACCs pre-treatment relative to non-responding depressed patients and controls (Davidson et al, 2002; Pizzagalli et al, 2001; Mayberg et al, 1997). In response to this finding, Davidson et al concluded that the signal sent from the rACC—for further processing in response to conflict—is related to the treatment response. In addition, Mayberg et al (1997) found that rACC activity, and no other measure, differentiated treatment responders from non-responders and was predictive of amount of treatment response. This suggests that hypermetabolism in the rACC is needed for a good treatment outcome, and is potentially a predictive tool for treatment response (Mayberg et al, 1997). Interestingly, in a more recent study by Mayberg et al (2005), chronic stimulation of rACC white matter led to decreased levels of activity in the region as well as a sustained remission in severely depressed patients who had not responded from other treatments.

Thus, a hyperactive rACC has been seen consistently in at least some subgroups of depression. This increased activity might be associated with responsiveness to treatment, and decreasing this activity is effective in significantly improving depressive
symptoms. This hyper-activation in the rACC fits with the increased emotional distress and sensitivity to affective conflict seen in these patients (Davidson et al, 2002).

Lesions to the ACC have long been used to treat anxiety and depression (Luu et al, 2000), although it is unclear to which subregion these lesions were targeted. It is likely that the rACC was lesioned, since these surgeries were reported to decrease the intensity and duration of negative affect but not to improve cognitive deficits like ruminations (Luu et al, 2000). Some researchers claim that these lesions remove the limbic influence from the patients’ emotions, thus alleviating the abnormal affective distress associated with depression and anxiety (Luu et al, 2000). These findings support the role of the ACC in generating affective distress, and also the fact that other brain regions must also be involved in cognitive control.

One study using a modified Stroop task and mood disorder patients (six depressed and five bipolar) found surprising results in regards to group differences in subregion activity. George et al (1997) analyzed brain activity and behavioral data of a mood disorder and control group during a standard Stroop task and a sadness-related Stroop task. Using a PET-scan, the researchers found that during the standard Stroop task, mood-disordered patients had less activation in the dACC than control subjects (referred to in the study as the mid-cingulate) (George et al, 1997). Mood-disordered patients instead showed activation in the left DLPFC and right rACC (referred to as the anterior cingulate) (George et al, 1997). The sad Stroop task, surprisingly, was not associated with group differences in ACC activity. While the mood-disordered patients, and not the controls, showed more brain activity during the sad Stroop than the standard Stroop, this activity was only in the cerebellum and the visual cortex (George et al, 1997).
mood-disordered group was marginally slower and marginally more accurate than controls on the sad Stroop.

The George et al study showed that mood-disordered patients show differential brain activity during two types of Stroop tasks, but the ACC subregion activity did not differ as expected. While patients did show less dACC activity and more rACC activity than controls during the standard Stroop, they did not show more rACC activity than controls during the sad Stroop. This might be due to the heterogeneous subject sample, or the fact that the standard Stroop was more difficult for everyone because the tasks provided more direct interference, and difficulty activates the rACC more than emotional content. Therefore, this study aimed to use two counting Stroop tasks that have been shown to activate the rACC and dACC and a solely depressed sample to see if more expected group differences in ACC activity are found.

This study aimed to determine whether state-related depression differences, or task-induced differences, in ACC activity affect either the strengths of error-related signals originating in the ACC or behavioral adjustment after errors.

The ERN in different patient populations

Because ACC function can be tested by examining the ERN electrical potential, many studies have compared the ERNs of various clinical and non/sub-clinical subject groups in an attempt to learn more about the neural processes underlying the cognitive and behavioral deficits seen in the disorders. One of the most consistent findings is that patients with Obsessive Compulsive Disorder (OCD) and college students with Obsessive Compulsive (OC) symptoms show a larger rERN than controls as well as a hyperactive
rACC in response to errors (Hajcak et al, 2003; Hajcak & Simons, 2002; Gehring et al, 2000; Fitzgerald et al, 2005). OC symptom severity has been shown to be correlated with rERN amplitude and OCD symptom severity has been shown to be correlated with rACC error-related activity (Gehring et al, 2000; Fitzgerald et al, 2005). Hyperactivity in the ACC also increases with symptom provocation and decreases with successful treatment, showing that this abnormal activity is state-dependent (Fitzgerald et al, 2005). Surgical ablation has also been shown to improve OCD symptoms, specifically decreasing the distress associated with obsessions (Fitzgerald et al, 2005; Hajcak et al, 2003).

In OC and OCD subjects, an increased rERN has been found for both correct and incorrect trials, leading researchers to conclude that these patients have a hyperactive action monitoring system, so that they are more likely to doubt their actions and worry about making mistakes, even on correct trials (Hajcak & Simons, 2002). Gehring explains this finding in terms of a malfunctioning comparator, such that the ACC in these subjects is reporting that something is in need of change in the environment when everything is in fact satisfactory (Gehring et al, 2000). This ACC hyperreactivity thus might be related to the constant checking seen so often in OCD.

There have been very few studies analyzing the fERN in OCD, but one study found a trend for the fERN to be larger in severe OCD patients and for rERNs to actually be smaller in these patients (Nieuwenhuis, 2005). This contradictory rERN finding could be due to the fact that a trial-and-error task was used where rules were not known at the beginning of the task—and this type of task has been associated with larger fERNs and smaller rERNs.
Hyperactivity in the ACC and abnormally large ERNs have also been found in other anxiety disorders. Hajcak et al (2003) found that college students who scored high on the Penn State Worry Questionnaire (PSWQ) showed a stronger rERN than phobic and non-anxious students after correct and incorrect responses on a standard Stroop task. Patients with panic disorder (PD) and simple phobia show abnormal ACC activity when they are experiencing anxiety symptoms, and patients with PTSD show abnormal rACC activation during an emotional Stroop task (Hajcak et al, 2003). Induction of anxiety in normal subjects is associated with increased blood flow in the ACC and stimulating the ACC can cause intense emotional experiences, including anxiety (Hajcak et al, 2003).

While OCD and other anxiety disorders like Generalized Anxiety Disorder (GAD) have very low rates of co-morbidity, enhanced ERNs in both of these patient populations and their subclinical counterparts indicates that they may share an underlying feature related to enhanced error-related and conflict-related brain activity (Hajcak et al, 2003). One possible connection between these disorders is the prevalence of high negative affect (NA), which is related to general emotional distress (including anger, disgust, guilt, and fear) and is usually measured along with positive affect (PA) using the Positive and Negative Affect Scale (PANAS) (Clark & Watson, 1991). Both of these variables have been studied in relation to the ERN, and these findings could provide insight into the relationship between symptoms, personality variables, and ERN amplitude in different types of disorders.

Several studies have associated high levels of NA with abnormal responses to errors. For example, Luu et al (2000) found enhanced rERNs in a high NA group in the first trial block of a cognitive interference task. Interestingly, this difference disappeared
after the first block of trials, and the researchers attributed this to disengagement on the part of the high NA group. Since then, researchers have confirmed that high NA is related to a higher ERN, and a smaller Pe (Hajcak et al, 2004).

Because of these findings of greater ERN amplitudes in patients with high NA, some researchers believe that the ERN is related to general negative affective experience (Hajcak et al, 2004). Because of the common comorbidity of depression and anxiety, and the presence of high NA and emotional distress in both disorders, it might be expected that depressed subjects would show greater ERNs than controls just as anxious subjects do. However, this has not been found consistently in depressed subjects, and this might be due to the lower PA seen in depression.

The ERN in depression

As expected, the few ERN studies that have been done on depression have had inconsistent findings. A larger fERN has been seen in patients with Major Depressive Disorder (MDD) compared to controls on a spatial compatibility task with delayed feedback based on response time (Tucker et al, 2003). Feedback consisted of an A, C, or F letter grade. While the depressed patients’ fERNs were enhanced for all three letter grades, they showed the greatest difference from controls after the F grade. No differences between groups were seen in the rERN. Interestingly, the greatest effects in this study were seen in the moderately depressed patients (BDI score of 7-15). These authors suggest that this moderate level of depression may sensitize the limbic structures to respond strongly to adverse events (Tucker et al, 2003).

This study therefore showed a greater fERN in depressed subjects, especially after negative feedback, but no group differences in the rERN. Because level of performance
was based on speed and not accuracy it is likely that subjects were not aware of their errors until feedback. Since higher error awareness and a focus on accuracy over speed have both been associated with greater ERNs, this lack of group difference for rERNs could be due to low rERN activity for all subjects. Also, the depressed subjects were in remission and were not screened for high anxiety—three of these subjects were diagnosed with anxiety disorders. Since earlier studies have shown enhanced error sensitivity in anxiety, the presence of anxiety symptoms in the depressed sample could have confounded the results—that is the group differences seen could have been due to anxiety differences and not depression. Other studies have shown that abnormal ACC activity is state-dependent in depression, and so it is also possible that different results would have been seen had subjects been tested during acute depressive stages.

A recent pair of studies analyzed the ERN of depressed patients in remission who were screened for anxiety disorders, including OCD. This depressed group and a control group completed two different cognitive interference tasks with continuous feedback and no significant group differences were found for the rERN or the fERN (Ruchsow et al, 2004; 2005). However, group differences were found on trials following errors. Both studies found that depressed patients had smaller fERNs on error trials after error trials, but not on error trials after correct trials. This suggests that it was cognitive processing related specifically to errors that differentially affected the fERN in these subjects. More specifically, depressed subjects in this study showed similar cognitive processing to controls after correct trials, but less processing than controls on trials after errors. The researchers suggested that this decreased post-error activity could be due to reduced activity in the PFC in these patients, reflecting an underactive central reward pathway.
and/or a deficit in strategic reasoning (Ruchسow et al, 2004). Since depressed subjects
did not have significant anxiety symptoms, these results seem to suggest that there are no
differences in the ERN on error trials attributable to depressive symptoms alone.
However, the lack of group differences on the error trials themselves could be attributed
to the fact that these subjects were in remission and not in acute phases of depression.

Therefore, some past findings show that depressed patients are abnormally
responsive to errors on either the error trial itself or the trial after an error, as measured by
the ERN. There are also indications that depressed subjects show abnormal error
processing on the actual error trial that is not measured by the ERN. It is possible that
these differences could be measured by the Pe, which has never been studied in
depression. It remains to be seen whether depressed subjects with acute symptoms and
no differences in anxiety symptoms show different ERN amplitudes than controls, and in
what direction this difference occurs consistently. Moderately depressed patients appear
to show amplified ERN differences (at least for the fERN). These findings are much less
consistent than those on anxiety disorders. One reason for these differences might relate
to the symptomatology that differentiates these two disorders.

