12-2020

Emotion Regulation and N2 Amplitude During a Go/Nogo Task: an ERP Study

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An Honors Thesis submitted in partial fulfillment of the requirements of Honors Studies in Psychology

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Fall 2020

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Acknowledgements

I would like to thank Dr. Connie Lamm for mentoring me through this process, endlessly editing my paper, and creating a welcoming and educational lab for students.

A special thank you to Arooj Abid for helping with data analysis, giving feedback, and having monthly zoom calls to help me with my thesis.

I would also like to thank Morgan Middlebrooks for supporting me during my time in the DCAP lab and always being available to answer questions.

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1. Abstract

Emotion regulation is how people respond to and manage their reactions to life experiences, including resolving conflict between variable responses. Past research has associated the N2, an event-related potential associated with resolving response conflict, with both emotion regulation and negative emotion. However, to the best of our knowledge, no one has assessed if different emotion-regulation strategies are differentially associated with N2 activation. To assess this question, we conducted an EEG study with 147 participants. Participants completed the Cognitive Emotion Regulation Questionnaire (CERQ) and then played the go/no-go game as their EEG data was collected. The relationship between N2 amplitude and self-reported emotion-regulation strategies on the CERQ, specifically rumination, catastrophizing, and self-blame, was assessed. No significant relationships between these CERQ subscales and the N2 amplitude were found. Future studies should investigate different ERPs, possibly in a clinically-anxious sample.

2. Introduction

Dysfunctional emotion regulation has been associated with a number of clinical disorders, such as anxiety disorders and depression (Zhu, et al., 2009; Joorman & Gotlib, 2010). Emotion regulation is an individual's modification of ongoing or initiation of new emotional responses (Gross & John, 2003). The ability to cope with unpleasant situations and exert control over one's emotional reaction is crucial to psychological well-being and healthy social interactions (Kim & Hamman, 2007).

The Gross model of emotion regulation postulates several emotion regulation strategies, including cognitive reappraisal and expressive suppression (Gross & John, 2003). Another model of emotion regulation breaks the process into unconscious (projection and denial) and conscious cognitive processes, such as other-blame, self-blame (Kalokerinos, Bastian & Kuppens, 2018). A study done by Lazarus stated that conscious cognitive emotion regulation was emotion focused coping and aims to change the meaning of a stressful event and lessen emotional distress (Lazarus, 1993). A study using a measure of cognitive emotion regulation indicated the important role the conscious strategies play while dealing with negative events and solving conflict (Garnefski et al., 2001). Together, these studies indicate that day-to-day we apply various emotion regulation strategies to navigate our social lives.

Many studies have shown that emotion regulation is essential for resolving conflicts (Mitchell, 2011; Rolls, 2005). Mitchell (2011) concludes that emotion regulation and decision making are bound by a similar functional objective and this decision making is how conflicts are resolved. Ben-Naim and colleagues (2013) conducted a study in which they randomly assigned couples to various emotion regulation strategies and then studied their ability to resolve conflict. They found significant differences between various emotion regulation strategies. Furthermore,

Halperin and Gross (2011) assessed the conflict resolution abilities of Israeli humanitarian aid workers and found that those applying emotion regulation strategies showed more support to innocent Palestinia citizens. These studies clearly show the importance of emotion regulation to resolving conflict; however, these important real-world studies are unable to explore the neural processes underlying our capacity for emotion regulation.

Numerous studies have shown that prefrontal cortical activation contributes to our ability to regulate our emotions (Romero-Rebollar et al., 2016; Doré et al., 2018). Neuroimaging studies have shown that individuals who often use expressive suppression have higher anterior insula volumes and those who use cognitive reappraisal have a higher dorsal anterior cingulate volumes, suggesting that different brain structures are associated with different regulation strategies (Cutuli, 2014). Other studies have shown the importance of the prefrontal cortex during expressive suppression (inhibiting responses), specifically the dorsomedial and dorsolateral regions of the prefrontal cortex (Ochsner, Silvers & Buhle, 2012). Some of these same prefrontal brain regions have been associated with resolving response conflict (Wendelken et al., 2009) and there is some evidence that neural efficiency in these regions can be altered with targeted treatment (Braver et al., 2009; Lewis et al., 2008). Given the importance of emotion regulation in resolving conflict, we explore if specific emotion-regulation strategies are associated with resolving response conflict using a computerized behavioral task.

