Neurocognitive Factors Related to Trauma Symptoms and Emotion Regulation

Marie Rodriguez
University of North Carolina Wilmington
Faculty Mentor: Kate Nooner
University of North Carolina Wilmington

ABSTRACT
Previous research has found that implicit or automatic emotion regulation (AER) is a less studied aspect of emotion regulation, and that it may also be useful for coping with negative emotions following traumatic experiences. Alterations in brain function and trauma symptoms have both been linked to deficits in emotion regulation; however, they have not been studied together with AER. We hypothesized that brain changes related to poorer AER would be more strongly associated with negative facial emotions than with positive ones. In addition, we hypothesized that higher trauma symptoms would also be associated with brain changes for positive and negative faces but not neutral. To test these hypotheses, the current study examined AER and trauma symptoms in twenty-three college students while measuring event-related potentials (ERP) with electroencephalography (EEG) during a cued implicit response inhibition task. Results indicated no differences in brain function as measured with ERP latencies at the two electrode sites for the positive, negative, and neutral faces. However, higher trauma symptoms were significantly related to shorter ERP latencies for neutral and negative faces but not for positive. Examining the ways in which trauma symptoms are related to implicit and explicit emotion regulation as well as brain function may help researchers develop novel interventions aimed at improving outcomes for individuals who have trauma symptoms.

Emotion regulation is the process of responding to life events and experiences with a range of emotions that are socially and individually appropriate to the situation. Effective emotion regulation is flexible and facilitates modifying and delaying emotional reactions according to the situation (Zaki & Williams, 2013). In traumatized individuals, particularly those with symptoms of post-traumatic stress disorder (PTSD), researchers have found that regulating emotions, especially negative ones, can be difficult (Etkin, Prater, Hoeft, Menon, & Schatzberg, 2010; Shepherd & Wild, 2014). Given the need for emotion regulation in everyday life, an aspect of emotion regulation that is emerging in research is automatic emotion regulation (AER), which is the implicit process of regulating emotions without consciously exerting any effort to do so (Mauss, Cook, & Gross, 2007; Zaki & Williams, 2013). Though most of the current research has focused on explicit emotion regulation, the current study is one of the first to examine implicit emotion regulation. Previous research has found that AER is necessary for individual wellbeing and can be useful in reducing the undesired or problematic impact of negative emotions, such as anger, in a more immediate and fluid manner than explicit emotion regulation (Mauss et al. 2007; Mauss, Bunge, & Gross, 2007). Previous electroencephalography (EEG) studies of brain function have found that
individuals with PTSD have difficulty regulating and reducing negative emotions and show marked deficits in AER (Felmingham, Bryant, Kendall, & Gordon, 2002; Shepherd & Wild, 2014). Learning more about both explicit and implicit emotion regulation should help individuals with trauma symptoms have more options for recovery.

**Post-traumatic stress disorder (PTSD).** PTSD is a widespread mental health diagnosis that can occur after experiencing a traumatic event. According to the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-V), yearly prevalence of PTSD among U.S. adults is about 3.5%. Depending on the severity of the disorder, individuals with PTSD may experience a range of symptoms. These include intrusion symptoms such as recurring distressing dreams, avoidance of anything surrounding the traumatic event, negative changes in thoughts and mood associated with the event, and variations in arousal and reactivity associated with the event (American Psychiatric Association, 2013). The directly experienced traumatic events in Criterion A of the DSM-V include, but are not limited to, exposure to war as a combatant or civilian, threatened or actual physical assault, threatened or actual sexual violence, being kidnapped, being taken hostage, terrorist attack, torture, incarceration as a prisoner of war, natural or human-made disasters, and severe motor vehicle accidents (American Psychiatric Association, 2013).

