# DIFFERENCES IN THE LATE POSITIVE POTENTIAL IN RESPONSE TO EMOTIONAL STIMULI IN TRAUMA-EXPOSED COLLEGE STUDENTS

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ABSTRACT OF THE THESIS

Differences in the Late Positive Potential in Response to Emotional Stimuli in Trauma-exposed People

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Despite the high prevalence of post-traumatic stress disorder (PTSD; Kilpatrick et al., 2013), much is still unknown about the etiology of this debilitating condition. Dysregulated reactivity to emotional stimuli has been implicated in the maintenance and onset of PTSD symptoms (McLean & Foa, 2017; Nawjin et al., 2015). Neurobiological markers of emotional reactivity in response to emotional stimuli can help elucidate the relationship between post-trauma psychopathology and emotional processing. The late positive potential (LPP) is an event-related potential that is associated with emotional reactivity to salient stimuli and can be used as neural measure of dysregulated emotional processes in clinical and subclinical populations (Hajcak et al., 2010). However, results of studies of the relationship between the LPP in response to emotional stimuli and PTSD symptoms in trauma-exposed people have been mixed (e.g., DiGangi et al., 2017; Lobo et al., 2014). The purpose of the current study was to test the relationship between emotional reactivity, as measured by the LPP, and PTSD symptoms in trauma-exposed people. We hypothesized that trauma-exposed people will have greater LPP amplitude in response to negative stimuli than to positive or neutral stimuli, and that greater LPP amplitude to negative stimuli will be associated with greater severity of PTSD symptoms. Data from 76 trauma-exposed undergraduates were used. Participants viewed a series of negative, neutral, and positive images while ERPs were recorded. Negative images produced the highest LPP amplitudes, followed by positive and neutral images. However, LPP amplitudes to emotional images did not predict PTSD symptoms. These results suggest that trauma-exposed people experience stronger emotional reactions to negative compared to positive images. More work is needed to understand the relationship between emotional reactivity and PTSD symptoms.

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# I. Introduction

Exposure to traumatic events is highly prevalent, with about 90% (Kilpatrick et al., 2013) of individuals in the U.S. reporting that, at some point in their lives, they experienced a Criterion A traumatic event (i.e., any event in which someone is exposed to actual or threatened death, serious injury, or sexual violence; American Psychiatric Association, 2013). About 9% of trauma-exposed people at a given time report symptoms consistent with a diagnosis of post-traumatic stress disorder (PTSD; Kilpatrick et al., 2013). Prevalence of trauma-exposure among college students is lower than in the general population, with 59% reporting having experienced a traumatic event at some point in their lives, but the rate of PTSD following a traumatic event is comparable, with about 8-9% of trauma-exposed undergraduates meeting criteria for PTSD at a given time (Elhai et al., 2012).

PTSD is comprised of four symptom clusters: intrusive symptoms (e.g., intrusive thoughts, nightmares, flashbacks), avoidance symptoms (e.g., avoidance of trauma reminders, avoidance of trauma-related thoughts or memories), negative alterations in cognitions and mood (e.g., anhedonia, difficulty experiencing positive emotions, negative assumptions about the world), and hyperarousal symptoms (e.g., hypervigilance, exaggerated startle response, sleep disturbance; APA, 2013). Together, these symptoms form a syndrome that can have a range debilitating effects on an individual. Although effective evidence-based treatments are available, up to 47% of patients fail to experience symptom relief from current gold-standard interventions (Bradely et al., 2005). Developing a better understanding of the processes involved in the etiology and maintenance of PTSD can help inform efforts to improve treatment.

Dysregulated (i.e., maladaptive) processing of emotional information (either trauma-related or not) is one such process that has been implicated in the etiology and maintenance of PTSD (McLean & Foa, 2017). Dysregulation in a number of emotional processes can contribute to PTSD, including maladaptive emotional reactivity and difficulty with emotion regulation (Fitzgerald et al., 2018; Seligowski et al., 2015). Emotional reactivity refers to the experiential, behavioral, and physiological changes that occur when an individual attends to and appraises a goal-related situation (Gross, 2010). Emotion regulation refers to any process that alters emotions, including which, when, where, and how emotions are experienced and expressed (Gross, 2010). If individuals encounter a stimulus that evokes a particularly strong negative emotion (i.e., reactivity), such as seeing a film that makes them especially sad, they may alter how they perceive or evaluate the situation, thereby diminishing their emotional response (i.e., regulation). For example, they could remind themselves that what they saw was a work of fiction.

In the modal model of emotion (Gross, 1998), attention to and appraisal of a situation contribute to emotional reactivity. Further, there is a bidirectional relationship between attention and appraisal (Yiend, 2010). For example, appraisals of stimuli as being more negative or more positive are associated with greater allocation of attention to those stimuli (Calvo & Lang, 2004). Having individuals alter their appraisals of emotional stimuli can lead to corresponding changes in the amount of attention they allocate to those stimuli (Moser et al., 2006; Ochsner & Gross, 2005). Similarly, allocating greater attention to a stimulus (e.g., rumination) can affect how it is appraised (Jenness et al., 2016; Spinhoven et al., 2015). Thus, alterations in one component can lead to alterations in the other.

**PTSD and Dysregulated Emotional Processing**

Heightened emotional reactivity to trauma reminders (i.e., internal or external cues that remind an individual of a trauma) is a cardinal symptom of PTSD (APA, 2013). However, trauma-exposure can also alter how people appraise non-trauma-related stimuli by making them more sensitive to potential threats (Yehuda & LeDoux, 2007). Thus, trauma-exposed people, compared to no-trauma controls, have more negative appraisals of negative stimuli in general (Buckley et al., 2000; Ehlers & Clark, 2000). This leads to greater attention to negative stimuli, regardless of trauma-relevance (e.g., Armstrong et al., 2013; Bryant et al., 1995; Kimble et al., 2010). Given that attention to and appraisal of stimuli are aspects of emotional reactivity (Gross, 2010), the ability of negative stimuli to capture and sustain the attention of trauma-exposed people constitute a major example of their heightened emotional reactivity. Indeed this heightened emotional reactivity is reflected in heightened sympathetic nervous system (SNS) reactivity to negative stimuli (e.g., Cascardi et al., 2015; McTeague et al., 2010).