This study analyzes the relationship between an event-related potential (ERP; averaged EEG) associated with resolving response conflict (Enrique-Geppert, Konrad, Pantev & Huster, 2010), i.e., the N2, and various emotion-regulation strategies. Response conflict is when neural signals support competing response alternatives (Wendelken et al., 2009). The N2 is a medial frontal component that occurs roughly 200 ms after stimulus onset. A number of studies by

Lewis, Granic, Lamm, and colleagues (e.g., Lamm et al., 2006; Granic et al., 2008; Lewis et al., 2006) explored if N2 amplitudes changed in the face of increased need for emotion regulation. Indeed, they found more negative N2s in the face of increased need for emotion regulation in two samples of typically-developing children (Lewis et al., 2006; Lamm et al., 2010) and in a sample of aggressive children (Lamm et al., 2011). However, these authors did not explore what emotion-regulation strategies these children applied to successfully complete the task. Thus, the current study explores this question.

This experiment used a go/no-go task to elicit N2 activation. A go/no-go task requires participants to press a button as fast and as accurately as possible for every letter (go stimuli) viewed on a computer screen, except for the letter "X" (the nogo stimulus). The go stimuli are presented more often than nogo stimuli. Therefore, when a participant views the nogo stimulus, they experience conflict between pressing the button (habitual response) and not pressing the button (required response). The current study examines the association between N2 amplitudes and specific maladaptive emotion regulation strategies, namely rumination, self-blame and catastrophizing, measured using the Cognitive Emotion Regulation Questionnaire (CERQ). The CERQ is a questionnaire designed to assess an individual's cognitive coping strategies separately from their coping behaviors (Kalokerinos, Bastain & Kuppens, 2018). Rumination refers to the tendency to repeatedly think about thoughts and feelings after an unpleasant event. Self-blame is associated with an individual blaming themselves for the events that have occurred, whereas catastrophizing is when someone over-emphasizes the negativity of a situation by assuming the worst. We predict that participants who report high usage of self-blame, rumination, and catastrophizing emotion regulation strategies will have larger N2s compared to participants who do not show elevated scores for these subscales, particularly because these specific emotion

regulation strategies are related to symptoms of psychopathology and are considered non adaptive emotion regulation (Kalokerinos, Bastain & Kuppens, 2018). In general, the N2 amplitude is larger when a participant has to change a planned response and carry out an unplanned response (Randall & Smith, 2011). Given that larger N2 amplitude reflects increased recruitment of neural resources, participants that use emotion regulation strategies characterized by over thinking are hypothesized to yield larger N2s than participants that do not use these strategies.

3. Methods

3.1 Participants

Participants (N=164) were recruited and screened through Sona systems. Participants were University of Arkansas undergraduate students and received General Psychology course credit for their participation. Participants were excluded if they reported a current psychiatric diagnosis or a current use of psychoactive medication, had uncorrected visual impairments, and if they were over the age of 65. Participants were also excluded if any of the task conditions contained less than 10 correct artifact free trials and if they failed to complete the CERQ questionnaire. The final sample size was 147 participants with a mean age of 19.4 years (SD = 2.6, range = 18-47; 70 males).

3.2 Procedure

The participant was welcomed to the lab and asked to read and sign an informed consent document. The participant then completed several questionnaires, including the CERQ. The participant was seated 67 cm from the computer screen and had the electrode sensor net applied. Impedances of electrodes were checked and when each electrode showed an impedance value below 50 KΩ, data collection began. The participant then completed the go/no-go task, during

which behavioral and EEG data were collected. When the tasks and questionnaires were completed, the participant was debriefed and thanked for their participation.

3.3. Go/No-go Task

The participants first completed a practice block of 10 trials of the go/no-go task after which they completed the actual task. The participant was presented with an asterisk as the fixation point for 100 ms and single letters in white on a black screen for 200 ms. Following the go or no-go stimulus presentation, a fixation screen appeared for 600 ms during which participants responded. There was also an inter-trial interval jittered for 0-500 ms (see Figure 1 for task details). The task instructed participants to press a "1" on a response pad as quickly and accurately as possible using their left thumb when presented with any letter (e.g., "T"; go stimulus) and to withhold a response and not press any key when they see the letter "X" (no-go stimulus). The task had five blocks of 53 trials each, in which 75% of the trials were go trials (control trials) and 25% were no-go trials (target trials) and each no-go trial was separated by a go trial.