**PTSD and emotion regulation.** For emotion regulation to be effective, it should help shape emotional reactions to fit the needs of the situation. Some commonly used emotion regulation strategies are reappraisal, distraction, expressive suppression, and distancing (Zaki & Williams, 2013). Interestingly, research has found evidence that emotion regulation processes are dysfunctional in individuals with PTSD. Both Cisler and Olatunji (2012) and Pitman et al. (2012) found more PTSD symptoms to be associated with increased deficits in emotion regulation. Most recently, Shepherd and Wild (2014) also found that PTSD was associated with difficulty regulating, and specifically reducing, negative emotions. However, despite the availability of research surrounding general emotion regulation and PTSD, there is limited research on how PTSD relates specifically to AER.

**Automatic emotion regulation (AER).** Much of the current research on emotion regulation focuses on the explicit side of it, but little attention has been given to the implicit side of emotion regulation (Etkin, et al., 2010). However, in the past decade AER has attracted considerable attention from researchers (Etkin et al., 2010; Mauss, Bunge, & Gross, 2007; Zhang & Lu, 2011). For example, while reviewing the available research on AER, Mauss, Bunge, & Gross (2007) found little evidence regarding the cognitive processes underlying AER in previous studies. To account for the lack of information on AER, they turned to past neuroscientific literature involving neuroimaging studies that may have found underlying mechanisms of AER. This analysis identified two distinct processes involved in AER: response-focused AER, which occurs after an emotional response, and antecedent-focused AER, which occurs earlier in the emotional response. Interestingly, these two types of AER appear to have separate neural correlates. It seems that response-focused AER may be supported by the subcallosal cingulate cortex, dorsal pathway, and cerebellum. There is evidence that antecedent-focused AER may be supported by neural pathways involving portions of the prefrontal cortex, the anterior and posterior cingulate cortices, and basal ganglia. The existence of two separate processes involved in AER may provide an explanation for why it has been associated with both adaptive and maladaptive emotional response patterns.

In a more recent study of 17 patients with generalized anxiety disorder (GAD) and 24 healthy controls, Etkin et al. (2010) examined abnormalities in implicit regulation of emotions using fMRI. Participants completed an emotional conflict task, in which they labeled facial affect while ignoring superimposed
AER and PTSD. One aspect of AER that may be important for individual health is helping individuals regulate negative emotions (Mauss, Cook, & Gross, 2007). If individuals develop PTSD following trauma, it is common for them to have difficulties regulating emotions, especially negative ones (Felmingham et al., 2002; Shepherd & Wild, 2014). Though limited research has been done on the direct relation between PTSD and automatic emotion regulation, we suspect that individuals with trauma symptoms will exhibit deficits in AER. These deficits may also contribute to a reduced ability to regulate negative emotions.

To examine if the relatively unexplored automatic processes in emotion regulation could effectively reduce negative emotions, Mauss, et al. (2007) conducted a two-part study. First, researchers investigated if it was possible to manipulate an implicit component of emotion regulation such as AER. Next, they examined the affective consequences of AER. In the first part of the study, researchers experimentally manipulated AER by priming either emotion control or emotion expression using a modified version of the Sentence Unscrambling Task and subsequently asking participants to report their anger. The second part of the study was conducted similarly, but researchers also measured the occurrence of overall negative emotion and cardiovascular responses. The second half of the study aimed to find out if AER could reduce anger. Researchers hypothesized that priming for emotion control would lead to less anger than priming for emotion expression. The findings from this study suggest that AER may lead to reductions in anger after anger provocation in the lab, which has real-world implications for the importance of AER in everyday social interaction and emotion regulation. These results are important not only for understanding how AER can effectively decrease negative emotions, but also how its absence may affect the emotional health of the individual.

Event-related potentials. The current study examined the relationship between PTSD and AER by measuring event related potentials (ERPs) captured using EEG. ERPs are small voltages produced in the brain in response to specific stimuli (Sur & Sinha, 2009). Following the presentation of a stimuli or event, these electrical potentials are observed as changes in EEG that are time-locked to the stimuli or events in cognitive tasks (Luck, 2014; Sur & Sinha, 2009). Researchers often study ERPs because ERPs can achieve a level of temporal accuracy that other measurements, such as fMRI imaging, do not (Karl, Malta, & Maercker, 2006). ERP waveforms can be described in terms of latency, which reflects time in milliseconds, or amplitude, which is measured in millivolts and reflects amount of mental effort exerted (Clayson, Baldwin, & Larson, 2013).