This heightened emotional reactivity to negative stimuli (reflected by altered appraisals and greater attention) has been implicated in the onset and maintenance of PTSD symptoms. Frequent, intense, and sustained emotional reactions to both trauma-related and non-trauma-related stimuli might lead to and maintain hyperarousal, negative cognitions (e.g., “the world is not safe”), and avoidance of perceived threats (Brewin & Holmes, 2003). Indeed, studies have shown that heightened emotional reactivity to negative stimuli in trauma-exposed people can predict greater PTSD symptom severity (Badour & Feldner, 2013; Kimble et al., 2010). Further, a meta-analysis revealed that greater emotional reactivity (as assessed by allocation of attention) was associated with greater psychopathology, including PTSD (Bar-Haim et al., 2007).

Although heightened emotional reactivity to negative stimuli in trauma-exposed people has been the focus, there is also evidence that the processing of positive and neutral stimuli can also be dysregulated in this population. Diminished reactivity to positive stimuli is reflected in symptoms related to negative alterations in cognitions and mood (e.g., anhedonia), and might also be involved in the development and maintenance of other PTSD symptoms (Nawjin et al., 2015). Trauma-exposed people have been found to be less sensitive to rewards (Jatzko et al., 2006; Lieberman et al., 2017), indicating diminished reactivity to positive stimuli. Less reactivity to positive stimuli, in turn, is associated with PTSD symptoms in trauma-exposed people (Amdur et al., 2000; Weiss et al., 2018). However, the relationship between diminished reactivity to positive stimuli and PTSD symptoms remains unclear and some of the results have been mixed (e.g., Brown et al., 2016). Overall, the role of reactivity to positive stimuli in PTSD is understudied.

Finally, reactivity to neutral stimuli has been especially understudied in trauma-exposed people, despite its relevance to PTSD symptomology. Trauma-exposed people show greater attention to neutral stimuli (Dalgleish et al., 2001; Yoon & Weierich, 2016) and are less able to discriminate between negative and non-negative (i.e., neutral or positive) stimuli (Chu et al., 2016; Kleshchova et al., 2019). This is reflected in hyperarousal symptoms in PTSD, and hypervigilance (i.e., heightened vigilance for potential threats even in safe environments), in particular, can reflect heightened attention to and potentially more negative appraisal of neutral stimuli (APA, 2013).

**The Role of Event-related Potentials in PTSD Research**

These dysregulations in emotional processing might reflect underlying maladaptive neural activity and, consistent with the National Institute of Mental Health's Research Domain Criteria (RDoC), studying the neural mechanisms that might underlie symptomology might help to guide treatment improvement (Insel et al., 2010). The electroencephalogram (EEG) is one brain-imaging tool that can be useful for affective neuroscience research in humans. Event-related potentials (ERPs) are neural responses to stimuli that are recorded using EEG, and are useful and cost-effective neural markers of cognitive and emotional processes that are becoming increasingly prominent in psychopathology research (Nusslock, 2016). Variations in the latency and magnitude of several ERP components that are related to attention have been found to be associated with PTSD symptoms in trauma-exposed people (Lobo et al., 2015).

One ERP component that is emerging as a potential neural marker of dysregulated emotional reactivity is the late positive potential (LPP). The LPP is an ERP component typically measured at central and parietal areas along the midline of the scalp (Foti et al., 2009). It typically begins around 500 ms after the onset of a stimulus and can last as long as an individual attends to a stimulus (Hajcak & Olvet, 2008). The LPP is a correlate of attention to motivationally salient stimuli, such that a higher LPP amplitude can indicate greater attentional allocation to a stimulus (Schupp et al., 2004). As such, higher LPP amplitude to an emotionally salient stimulus can indicate a stronger emotional response to that stimulus (Hajcak et al., 2010). Changing one's appraisals of a stimulus (i.e., a form of emotion regulation that involves reinterpreting the meaning of the content of an image so that one's emotional reaction increases or decreases) can lead to corresponding alterations in LPP amplitude (Hajcak & Nieuwenhuis, 2006; Moser et al., 2006). Thus, the LPP can be a marker of the interconnected appraisal and attention components of emotion (Gross, 2010). Finally, the LPP is associated with activity in brain areas involved in emotional and visual processing that have been implicated in anxiety disorders, including the occipital cortex, the parietal cortex, the medial prefrontal cortex, and the amygdala (Liu et al., 2012; Sabatinelli et al., 2007).

**The Late Positive Potential and Trauma-exposure**

Given evidence pertaining to the significance and utility of the LPP in affective neuroscience, it might be a marker of the kind of dysregulated emotional reactivity that is cardinal to PTSD symptomology in trauma-exposed people. However, studies of the LPP in trauma-exposed people have found mixed results. A series of studies have found that veterans with PTSD, compared to those without, experience smaller LPPs to angry faces (MacNamara et al., 2013), suggesting a blunted emotional reactivity negative social cues in people with PTSD. However, another study by the same group found no independent relationship between PTSD symptoms and LPP amplitude to angry faces, but did find an interaction wherein individuals with greater perseverative errors, indexed by the Wisconsin Card Sorting Test (WCST), and relatively greater LPP amplitude to angry faces, also had relatively  more PTSD symptoms (DiGangi et al., 2017). Further analysis revealed no differences in LPP amplitude to fearful faces between veterans with and without PTSD, but there was an association between LPP amplitude to fearful faces and greater intrusive symptoms (DiGangi et al., 2017). DiGangi et al. (2018) found that greater post-deployment stress in veterans was associated with greater LPP amplitude to faces of all emotion types, while PTSD symptoms were associated with attenuated LPP amplitude in response to all emotion types. These mixed results suggest that there might be multiple moderators that affect the relationship between LPP amplitude to negative social cues and PTSD symptomology. Finally, contrary to studies of veterans, a study of adults with a history of childhood abuse found that those individuals, compared to no-trauma controls, exhibited higher LPP amplitudes to fearful faces but not to angry faces (Sandre et al., 2018). This suggests that trauma type might also be an important moderator of the LPP in response to emotional social cues.

Social cues, however, are only one type of emotional stimulus, and PTSD is characterized by dysregulated emotional processing in response to a variety of stimuli. A handful of studies have examined the effects of trauma-exposure on the LPP response to other kinds of stimuli. A study that used images taken from the International Affective Picture System (IAPS; Lang et al., 2008) found no differences in LPP amplitude in response to negative images between veterans with and without PTSD (Fitzgerald et al., 2016). This study also found no differences in the change in LPP amplitude during down-regulation of emotion. However, diminished ability to down-regulate reactivity to negative stimuli, as indexed by LPP amplitude, predicted greater symptoms of PTSD upon 6-month and 12-month follow-up (Fitzgerald et al., 2018). Another study that used unpleasant odors as a stimulus to invoke the LPP found no difference in LPP amplitude between veterans with and without PTSD but, within the PTSD group, greater PTSD symptomatology was associated with greater LPP response (Bedwell et al., 2018). Taken together, these results suggest that emotional reactivity and regulation ability, as measured by LPP amplitude, might not be predictive of a PTSD diagnosis, but it might predict prospective fluctuations in symptomatology, consistent with an RDoC approach.