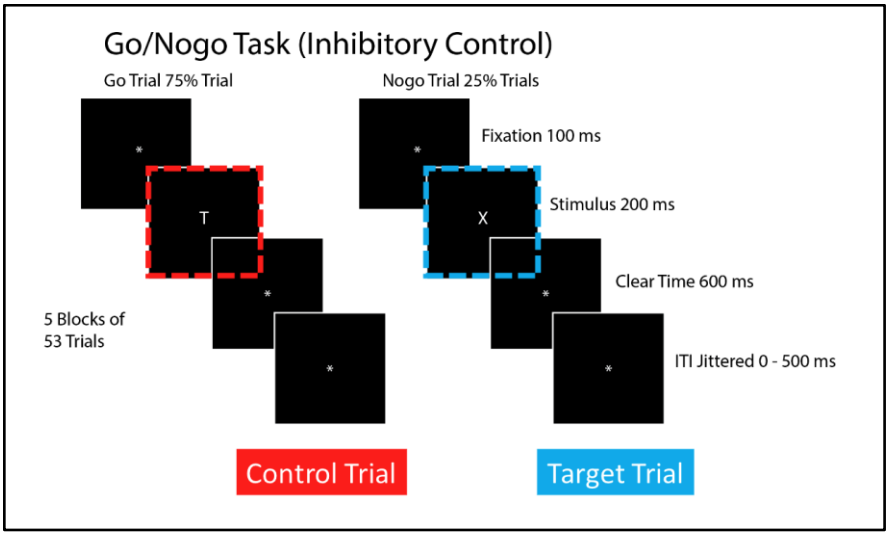

Figure 1. Go/no-go task diagram.

3.4 Cognitive Emotion Regulation Questionnaire (CERQ)

The CERQ (Kalokerinos, Bastain & Kuppens, 2018) measures nine different types of emotion regulation strategies established by the clinical psychology literature: Self Blame, Acceptance, Rumination, Positive Refocusing, Refocus on Planning, Positive Reappraisal, Putting thoughts into Perspective, Catastrophizing, and Other Blame. The CERQ has a total of 36 self-report questions and each coping strategy has four questions dedicated to assessing the extent to which a person uses that strategy in response to distressing experiences. The participant is asked to indicate on a scale from 1 (almost never) to 5 (almost always) how often they generally feel or think a certain way. An example of a question on this survey is, "I feel that I am the one to blame for it" which is assessing the frequency of self-blame as an individual's coping strategy. Each participant obtained a score ranging between 4 (low frequency of specific subscale) and 20 (high frequency) on each of the subscales, from which the self-blame, rumination, and catastrophizing subscales were extracted and analyzed for the purpose of this study.

3.5 EEG data collection and processing

EEG data were recorded through a 128-channel Geodesic Sensor Net and sampled at 250 Hz, using EGI software (Net Station, Electrical Geodesic, Inc., Eugene Oregon). Procedure and technique for EEG data collection and processing were consistent with Lamm et al., (2013). Data was not recorded until all channel impedances were below 50 kΩ. Half-way through data collection, channel impedances were checked and once again reduced to below 50 kΩ. All channels were references to Cz and then re-referenced to the average reference after data collection was completed.

MATLAB's processing toolbox EEGLAB was used to pre-process collected EEG data (Delorme & Makeig, 2004; http://www.sccn.ucsd.edu/eeglab). Data was filtered to exclude data with a bandwidth outside of 0.1-35 Hz and down sampled to 125Hz. If EEG channels were four standard deviations above the mean, they were removed then later recalculated to fit the average. Data was segmented from 300 ms before to 900 ms after stimulus presentation and was stimulus-locked to go and no-go stimulus trials. Infomax ICA was used to clean the data set using runica (Makeig, Jung, Bell, Ghahremani, & Sejnowski, 1997) and the ADJUST plugin (Mognon et al., 2011) to remove eye blinks and movement from the EEG data. Data was then rejected if artifacts surpassed ±140 μV (peak-to-peak). The N2 was most negative at FCz (electrode 6) between 232 and 288 ms after stimulus (go and no-go) onset. The N2 component means were only extracted from correct trials.

3.6 Analyses

To investigate the relationship between participants' self-reported emotion regulation strategy and ERP amplitudes (specifically N2), we ran regression analyses between CERQ subscale scores (specifically, rumination, catastrophizing, and self-blame scores) and no-go N2s, while controlling for go-N2s and no-go trial count. The mean number of correct go trials was 152.46 (SD = 24.01, min = 75, max = 180) and the mean number of correct no-go trials was 31.34 (SD = 8.48, min = 10, max = 55).