According to the tripartite model of depression and anxiety, depression and
anxiety can be differentiated by relative levels of two sets of symptoms—Negative Affect
(NA) and Positive Affect (PA) (Clark & Watson, 1991). NA refers to general distress
and unpleasant mood and is seen in elevated levels in depression and anxiety (Beck et al
2001; Clark & Watson, 1991). Therefore, if NA is an important predictor of larger
ERNs, one would expect depressed subjects to have larger ERNs like anxious subjects
do. However, PA is low only in depression, which manifests as decreased arousal,
energy and activity as well as hopelessness and a lack of motivation (Beck et al 2001; Clark & Watson, 1991). Perhaps this difference in PA could help explain the inconsistent ERN findings in depression, since these symptoms are generally the opposite of the hypervigilance seen in OCD and anxiety disorders. Therefore, perhaps it is only high NA in the presence of high PA that causes greater ERN amplitude (Hajcak et al, 2003). In other words, perhaps the low PA in the depressed subjects counteracts the effect of high NA on the rERN, and either removes the group difference in rERN amplitude or, as proposed in the current study, actually reverses the effect and decreases the rERN in the depressed subjects relative to controls. So far, however, in the few ERN studies that have measured PA, PA has not been found to have an effect on the ERN (Luu et al, 2000; Tucker et al, 2003). It remains to be clarified what effects PA and NA have on the ERN, to see whether these measures, and specifically the PA, provide a way of differentiating anxiety from depression.

Despite inconsistencies, there is ample evidence that depressed subjects show an abnormal response to errors, and one key question is how the actual performance of these subjects is affected by this abnormal error processing, in general and more importantly after errors.

**Post-error Behavioral Adjustment in Depression**

There are many important factors related to post-error behavioral adjustment in depression. When studying post-error behavioral adjustment in depression, it is important to consider the ACC’s role in cognitive control and its connection with the PFC, as well as group differences in activity in these regions in depression. It is also
important to consider depressed subjects’ behavior on post-error trials, and the cognitive styles and motivational differences seen in depression that might relate to post-error behavior.

PFC abnormalities have consistently been found in depressed subjects, and these abnormalities have been associated with cognitive deficits seen in these patients. Both reduced activity and gray matter volume have consistently been found in the PFC of depressed patients; especially in the left hemisphere (Drevets et al, 1998; George et al, 1997). Hypoactivity in the left PFC is correlated with depression severity and activity here has been shown to increase during remission, suggesting it is a state-dependent trait (Luu et al, 2000). Lowered PFC activity has been linked with problems with goal-directed behavior, strategic reasoning, new rule generation, and exerting cognitive control—all processes needed to respond effectively to errors (Drevets et al, 1998).

While it is clear that the PFC is related to cognitive control, and that the ACC and PFC are anatomically connected, because of mixed findings it is not clear how directly the ACC is involved in modulating cognitive control (Davidson et al, 2002). For example, an intact dACC is not necessary for normal cognitive control—including adjusting behavior after task manipulation and instruction, and slowing of reaction time after errors (on a cognitive interference task) (Fellows & Farah, 2005; Gehring & Taylor, 2004). However, amount of dACC activity after negative feedback has been found to predict the accuracy of the subsequent response (Gehring & Taylor, 2004). Also, when the dACC is damaged, subjects are worse at changing their responses, especially after a reduced reward cue (Gehring & Taylor, 2004). However, subjects in this study were OCD patients tested immediately before and after brain surgery, so they are not a
representative sample (Gehring & Taylor, 2004). Therefore, more research needs to be
done investigating the ACC’s specific role in cognitive control and behavioral adjustment
after errors.

Although the ACC’s direct role in cognitive control is not clear, ACC and frontal
cortex activity have consistently been found to be negatively correlated, and it is this
relationship which might be abnormal in depression. While ACC activity tends to
precede PFC activity, while the PFC is active the limbic system, including the ACC,
shows low activity, appearing to be inhibited (Tucker et al, 2003). Since depressed
patients have been shown to have less active and smaller PFCs it follows that this
inhibition might be abnormally low in these patients, so that limbic system and ACC
activity is abnormally high following errors (which agrees with high levels of emotional
distress seen after negative events and errors) (George et al, 1997). Interestingly, this
decreased inhibition appears to normalize upon recovery from depression; subjects who
have recovered from depression who are induced to feel normal sadness show decreased
rACC activity and increased PFC activity (so the PFC is more efficiently inhibiting ACC
connectivity has also been found in these patients. Pizzagalli et al (2003) found that
depressed patients did not show the normal correlation between ACC activity and frontal
cortex activity, suggesting a “disruption of functional connectivity.” Thus it appears that
either differences in activity of the entire circuit or the PFC’s inhibitory control of the
ACC is contributing to the deficits seen in cognitive control and post-error adjustment in
depression. These findings support the role of the ACC in post-error adjustment and the
expectation that this role should be different in depressed individuals.
In terms of actual behavioral adjustments following errors, depressed patients have consistently shown deficits relative to controls. In general, most subjects performing speeded choice reaction time tasks show slower reaction times on trials following errors. Researchers tend to view this as a cognitive adjustment made in order to decrease the likelihood of another error. Studies on depression have found that these patients have either reduced post-error slowing or no slowing at all (Luu et al, 2000; Elliott et al, 1997). Elliott et al (1996; 1997) also found that negative feedback had a detrimental effect on patients’ accuracy on trials after errors. Thus, depressed subjects who made an error on trial x were more likely than controls to fail on trial x+1. The authors referred to this effect as a “catastrophic response to perceived failure” (Elliott et al, 1997). This effect was found during depression and subsequent recovery, was specific to depression and not other psychiatric disorders, and was not a reaction to higher error rates overall. Responses to positive feedback were not analyzed. Anxiety has not been found to affect post-error slowing, supporting the fact that this effect is specific to depression (Hajcak et al, 2003).

Thus, while healthy subjects respond to errors by improving performance on the next trial, depressed patients do not show as much of an improvement (Elliott et al, 1997). Therefore, these patients show an abnormally small cognitive response to errors. The neurological basis for this is unknown. These deficits could be caused by rumination on past errors, lack of motivation, or several other abnormal cognitive or affective processes.

The cognitive styles and motivational levels of depression might provide clues to why depressed subjects do not tend to successfully adjust their behavior after errors. It is
possible that depressed patients’ ruminations on the previous negative event distract them from processing the current event. It is also possible that, since depressed patients have been shown to be less motivated by potential reward (and show less motivation more generally), they are less motivated to adjust their performance (although this would lead one to expect generally worse performance by depressed patients which is not seen) (Elliott et al., 1997; Davidson et al., 2002). It remains to be seen why exactly depressed subjects show this deficiency in post-error adjustment, and whether it is more due to excessive/hyperactive emotional processing or hypoactive cognitive processing, or both.

This abnormal reaction to errors and negative events more generally has important clinical/real-life ramifications for these patients. Abnormal response to failure is sometimes more related to whether a patient is admitted to the hospital than levels on clinical scales (Elliott et al., 1997). High levels of criticism also increase relapse rate to depression (Elliott et al., 1997). In addition, depressed patients are more likely to view events negatively and their performance as inadequate, so that they feel the above-mentioned catastrophic response much more often than healthy controls (Elliott et al., 1997). Therefore, more research is warranted on the ACC in depression to help determine the neurological underpinnings of this abnormal reaction to and adjustment after errors as it may help explain the etiology and exacerbation of the disorder.

**Hypothesis and Importance Based on Past Studies**

Measuring activity in the ACC along with behavioral adjustments could be a way to tie together the emotional and cognitive deficits seen in depression, since no unifying theory has yet been proposed (Elliott et al., 1997). In the current study, it was
hypothesized that the rERN will be reduced in the high depression group. The dACC is sensitive to tasks with high response conflict (both correct and incorrect trials). Depressed patients have been shown to have less activity in their dACC. Therefore, these subjects should show less activity in the dACC in response to high response conflict (on correct trials and error trials). The rERN has been localized to the dACC (and possibly also the PFC—which has been shown to be less active and smaller in depressed subjects)—so depressed subjects should show a lower amplitude rERN (less negative) than controls after high conflict trials. Depressed patients also tend to be less aware of their errors (and the rERN is greater the more sure you are of having made an error) so a smaller rERN would fit with this finding (if there is any doubt in the task).

The rACC on the other hand is only active in response to errors and is more associated with affective processing. Subgroups of depressed patients have been shown to have more activity in their rACC. Therefore, depressed subjects should show more rACC activity in response to errors. The Pe has been localized to the rACC—so depressed subjects should show a greater Pe than controls after errors.

The fERN appears to originate from both the rACC and dACC, and so predictions for it are not as clear-cut. However, if the affective component from the rACC is stronger, at least in depressed subjects (and studies have shown that the fERN is more affected by emotional distress) then this group should show a greater fERN (Tucker et al, 2003). This is therefore expected since these patients have shown a “catastrophic reaction to failure” as long as they are sure of their error—made sure by feedback, as well as a hypersensitivity to criticism—which is very similar to negative feedback. This effect should be exaggerated during the ecStroop since the more depressed subjects should be
less successful at ignoring the emotional stimuli. A greater fERN would also fit the past findings that depressed patients show greater emotional distress in general and related to negative events and pain. Control subjects might also show a greater fERN in the emotional vs. cognitive task, since their rACC should be more activated, making them more similar to the depressed group.

Post-error adjustment is agreed to be a function of the prefrontal cortex (PFC) and possibly also the dACC. Depressed patients have shown less activity in both of these areas as well as deficient post-error adjustment—in the form of less RT slowing on the trial following an error (Elliott et al, 1997). The present study aimed to replicate that finding. It was also hypothesized that depressed subjects would show even less post-error adjustment than controls on the emotional task, when they are more emotionally distressed and this is interfering with their cognitive processes (Elliott et al, 1997).

All in all, it was postulated that in response to errors, depressed subjects would show a hyperactive emotional response from the rACC. In response to all high conflict trials, depressed subjects were expected to show a hypoactive cognitive response from the dACC. These different activity levels should be reflected by a lower rERN, a higher Pe and fERN, and less post-error slowing than controls. Further, group differences should be more pronounced on the emotional task.