Much of the work on trauma-exposure and the LPP to date has focussed on combat-exposed veterans, and few studies have tested the LPP-PTSD symptom relationship in other trauma-exposed populations. A study of trauma-exposed youth found no differences in LPP amplitude between those with PTSD and those without, but did find that the sample overall exhibited greater LPP amplitude in response to negative images irrespective of trauma exposure (Grasso & Simons, 2012). However, a different study that used emotional words found that youth with PTSD showed greater LPP amplitudes to social-threat words compared to controls (Klein et al., 2019). A study of trauma-exposed college students found that people with more post-traumatic stress symptoms had greater neural reactivity to negative compared to neutral images, as indexed by the LPP (Lobo et al., 2014).

The current literature on the LPP as a predictor of PTSD symptoms is inconsistent. Methods and stimulus types have varied between studies, and much of the work thus far has focussed on veterans and combat-related trauma. Fewer studies have tested the relationship between the LPP and PTSD symptoms in other populations, and those that have have limited their focus to a specific type of trauma (e.g., history of childhood abuse; Sandre et al., 2018). Further, many studies have only tested differences between trauma-exposed individuals with and without PTSD, rather than taking a dimensional approach to psychopathology that would be more consistent with RDoC principles (Insel et al., 2010) and might be more appropriate for studying maladaptive neural processes related to PTSD symptoms (Lobo et al., 2015).

**The Current Study**

The purpose of the current study was to test the associations between LPP amplitude and PTSD symptoms in a sample of undergraduates who reported experiencing a variety of Criterion A traumas. We used positive, negative, and neutral IAPS images in order to represent a wide variety of emotional stimuli, beyond simple social cues (i.e., emotional faces). This allowed us to determine whether neural reactivity to neutral and positive stimuli, not just negative stimuli, can also be predictive of PTSD symptoms. Finally, we took a dimensional approach to psychopathology in our analyses, rather than testing for differences between individuals who meet diagnostic criteria or not. We hypothesized that trauma-exposed people would exhibit the greatest emotional reactivity, indexed by higher LPP amplitude, to negative images, compared to neutral and positive images. We also hypothesized that emotional reactivity to negative images, indexed by LPP amplitude, would predict PTSD symptoms. We did not have a priori hypotheses about specific PTSD symptoms clusters, or about the relationships between PTSD symptoms and neutral or positive images.

# II. Method

## Participants

Students from an undergraduate introductory psychology class, who reported on a prescreen that they had experienced a traumatic event, were recruited as participants for this study. Participants who did not report experiencing a traumatic event themselves were not included in analyses. Participants who reported taking any psychoactive medications, a history of neurological disease, or regular use of tobacco, cannabis, or other substances were excluded. Participants were instructed to refrain from consuming caffeine, alcohol, or illicit drugs, smoking, and exercising two hours prior to the start of the study. A total of 112 people participated. Data from 16 were excluded due to poor or no EEG recordings; data from 8 were excluded due to self-reported psychotropic medication use; and data from 12 were excluded due no self-reported experience of traumatic events upon reassessment during the experimental session. The final sample size for the current study was 76. Participant demographics are displayed in Table 1 and clinical data are displayed in Table 2.

## Self-report Measures

### *Life Events Checklist for DSM-5 (LEC-5)*

The LEC-5 (Weathers et al., 2013) was used to determine the trauma history of participants. The LEC-5 is 17-item, self-report questionnaire that asks participants to indicate whether they a) experienced a traumatic event themselves, b) witnessed the event, c) learned about the event happening to someone close to them, d) were exposed to the event as part of their job, e) are not sure if they experienced the event, or f) the event does not apply to them. The LEC-5 has been shown to be a reliable and valid measure of likely trauma-exposure according to DSM-5 criteria (Weathers et al., 2013).

*PTSD Checklist for the DSM-5 (PCL-5)*

The PCL-5 (Weathers et al., 2013), a 20-item, self-report questionnaire, was used to assess PTSD symptomology related to a traumatic event. Participants rated how much each DSM-5 symptom of PTSD has bothered them over the past month using a scale that ranged from 0 (“not at all”) to 5 (“extremely”). The PCL-5 can be used to assess total PTSD symptom severity, as well as severity of symptoms in each of the four DSM-5 PTSD symptom clusters (i.e., re-experiencing, avoidance, negative changes in mood and cognitions, and hyperarousal). The PCL-5 has been shown to be a reliable and valid measure of DSM-5 PTSD symptoms in trauma-exposed undergraduates (Blevins et al., 2015). Participants were instructed to complete the PCL-5 in regard to symptoms related to the traumatic event that affected them the most.

*Center for Epidemiological Studies Depression Scale (CES-D)*

The CES-D (Radloff, 1977), a 20-item, self-report questionnaire, was used to assess depressive symptomology in the past week. Total scores range from 0 to 60, and a score of 16 or higher indicates greater than mild depressive symptoms. The CES-D has been shown to be a reliable and valid measure of depressive symptoms (Radloff, 1977).

*Beck Anxiety Inventory (BAI)*

The BAI (Beck et al., 1988), a 21-item, self-report questionnaire, was used to assess anxiety symptom severity in the past month. Total scores range from 0 to 63 and a score of 19 or higher indicates greater than mild anxiety. The BAI has been shown to be a reliable and valid measure of anxiety symptoms (Beck et al., 1988).

**IAPS Task**

The International Affective Picture System (IAPS; Lang et al., 2008) is a standardized set of images, rated on valence and arousal by large numbers of subjects, that is commonly used in affective science research. Stimuli for this study included 80 positive images (40 medium arousal, 40 high arousal), 80 negative images (40 medium arousal, 40 high arousal), and 80 neutral images (40 low arousal, 40 medium arousal). This stimulus set has been shown to reliably evoke the late positive potential (LPP) in a variety of populations and across developmental stage (Hajcak et al., 2010). Images from the IAPS were selected for this study to have valence and arousal ratings comparable to those of other studies (e.g., Fitzgerald et al., 2016). A list of all the images used in this study is available in Appendix B. Images were presented at random for 2000 ms each. A fixation cross appeared on screen for 500 ms prior to each stimulus onset. The task was divided into 3 blocks, consisting of 80 images each, and participants were given the opportunity to take a short break before continuing to the next block.