P300 Waveform. The P300, or P3, waveform is one of the most commonly studied ERP components. It is the third positive peak in an ERP component that occurs following a task-related stimulus (Clayson et al., 2013). It may also be elicited by the absence of a stimulus, if participants are instructed to respond to the absence of a certain stimulus. The P3 tends to occur post-stimulus around 300ms, which is how it was termed the “P300” (Luck, 2014). However, latencies for P3s can be variable, ranging anywhere from 200ms to 700ms, leading researchers to refer to it simply as the P3 (Luck, 2014). In general, P3 latency is a more reliable sign of cognitive dysfunction than P3 amplitude, because it is less altered by attention (Picton, 1992). A common way to measure the P3 component of the ERP using EEG is to use an oddball paradigm, in which a participant identifies infrequently presented target stimuli in a task consisting primarily of standard non-target
stimuli. Typically, the infrequent stimuli will generate a larger P3 wave than the frequent stimuli (Luck, 2014).

**N200 Waveform.** The N200, or N2 waveform is one of the most frequently studied negative-going ERP waveforms. It is commonly referred to as the N2 because it peaks approximately 200ms after onset of a task-related stimulus. Often, it is evoked during stimulus discrimination tasks, in which the participant must ignore one stimulus and focus on another. Like the P3, the N2 is commonly evoked using the oddball paradigm (Felmingham et al., 2002). However, both P3 and N2 waveforms have been evoked in previous studies using a version of the Go/Nogo paradigm in which there are equal presentations of target and non-target stimuli (Zhang & Lu, 2012; Luck, 2014).

**PTSD and P3 Latencies.** The relationship between P3 waveforms and PTSD has been studied in previous literature, but the results have been largely inconclusive (Johnson et al., 2013). While some studies have found P3 latencies to be shorter in populations with PTSD than healthy populations, many studies have also found them to be prolonged (Felmingham et al., 2002; Johnson et al., 2013; Schaefer & Nooner, 2017; Stanford et al., 2001). Two commonly selected electrode sites used for analysis of ERP data include Cz and Fz. Both of these midline electrode sites have been widely used by researchers to examine P3 latency. For example, Stanford et al. (2001) found that when presented with negative, trauma-relevant pictures, participants with PTSD exhibited shorter P3 latencies at electrode site Cz than a control group without PTSD. Nooner and Schaefer (2017) found evidence of shortened P3 latencies at site Cz in a high trauma symptom group compared to the low trauma symptom group. Conversely, Felmingham et al. (2002) used an auditory oddball paradigm to elicit P3s from a group with PTSD and a control group who were exposed to 40 target tones and 247 common tones. The results of this study found evidence of longer P3 latencies at electrode site Fz in the PTSD group than the control group (Felmingham et al., 2002). More recently, Johnson et al. (2013) conducted a meta-analysis of 22 studies found on Psych Info and PubMed regarding ERP components and PTSD. Researchers used a random effects model and included all studies looking at P3 components and PTSD between 1990 and 2010. Specifically, they wanted to further the existing literature on the clinical usefulness of the P3 ERP and its components, including less commonly studied parts of the P3 including the P3a, P3b, and P3wm (working memory). The P3a is a subcomponent of the P3 that tends to be elicited when the brain is processing novelty, and is usually invoked using infrequent target stimuli. The P3b is another subcomponent that can be elicited by novelty, and is often used to measure cognitive load. The P3wm is a third component that measures working memory during a task (Johnson et al., 2013). This analysis found that P3a latency in response to neutral distracters was longer in patients with PTSD compared to non-PTSD patients, but these results were not statistically significant for sites Fz, Pz, or Cz. This meta-analysis also did not reveal any significant differences in P3b latency between the PTSD and control groups at sites Fz, Cz, and Pz. Finally, the effect size for P3wm latency for Fz, Cz, and Pz was not significant. Based on this meta-analysis, the current literature on the relationship between P3 latency and PTSD appears to be inconclusive. Further research is necessary to determine if there are differences in P3 latencies between PTSD patients and healthy controls.