In order to isolate the effects of depression, subjects in the current study were depressive college students and an anxiety-matched control group. Subjects completed two versions of the Stroop task—the counting Stroop (cStroop) which has been shown to activate the dACC, and the emotional counting Stroop (ecStroop) which has been shown to activate the rACC (Whalen et al, 1998; Bush et al, 1999). This was the first study to
use these tasks in conjunction with ERP measurements, and with depressed subjects. By doing so, researchers were attempting to determine whether selective activation of these subregions by task had an effect on error signals and post-error behavior, and whether this effect was different in depressed subjects—attempting to determine how past imaging research on the rACC and dACC in these subjects relates to their error responsiveness. All past ERN studies on depression have used responses to neutral material. While these studies are useful, it is important to analyze these subjects’ responses to emotional stimuli, since it is these emotional events that are likely to play an important role in the etiology and exacerbation of depression. The current study was the first to study the Pe in depression, as well as the first to compare the rERN, fERN, and Pe in the same subject sample, comparing all three signals to post-error behavior. Therefore, this study aimed to investigate how different ACC responses affect cognitive adjustment.

By controlling for anxiety symptoms in this sample of depressed and control college students, the current findings will hopefully lead to a better understand of the specific effects of depressive symptoms and help to interpret the conflicting ERN studies in this group. These results can also be compared to past ERN studies in anxiety to further explain the specific similarities and differences between anxiety and depression in their error responsiveness. By measuring NA and PA along with depression and anxiety symptoms, this study aimed to test which personality variables most determined subject differences in error signals and post-error behavior, trying to tie together past findings for these two groups of disorders. Past studies have focused on clinically depressed subjects, usually in remission, and rarely excluding anxious subjects. The present study thus investigated the effects of current depressive symptoms, which is important because of
the state-dependent ACC abnormalities seen in depressed patients. Anxiety and OCD studies on the ERN have been replicated in the college sample but depression studies have not. It is known that “negative cognitive styles” are a risk factor for depression, and a way to visualize these differences could help in diagnosis and possibly prevention of this disorder (Gibb et al, 2004).

Comparing the signal across different task types was meant to compare this signal’s strength in response to emotional and neutral stimuli, to see if abnormal error responsiveness in depression is more pronounced in response to emotional stimuli. A main question for this study is how the emerging research on the rACC and dACC fit together and relate to error responsiveness and ERN amplitude in general, as well as in depressed subjects.

Instead of being generally hypersensitive to their errors, it was hypothesized that depressed subjects would show an abnormally strong emotional response but an abnormally weak cognitive response to their errors. Considering the rACC hyperactivity seen in some subgroups of depressed subjects, as well as their hypersensitivity to negative events, the current researchers expected that the Pe would be enhanced in depressed subjects (Elliott et al, 1996; 1997). Studying the Pe along with the ERN will add an important new method of measuring what appears to be abnormal error processing in these depressed subjects. The fERN has been shown to originate from both the rACC and dACC, and thus is also thought to have a strong affective component (Tucker et al, 2003). Because of the “catastrophic” response to errors seen in depressed subjects as well as their sensitivity to criticism, it was hypothesized that this signal would be larger in depressed subjects. However, depressed patients have been shown to have both lower
dACC and PFC activity, and therefore it was hypothesized that they will show an abnormally low cognitive response to errors—in the form of a lower rERN from the dACC, less post-error slowing, and lower accuracy on trials after errors relative to controls.

In summary, it was hypothesized that depressed patients’ reactions to errors is one of inappropriate emotional distress, which disrupts post-error cognitive adjustment but is not necessarily reflected by the rERN. Instead, this affective response might be reflected more by the Pe and possibly the fERN. Thus, depressed subjects’ response to errors is emotionally distressing but not constructive in bringing about appropriate cognitive adjustments. PA is a personality variable which differs between anxious and depressed patients, and these differences in PA may help explain why depressed subjects do not always show larger ERNs than controls like anxious subjects do.

**Methods**

**Subjects and Personality Measures**

Subjects were recruited from two college populations to fill out an online screening questionnaire consisting of the Beck Depression Inventory (BDI) and the Penn State Worry Questionnaire (PSWQ) (see Appendix A). Students who completed the questionnaire were entered into a lottery to win two prizes of fifty dollars, and those students in Introductory Psychology received course credit. Of those subjects that completed the questionnaire, individuals were excluded who had abnormal vision, a neurological disorder, a learning disability, or who were currently using medication or non-medical substances that affected the central nervous system (excluding moderate use of alcohol, caffeine or cigarettes). From the remaining group of screened subjects,
specific subjects were selected to participate in the lab session based on their BDI and PSWQ scores.

The high depression group consisted of 11 subjects scoring a 20 or higher on the BDI (considered moderate to severe depression) (see Table 1) (Beck & Steer, 1988). This high depression group included six moderate to severely depressed subjects and five severely depressed subjects, according to ranges cited by Beck & Steer (1988). The low depression group consisted of 13 subjects matched to the depressed group by PSWQ score, but having low scores on the BDI. Matching the groups on PSWQ scores meant that the groups varied only on one variable, so that conclusions could be made about the groups specifically related to the BDI. Both groups had three males, and the rest female.

<table>
<thead>
<tr>
<th></th>
<th>BDI</th>
<th>PSWQ</th>
<th>PA</th>
<th>NA</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>High depression</strong></td>
<td>27.27 ± 8.00</td>
<td>58.37 ± 12.45</td>
<td>27.30 ± 7.54</td>
<td>26.7 ± 7.06</td>
</tr>
<tr>
<td><strong>Low depression</strong></td>
<td>5.30 ± 3.58</td>
<td>61.80 ± 9.01</td>
<td>30.79 ± 5.85</td>
<td>18.21 ± 3.72</td>
</tr>
</tbody>
</table>

Table 1. Means of personality variables for high and low depression groups.

Once the two groups were selected, the subjects were contacted and asked to participate in the second phase of the study. These subjects received 20 dollars for a one hour session. After subjects gave their informed consent they were fitted with an EEG cap and completed two 15 minute computerized tasks (described later), followed by a computerized questionnaire including the Positive and Negative Affect Scale (PANAS) about the past week and a family history questionnaire (see Appendix C). The family history questionnaire asked subjects whether any of their first-degree relatives had been diagnosed with depression, bipolar disorder, anxiety, or ADD.
**Apparatus**

![Figure 1. Scalp placement of electrodes.](www.heartcoherence.com/userguide/pro04UserGuide)

Subjects were fitted with a stretchy EEG cap. Electrical activity was measured at the Fz, Cz, and FCz sites referenced to right mastoid (see Figure 1). The ERN has been localized to these sites in past studies, which are thought to reflect activity in the ACC (Luu et al, 2004; Yeung et al, 2004). Electrodes were also placed above and below the left eye to measure vertical eye movement and on each temple to measure horizontal eye movement. Data were collected with a Quik-Caps system connected to a NuAmps amplifier which filtered through a bandpass of 0.1-30 Hz. The electrodes recorded continuously with a 1000 Hz sampling rate.
Tasks

Tasks were presented by E-prime software on a Dell Dimension desktop computer. Each subject performed two tasks—the cStroop and a modified ecStroop. The only difference between these two tasks was the type of words used. The order of these tasks was counterbalanced across subjects ($\chi^2 (1, N = 24) = 1.92, p > .05$). At the beginning of the experiment, subjects were given instructions on how to complete the task and were instructed to be as fast and accurate as possible. They were then given eight practice trials, after which the experimenter answered any questions the subjects had. Each task was composed of five blocks of 72 trials, for a total of 360 trials for each task. There was a break in between each trial block when subjects were allowed to rest their eyes and were told to switch the hand they were responding with. Words were white on a black background.

Subjects reported, by pushing a key on a computer keyboard, the number of words on the screen (from one to four), regardless of word meaning (see Appendix B). With four fingers of their hand (not including their thumb) placed on the f, g, h, and j keys of the computer keyboard, subjects pressed the “f” key if the word appeared once, the “g” key if the word appeared twice, the “h” key if the word appeared three times, and the “j” key if the word appeared four times. To increase the difficulty of the task, a string of “X”s the same length as the given word was used to fill the open space where words were not listed if the word was repeated less than four times.

For the cStroop, the word was either a number word (one, two, three, four) or a neutral word (dog, cat, bird, mouse). These were the same words used by Bush et al (1998). The number words were considered the interference trials, since the number was
never the same as the appropriate response, and these trials were meant to provide more
cognitive interference and difficulty than the neutral trials (see Appendix B). For
example, in an interference trial for the cStroop, the word “three” would be listed four
times on the screen and the subject must respond by pressing “j” corresponding to four.

For the ecStroop, the neutral words were “cup,” “bowl,” “fork,” and “glass” and the
interference words were “sad,” “hurt,” “fail,” and “grief.” These interference words
were altered from the anxiety-related words used by Bush and Whalen, some taken from
words used by George et al for the sad Stroop, in order to target the depressed group
specifically (George et al, 1997; Bush et al, 1998; Whalen et al, 1999). In this task, the
emotional valence of the words provided the interference (Whalen et al, 1998). For each
task, each word was presented three times with three different frequencies. Each
frequency (one, two, three, or four) was presented in the same position on the screen.

A black fixation screen was presented for 500 ms, followed by the stimulus for
100 ms. After the stimulus disappeared, subjects were allowed 2000 ms to respond so as
to provide modest time pressure and limit the number of omission errors. Immediately
after the subject’s response, there was a 600 ms pause followed by feedback. This
prevented the rERN from being influenced by task feedback, so that the rERN and fERN
could be analyzed separately. Feedback lasted for 500 ms and consisted of either
“Correct!” presented in white or “Incorrect!” in red, both on a black background. This
feedback was given after every trial to control for possible differences in error awareness
that have been found in depressed patients and patients with high NA (Hajcak et al, 2003;
Luu et al, 2000). The stimuli presentation duration was set at 100 ms so that it was as
quick as possible while still being readable, to increase the difficulty of the task—based
on Falkenstein et al’s findings that moderate time pressure led to the greatest ERN amplitudes (Falkenstein et al, 2000). The procedures for this study were approved by the college’s Institutional Review Board.

**EEG Data Preparation**

After data collection, voltages were re-referenced to the average of both mastoids to balance out any asymmetries in reference-site activity. Gross artifacts were removed manually as were portions of the record corresponding to task breaks. A regression-based blink reduction algorithm was used to remove any remaining artifacts. For each subject, all epochs of a given trial type were averaged into a single waveform. The time of each response and each feedback screen presentation was marked by E-prime on the EEG record so that the waveforms could be analyzed around specific time windows relative to these events. For each average waveform, the rERN was defined as the most negative peak between -50 and 150 milliseconds (ms) after the subject’s response. The Pe was defined as the most positive peak between 200 and 400 ms after the response and the fERN was defined as the most negative peak between 300 and 500 ms after task feedback, and (van Veen et al, 2002). Data from each scalp site (Fz, Fcz, and Cz) remain separate for most of the data analysis, in order to include as many data points as possible and to compare the effects at the different sites. One male subject from the depressed group was removed from the dataset for analysis due to high error rates and noisy ERP data. For data analysis including task and accuracy one male control subject was removed due to low error rates.
Results

Error Signals:

rERN

It was expected that the rERN amplitudes for all subjects, as defined above, would be higher (more negative) for incorrect trials than for correct trials. This was confirmed using a 3 x 2 x 2 x 2 mixed ANOVA examining the effects of EEG site, accuracy, task, and group on rERN amplitudes. There was a significant main effect of accuracy (F(1,21) = 55.90, p<.001), showing that the EEG sites chosen were indeed able to detect this specific error-related neural activity, and that the time frames and tasks used were able to elicit an rERN (as described in past literature) (see Figure 2 between 0 and 100 ms).