*ERP Recording and Processing*

Electroencephalograph data were recorded using Neuroscan Synamps 2 (Herdon, VA) amplifiers and an electrode cap (Electro-Cap International, Eaton, OH) with 19 scalp electrode sites based on the International 10/20 System (AFz, Fz, F3, F4, F7, F8, FCz, Cz, C3, C4, T3, T4, T7, T8, Pz, P3, P4, O7, O8) and 4 additional electrodes for the left and right mastoids (M1 and M2) and above and below the left eye (VEOG). The sampling rate was 1000 Hz. Signals were referenced online to M1 and re-referenced offline to the average of M1 and M2. AFz was used as the ground electrode. Impedance at each elecotrode site was kept below 5 kΩ.

Eye-blink artifact was corrected using a previously validated linear regression method (Gratton et al., 1983). An offline, band-pass filter was applied at 0.1-30 Hz. Trial data were separated into epochs starting 500 ms prior to stimulus onset and ending 2000 ms after stimulus onset. Each epoch was baseline corrected using a baseline period of -50 ms to 0 ms. Artifact detection procedures were only applied to Pz because this was the only site used to measure the LPP. A moving window artifact detection algorithm in the EEGLab signal processing software package was used to detect and delete trials with a voltage change of 100 μV within a 200 ms window. In addition, trials with amplitudes greater than 75 μV or lower than -75 μV were also deleted. The mean amplitude of the late positive potential (LPP) was measured between 500 ms after stimulus onset and 2000 ms after stimulus onset at Pz. Grand average ERPs are displayed in Figure 1.

**Procedure**

After providing informed consent, participants completed a series of questionnaires to assess trauma status and PTSD symptoms. Participants were then fitted with an EEG cap by a trained experimenter. Participants were also fitted with 3 electrocardiogram (EKG) electrodes (one on each wrist and one on the left ankle) as part of data collection unrelated to the current study. A set of resting-state physiological recordings that lasted for 8 min preceded the IAPS task. Prior to starting the ERP task participants were told: “You will be shown a series of different pictures on the screen. The pictures can range from very pleasant to very unpleasant. Just look at the pictures and try to take in as much information about each image as possible.” Participants freely viewed each image as it was presented on a computer screen in front of them while EEG data were recorded.

III. Results

**Differences in the Late Positive Potential by Valence**

Mean LPP amplitudes are displayed in Table 3. We conducted a within-subjects ANOVA to test for differences in LPP amplitude in response to negative, neutral, and positive images. Mauchly's Test of Sphericity indicated that the assumption of sphericity had not been violated, $\chi^2$ (2) = 0.367, $p$ = 0.833.There was a significant effect of valence, $F$ (2, 150) = 12.89, $p < 0.001$, $\eta_p^2 = 0.147$. Paired-samples $t$-tests tests revealed that LPP amplitude to negative images was higher than that to neutral images, $t$ (75) = 5.11, $p <$ 0.001, $d$ = 0.429, and positive images, $t$ (75) = 2.37, $p$ = 0.021, $d$ = 0.201. LPP amplitude to positive images was also higher than that to neutral images, $t$ (75) = 2.71, $p$ = 0.008, $d$ = 0.221. Means are displayed in Figure 2.

**Association between LPP amplitude and PTSD symptoms**

Hierarchical regression analyses were conducted to test whether LPP amplitude predicts PTSD symptoms. Gender, depression symptoms, and anxiety symptoms were entered into step 1, and LPP amplitude to negative, neutral, and positive images were entered into step 2. Correlations between variables are presented in Table 5. The summaries of the regression analyses are presented in Tables 6-10. For total PTSD symptoms, the only significant predictor in step 1 was depressive symptoms, $\beta$ = 0.457, $p$ < 0.001. In step 2, the only significant predictor, again, was depressive symptoms, $\beta$ = 0.516, $p$ < 0.001. Regression analyses for each independent PTSD symptom cluster revealed similar results. For intrusive symptoms, the only significant predictor in step 1 was depressive symptoms, $\beta$ = 0.299, $p$ = 0.028. In step 2, the only significant predictor, again, was depressive symptoms, $\beta$ = 0.317, $p$ = 0.028. For avoidance symptoms, the only significant predictor in step 1 was depressive symptoms, $\beta = 0.494$, $p = 0.001$. In step 2, the only significant predictor, again, was depressive symptoms, $\beta = 0.520$, $p = 0.001$. For cognition and mood symptoms, the only significant predictor in step 1 was depressive symptoms, $\beta = 0.494$, $p < 0.001$. In step 2, the only significant predictor, again, was depressive symptoms, $\beta = 0.494$, $p = 0.001$. For hyperarousal symptoms, the only significant predictor in step 1 was depressive symptoms, $\beta = 0.445$, $p = 0.001$. In step 2, the only significant predictor, again, was depressive symptoms, $\beta = 0.492$, $p = 0.001$. LPP amplitude to negative, neutral, or positive images did not predict total PTSD symptoms or any PTSD symptom clusters, $p > 0.05$.

## IV. Discussion

The purpose of the current study was to test the relationship between emotional reactivity, indexed by LPP amplitude, and PTSD symptoms, in trauma-exposed individuals. We used negative, neutral, and positive images from a standardized stimulus set and assessed LPP amplitude to each image type as a predictor of self-reported PTSD symptoms. Consistent with recent approaches to psychopathology (Insel et al., 2010; Kotov et al., 2017), we sought to assess PTSD symptoms dimensionally, rather than based on diagnostic criteria. We also assessed the relationships between emotional reactivity and each PTSD symptom cluster individually, in addition to examining total PTSD symptoms.

Consistent with our first hypothesis, trauma-exposed participants experienced different LPP amplitudes to different image valences, such that negative images elicited the highest amplitudes, positive images the second highest, and neutral images the lowest. This is consistent with prior studies that have also found higher reactivity to negative stimuli in trauma-exposed adults (Lobo et al., 2014) and trauma-exposed youth (Grasso & Simons, 2012). Although some prior studies have found blunted reactivity to negative stimuli compared to positive stimuli (e.g., DiGangi et al., 2017), this effect might be unique to combat veterans responding to emotional faces. Studies of veterans that have used stimuli similar to those used in the current study (i.e, IAPS images) have found that veterans experience greater reactivity to negative stimuli compared to neutral stimuli (e.g., Fitzgerald et al., 2016; Woodward et al., 2015). Further, non-veteran trauma-exposed people also experience greater reactivity to negative faces (Sandre et al., 2018). The results of the current study add to these findings and suggest that non-veteran trauma-exposed people also experience heightened reactivity to negative stimuli, compared to positive and neutral stimuli.