**Go/NoGo task.** To study the time course of AER, Zhang and Lu (2012) measured ERPs during an implicit Go/NoGo emotion task. Researchers recorded ERPs from 20 participants during the task with positive, negative, and neutral faces. The task was gender-cued, meaning that participants were presented with a gender word (“male” or “female”) and then presented with a face to respond to. The participant was instructed to press the space bar if the face matched the gender of the preceding cue (“Go” condition), and to do nothing if it did not.
if it did not (“NoGo” condition). The findings of this study suggested that N200 amplitudes and latencies in the Go condition following positive and negative faces decreased more than those following neutral faces, but N200 amplitudes and latencies in the NoGo condition didn’t change with valence. Positive and negative faces created larger P3 amplitudes and smaller P3 latencies than neutral faces in all trials (Zhang & Lu, 2012). The current study replicated Zhang and Lu’s (2012) Go/NoGo task to examine similar neural processes and extend these results to encompass the relationship between trauma symptoms and AER. To replicate Zhang and Lu’s study as closely as possible, the current study utilized a Go/NoGo with equal presentations of “Go” and “NoGo” conditions, instead of a typical oddball paradigm.

**Current Study.** Explicit emotion regulation has been a popular topic in previous literature, but implicit emotion regulation has remained elusive. Two of the important functions of this implicit regulation, known as AER, include reducing negative emotions and contributing to better outcomes in individuals following trauma. Previous studies have shown that individuals with trauma symptoms exhibit marked deficits in AER, perhaps contributing to a reduced ability to regulate their negative emotions (Felmingham et al., 2002; Shepherd & Wild, 2014). However, the previous literature on the relationship between AER and trauma is limited, and because of that the current study aims to examine AER as it relates to trauma symptoms. Specifically, we replicated a previous study to examine the relationship between trauma symptom scores and P3 latencies in three separate emotional valence conditions (positive, neutral and negative). Based on more recent findings by Zhang and Lu (2012), we first hypothesized that negative and positive faces would elicit briefer P3 latencies than neutral faces. Then, we hypothesized that shorter P3 latencies in the negative valence condition in the frontal and central cortices as measured by sites Fz and Cz would be associated with more trauma symptoms. Next, we hypothesized that shorter P3 peak latencies in the neutral valence condition in the central cortex as measured by Cz would be associated with more trauma symptoms. Finally, we hypothesized that participants would exhibit shorter P3 latencies at sites Fz and Cz when presented with negative and positive faces but not neutral faces.

**Method**

**Participants**

Twenty-three volunteers (16 women, 7 men, \( M_{\text{age}} = 20.087 \) years, age range 18 - 33 years) were recruited from the University of North Carolina Wilmington using flyers. All participants had normal visual acuity and no history of neurological deficits at the time of the experiment. Participants signed an informed consent prior to the experiment and received no compensation. All study materials were approved by the UNCW Institutional Review Board (IRB). Including informed consent, the study took one hour or less to complete. Participants received two student research credits for volunteering.

**Materials**

**Behavioral Measures**

**TSC-40.** Participants completed the 40-item Trauma Symptom Checklist (TSC-40) trauma questionnaire. The TSC-40 is a 40-item self-report instrument for adults containing six subscales: Anxiety, Depression, Dissociation, Sexual Abuse Trauma Index (SATI), Sexual Problems, and Sleep Disturbance, as well as a final overall score. Items are rated according to frequency of occurrence over the prior two months, using a four-point scale ranging from 0 (never) to 3 (often). It takes approximately 10-15 minutes to complete the TSC-40, and it can be scored in approximately 5-10 minutes. Previous studies using the TSC-40 indicate that it is a relatively reliable measure (subscale alphas typically range from .66 to .77, with mean alphas for the full scale between .89 and .91). The TSC-40 has predictive validity regarding a wide range of traumatic experiences (Elliott & Briere, 1992).

**Demographics**

Participants completed a demographics survey,
indicating race, ethnicity, gender, and age.