Figure 2. Graph showing the main effect of accuracy on both rERN amplitude (0 to 100 ms) and Pe amplitude (200-400 ms) at the Fz site for all subjects. Subject response is at 0 ms.

Using the same ANOVA, a main effect of task was found (F(1,21)=5.11, p<.05) (ecStroop: M = -5.03, SD = .72; cStroop: M = -3.83, SD = .72) (see Figure 3).
Therefore, subjects showed greater rERN amplitudes on the ecStroop than the cStroop. There was also a marginally significant interaction of accuracy and task \((F(1,21)=3.35, p=.081)\), meaning that accuracy had more of an effect on rERN amplitude during the ecStroop (see Figure 3). Incorrect trials differed marginally more between tasks than the correct trials. Errors on the ecStroop had marginally higher amplitudes then errors on the cStroop. This shows that the rERN was somewhat more affected by the emotional task than the cognitive one.

![Figure 3](image.png)

**Figure 3.** Graph showing the main effect of task and marginal interaction of accuracy and task on rERN amplitude, with no group effects or interactions.

This ANOVA was also used to test the hypothesis that depressed subjects would have lower rERN amplitudes than controls. There was no main effect of group \((F<1)\), or an interaction of group and accuracy \((F<1)\). Therefore, the rERN amplitudes of the high depression group and the low depression group did not differ significantly overall, or
between correct and incorrect trials (no group x accuracy effect). There was also no interaction of task and group (F(1,21)=2.10, p>.1) or of accuracy, task and group (F(1,21)=6.7, p>.1), so the two tasks did not affect the groups differently.

In summary, the rERN was found to differ significantly between correct and incorrect responses, but no group differences in amplitude were found, on all trials or on incorrect trials. There were however interesting effects of task. All subjects showed greater rERN amplitudes during the ecStroop. This difference was due to higher amplitudes for both the neutral and high conflict trials of the ecStroop. There was also a marginal interaction between accuracy and task, so that there was a bigger difference between correct and incorrect trial amplitudes in the ecStroop than the cStroop. These results suggest that the rERN is in fact related to error monitoring and that there is more active error monitoring in the emotional task, and specifically errors on the emotional task.

**fERN**

It was expected that subjects’ fERNs would be higher for errors than correct trials, and that the high depression group would show higher fERNs than the low depression group. In a 3 x 2 x 2 mixed ANOVA of site, accuracy and group’s effects on fERN amplitude, there was no main effect of accuracy (F<1) or group (F<1) or an interaction of accuracy and group (F<1). Therefore there was no difference in fERN amplitude after correct and incorrect feedback or between groups. There was also no main effect of task (F<1) or any significant interactions with task (F<1).
Because the Pe is thought to reflect more conscious affective processing, it was expected that the high depression group’s Pe amplitudes would be greater than the low depression group’s and that this difference would be greater on the(ecStroop than the cStroop. In a 3 x 2 x 2 x 2 mixed ANOVA examining the effects of site, accuracy, task and group on Pe magnitude, there was a main effect of accuracy ($F(1, 21)=30.56, p<.001$) (see Figure 2 between 200 and 400 ms, and Figure 4). The amplitude of the Pe signal was therefore greater (more positive) on incorrect trials than correct trials.

Figure 4. Graph showing the main effect of accuracy but no group or group x accuracy effect.

There was also a main effect of site ($F(2,42)=8.71, p=.001$) as well as an interaction of accuracy x site ($F(2,42)=9.02, p=.001$) (see Table 2). Therefore, Pe
amplitudes were significantly different across the three sites, and on correct and incorrect trials were more similar at the Fz site than at the other two sites.

<table>
<thead>
<tr>
<th>Accuracy</th>
<th>Site</th>
<th>Mean Amp (µV)</th>
<th>St. Dev.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Correct</td>
<td>Fz</td>
<td>4.440</td>
<td>.757</td>
</tr>
<tr>
<td></td>
<td>FCz</td>
<td>3.860</td>
<td>.862</td>
</tr>
<tr>
<td></td>
<td>Cz</td>
<td>1.674</td>
<td>.897</td>
</tr>
<tr>
<td>Incorrect</td>
<td>Fz</td>
<td>11.393</td>
<td>1.116</td>
</tr>
<tr>
<td></td>
<td>FCz</td>
<td>13.256</td>
<td>1.361</td>
</tr>
<tr>
<td></td>
<td>Cz</td>
<td>11.619</td>
<td>1.326</td>
</tr>
</tbody>
</table>

**Table 2.** Mean and st dev of Pe amplitudes at three sites for correct and incorrect trials, showing the main effect of accuracy and site and accuracy x site interaction.

There was no main effect of group across all sites (F(1,21)=1.05, p>.3) but there was a marginal interaction of site x group (F(2,42)= 3.02, p=.059) meaning that there was a slightly bigger difference between groups at the most frontal site (Fz) than at the other two sites (see Table 3). In fact, at the Fz site alone, there was a main effect of group (F(1,21)=4.37, p<.05). Therefore, at the Fz site, the high depression group had significantly lower Pe amplitudes than the low depression group.

<table>
<thead>
<tr>
<th>Group</th>
<th>Site</th>
<th>Mean Amp (µV)</th>
<th>St. Dev.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low depression</td>
<td>Fz</td>
<td>9.226</td>
<td>.825</td>
</tr>
<tr>
<td></td>
<td>FCz</td>
<td>9.000</td>
<td>.904</td>
</tr>
<tr>
<td></td>
<td>Cz</td>
<td>6.872</td>
<td>1.023</td>
</tr>
<tr>
<td>High depression</td>
<td>Fz</td>
<td>6.608</td>
<td>.941</td>
</tr>
<tr>
<td></td>
<td>FCz</td>
<td>8.116</td>
<td>1.030</td>
</tr>
<tr>
<td></td>
<td>Cz</td>
<td>6.420</td>
<td>1.166</td>
</tr>
</tbody>
</table>

**Table 3.** Mean and st dev of Pe amplitudes at three sites for high and low depression groups, showing marginal site x group interaction and main effect of group at Fz site.
No overall task differences were expected for Pe amplitude, and there was in fact no main effect of task (F(1,21)=2.95, p>.1). It was also expected that the Pe amplitudes of the high depression group would differ more between tasks than the controls since they would be more affected by the ecStroop than controls. The data did not support this, as there was no task x group interaction (F<1). Across all subjects, there was a trend for the Pe to be larger for the cStroop than the ecStroop at the Fz site (marginal task effect at the Fz site). Therefore, at the frontal site, Pe amplitude was somewhat larger during the cognitive task than the emotional one.

In summary, as expected, the Pe signal was larger for incorrect than correct trials, although the magnitude of this effect varied somewhat across sites. At the Fz site, the Pe was unexpectedly smaller for the depressed group than the control group. Both groups’ Pe amplitudes were equally affected by the two tasks and by correct and incorrect trials. There was a trend for both groups to show larger Pe amplitudes at the Fz site for the cStroop than the ecStroop. There were no significant Pe differences between tasks and the two groups’ Pe amplitudes were not affected differently by each task.

**Behavioral Data**

**Reaction Time**

The researchers expected that reaction times (RT) overall would not differ between groups. Indeed, in a 2 x 2 x 2 mixed ANOVA analyzing the effects of task, trial type, and group on RT, there was no main effect of group (F(1,22)=1.783, p>.1). There was, however, a main effect of task (F(1,22)=27.76, p<.001) (ecStroop: \( M = 600.20, SD = 19.29 \); cStroop: \( M = 670.16, SD = 23.35 \)), where subjects took longer to respond on
the cStroop than the ecStroop, showing that the cStroop trials provided more cognitive interference (causing them to be more difficult).

Within the tasks, it was expected that all subjects would take longer to respond on the interference trials than neutral trials. In the above-mentioned ANOVA, there was a main effect of trial type as expected (F(1,22)=31.30, p<.001) (neutral: $M = 562.17$, $SD = 17.90$; interference: $M = 588.15$, $SD = 16.66$). Interestingly, there was also a task x trial type effect (F(1,22)=36.60, p<.001), meaning that the effect of trial type was more pronounced for the cStroop than the ecStroop (see Figure 5). This is understandable since the number trials on the cStroop directly conflicted with the correct response, while the emotion word trials on the ecStroop did not. In other words, the interference trials were more difficult than the neutral trials on the cStroop than the ecStroop.

![Figure 5. Graph showing the main effect of task and trial type and the task x trial type interaction and task x trial type x group interaction. The effect of task, trial type, and group on RT.](image-url)
It was expected that the high depression group would be more affected by the interference trials of the ecStroop than the control group would be (in other words that 1) the depressed group would have longer RTs on the interference ecStroop trials than the control group, and 2) that the depressed group would have more of a difference of trial type effect between both tasks than the control group). There was no group x trial type interaction meaning that in general both groups reacted similarly to both types of trials across both tasks. However, the difference in trial type effect did differ between groups, in the form of a task x trial type x group interaction (F (1,22)=5.98, p<.05) (see Figure 5). Therefore, in the ecStroop, the high depression group was slower for emotional words than for neutral words, but the control group was not. In other words, both groups were more slowed by interference on the cStroop than the ecStroop, but this difference between tasks was smaller for the depressed group. So for the high depression group, the emotional interference effect approached the large cognitive interference effect. The task x trial type interaction for the depressed group (F (1,10)=9.45, p=.012) was less significant than the task x trial type interaction for the control group (F (1,13)=38.07, p=.000).

Past studies have shown that subjects sometimes disengage as the session continues, slowing their RT (Luu et al, 2000). This behavior has also been shown to vary based on personality variables including NA (Luu et al, 2000). Disengagement was analyzed by separating the entire testing session into 10 trial blocks and running a 2 (group) x 2 (order) x 10 (trial block) mixed ANOVA on reaction time. This analysis showed a main effect of block (F(9,180)= 13.12, p<.001). Therefore, for this study, subjects actually responded more quickly as the session progressed. This disengagement
did not differ between groups (no block x group interaction), so it can be assumed that basic motivation and attention were similar for both groups throughout the session. Since there were so few errors made in the entire session changes in accuracy across the session was not analyzed.