It is unclear whether this heightened reactivity to negative stimuli is specific to trauma-exposed individuals. Prior studies have found that in normative samples, non-trauma-exposed people also experience greater reactivity to emotional stimuli (Schupp et al., 2004). However, results have been inconsistent as to whether there are differences in reactivity to negative and positive images in normative samples. Some studies have found no difference (e.g., Schupp et al., 2000), while others have found greater reactivity to negative images (e.g., Bradley et al., 2007). There are likely individual differences that play a role in both emotional reactivity and risk for psychopathology (e.g., trait neuroticism; Hill et al., 2019).

Contrary to our second hypothesis, LPP amplitude to neither negative, neutral, nor positive images predicted overall PTSD symptoms. Further, LPP amplitude to each valence failed to predict PTSD symptoms in any of the individual PTSD symptom clusters. In our model, gender, depressive symptoms, and anxiety symptoms explained 38.1% of the variability in PTSD symptoms, and the addition of reactivity to negative, neutral, and positive images only explained an additional 0.7%. Depressive symptoms was the only variable that significantly predicted overall PTSD symptoms and each PTSD symptom cluster. This is consistent with the high comorbidity between major depressive disorder and PTSD (APA, 2013; Elhai et al., 2011). These results suggest that reactivity to emotional stimuli might not predict PTSD symptoms in a non-clinical, trauma-exposed sample, or that such a relationship varies depending on sample characteristics or methodological factors, given the inconsistencies in the literature

Results from prior studies testing the LPP as a predictor of PTSD symptoms have, indeed, been inconsistent. Studies that found a blunted LPP to negative emotional faces also found an inverse relationship with PTSD symptoms (DiGangi et al., 2017; MacNamara et al., 2018). However, other studies that found higher reactivity in response to negative images (Lobo et al., 2014) and other stimuli (Bedwell et al., 2018) also found that greater reactivity was associated with greater PTSD symptoms. Still other studies have found no associations between emotional reactivity and psychopathology (Sandre et al., 2018).

Results from several studies suggest that there might be moderators that can influence the relationship between emotional reactivity and psychopathology. For example, higher perseverative thinking might interact with greater emotional reactivity to predict greater PTSD symptoms (DiGangi et al., 2017). Further, greater emotional reactivity might lead to greater psychopathology when post-trauma stress is high (DiGangi et al., 2018; Kujawa et al., 2016). The current study did not test any of these potential moderators. The inconsistencies in the literature might also be explained by methodological differences. Differences in trauma type (i.e., combat exposure vs. non-combat exposure) might reflect differences in processing of emotional information. Similarly, reactivity to certain types of stimuli (e.g., faces vs. scenes) might be better predictors of PTSD symptoms. Further research is required to determine what type of stimulus can best be used to predict psychopathology for which subpopulations of trauma-exposed people.

## Limitations

The present study had several limitations. First, the study did not include a no-trauma control group. Although the pattern of higher reactivity to negative compared to non-negative images in this study is similar to that of studies of non-trauma-exposed people (e.g., Schupp et al., 2004), there is evidence to suggest that trauma-exposed people experience greater reactivity to negative stimuli compared to no-trauma controls (Armstrong et al., 2013; Patel et al., 2012). Thus, we were not able to test for the specific effects of trauma-exposure on emotional reactivity to emotional scenes. Second, our sample consisted of individuals that experienced a variety of different traumas, and although one of our goals was to test whether prior findings generalize to non-veterans populations, we were not able to control for the potential effects that type of trauma can have on the LPP. Third, although our sample represented a range of PTSD symptomology, and 25% percent of our sample had PCL-5 scores consistent with a likely PTSD diagnosis, it is unclear whether our sample was relatively higher in functioning compared to a clinical-seeking population. Finally, the current study used self-report measures of trauma-exposure that although have been found to be valid and reliable (Blevins et al., 2015; Weathers et al., 2013), might not have the same diagnostic accuracy as gold-standard structured interviews.

## Implications

The mixed results found in studies of the LPP in trauma-exposed people might reflect differing neurobiological profiles that result in PTSD (Michopoulos et al., 2015). Given how heterogeneous PTSD is in terms of potential symptom profiles (Galatzer-Levy & Bryant, 2013), it is possible that the neurobiological mechanisms that underlie these different phenotypes are similarly heterogeneous. As such, future studies should work to determine how factors such as trauma type, time since trauma, and gender or sex, can affect emotional reactivity as it is represented by brain activity. Further, the time-course of attention to emotional stimuli might be relevant to the development of PTSD symptoms (Lazarov et al., 2019). Thus, potential differences in the LPP at different points in viewing a stimulus should also be tested. Maladaptive reactivity to trauma-related stimuli might also be more relevant for the development of PTSD symptoms than reactivity to general threat stimuli (Thomas et al., 2013). However, this relationship remains untested using the LPP and future studies should incorporate trauma-related stimuli. Finally, given that the LPP can be used to assess emotion regulation (Hajcak et al., 2010), it might be best used to assess maladaptive processes that underlie psychopathology transdiagnostically.

**Conclusion**

This study sought to test differences in reactivity to emotional stimuli, and the relationship between emotional reactivity and PTSD symptoms, in trauma-exposed people. We found that trauma-exposed people experience greater reactivity to negative stimuli, compared to positive and neutral stimuli, but that this reactivity does not predict PTSD symptoms. Our study contributes to the literature by taking a dimensional approach to psychopathology, and by focusing on a non-veteran sample. Further research is required to determine whether the LPP can serve as a useful marker of neurobiological processes that underlie or maintain psychopathology.

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Appendix A

Table 1.

*Demographics*

| | |
|---|---|
| Age, *M* (*SD*) | 19.2 (3.3) |
| Sex, *n* (%) | |
| Male | 32 (42.1%) |
| Female | 44 (57.9%) |
| Race/ Ethnicity, *n* (%) | |
| African-American/ African | 5 (6.6%) |
| Asian | 40 (52.6%) |
| Caucasian | 11 (14.5%) |
| Hispanic/ Latino | 15 (19.7%) |
| Multiple | 2 (2.6%) |
| Other | 3 (3.9%) |

Table 2.