**Cued Implicit Response Inhibition Task**

**NimStim Set of Facial Expressions.** The NimStim set consists of 43 professional actors and 672 total stimuli, including 18 female and 25 male faces between ages 21-30. Four ethnicities are represented in the NimStim, including African (N = 10), Asian (N = 6), European (N = 25), and Latino-American (N = 2). Each actor exhibited a happy, sad, disgusted, fearful, angry, surprised, neutral, and calm expression, including open and closed mouth versions of all except surprise (Tottenham et al., 2011). One hundred and twenty-six of these faces met the criteria for our study, meaning they exhibited either a positive (happy), negative (angry), or neutral valence. Each positive, negative, and neutral expression for each actor was presented twice during our task: once in the Go condition and again in the NoGo condition. The final task included 84 presentations of positive faces, 84 presentations of negative faces, and 84 presentations of neutral faces. We used 10 African American, 6 Asian, 24 European, and 2 Latino-American faces. Finally, our faces included 24 men and 18 women. The NimStim set was chosen over other sets because it provided racial diversity, showed high validity, and included neutral expressions, which some sets do not (Tottenham et al., 2011). We acquired the NimStim by contacting Nim Tottenham, the creator of the set and the researcher in charge of the Developmental Affective Neuroscience Lab at Columbia University, and receiving her permission to use the set in our task.

**Go/NoGo Task.** To find out if PTSD is correlated with ERP dysfunction, we measured ERPs collected during a cued emotional Go/NoGo task that was modified from another study on AER (Zhang & Lu, 2012). We sought to replicate Zhang and Lu’s (2012) Go/NoGo task using Paradigm, a psychology research software program. The final cued emotional Go/NoGo task consisted of 42 validated and standardized faces from the NimStim facial stimulus set (Tottenham et al., 2011). Participants pressed the space bar when the gender word matched the gender of the face that immediately followed it (Go condition). They abstained from pressing the space bar if the gender word did not match the face that immediately followed it (NoGo condition). During the task, a blank screen appeared for 1200 ms between each trial. Each trial began with a small black cross (+), which was visible for 500 ms. This was followed by the word for a gender cue (“male” or “female”) presented on the computer screen, which appeared for 1000 ms. A face was presented at random for a random duration between 150-250 ms and participants had the opportunity to push the space bar or not depending on their perception of the condition as a Go or NoGo. The entire experiment consisted of 21 randomized blocks, with 12 randomized trials each. There was a 20 second resting baseline every 9 blocks to allow participants to have a break from the stimuli. In accordance with Zhang and Lu’s (2012) task, an equal number of Go and NoGo trials were presented to account for the novelty of NoGo cues. The task took approximately 21 minutes.

**EEG.** The current study used a Food and Drug Administration (FDA) approved 64-channel Biosemi EEG system with ActiveTwo Pin-type electrodes provided by Cortec Solutions to assess brain activity while participants completed the Go/NoGo task. Participants’ heads were measured in centimeters using a tape measure before the experiment to determine the appropriate EEG cap size to use. We also used four reference electrodes, referenced to the left or right mastoid. We also included references on the left and right cheekbone to detect eye blinks. EEG data were acquired simultaneously with Paradigm stimulus presentation.

**Procedure**

After arriving at the laboratory, each participant signed an IRB-approved informed consent. Participants completed the Trauma Symptom Checklist (TSC–40; Elliot & Briere, 1992) and a demographics survey. Directly after completing these assessments, participants completed the Go/NoGo task.
while the EEG activity was recorded. A licensed psychologist was present to monitor clinical safety. For confidentiality, participant identifiers were removed, including names, birth dates, and assessment dates. All data was secured using an encrypted, computerized data collection and management system, the Collaborative Informatics and Neuroimaging Suite.

