Distribution Agreement

In presenting this thesis as a partial fulfillment of the requirements for a degree from Emory University, I hereby grant to Emory University and its agents the non-exclusive license to archive, make accessible, and display my thesis in whole or in part in all forms of media, now or hereafter now, including display on the World Wide Web. I understand that I may select some access restrictions as part of the online submission of this thesis. I retain all ownership rights to the copyright of the thesis. I also retain the right to use in future works (such as articles or books) all or part of this thesis.

Irina Lucaciu April 14th, 2015

Emotional and Cognitive Content of Autobiographical Memories of Trauma in Women with Posttraumatic Stress Disorder: An fMRI and Narrative Analysis Study

by

Irina Lucaciu

Robyn Fivush

Adviser

Neuroscience and Behavioral Biology

Robyn Fivush

Adviser

Jennifer Stevens

Committee Member

Kerry Ressler

Committee Member

Tanja Jovanovic

Committee Member

2015

Emotional and Cognitive Content of Autobiographical Memories of Trauma in Women with Posttraumatic Stress Disorder: An fMRI and Narrative Analysis Study

By

Irina Lucaciu

Robyn Fivush

Adviser

An abstract of

a thesis submitted to the Faculty of Emory College of Arts and Sciences

of Emory University in partial fulfillment

of the requirements of the degree of

Bachelor of Sciences with Honors

Neuroscience and Behavioral Biology

2015

Abstract

Emotional and Cognitive Content of Autobiographical Memories of Trauma in Women with Posttraumatic Stress Disorder: An fMRI and Narrative Analysis Study

By Irina Lucaciu

Posttraumatic Stress Disorder (PTSD) is a debilitating memory disorder that develops in the aftermath of trauma. Nevertheless, some individuals who suffer comparable trauma become resilient to the disorder. To better understand resilience, previous studies have investigated differences in the emotional arousal experienced by individuals diagnosed with PTSD and resilient individuals when recalling trauma memories. A differential use of affective and cognitive language in trauma memories, as well as differential activation in amygdala and medial prefrontal cortex (mPFC), have been previously identified. Since no studies have examined emotional processing in traumatized individuals by simultaneously using narrative analysis and brain imaging, the present study aims to merge the two approaches. For this purpose, 40 African-American women (15 with PTSD and 25 trauma controls) were recruited from a publicly funded hospital in Atlanta, Georgia. A trained clinician interviewed all participants, and the narratives of the most traumatic childhood and adult events were transcribed and analyzed using the Linguistic Inquiry and Word Count (LIWC) program. A subset of 30 participants also viewed emotional images from the International Affective Picture System during functional magnetic resonance imaging. As predicted, participants with PTSD used significantly more affective words, and specifically more negative emotion words, than trauma controls ($ps < .05$) in their trauma narratives, but no difference was found in cognitive word use. Use of affective words was correlated to PTSD symptoms. There was a trend for less amygdala activation for the PTSD group ($p = .076$). No difference in mPFC activation was found ($p > .05$). However, we found that the more mPFC activation to positive images participants showed, the lesser affective and specifically negative emotion words they used in their trauma narratives. In conclusion, differences in emotional processing exist between traumatized individuals with or without PTSD, and they are evident in both the way they narrate their traumas and in their brain's reaction to emotional stimuli. This is the first study that we are aware of to examine the emotional content of trauma memories using both fMRI and narrative analysis methodologies.

Emotional and Cognitive Content of Autobiographical Memories of Trauma in Women with Posttraumatic Stress Disorder: An fMRI and Narrative Analysis Study

By

Irina Lucaciu

Robyn Fivush

Adviser

A thesis submitted to the Faculty of Emory College of Arts and Sciences

of Emory University in partial fulfillment

of the requirements of the degree of

Bachelor of Science with Honors

Neuroscience and Behavioral Biology

2015

Acknowledgements

I would like to thank my advisor, Dr. Robyn Fivush, for the continuous guidance, support, and feedback throughout the process. It has truly been an honor to be her student. All that I have learned from her during the past year as well as the intellectual confidence she has encouraged me to develop are a priceless gift.

I would also like to thank my committee members, Dr. Tanja Jovanovic, Dr. Kerry Ressler, and Dr. Jennifer Stevens, for making this thesis possible and for shaping my academic thinking and interests. I can only express my deepest gratitude to Dr. Stevens, who has been the most patient, understanding, and motivating research mentor I could have had during my time at Emory. My involvement in this project would not have been possible if Dr. Ressler would not have graciously carved time out of his unbelievingly busy schedule to talk to me about the Grady Trauma Project. Dr. Jovanovic has been a wonderful mentor, and her comments and feedback were very valuable.