*Clinical Characteristics*

| | |
|---|---|
| PCL-5, *M* (*SD*) | |
| Intrusive symptoms | 5.38 (4.46) |
| Avoidance symptoms | 3.50 (2.58) |
| Cognitive/ Mood symptoms | 8.09 (6.42) |
| Hyperarousal symptoms | 5.27 (4.79) |
| Total symptoms | 22.20 (15.55) |
| PCL-5 ≥ 33, *n* (%) | 20 (26.3%) |
| CESD, *M* (*SD*) | 20.3 (10.83) |
| BAI, *M* (*SD*) | 13.56 (9.44) |
| Trauma Experienced, *n* (%) | |
| Natural disaster | 43 (56.6%) |
| Fire or explosion | 6 (7.9%) |
| Transportation accident | 31 (40.8%) |
| Serious accident or injury | 15 (19.7%) |
| Exposure to toxic substance | 5 (6.6%) |
| Physical assault | 43 (56.6%) |
| Assault with weapon | 10 (13.2%) |
| Sexual assault | 16 (21.1%) |
| Other unwanted sexual experience | 29 (38.2%) |
| Combat or warzone exposure | 1 (1.3%) |
| Captivity | 0 (0%) |
| Life-threatening illness or injury | 5 (6.6%) |
| Severe human suffering | 2 (2.6%) |
| Sudden violent death | 4 (5.3%) |

| | |
|---|---|
| Sudden accidental death | 3 (3.9%) |
| Serious injury or death to another | 6 (7.9%) |

Note: PCL-5 - PTSD Checklist - 5, CESD - Center for Epidemiological Studies Depression Scale, BAI - Beck Anxiety Inventory, percentages do not sum to 100% because most participants reported experiencing multiple traumatic-events

Table 3.
*Mean Valence and Arousal Ratings for IAPS Images*

| Image Type *M, (SD)* | Valence Ratings | Arousal Ratings |
|---|---|---|
| Negative | 2.73 (1.63) | 5.52 (2.16) |
| Neutral | 4.92 (1.40) | 3.46 (1.97) |
| Positive | 7.23 (1.60) | 5.37 (2.28) |

Note: Values are normative ratings from Lang et al., 2008.

Table 4.
*Mean LPP Amplitudes*

| Image Type *M, (SD)* | |
|---|---|
| Negative | 2.79 (2.66) |
| Neutral | 1.67 (2.54) |
| Positive | 2.25 (2.68) |

Note: Values are measured in μV

Table 5

*Correlations between Study Variables*

| | Gender | CESD | BAI | PCL total | PCL B | PCL C | PCL D | PCL E | LPP Negative | LPP Neutral | LPP Positive |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Gender | - | | | | | | | | | | |
| CESD | 0.194 | - | | | | | | | | | |
| BAI | 0.294* | 0.644*** | - | | | | | | | | |
| PCL total | 0.192 | 0.598*** | 0.491*** | - | | | | | | | |
| PCL B | 0.206 | 0.469*** | 0.479*** | 0.805*** | - | | | | | | |
| PCL C | 0.166 | 0.439*** | 0.247* | 0.799*** | 0.799*** | - | | | | | |
| PCL D | 0.161 | 0.546*** | 0.389* | 0.903*** | 0.556*** | 0.695*** | - | | | | |
| PCL E | 0.133 | 0.572*** | 0.486*** | 0.863*** | 0.606*** | 0.554*** | 0.711*** | - | | | |
| LPP Negative | 0.207 | 0.034 | 0.022 | -0.020 | 0.017 | -0.104 | -0.041 | 0.036 | - | | |
| LPP Neutral | -0.009 | -0.196 | -0.188 | -0.118 | -0.100 | -0.156 | -0.136 | -0.029 | 0.732*** | - | |
| LPP Positive | -0.049 | 0.023 | -0.105 | -0.086 | -0.088 | -0.136 | -0.046 | -0.060 | 0.725*** | 0.746*** | - |

$^*p \leq 0.05$, $^{**}p \leq 0.01$, $^{***}p \leq 0.001$

Note: $n = 76$, PCL-5 - PTSD Checklist, PCL B - intrusive symptoms, PCL C - avoidance symptoms, PCL D - mood and cognitive symptoms, PCL E - hyperarousal symptoms, CESD - Center for Epidemiological Studies Depression Scale, BAI - Beck Anxiety Inventory

Table 6.
*Hierarchical Regression Analysis of Total PTSD symptoms*

| | PCL Total | | | | |
|---|---|---|---|---|---|
| | β | *t* | $sr^2$ | $R^2$ | $\Delta R^2$ |
| Step 1 | | | | 0.381 | 0.381 |
| Gender | 0.039 | 0.392 | 0.001 | | |
| CESD | 0.487 | 3.938*** | 0.139 | | |
| BAI | 0.165 | 1.303 | 0.015 | | |
| Step 2 | | | | 0.388 | 0.007 |
| Gender | 0.032 | 0.300 | | | |
| CESD | 0.516 | 3.949*** | 0.144 | | |
| BAI | 0.161 | 1.238 | 0.014 | | |
| LPP Negative | 0.002 | 0.015 | $1x10^{-6}$ | | |
| LPP Neutral | 0.120 | 0.778 | 0.005 | | |
| LPP Positive | -0.097 | -0.648 | 0.004 | | |

*p ≤ 0.05, **p ≤ 0.01, ***p ≤ 0.001

Table 7.
*Hierarchical Regression Analysis of Intrusive Symptoms*

| | PCL Cluster B | | | | |
|---|---|---|---|---|---|
| | β | *t* | $sr^2$ | $R^2$ | $\Delta R^2$ |
| Step 1 | | | | 0.285 | 0.285 |
| Gender | 0.057 | 0.539 | 0.003 | | |
| CESD | 0.299 | 2.246* | 0.052 | | |
| BAI | 0.270 | 1.977 | 0.040 | | |
| Step 2 | | | | 0.290 | 0.005 |
| Gender | 0.042 | 0.364 | 0.001 | | |
| CESD | 0.317 | 2.252* | 0.055 | | |
| BAI | 0.265 | 1.885 | 0.038 | | |
| LPP Negative | 0.051 | 0.319 | 0.001 | | |
| LPP Neutral | 0.067 | 0.403 | 0.002 | | |
| LPP Positive | -0.900 | -0.554 | 0.003 | | |

*p ≤ 0.05, **p ≤ 0.01, ***p ≤ 0.001

Table 8.