Accuracy

On a 2 x 2 x 2 ANOVA analyzing the effects of group, task, and trial type on percent correct, all subjects made more errors on interference trials than neutral trials (main effect of trial type) (F (1,22)=5.80, p<.05) (neutral: $M = .948$, $SD = .012$; interference: $M = .942$, $SD = .012$). As in the RT data, there was a task x trial type interaction, such that trial type had a bigger effect on accuracy for the cStroop than the ecStroop (F(1,22)=41.16, p<.001). In other words, the number trials on the cStroop were the most difficult out of any trials. It was not expected that groups would differ in overall accuracy. Indeed, there was no main effect of group (F<1). There was also no difference between groups with respect to accuracy on each task (no task x group interaction) (F<1).

Post-error Behavioral Compensation

Studies have consistently found that on the trial after an error, subjects take longer to respond. For this analysis, behavioral data were coded based on the accuracy of the previous trial. In a 2 x 2 x 2 mixed ANOVA analyzing the effects of previous trial accuracy, task, and group on RT, there was a main effect of previous accuracy (F (1,21)=19.90, p<.001) (previous correct: $M = 569.89$, $SD = 18.25$; previous incorrect: $M = 700.47$, $SD = 29.31$), showing that subjects took longer to respond when they had made an error on the previous trial. All subjects showed more post-error slowing on the
cStroop than the ecStroop, which fits with findings showing that the cStroop was more difficult (task x previous accuracy interaction: F (1,21)=4.77, p<.05). The high depression group did not show less post-error slowing than controls, as has been found in past studies (F (1,21)=1.27, p>.05).

On trials following errors, subjects also tend to be more accurate. However, in this study this was not found. Surprisingly, on a 2 x 2 x 2 mixed ANOVA analyzing the effects of previous accuracy, task, and group on percent accuracy, subjects were actually less accurate on trials following errors than they were on trials following correct responses (main effect of previous accuracy, F (1,21)=6.55, p<.025). Therefore, subjects were more likely to perform as they did on the last trial than to improve their performance following errors. The high depression group did not behave differently from controls in this regard.

Compensation and Error Signals

There have been conflicting findings on whether amplitude of various error signals correlates with the amount of behavioral compensation following an error. In this study there was a positive correlation between rERN amplitude after errors (at the Fz site and the Cz site) and amount of post-error slowing, defined as the difference in RT between the current trial and the previous error trial (Fz: r(24)= -.662, p=.001; Cz: r(24)= -.475, p<.02). Larger rERNs were thus associated with more post-error compensation. There was also a positive correlation between the average amplitude of the Pe at all three sites after errors and the average amount post-error slowing (Fz: r(24)= .513, p<.02; FCz: r(24)= .634, p=.001; Cz: r(24)= .474, p<.02). There was no correlation with the amplitude of the fERN. Therefore, greater rERN and Pe amplitudes were shown to
predict more behavioral compensation, thus validating the link between ERP signals and actual cognitive control.

It was expected that, since depressed subjects have been shown to have deficits in post-error compensation, that there might be less of a connection between the error signals and amount of post-error compensation— that is, that depressed subjects aren’t using information well to adjust to errors. However, when separated by groups, the correlations only remained in the depressed group for the rERN and Pe and not the control group (for dep group: rERN: Fz: r(10)= -.807, p=.005; Cz: r(10)= -.744, p<.02, for control group: p>.2 for every site) (for dep group: Pe: Fz: r(10) = .747, p<.02; FCz: r(10) = .853, p=.002; Cz: r(10)= .832, p=.003, for control group: r(14) = .15, .31, .82). This is the opposite of what was expected. Neither group showed correlations with the fERN and post-error compensation.

**PA and NA**

Given that group effects were not as predicted, the current researchers analyzed whether NA and PA were better predictors of error responsiveness and behavior. The PANAS provided a chance to describe and group the same subject sample in a different manner. It was expected that, based on Clark & Watson’s tripartite model of depression and anxiety, that both groups would have equally high NA, but the high depression group would have lower positive affect (PA). However, the opposite was true: an independent samples t-test was performed to test whether there was a significant difference in PA and NA between the high and low depression groups. The results revealed that the mean NA score was significantly higher in the depressed group than the control group (t(22)=...
-3.84, p<.01), but no differences were found between mean PA scores (t(22)= 1.28, p>.1) (see Table 1).

It was expected that NA scores would correlate positively with both BDI and PSWQ scores (which would be positively correlated with each other), and the PA scores would be negatively correlated with BDI scores. In the present subject sample, only the PA and NA scores were significantly correlated with BDI scores (PA: r(24)= -.447, p<.05, NA: r(24)= .627, p<.01). Therefore, as expected, higher BDI scores were associated with higher NA and lower PA scores. However, the BDI and the PSWQ were not correlated.

Past studies have shown correlations between NA and the rERN, while PA levels have not yet been studied in this way. In this study, Pe signal amplitude on correct trials was negatively correlated with NA (r(24)= -.481, p<.02). Therefore, the lower a subject’s NA, the greater their average Pe amplitude on correct trials. Pe amplitudes did not correlate with NA on incorrect trials or with PA on any trials. rERN amplitude on correct and incorrect trials was marginally negatively correlated with PA (corr: p(24)= .338, p=.106; inc: p(24)= .345, p=.099) but not NA (corr: p(24)= .121, p>.5; inc: p(24)= .096, p>.5). Therefore, subjects with lower PA showed somewhat higher rERN amplitudes on both correct and incorrect trials. fERN amplitude was not correlated with either NA or PA.

Since the groups did not differ on PA as was expected, and PA was found to be marginally correlated with rERN, the entire subject pool was divided by a median split into high PA and low PA groups to see if error signals would vary according to these groups. In a 3 x 2 x 2 mixed ANOVA of the effects of site, accuracy, and PA group on
rERN amplitude, there was a significant task by site by PA group interaction (F(2,42)=7.83, p=.001) as well as a task by site by PA group by accuracy interaction (F(2,42)=6.64, p<.01) (see Figure 6). When the correct and incorrect trials were compared, this task x site x PA group interaction was only significant for the incorrect trials and not the correct trials. The greatest rERN amplitudes were seen when the low PA group responded to errors on the emotional task. The lowest rERN amplitudes were seen when the high PA group responded to errors on the counting task. rERNs varied the most between the two groups at the Cz site for the emotional task and at the Fz site for the cognitive task (see Figure 6).

When looking at EEG sites together, there were no significant PA group effects or PA group interactions. However, at the Fz site, there was a marginal PA group effect (F(1,21)= 2.68, p=.116) and a marginal accuracy by task by PA group interaction (F(1,21)= 3.67, p=.069). Therefore, at the Fz site, on the cStroop, the rERN amplitudes of the high PA group increased more for incorrect trials vs. correct trials than the low PA group (see Figure 6). There were no significant effects of PA groups for fERN amplitude.
Figure 6. Graph showing rERN amplitude on error trials, separated by site, task, and PA group. Graphs show the task x site x PA group interaction and the task x site x PA group x accuracy interaction.
Discussion

The main objective of this study was to compare the rERN, fERN and Pe error signals, as well as post-error behavioral adjustment, of a high and low depression group. The goal was to try to relate these findings to past studies of the rACC and dACC in depressed patients and studies of error signals in other different patient groups. It was expected that the high depression group would show greater amplitudes of the more emotional signals (fERN and Pe), lower amplitudes of the cognitive signal (rERN), and less post-error behavioral adjustment than controls.

rERN

As expected, all subjects showed a more negative peak at the three scalp sites after error trials than after correct trials. This validates the current methodology used to elicit, record, and quantify the rERN. The high and low depression groups did not show differences in rERN amplitude on errors or across all trials. In other words, the personality variables tapped by the BDI did not affect rERN amplitude. This was an interesting finding, since many researchers have found an increased rERN in various anxiety disorders (Hajcak et al, 2003; Hajcak & Simons, 2002; Gehring et al, 2000; Fitzgerald et al, 2005). This suggests that these depressed subjects did not show the same hyperactive conflict monitoring as anxious subjects have shown. This lack of difference has also been seen by Ruchsow et al (2004), who found that depressed subjects without anxiety did not show significantly different rERN amplitudes than controls on a continuous feedback task.
However, trends in this data suggest that depression is related to greater rERN amplitude. In the present study PA was marginally negatively correlated with rERN amplitude on all trials, suggesting that subjects with low PA (usually common in depressed individuals) showed a somewhat greater rERN. Therefore, in this study, subjects with low PA showed more similar error responsiveness to anxious subjects in past studies than those subjects that were high in BDI, suggesting that PA might be more related to error processing than symptoms measured by the BDI.

Groups in this study were matched according to PSWQ scores, intending to control for general level of anxiety. However, the groups differed unexpectedly on NA, which is usually high in anxiety disorders as well as depression. In the current subjects, the high depression group showed significantly greater NA scores. Since high NA has been associated with a higher rERN (Luu et al, 2000; Hajcak et al, 2004), it is surprising that no increase in rERN was seen in the high depression group in the present study. It is possible that, while PA was equally high in both groups, it was the differences in BDI scores that counteracted this effect of NA (the high NA group had high BDI scores and studies of depressed patients not excluding anxiety have found no difference in the rERN (Tucker et al, 2003).

It was expected that the depressed subjects would show a different pattern of error responsiveness than anxious subjects in past studies. In fact, since the rERN is thought to be more of a cognitive or vigilance signal, and since it originates in the dACC and possibly the PFC, it was expected that depressed subjects would show smaller rERN amplitudes than controls. This was also not true, showing that according to rERN, the depressed subjects in the current study were just as cognitively responsive to their errors
as controls. This also suggests that the high depression group did not differ in dACC reactivity after errors. It is possible that the high depression group showed a lower baseline level of activity in this region, but were just as reactive to errors as controls. It is also possible that this lack of group effect was due to the fact that the depressed group was not clinically depressed and that all subjects were high-functioning college students. Therefore, it is unlikely that any subjects had severe cognitive deficits.