4. Results

Results indicated that the relationship between the N2 amplitude and maladaptive emotion-regulation strategies were non-significant. Specifically, N2 and rumination were non-significant, r = 0.091, p = 0.275. Similarly, N2 amplitude and catastrophizing scores were non-

significant r = 0.133, p = 0.109. Lastly, there was no relationship between the N2 and self-blame scores either, r = 0.096, p = 0.247. We also ran partial correlations controlling for gender, but all effects were still non-significant.

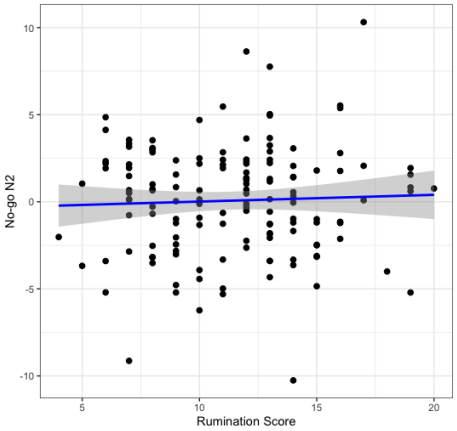

Figure 2. Relationship between rumination and the N2

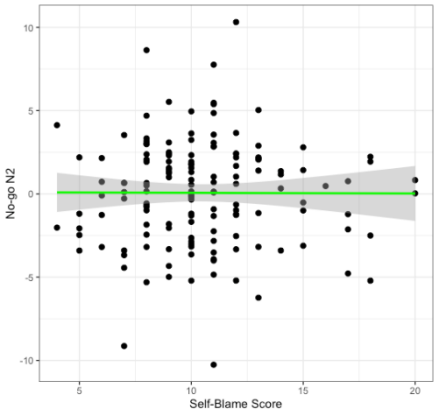

Figure 3. Relationship between Self-Blame and N2

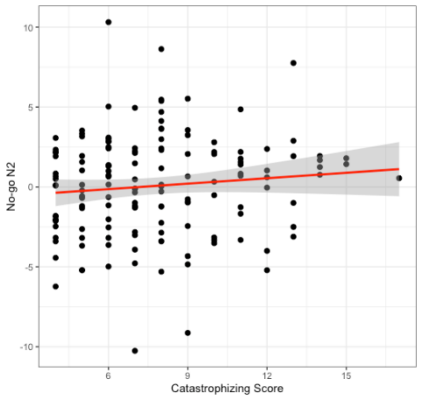

Figure 4. Relationship between Catastrophizing and N2

5. Discussion

The goal of the current study was to investigate the relationship between emotion-regulation strategies (specifically, rumination, catastrophizing, and self-blame) and N2 amplitude. N2 activation was measured while participants played a Go/No-go task, which elicits conflict between the frequent go trials and the infrequent nogo trials.

Past research has related N2 activation with emotion regulation (Lamm et al., 2006 & Megías et al., 2017) as well as with negative emotion (Krendl et al., 2017; Lewis et al., 2006). These studies indicated that the N2s were more negative after negative emotions were induced and emotion regulation was used. The current study explored if different emotion-regulation strategies differentially correlate with N2 amplitudes in a non-clinical population. The Go/No-go task elicits response conflict and research suggests that when more cortical resources are used to solve a conflict, the N2 is larger (Sehlmeyer et al., 2010). Based on this past literature, we

predicted that the N2 would be more negative when a participant reports a high frequency of (obtains a higher score) using catastrophizing, self-blame, and rumination on the CERQ compared with low frequency. Contrary to what was predicted, results revealed no significant relationship between N2s and emotion regulation strategies.

The current study failed to support the hypothesis that individuals who have a tendency to overthink when presented with a conflict will use more cortical resources to resolve the conflict. It is unclear as to why we did not find an association between N2s and any of the emotion regulation strategies. It may be that our Go/No-go task was not a good "imitation" of real-life conflict. Future research should explore patterns of EEG frequency in real-world interactions to see if theta activity, an EEG frequency associated with response conflict (e.g., Cohen & Cavanagh, 2011), is elevated during real-life conflict. It is also possible that context plays a role. Lewis and colleagues (e.g., Lamm et al., 2006; Lewis et al., 2008; Lewis et al., 2006) examined N2 activation in the context of negative emotion. Thus, our relatively unemotional task may have not elicited the same neural correlates as found by Lewis and colleagues. Lastly, it is also possible that our data showed a restriction of range due to not being a clinical sample, and thus lacked the variance in order to find significant effects. Nevertheless, the current study helps fill a gap in the literature by demonstrating how emotion regulation and the N2 may not necessarily be related.

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