*Hierarchical Regression Analysis of Avoidance Symptoms*

| | PCL Cluster C | | | | |
|---|---|---|---|---|---|
| | β | *t* | $sr^2$ | $R^2$ | $\Delta R^2$ |
| Step 1 | | | | 0.209 | 0.209 |
| Gender | 0.074 | 0.662 | 0.005 | | |
| CESD | 0.494 | 3.530*** | 0.143 | | |
| BAI | -0.930 | -0.650 | 0.005 | | |
| Step 2 | | | | 0.222 | 0.013 |
| Gender | 0.088 | 0.734 | 0.006 | | |
| CESD | 0.520 | 3.530*** | 0.147 | | |
| BAI | -0.105 | -0.715 | 0.006 | | |
| LPP Negative | -0.101 | -0.600 | 0.004 | | |
| LPP Neutral | 0.080 | 0.457 | 0.003 | | |
| LPP Positive | -0.079 | -0.465 | 0.003 | | |

*p ≤ 0.05, **p ≤ 0.01, ***p ≤ 0.001

Table 9.
*Hierarchical Regression Analysis of Mood and Cognitive Symptoms*

| | PCL Cluster D | | | | |
|---|---|---|---|---|---|
| | β | *t* | $sr^2$ | $R^2$ | $\Delta R^2$ |
| Step 1 | | | | 0.296 | 0.296 |
| Gender | 0.043 | 0.408 | 0.002 | | |
| CESD | 0.494 | 3.741*** | 0.143 | | |
| BAI | 0.058 | 0.427 | 0.002 | | |
| Step 2 | | | | 0.296 | 0.000 |
| Gender | 0.049 | 0.428 | 0.002 | | |
| CESD | 0.494 | 3.526*** | 0.132 | | |
| BAI | 0.060 | 0.432 | 0.002 | | |
| LPP Negative | -0.023 | -0.145 | $2x10^{-4}$ | | |
| LPP Neutral | 0.011 | 0.069 | $5x10^{-5}$ | | |
| LPP Positive | 0.017 | 0.107 | $1x10^{-4}$ | | |

*p ≤ 0.05, **p ≤ 0.01, ***p ≤ 0.001

Table 10.
*Hierarchical Regression Analysis of Hyperarousal Symptoms*

| | PCL Cluster E | | | | |
|---|---|---|---|---|---|
| | β | *t* | $sr^2$ | $R^2$ | $\Delta R^2$ |
| Step 1 | | | | 0.343 | 0.343 |
| Gender | -0.005 | -0.047 | $3x10^{-5}$ | | |
| CESD | 0.445 | 3.320*** | 0.106 | | |
| BAI | 0.070 | 0.186 | 0.017 | | |
| Step 2 | | | | 0.379 | 0.036 |
| Gender | -0.040 | -0.375 | 0.001 | | |
| CESD | 0.492 | 3.613*** | 0.125 | | |
| BAI | 0.179 | 1.285 | 0.016 | | |
| LPP Negative | 0.108 | 0.697 | 0.005 | | |
| LPP Neutral | 0.208 | 1.299 | 0.016 | | |
| LPP Positive | -0.216 | -1.417 | 0.019 | | |

*p ≤ 0.05, **p ≤ 0.01, ***p ≤ 0.001

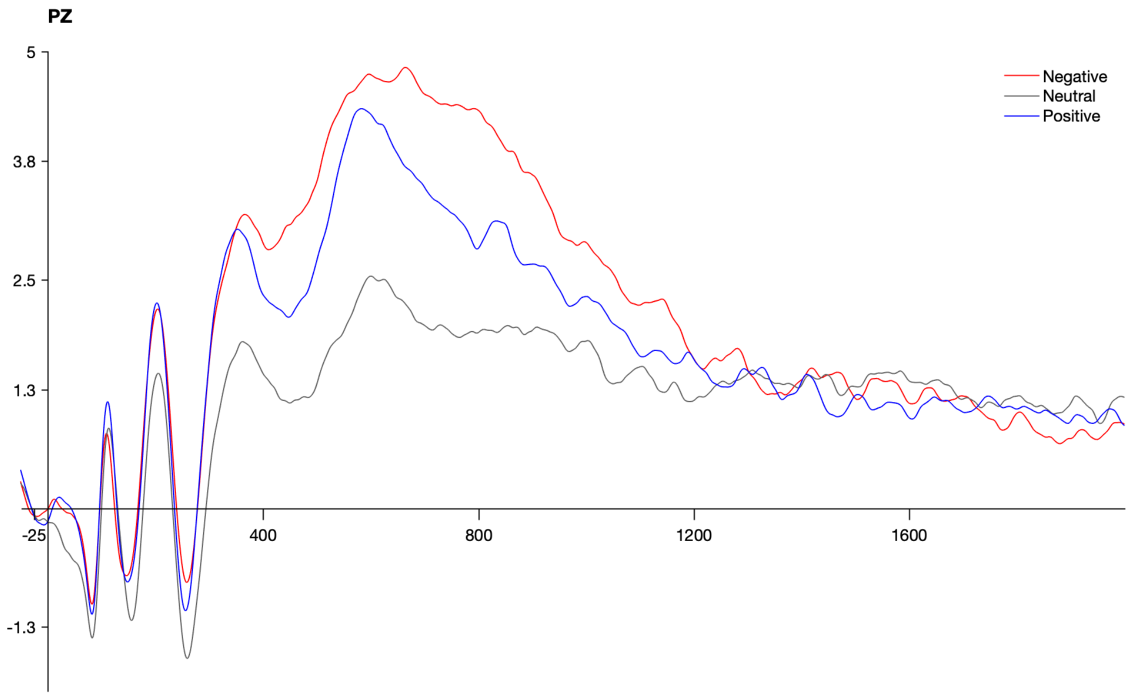


*Figure 1*. Grand average ERPs for negative, neutral, and positive scenes at electrode site Pz.