When subjects were divided into high PA and low PA groups, there was only a marginal effect of group (discussed earlier) but there was an interaction which showed that on the cStroop, the high PA group had less of an increase in rERN amplitude for incorrect trials (relative to correct trials) than the low PA group. Therefore, the group with low PA (which is normally seen in depression) showed a bigger increase in rERN amplitude than those with a high PA on a cognitive task. This is the opposite of what was expected, since it has been found that depressed subjects do not show the normal increase in dACC activity with increasing difficulty of a task (Elliott et al, 1997). This suggests that subjects with low PA are actually more reactive to errors on a cognitive task than subjects with high PA. In this way, low PA subjects were more similar to anxious subjects in past studies. While this result was not expected, it does agree with the correlational finding that low PA was associated with greater rERNs.

Besides group differences, there were several interesting effects of task on the rERN. All subjects showed higher rERN peaks on the ecStroop than the cStroop. Also, accuracy affected the rERN marginally more on the emotional task than the cognitive task. Upon further analysis, the incorrect trials appeared to be causing this interaction, since these trials differed more between tasks than did the correct trials. (Incorrect trials
on the ecStroop had higher rERN amplitudes than those on the cStroop.) These task differences were not expected since the rERN has been localized to the dACC and thus is thought to be more of a cognitive signal. This finding is also surprising since the cStroop was the more difficult task, and in normals, the dACC has been shown to be more active during more difficult tasks (Kiehl et al, 2000; Carter et al, 1998; Fellows et al, 2005).

There are several possible reasons for these unexpected results. For one, since ERP signals were not localized to specific brain regions, it is possible that the rERN activity was originating in a different area than the dACC, or multiple different areas. This could be likely since larger rERNS were not seen on the more difficult task, the cStroop, suggesting that the signal was not sensitive to greater cognitive interference, as the rERN has been shown to be in the past. However, the rERN might have been signaling motivational differences, which have been found to influence the signal in the past (Gehring et al, 1993). Since focusing on accuracy has been shown to increase the size of the rERN, it is possible that the emotional words (such as “fail”) caused all subjects to focus more on their accuracy during this task relative to the counting task. It is interesting that, while there was the biggest effect of trial type on the cStroop (for reaction time and accuracy), there was not a bigger difference in rERN amplitude for trials on this task. Thus, the cognitive interference indicated by slowed RT and reduced accuracy on interference trials was different than the error monitoring measured by the rERN. It is also interesting that the rERN amplitudes on neutral trials of the ecStroop were also elevated compared to the neutral trials of the cStroop, meaning that the emotional task caused more error monitoring activity during both the neutral and interference trials.
Because an rERN-type signal has been found after correct, high conflict trials, average waveforms for all neutral and interference trials were visually analyzed, regardless of accuracy. These waveforms were not significantly different—showing that in this study the rERN appeared to reflect error monitoring as opposed to conflict monitoring.

fERN

Bigger fERN amplitudes were not seen on incorrect trials compared to correct trials in either group or task. Therefore, whatever accuracy effects were seen in the rERN were not present by the time the fERN peaks emerged. In fact, upon further analysis, it was found that the fERN and rERN amplitudes were not correlated for all subjects, showing that the difference in processing for incorrect vs. correct trials seen in the rERN did not continue in this later time window after feedback. This suggests that the subjects were already aware of their errors and thus the feedback did not elicit additional error processing. In other words, subjects were not relying on feedback for their performance on this task, and so they did not process the feedback differently if it indicated correct or incorrect performance—that cognitive processing had already occurred when they realized independently that they had made an error. Regardless of whether the signal was greater for errors than correct trials or subjects were using the feedback to alter their performance, it is interesting that the high depression group still did not show more of a reaction to negative feedback than controls, considering this group’s sensitivity to criticism and to negative events. This lack of group effect suggests that the groups showed equal error awareness. Therefore, it seems that when feedback is redundant and
not crucial for performance monitoring, it is not reacted to abnormally by depressed subjects—that it might only be meaningful feedback that is responded to differently.

Very few other studies have examined the fERN on a continuous feedback task, and so it is possible that this signal is less sensitive to errors (or not at all) when feedback is continuous. In fact, it has been shown that in tasks with rules that are easy to learn, there are smaller fERNs, or sometimes none, compared to tasks that are more trial-and-error where subjects rely more heavily on feedback (Nieuwenhuis et al, 2005; Holroyd et al, 2004). Therefore, while using continuous feedback controlled for error awareness, it was not ideal for eliciting an error-sensitive fERN.

There was also no main effect of group or task, or any significant interactions with either. Other studies that have found an error-sensitive fERN have also not found group effects. Ruchsow et al (2004) used continuous feedback and did not see differences in depressed, non-anxious subjects compared to controls. Past studies have also shown that subjects with NA scores of higher than 20 have attenuated fERNs. Thirteen out of 24 of the current subjects had NA scores above 20, indicating that the high NA levels in the current subject group might have reduced the fERN signals for all subjects, and potentially diminished the group effect since the high depression group had higher NA levels. The subjects in Tucker et al’s 2003 study with BDIs similar to the current high depression group showed small or moderate fERNs. The authors attributed this lack of group effect in more severe depression as blunted emotional sensitivity to feedback (which is associated with the apathy and anhedonia seen in these patients).

Therefore, the task used in this study was not effective in eliciting an error-specific fERN. There were also no group differences found in the signal size. Therefore,
additional research is needed to see if group differences are indeed seen for the fERN on
tasks that do elicit this signal effectively.

**Pe**

The Pe did show the expected difference between correct and incorrect trials that
has been shown in past studies (Falkenstein, 2000; Nieuwenhuis, 2001). That is, Pe
amplitudes were greater following errors than correct trials—again reaffirming the
methods used to elicit and measure the signal. This is the first study of the Pe in
depression, and thus the first finding that this signal differed at the Fz site between a
depressed group and a control group. However, the difference was in the opposite
direction from what was expected—that is, the high depression group showed a lower Pe
at this site than controls. If the Pe does reflect affective processing of errors like past
studies have shown, and the signal is localized to the rACC (van Veen & Carter, 2002;
Falkenstein et al, 2000; Kiehl et al, 2000), then the high depression group should show
higher Pe signals since they should be more emotionally reactive to errors and have been
found to have hyperactive rACCs. There is no unified theory of what the Pe reflects. For
instance, some researchers claim that it reflects cognitive processes like conscious error
processing or updating of error context (Nieuwenhuis et al, 2001; Leuthold & Sommer,
1999; Fallgatter, 2004). Since there were no differences in overall performance between
groups, the Pe did not appear to reflect any performance-related cognitive processes.

Both the rACC and the Pe have been implicated with affective processing, but
these group differences suggest that either this high depression group showed less
affective processing of errors, or that this signal reflects a different kind of cognitive
processing. Since the Fz site was the closest to the rACC, these findings support the past
localizations of the Pe to the rACC, and suggest that the Pe is in fact involved in affective processing/distress (see Appendix D). Therefore, if the Pe measured was originating in the rACC, these depressed subjects showed less activity here and than controls. If the Pe does reflect affective processing, it appears that the high depression group showed a lower affective response to their errors than controls. It is possible that this is due to the blunted affective processing reported in more severely depressed individuals, which has been associated with anhedonia and apathy (Tucker et al, 2003). Although there were no differences in overall performance, it is possible that the depressed group in this study was less motivated by positive feedback, and thus were less reactive than controls when they did not receive it. These unexpected findings could perhaps be better explained by looking at task effects to see if the Pe was stronger in response to emotional stimuli.

Since there was no main effect of task, the presence of emotional words did not affect the Pe amplitude and did not affect accuracy’s effect on Pe amplitude. Moreover, for all subjects the Pe was marginally larger during the cStroop than the ecStroop at the Fz site. Since these tasks varied significantly by difficulty (increased RT and decreased accuracy), it is not clear whether this marginal effect was due to increased difficulty or the different word meaning on the cStroop. One explanation is that the cStroop caused more affective processing because it was more difficult. That is, difficulty might have had more of an effect on Pe size than affective content.

While there was no way to localize the ERP signals to specific brain regions, the three scalp sites used can be used to compare relative distance from the rACC and dACC. The most frontal site is the closest to both the rACC and dACC. The finding that there was a bigger group difference at the Fz site for the Pe might be due to this site’s closer
proximity to the rACC, the suspected source of the Pe. However, this would mean that the depressed group had a less reactive rACC than the control group. If true, this would differentiate the depressed group from past anxiety groups studied, since OCD patients have shown more reactive rACCs than controls (Nieuwenhuis et al, 2005). There was less of a difference between Pe amplitude for correct and incorrect trials at the Fz site, which goes against past findings that the rACC activates selectively after errors, and could suggest that Pe activity was coming from additional areas.

Since the high depression group showed higher NA, the presence of lower Pe signals in this group supports the past finding that subjects with higher NA have lower Pe signals (Hajcak et al, 2003). If the signal from the rACC is related to treatment response as some researchers have postulated (somehow calling for further processing in response to conflict, or a “will to change” when something is not going well) then differences in this signal could be related to coping differences (Davidson et al, 2002). Therefore, instead of less distress in response to errors, depressed subjects are showing less of a “will to change” than the control group, and either their affective distress is not different or is not accurately reflected by this signal. However, both groups performed equally well on the task, and so this weaker signal was not sufficient enough to worsen the depressed group’s performance on the task. This might relate to the fact that all subjects were high-functioning with no apparent cognitive deficits, and so it is possible that other brain areas responsible for cognitive control (the PFC, etc.) were functional enough that this difference in input from the ACC did not have a detrimental effect on performance.

It was expected that the depressed group’s Pe amplitudes would have differed more between the emotional and cognitive tasks than the control group, because they
should be more reactive to emotional stimuli. However, this task x group interaction was not seen. One possible reason for this is that the ecStroop had a normalizing effect on the two groups rather than an exaggerating effect—such that the negative emotional words put the control group in a depressive mindset, and theoretically activated their rACC more than baseline, which could have also increased their error reactivity as measured by the Pe. It is possible that this task did not have such a strong effect in the depressed group because it was not as different from their normal mindset.

This preliminary study of the Pe in depression shows interesting effects of group and task that could possibly lead to different interpretations of the signal’s meaning and could provide a new perspective on error responsiveness in depression. More studies need to be done to replicate the group differences found for this signal, since it was in the opposite direction as was expected.

**Behavioral Data**

**RT and Accuracy**

There was no difference in overall RT or accuracy between groups, as expected. However, all subjects showed slower RTs on the cStroop than the ecStroop, showing that the trials in this task provided more interference. There was a task by trial type effect for RT and accuracy such that interference trials were more difficult for all of the subjects and trial type made a bigger difference in the cStroop than the ecStroop—in other words the interference trials on the cStroop were the most difficult type of trial.