EEG Data Processing. We processed data using the EEGLab and ERPLab plug-ins designed for MATLAB following the methods outlined on the ERPLab Toolbox Web site (Lopez-Calderon & Luck, 2014). The EEG data were divided into epochs, which are windows of time during stimulus presentation generated from -200 ms to 1000 ms relative to the onset of stimuli. Then we modified the EEG data sets in MATLAB to eliminate bad channels, electrical noise, and unnecessary artifacts such as blinks, which can distort brain waves. We analyzed the data using steps from the ERPLab Toolbox tutorial, which outlines the best steps for analyzing EEG and ERP data (Lopez-Calderon & Luck, 2014). ERPLab was used to create ERPs without artifacts to examine P3 latency at two electrode sites: Fz (frontal) and Cz (central). We chose the P3 component of the ERP and the midline electrode sites because both were used frequently in the previous literature (Zhang & Lu, 2012).

Results

Scores from the Trauma Symptom Checklist were used as a continuous variable in our analyses. Descriptive statistics for the TSC-40 (M = 30.53, SD = 14.09) indicated that our sample fell below the clinical range, which is 65 and above.

A one-way repeated measures ANOVA indicated a trend in the Cz latencies between positive, negative or neutral stimuli F(2, 28) = 3.128, p = .059 (see Figure 1). Post-hoc comparisons at site Cz indicated that latencies to positive stimuli were marginally longer than those to negative stimuli and that latencies to neutral stimuli did not differ from those to positive or negative stimuli. A one-way repeated measures ANOVA indicated no difference in the Fz latencies between positive, negative or neutral stimuli, F(2, 28) = .473, p = .78 (see Figure 2).

We found a strong, significant negative correlation between P3 peak latency at site Cz with a neutral valence and trauma symptom score, which indicated that those with shorter P3 latencies tended to report more trauma symptoms, r(13) = -.592, p < .01. A strong, significant negative correlation between P3 peak latency at site Fz with a negative valence and trauma symptom score indicated that those with shorter P3 latencies tended to report more trauma symptoms, r(13) = -.529, p < .05. A strong, significant negative correlation between P3 peak latency at site Cz with a negative valence and trauma symptom score indicated that those with shorter P3 latencies tended to report more trauma symptoms, r(13) = -.650, p < .01. We found no significant correlation between P3 peak latency at site Cz with a positive valence and trauma symptom score. We also found no significant correlation between P3 peak latency at site Fz with a positive valence and trauma symptom score.

Tests of the three hypotheses were conducted using Bonferroni adjusted alpha levels of .017 per test (.05/3). Three separate simple regression analyses were calculated to test if P3 peak latency at electrode sites Fz and Cz with negative and neutral emotional valences significantly predicted participant score on the TSC-40 (see Table 1). The first simple linear regression was calculated to predict trauma symptom score based on P3 peak latency at electrode site Cz with a neutral valence. A significant regression equation indicated smaller Cz peak latencies when viewing neutral stimuli predicted higher trauma symptoms with R^2 = .300, p < .02. The second simple linear regression was calculated to predict trauma symptom score based on P3 peak latency at electrode site Cz with a neutral valence. A significant regression equation indicated smaller Fz peak latencies when viewing neutral stimuli predicted higher trauma symptoms with R^2 = .224, p < .02. The last simple linear regression
was calculated to predict trauma symptom score based on P3 peak latency at electrode site Cz with a negative valence. A significant regression equation indicated smaller Cz peak latencies when viewing negative stimuli predicted higher trauma symptoms with $R^2 = .377$, $p < .01$.

**Discussion**

While emotion regulation has been regarded as an important topic for some time, most of this research has focused on explicit or skill-based emotion regulation that is used following trauma or a challenging life experience (Etkin et al., 2010). However, the implicit aspect of emotion regulation, known as AER, may also be important for reducing negative emotions and contributing to better outcomes for individuals suffering from trauma, particularly following a traumatic event (Shepherd & Wild, 2014).