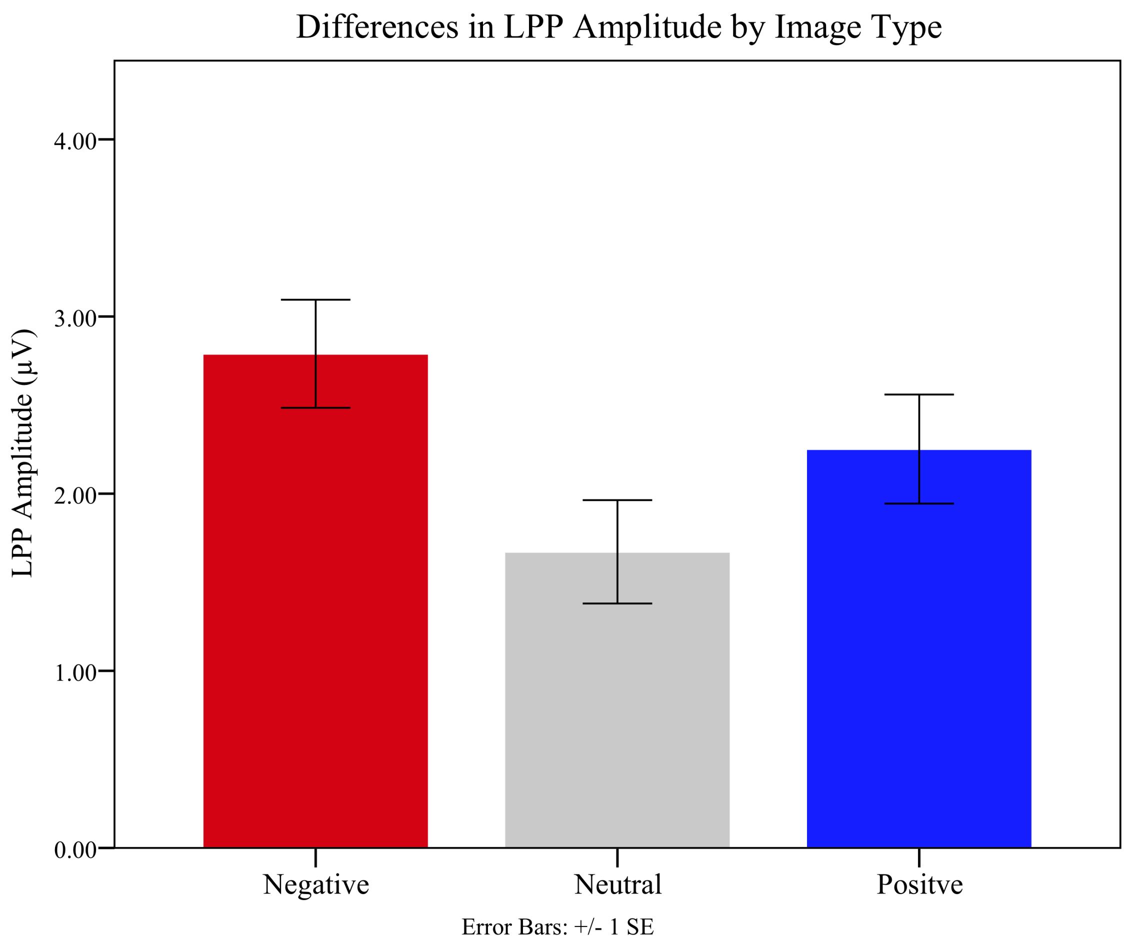


*Figure 2*. Bar graph representing mean LPP amplitudes for negative, neutral, and positive images.

Appendix B

Table 11.

*Negative images used for IAPS task*

| | | | | |
|---|---|---|---|---|
| 1111 | 1120 | 1201 | 1205 | 1275 |
| 1280 | 2053 | 2095 | 2110 | 2120 |
| 2141 | 2205 | 2276 | 2312 | 2375.1 |
| 2399 | 2455 | 2490 | 2683 | 2688 |
| 2692 | 2694 | 2710 | 2800 | 3030 |
| 3051 | 3071 | 3102 | 3160 | 3180 |
| 3230 | 3280 | 3300 | 3350 | 3530 |
| 6212 | 6230 | 6260 | 6311 | 6370 |
| 6415 | 6510 | 6550 | 6561 | 6570 |
| 6571 | 6830 | 6831 | 7380 | 8230 |
| 8485 | 9001 | 9040 | 9041 | 9050 |
| 9090 | 9101 | 9120 | 9140 | 9181 |
| 9250 | 9265 | 9331 | 9410 | 9415 |
| 9432 | 9440 | 9530 | 9584 | 9592 |
| 9611 | 9810 | 9910 | 9911 | 9921 |

Note: Stimuli taken from the International Affective Picture System (Lang et al., 2008.)

Table 12.

*Neutral images used for IAPS task*

| | | | | |
|---|---|---|---|---|
| 1121 | 1935 | 1945 | 1947 | 2025 |
| 2190 | 2191 | 2200 | 2206 | 2215 |
| 2220 | 2221 | 2271 | 2280 | 2372 |
| 2381 | 2385 | 2393 | 2394 | 2410 |
| 2440 | 2441 | 2480 | 2487 | 2499 |
| 2575 | 2579 | 2595 | 2600 | 2795 |
| 2840 | 2880 | 4605 | 5120 | 5130 |
| 5395 | 5455 | 5534 | 5535 | 5731 |
| 5740 | 7000 | 7004 | 7006 | 7010 |
| 7025 | 7030 | 7031 | 7035 | 7036 |
| 7040 | 7140 | 7150 | 7175 | 7190 |
| 7207 | 7211 | 7217 | 7224 | 7233 |
| 7234 | 7235 | 7283 | 7491 | 7504 |
| 7560 | 7590 | 7620 | 7705 | 7950 |
| 8010 | 8211 | 8232 | 9070 | 9080 |
| 9171 | 9190 | 9210 | 9360 | 9913 |

Note: Stimuli taken from the International Affective Picture System (Lang et al., 2008.)

Table 13.

*Positive images used for IAPS task*

| | | | | |
|---|---|---|---|---|
| 1340 | 1440 | 1460 | 1510 | 1601 |
| 1650 | 1710 | 1721 | 1750 | 1811 |
| 1920 | 2030 | 2040 | 2057 | 2070 |
| 2080 | 2091 | 2160 | 2209 | 2216 |
| 2222 | 2299 | 2310 | 2389 | 2530 |
| 2550 | 4150 | 4220 | 4250 | 4255 |
| 4503 | 4520 | 4532 | 4533 | 4599 |
| 4608 | 4609 | 4625 | 4626 | 4653 |
| 4660 | 4689 | 5260 | 5450 | 5460 |
| 5470 | 5480 | 5600 | 5621 | 5623 |
| 5628 | 5629 | 5700 | 5831 | 5910 |
| 7230 | 7260 | 7270 | 7284 | 7330 |
| 7350 | 7430 | 7502 | 8021 | 8030 |
| 8032 | 8040 | 8080 | 8116 | 8161 |
| 8162 | 8185 | 8200 | 8210 | 8260 |
| 8341 | 8370 | 8470 | 8490 | 8496 |

Note: Stimuli taken from the International Affective Picture System (Lang et al., 2008.)