While there were no overall group differences, there were interesting differences between the groups related to task. While all subjects showed a high level of interference on the cStroop, the emotional interference caused by the ecStroop was closer to this level
in the depressed group than the control. The depressed group was also slower for emotional words than neutral words on the ecStroop but the control group was not. (The control group was marginally faster for emotional trials than the high depression group, showing that the emotional interference was seen more in the depressed group, which was expected.) Therefore, the emotional words were harder for the high depression group to ignore, but they did not affect the RT of the/not for the control group. This interaction was not present in the accuracy data, meaning that while the depressed group slowed down more on these trials, they did not make more mistakes. Therefore, the depressed group was not slower or less accurate on the ecStroop than the control group, but their interference effect (as seen in RT differences) was greater during the ecStroop than the control group, relative to the cStroop.

As opposed to some past studies, subjects in this study did not show a slowing of RT as the task went on, showing that the current subjects did not disengage from the task in this fashion. Hence, the overall difference in motivation and attention (as measured by RT and accuracy) stayed constant for both groups across the testing session. In fact, all subjects showed decreasing reaction times across the session—which has been seen in previous studies using a similar cognitive task (Bush et al, 2000?). These authors contributed this effect to learning, so that the interference effect diminished with time.

Therefore, while error signals did not show the expected group or group x task effects, there were several differences in interference seen through RT differences. This suggests that in such a high functioning sample, RT might be a more sensitive measure of cognitive interference. Interestingly, it has been shown that as the interference effect on
RT diminished, the difference in dACC activity also decreases, showing that RT can reflect important functional changes, and warrants future research (Bush et al, 1998).

**Post-Error Compensation**

Subjects did show the characteristic slowing of RT following errors, but they did not show increased accuracy on the post-error trials. In fact, subjects were actually more likely to make an error having just made one on the previous trial. It is possible that for all subjects they responded well to positive feedback but the negative feedback somehow caused them to perform more poorly. While Elliott et al (1997) have found this tendency to fail having just failed in a clinically depressed group, it is not clear why this would be true in both the depressed and control groups in the current study. One explanation is that the speed of this task made it so that it was easy to be distracted by an error just committed, causing subjects to perform worse on the next trial. In a sense, the speed of the stimuli presentation might have made it easier to be distracted by an error on a subsequent trial—thus making the control subjects more similar to depressed patients in past studies. In other words, perhaps all subjects in this study were more likely to show the “catastrophic” response to failure that has been seen in depressed patients.

It was expected that the high depression group would show deficient post-error compensation. Surprisingly, post-error behavior did not vary between groups. Therefore, both groups reacted just as well behaviorally to their errors, and thus did not appear to differ in rumination or coping. This suggests that the ACC/PFC circuit and the PFC itself were not functioning any differently in the high depression group. Two past studies also failed to find a difference in post-error slowing in depression (Tucker et al, 2003; Ruchsow et al, 2004). While the lack of group differences found in the current
study might be due to the high functioning subject sample, the fact that these two past studies also did not show differences suggests that not all depressed patients show deficits in this post-error slowing. More studies need to be conducted in order to investigate if this behavioral deficit is only seen in patients with more severe cognitive deficits.

**Compensation and Error Signals**

Past studies have shown mixed results for the correlation of error signals with post-error behavior. Luu et al (2003) found that there was a correlation of fERN strength on “C” feedback with RT on next trial (and not with “A” or “C” feedback, and feedback was delayed). Falkenstein et al showed that Pe amplitude was not correlated with post-error slowing. Hajcak et al (2003) found that while rERN strength was not correlated with post-error slowing, Pe strength was.

In the present study, both rERN and Pe amplitudes were found to correlate positively with post-error slowing, showing that the underlying error-monitoring processes reflected by these signals (activity in the ACC) were somehow linked with behavioral adjustment. (No correlations were found with fERN amplitude.) While it was expected that this link would be less efficient in the depressed group, when subject groups were analyzed separately, the correlations disappeared for the control group but remained for the depressed group. Therefore, it appears that there was a stronger connection between error responsiveness and post-error adjustment for the depressed group than for controls—so that, while depressed subjects were not better at adjusting behavior overall, their rERN and Pe amplitudes were better predictors of post-error adjustment. This conflicts with past studies that have found that depressed subjects to be
worse than controls at post-error adjustment. Since controls compensated just as well as the depressed group after errors, this suggests that different processes might be driving post-error compensation, or they might show a more constant level of brain activity associated with compensation, relying less on reactivity after errors.

The fact that the rERN and Pe amplitudes correlated with post-error behavioral adjustment shows that these signals, and the ACC activity they measure, are directly related to how subjects cope with errors. Therefore abnormal activity in the ACC is likely to be directly related to an individual’s behavioral compensation after errors. More studies are needed to determine the exact mechanism of this effect.

**PA/NA**

Past studies have found higher rERN amplitudes and lower Pe amplitudes in subjects with high NA, but no studies have found effects of PA on these error signals. Thus, the current study was the first to find an effect of PA on the ERN. In the current study, it was found that subjects with high PA had less of rERN amplitude increase on incorrect trials on the cognitive task than the low PA group. No differences were found between the high and low PA groups on the fERN. Pe signals on correct trials were negatively correlated with NA but not PA. The Pe therefore appears to be more influenced by NA and the rERN by the PA. Based on these findings, and according to the tripartite model, depressed subjects should show a low Pe (on correct trials) and a greater increase for the rERN on incorrect trials. Since depressed subjects in this study did in fact show a lower Pe than controls (although no greater rERNs) the NA might be a useful measure in determining or comparing Pe amplitudes.
Hajcak et al predicted that the rERN was specific to anxiety, and so large amplitudes should only be seen in subjects with high NA and moderate to high PA. The high depression group in this study had both of these elevated levels but did not show enhanced rERNs, suggesting that more qualifiers are needed to show an enhanced rERN—such as higher anxiety-specific scores or a clinical diagnosis of anxiety.

Since there were interesting differences revealed when subjects were compared based on the PA and NA, perhaps moreso than groups based on BDI and PSWQ scores, this supports the comparison of subjects based on these variables, as well as the collection of multiple personality variables in order to pinpoint the set of symptoms which most directly relate to these error signals. These differences based on PA suggests that general level of motivation, arousal, and hopefulness might be more related to these signals than the various symptoms measured by the BDI. More studies of high-functioning college students are needed to replicate these findings.

**Limitations and Future Directions**

While the current study used a non-clinical sample, the subjects’ scores on the BDI are considered moderate to severe according to Beck & Steer (1988). Past findings in other populations have suggested that moderately depressed subjects (as measured by the BDI) show an increased fERN relative to controls than severe or low depressed groups (Tucker et al, 2003). Therefore, the lack of group effects seen in this study could be due to the fact that so many of the subjects in the depressed group were considered moderately to severely depressed (six subjects) or severely depressed (five). Future studies with larger subject samples (and thus a larger range of BDI scores) would be useful in determining the more specific effects of this variable on ERN group differences.
One potential problem relating to personality variables is that pre-screening (including completion of the BDI and PSWQ) was completed weeks earlier than testing, and during a finals period where the subjects’ stress levels were likely to be higher than normal. However, BDI scores have been shown to be very stable: in undergrads, stability has been shown to range from .6 (testing 4 months apart) to .9 (2 weeks apart) (Beck & Steer, 1988). Also, the fact that PA and NA levels (which were measured at testing) were highly correlated with BDI scores shows that there was stability across the pre-screening and testing sessions.

The ecStroop and the cStroop devised by Bush, Whalen, and colleagues have been shown to activate the rACC and dACC differentially in healthy subjects (Bush et al, 1998; Whalen et al, 1998). In this study, however, alterations were made to the tasks in order to increase the difficulty of the task and to target the ecStroop specifically to depressed subjects. These changes included using different words for the ecStroop, a different response method for both tasks, including “X” strings in the stimuli, changing the timing of the presentation, and randomizing the neutral and interference trials instead of alternating by trial block. Therefore, there is no direct evidence that the altered tasks used in this study activate the dACC and rACC as effectively as the original tasks. Future imaging studies would be needed to determine this.

Another issue with these tasks is that, while there has been much research on the general levels of activity in the rACC and dACC in depression, there has not been much research done on the reactivity of these regions, or the effect of activating these regions in controls. Therefore, even though some depressed patients have been shown to have greater baseline levels of rACC activity than controls, they may not show more reactive
rACCs than controls in response to errors. Additionally, it is not clear how this greater baseline activity would be affected by a task activating this area. In other words, higher reactivity does not necessarily follow from higher baseline levels of activity, or vice a versa. Evidence for this is that the Pe was not greater during the ecStroop than the cStroop, possibly suggesting that activating the rACC of all subjects does not increase the reactivity of this subregion to errors for all subjects. It is still not clear whether this selective activation by task accentuated the differences between the groups or diminished them.

In future studies, it would be ideal to either use more sites for EEG recording, or use concurrent brain imaging, to localize the error signals to specific areas of the brain, and to specific subregions of the ACC. This is a key missing link between the imaging and ERP research done on the ACC and its role in depression. It would also be interesting to use measures of ACC activity or error signals to track progress or prognosis for depressed subjects. For instance, to see whether, like with OCD, successful Cognitive Behavioral Therapy decreases rACC activity in depressed subjects (Fitzgerald et al, 2005). It would also be interesting to see how rACC and dACC activity affect each other.

Depressed patients who respond to treatment have been shown to have greater pre-treatment rACC reactivity, and researchers have compared error activity here to this treatment response (Davidson et al, 2002). If the Pe is a signal that enlists further processing in the face of conflict, then depressed subjects in this study showed a weaker signal of this type. This would fit with the idea of poorer adjustment to errors and worse coping with negative events. However this difference did not manifest as deficits in post-
error adjustment. Perhaps in this high functioning group, brain regions more devoted to
cognitive control, like the PFC, are normally functional and therefore do not rely on a
normal triggering signal from the ACC to compensate after errors. However, in more
severely depressed individuals, where there might be further PFC damage or
hypoactivity, this entire circuit is lower functioning and the appropriate further
processing is not performed.