The current study examined AER in an implicit emotion regulation task. The task was explicitly a response inhibition task known as a Go/NoGo task in which participants had to match gender words to gender pictures. However, the pictures also had emotional valences of positive, negative, or neutral, which was the implicit portion of the task. Participants were told to match faces but were not told anything about the emotions on the faces, nor were they asked to do anything with the emotions on the faces. We were interested in seeing if response times (i.e., latencies) in the Go/NoGo task differed as a function of the emotion of the face presented. Our hypotheses that positive faces would elicit shorter P3 latencies at sites Fz and Cz were not supported. Our next hypothesis (i.e., shorter P3 latencies with negative faces at sites Fz and Cz would be associated with more trauma symptoms) was supported. For negative faces, we found that individuals with shorter P3 latencies at electrode sites Fz and Cz were more likely to report greater trauma symptoms. Our last hypothesis, (i.e., shorter P3 latencies with neutral faces at sites Cz and Fz would not be associated with more trauma symptoms), was supported for site Fz but not Cz. For neutral faces, we found that individuals with shorter P3 latencies at electrode sites Cz were more likely to report greater trauma symptoms.

The results of our study are supported by findings in previous literature. Stanford et al. (2001) found that when presented with negative, trauma-relevant stimuli, participants with PTSD exhibited shorter P3 latencies at electrode sites Cz and Pz than a control group without PTSD. Previous research has also found evidence of longer P3 latencies at electrode site Fz in response to negative visual stimuli (Felmingham et al., 2002), which would explain why P3 latencies at site Fz did not reach significance in our sample. Karl et al. (2006) found that individuals with PTSD exhibited shorter P3 latencies at site Cz in response to neutral stimuli, which supports our findings. Johnson et al. (2013) conducted a meta-analysis of a decade of P3 component research and found nothing of significance for PTSD and P3 latency at midline electrode sites Fz and Cz, but our findings suggest that more trauma is related to shorter latencies at sites Fz and Cz.

It is important to note that our findings add support to previous studies that have found evidence of shorter P3 latencies at sites Cz and Fz in response to negative stimuli. However, to account for inconsistency in results of previous research, it is clear that further study is needed to determine the relationship between trauma and P3 latency.

Our findings at site Cz (i.e., that latencies were shorter for neutral faces) were not supported in Zhang and Lu’s (2012) study. However, it is possible that participants in our sample reporting high trauma might have suffered from a negativity bias, resulting in shorter latencies in response to both neutral and negative faces. Etkin et al. (2010) found that in a sample of women with PTSD and a control group, women with PTSD were unable to process differences between negative and neutral faces because of their trauma. Unless the faces had a clearly positive valence, they were processed as negative faces. Because the neutral faces in the NimStim set appear to have a more negative
valence than positive, it is possible that participants with more trauma symptoms in our sample exhibited this negativity bias at site Cz. In cases such as these, it is possible that latency would decrease in response to negative and neutral faces compared to positive, because individuals with trauma symptoms process all stimuli that isn’t clearly positive as negative as a result of their trauma (Etkin et al., 2010; Tottenham et al., 2011).

**Limitations.** This study included several limitations that should be considered for future research. One limitation of the current study was our relatively small sample size. While we recruited a total of 23 participants, only 15 of those participants were included in our final analyses. Six participants were excluded from our analyses because of difficulties with EEG data collected while presenting the Go/NoGo task using Paradigm. Because these six data files lacked markers to indicate when participants were presented with positive, neutral, and negative stimuli, we were unable to track their response times and include their data. Two other participants were also excluded because their EEG data contained too much noise to use for analysis, especially at site Fz which is close to eyebrows and forehead muscles that tend to move even at rest.

It is common for researchers to elicit P3 waveforms using an oddball paradigm, in which infrequent target stimuli are randomly interspersed among more frequent non-target stimuli. Indeed, much of the previous literature used an oddball paradigm to elicit P3 amplitudes and latencies (Karl et al., 2006). Thus, it may have been a limitation to our study that we attempted to study P3 latencies without using an oddball paradigm. However, the study we replicated (Zhang & Lu, 2012) is one of the first to successfully use affective stimuli without using an oddball paradigm to elicit P3s, which was intentional. Zhang and Lu (2012) specifically chose to have an equal number of target and non-target stimuli in their Go/NoGo task, to control for the novelty of NoGo cues. To replicate their task as closely as possible, we chose to present an equal number of target and non-target stimuli in our task as well. At this time, it is impossible to know the effects of using an oddball versus a traditional Go/NoGo task to examine AER. Future research should examine differences between the two methods.