Distress in response to pain or errors is an important signal from the brain that
behavior needs to be changed. In order to react appropriately to the event and recruit the
appropriate cognitive control processes needed to improve the situation, this distress
signal needs to be great enough to be motivational and to lead to avoidance of the specific
stimuli in the future, but not great enough so that it is crippling and causes ruminations
and detriments in future behavior. It is clear from both brain imaging and some ERP
studies, as well as anecdotal evidence, that depressed patients show abnormal response to
both pain and errors. Once it is clear exactly how this response varies from controls, then
it might be possible to more effectively treat this problematic symptom, and possible
cause, of depression, and possibly to track the effectiveness of treatment. More
generally, emotions motivate us to behave in certain ways, and if we can better
understand the mechanism of this motivation to change, we might be better able to
understand coping in the face of negative events, and possibly help depressed patients
and others to do so more effectively.
Acknowledgements

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References


Appendix A
Web Questionnaire: PSWQ and BDI

The purpose of the questionnaire below is to help determine if you are eligible for a senior thesis study that will be conducted in the spring semester by Min Lin ’06 and Gray Vargas ’06, supervised by Prof. Rebecca Compton in the Haverford Psychology Department. If you complete the questionnaire below, which should take only about 5-10 minutes, we’ll enter your name into a lottery drawing for two $50 cash prizes. As long as you complete this questionnaire, your name will be entered into the lottery, even if your responses on this questionnaire make you ineligible for participating in the full study later on.

If your responses indicate that you are eligible for the full study, we will contact you within a few weeks to tell you more about what the full study involves, and to see if you might be interested in participating. If you complete this questionnaire, it does not mean that you are committing yourself to completing the full study; you can make that decision later if it turns out that you’ve met the eligibility requirements. The ultimate purpose of the full study is to better understand how individual differences in personality affect the brain’s response to different types of visual images.

Many of the questions on this questionnaire are concerned with understanding the range of individual differences in anxiety and depressive mood tendencies. We would appreciate it if you would answer the questions below as honestly as possible, but you may leave questions blank if you are uncomfortable answering them. If you choose to leave a question blank, it will not affect your entry into the lottery for the cash prizes.

For the duration of the study (about 6 months), your responses will be stored on a password-protected computer server that is accessible only to the investigator, Prof. Compton. Prof. Compton will be able to view all of your responses, including your name, though she will keep these responses completely confidential. The student investigators (Min Lin and Gray Vargas) will only have access to the questionnaire responses after the names have been removed. At the end of the study, your name will be removed from the permanent record so that nobody, not even the investigators, would be able to tell which person gave which questionnaire responses. Your responses will be kept confidential at all times and will not be shared with any outside parties. If you have any questions about the study, please contact Prof. Compton at 610-896-1309 or rcompton@haverford.edu. If you have questions or concerns about your rights as a research participant, you may also contact Prof. Rob Scarrow (610-896-1218, rscarroll@haverford.edu); Prof. Scarrow is chair of the Haverford College IRB, which oversees the protection of research participants.

Please check the box below to indicate that you have read the above instructions and that you voluntarily consent to have your responses below included in the dataset for this study.

______
Thank you for filling out our questionnaire!

Gender
_____ male  ______ female

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

* I have abnormal vision that is not corrected by glasses or contact lenses (e.g., color blindness, glaucoma, etc.)
* I have a history of neurological problems, such as epilepsy (seizures), head injury, stroke, brain tumor, multiple sclerosis, etc.
* I regularly take medication that is known to affect the central nervous system.
* I regularly consume non-medical substances that are known to affect the central nervous system (e.g., substances such as marijuana, cocaine, heroin, ecstasy, etc.; do not include moderate use of alcohol, caffeine, or cigarettes).
* I have a learning disability.

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

_____ No, none of the above statements describes me.

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

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

_________________________________________________________________

Self-Evaluation Questionnaire—Part 1

Please indicate the number from the scale below that best describes how typical or characteristic each of the 16 items is of you.

<table>
<thead>
<tr>
<th>Not at all typical</th>
<th>Somewhat typical</th>
<th>Very typical</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
</tbody>
</table>
____  1. If I don't have enough time to do everything, I don't worry about it.
____  2. My worries overwhelm me.
____  3. I don't tend to worry about things.
____  4. Many situations make me worry.
____  5. I know I shouldn't worry about things, but I just can't help it.
____  6. When I am under pressure, I worry a lot.
____  7. I am always worrying about something.
____  8. I find it easy to dismiss worrisome thoughts.
____  9. As soon as I finish one task, I start to worry about everything else I have to do.
____ 10. I never worry about anything.
____ 11. When there is nothing more I can do about a concern, I don't worry about it anymore.
____ 12. I've been a worrier all my life.
____ 13. I notice that I have been worrying about things.
____ 14. Once I start worrying, I can't stop.
____ 15. I worry all the time.
____ 16. I worry about projects until they are done.

Self-evaluation questionnaire- Part 2

Please read each group of statements carefully. Then pick out the one statement in each group that best describes the way that you have been feeling the past week, including today. Be sure to read each statement in each group before making your choices. [each group was accompanied by radio buttons on the web form]

I do not feel sad.
I feel sad.
I am sad all the time and I can’t snap out of it.
I am so sad or unhappy that I can’t stand it.

I am not particularly discouraged about the future.
I feel discouraged about the future.
I have nothing to look forward to.
I feel that the future is hopeless and that things cannot improve.

I do not feel like a failure.
I feel I have failed more than the average person.
As I look back on my life, all I see are a lot of failures.
I feel I am a complete failure as a person.

I get as much satisfaction out of things as I used to.
I don’t enjoy things the way I used to.
I don’t get any real satisfaction out of anything anymore.
I am dissatisfied or bored with everything.

I don’t feel particularly guilty.
I feel guilty a good part of the time.
I feel guilty most of the time.
I feel guilty all of the time.

I don’t feel I am being punished.
I feel I may be punished.
I expect to be punished.
I feel I am being punished.

I don’t feel disappointed in myself.
I am disappointed in myself.
I am disgusted with myself.
I hate myself.

I don’t feel I am any worse than anybody else.
I am critical of myself for my weaknesses or mistakes.
I blame myself all the time for my faults.
I blame myself for everything bad that happens.

I don’t have any thoughts of killing myself.
I have thoughts of killing myself but I would not carry them out.
I would like to kill myself.
I would kill myself if I had the chance.

I don’t cry any more than usual.
I cry more now than I used to.
I cry all the time now.
I used to be able to cry, but now I can’t cry even though I want to.
I am no more irritated now than I ever am.
I get annoyed or irritated more easily than I used to.
I feel irritated all the time now.
I don’t get irritated at all by the things that used to irritate me.

I have not lost interest in other people.
I am less interested in other people than I used to be.
I have lost most of my interest in other people.
I have lost all of my interest in other people.

I make decisions about as well as I ever could.
I put off making decisions more than I used to.
I have greater difficulty in making decisions than before.
I can’t make decisions at all anymore.

I don’t feel I look any worse than I used to.
I am worried that I am looking old or unattractive.
I feel that there are permanent changes in my appearance that make me look unattractive.
I believe that I look ugly.

I can work about as well as before.
It takes extra effort to get started at doing something.
I have to push myself very hard to do anything.
I can’t do any work at all.

I can sleep as well as usual.
I don’t sleep as well as I used to.
I wake up one to two hours earlier than usual and find it hard to get back to sleep.
I wake several hours earlier than I used to and cannot fall back to sleep.

I don’t get more tired than usual.
I get tired more easily than I used to.
I get tired from doing almost anything.
I am too tired to do anything.

My appetite is not worse than usual.
My appetite is not as good as it used to be.
My appetite is much worse now.
I have no appetite at all anymore.

I haven’t lost much weight, if any, lately.
I haven’t lost more than 5 pounds.
I have lost more than 10 pounds.
I have lots more than 15 pounds.
I am no more worried about my health than usual.  
I am worried about physical problems such as aches and pains, or upset stomach, or constipation.  
I am very worried about physical problems and it's hard to think of much else.  
I am so worried about my physical problems that I cannot think of anything else.

I have not noticed any recent changes in my interest in sex.  
I am less interested in sex than I used to be.  
I am much less interested in sex now.  
I have lost interest in sex completely.

Thank you very much for completing this survey, which will help us to identify possible participants for our main study in the spring semester.

Many of the questions on this survey were related to depressive tendencies. If you are interested in learning more about depression, please see the NIH website at: http://www.nimh.nih.gov/healthinformation/depressionmenu.cfm

If any of the questions caused you concern about your own well-being, you are encouraged to contact the college’s free Counseling and Psychological Services center, 610-896-1290; http://www.haverford.edu/caps/

In order for your name to be entered into the lottery for the cash prizes, you must enter your name and e-mail address in the boxes below. We will also use this information to contact you if your responses indicate that you are eligible for our main study in the spring.

Name ______________

E-mail ______________

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

[Submit button]
# Appendix B

## Sample Task Stimuli

### cStroop

<table>
<thead>
<tr>
<th>Neutral trial</th>
<th>Interference trial</th>
</tr>
</thead>
<tbody>
<tr>
<td>dog</td>
<td>two</td>
</tr>
<tr>
<td>dog</td>
<td>two</td>
</tr>
<tr>
<td>dog</td>
<td>two</td>
</tr>
</tbody>
</table>

### ecStroop

<table>
<thead>
<tr>
<th>Neutral trial</th>
<th>Interference trial</th>
</tr>
</thead>
<tbody>
<tr>
<td>fork</td>
<td>fail</td>
</tr>
<tr>
<td>fork</td>
<td>fail</td>
</tr>
<tr>
<td>fork</td>
<td>fail</td>
</tr>
</tbody>
</table>
Appendix C
PANAS and Family History

Brief Questionnaire
Haverford College 2006

Subject number:  

This questionnaire is designed to assess people's moods and emotions. It contains a number of words that are often used to describe moods, and you will be asked to indicate how much each word describes how you have felt THIS PAST WEEK. All the data collected in this questionnaire are anonymous, and no identifying information (e.g. your name or student ID) will be obtained or stored.

☐ Please check this box to indicate that you have read the above instructions and that you voluntarily consent to have your responses below included in the dataset for this study.

Gender: Male ☐ Female ☐

Please read each item and then select the appropriate answer in the pull-down menu next to the word. Indicate to what extent you have felt this way in the PAST WEEK.

[Choices were: Very Slightly/Not at all, A Little, Moderately, Quite a Bit, and Extremely]

ATTENTIVE

STRONG

IRRITABLE

INSPIRED

AFRAID

ALERT

UPSET

ACTIVE
GUILTY
NERVOUS
EXCITED
HOSTILE
PROUD
JITTERY
ASHAMED
SCARED
ENTHUSIASTIC
DISTRESSED
DETERMINED
INTERESTED

If you do not feel comfortable answering any of the following questions, please leave them blank.

Has anyone in your immediate family ever been diagnosed with:

- Depression? yes no
- Bipolar Disorder? yes no
- Anxiety? yes no
- ADD? yes no

When you have answered all of the questions please click on the submit button below so that your data can be recorded.

Submit  Reset
Appendix D

Approximate placement of the EEG scalp electrodes relative to the dACC and rACC.