Finally, limited statistical power because of the modest sample size in the present study ($n = 15$) may have played a role in limiting the significance of some of the statistical tests that were conducted. A post hoc power analysis was conducted using G*Power on the basis of the mean for the 15 participants. The between-groups comparison effect size observed in the present study was less than 1 standard deviation ($d(13) = .74$ to .93). With a recommended cutoff of .80 (Cohen, 1988), the effect sizes for Cz neutral ($d(13) = .93$) and Cz negative ($d(13) = .85$) were sufficiently powered to detect a between-groups effect, while Fz negative ($d(13) = .74$) was not.

There are several areas of interest future research should consider. One of these would be looking at AER in a population with a PTSD diagnosis. While the TSC-40 is a reliable measure used to gauge severity of trauma symptoms, further exploration of the relationship between AER and PTSD would require participants with a formal diagnosis, which we were unable to attain. Furthermore, the clinical applications of AER would be greater if a similar relationship could be established between PTSD and AER instead of just AER and trauma. Another point of interest may be examining gender differences in AER and trauma, which we were unable to do. Unfortunately, much of the data we had to exclude from analysis was from male participants, leaving us with an unbalanced sample of 11 women and 4 men. Partly due to this gender imbalance, we decided not to include gender as a variable in our analyses. We also chose not to include gender because it was not found to be significant in Zhang and Lu’s (2012) analyses of their original Go/NoGo task. Future research might incorporate parietal areas of the brain by using electrode
site Pz, which is often included in analyses of midline electrodes like Cz and Fz, to see if there are any noticeable differences in latency at that region of the brain. While our analysis did not include site Pz, much of the previous literature found significant evidence that site Pz is related to trauma symptoms.

**Conclusion.** The goal of the current study was to examine emotion regulation, specifically implicit regulation known as AER, as it relates to trauma symptoms using an implicit Go/NoGo task. We found in frontal regions of the brain, latencies did not significantly vary by face type. However, in central areas of the brain, latencies for positive faces were longer than for negative faces. We also found that briefer latencies in central areas of the brain in response to negative and neutral faces were associated with more trauma symptoms. Finally, we found that briefer latencies in frontal areas of the brain in response to negative faces were associated with more trauma symptoms. Our results suggest that individuals with trauma symptoms may exhibit difficulties with implicit regulation of negative emotions, and may be subject to a negativity bias in which they process all stimuli that isn’t clearly positive as negative. These findings add to the existing literature on brain function, trauma, and differences in emotion regulation. Our study is also one of the first to address the implicit side of emotion regulation as it relates to trauma using affective stimuli. Examining this relationship further may help to develop new interventions to improve prognoses for individuals with trauma symptoms, and perhaps in the future, PTSD.

**Figure 1:** P3 latency in milliseconds at site Cz in positive, neutral, and negative emotional valence conditions. No statistically significant differences were found between emotional valences.
Figure 2: P3 latency at site Fz in positive, neutral, and negative emotional valence conditions. No differences were found between emotional valences.

Table 1: Summary of Simple Linear Regression Analysis for TSC-40 Score and P3 Latency at Cz and Fz

<table>
<thead>
<tr>
<th>Sites</th>
<th>Valence</th>
<th>$R^2$</th>
<th>Sig. ($p$)</th>
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<tbody>
<tr>
<td>Cz</td>
<td>Negative</td>
<td>.377</td>
<td>&lt;.02</td>
</tr>
<tr>
<td>Cz</td>
<td>Neutral</td>
<td>.300</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Fz</td>
<td>Negative</td>
<td>.224</td>
<td>&lt;.02</td>
</tr>
</tbody>
</table>

Note. Positive valence was not included for either site because P3 latency and TSC-40 score were not significantly related in relation to positive stimuli. Neutral valence was not included for site Fz because P3 latency and TSC-40 score were not significantly related.
REFERENCES