I hold much gratitude for the Grady Trauma Project team, who has taught me compassion and dedication, and to the members of the Fivush Lab, who inspired me with their dedication to research. I also have special thanks to Timothy Eli for the much-appreciated help with analyzing the imaging data.

Finally, my family and friends were a crucial part of this process. I would like to highlight my mother for mastering the patience game during the many hours I talked to her about the project. I would like to extend special thanks to my roommate, Aneyn O'Grady, and my roommate from the previous year, Anusha Ravi, for making sure I stayed positive and optimistic during the transcription of numerous heartbreaking trauma stories. I would also like to thank Dr. Michael Crutcher, Dr. Paul Lennard, Nadia Brown, and Alan Weinstein from the Neuroscience and Behavioral Biology Department, and Andy Kim for his contagious energy around the topic of research. Thank you to the numerous other friends who have inquired about this thesis and showed interest in the topic.

Table of Contents

Introduction

Negative life events are part of human experience. The pain of losing a loved one, being physically hurt, or feeling lonely and abandoned is as disturbing as it is unavoidable for most individuals. In a majority of cases, the effects of negative life events on the social relations, behaviors, and mental wellbeing of the affected individuals diminish with the passage of time (Bonanno, 2004). Sometimes, however, negative life events can be highly arousing and have long-term consequences on the mental and emotional state of the individual. For the purpose of this research, the word "trauma" will refer to negative life events that include threats to an individual's physical and emotional integrity, situations in which the individual felt like his or her life was in danger, or situations that caused the individual to experience feelings of terror, horror, and helplessness (DSM-IV, American Psychiatric Association, 2000).

Some individuals who experience trauma have difficulties processing the memories of the traumatic event and develop Posttraumatic Stress Disorder (PTSD), a mental disorder that impacts their social and emotional health. Others, despite experiencing comparable amounts of trauma of similar intensity, are nevertheless able to become resilient to the pain and distress of trauma (Rothbaum, Foa, Riggs, Murdock, & Walsh, 1992; Bonanno, 2004). The objective of this study is to use both narrative analysis and functional imaging techniques to understand the way in which highly traumatized individuals cope with the traumatic events in their lives. Since PTSD is a disorder that disrupts memory, a first goal of this research is to determine whether resilient individuals differ from those who suffer from PTSD in the way they remember and narrate their most traumatic experiences. Because there is an overlap between the

neural regions involved in retrieving autobiographical trauma memories and regions involved in general emotional and cognitive processing (St. Jacques, Kragel, & Rubin, 2013), a second goal is to investigate whether PTSD manifests with differential processing of emotional stimuli in the emotional and cognitive centers of the brain. To our knowledge, there has been no previous research employing both the tools of narrative analysis combined with those of functional imaging to explore the way in which traumatized individuals process emotionally arousing stimuli following trauma. This research will shed light on the memory processes associated with PTSD by analyzing the narrative output of evoked traumatic memories in both diagnosed patients and resilient trauma controls. It will also advance the field by seeking to understand the way in which the activity of emotional and cognitive centers of the brain is affected by the experience of a major trauma. Additionally, I will explore the link between brain activation to emotional stimuli and narrative markers of emotion and cognition. The aim of this project is to ultimately support better protocols to treat and potentially prevent the development of PTSD by contributing to a more complete understanding of memory processes associated with the disorder.

To provide a framework for this project, I first define and describe the clinical characteristics and the social impact of PTSD by outlining the DSM-IV Criteria for diagnosis and by referring to previous studies assessing the risk and prevalence of the disorder. The second section explicates PTSD as a memory disorder, focusing on the processes involved in the processing of traumatic memories. The third section explains why looking at trauma narratives could give a glimpse into the content of trauma memories in PTSD-diagnosed and resilient participants. The fourth outlines current

knowledge about the neurobiology of PTSD. The fifth section explains why the parallel analysis of amygdala activation and narrative analysis is relevant to the study of PTSD, and the last section outlines the specific objectives and hypotheses of this study.

Clinical characteristics and social impact of PTSD

DSM-IV Criteria. For the purposes of this study, PTSD was defined using DSM-IV criteria. Criteria have since changed with the introduction of the DSM-5 (DSM-5, American Psychiatric Association, 2013). Post-traumatic stress disorder (PTSD) is a psychiatric disorder belonging to the "trauma and stressor related disorders" in the DSM-IV (DSM-IV, American Psychiatric Association, 2000). To be diagnosed with PTSD, a person must have experienced, witnessed, or been confronted with a stressor such as a death threat, a serious injury, or sexual violence, and have felt intense fear, helplessness, or horror as a result (Criterion A); experience intrusive thoughts such as flashbacks or nightmares (Criterion B); make efforts to avoid trauma-related stimuli (Criterion C); show increased arousal, such as difficulties falling or staying asleep, irritability, and hypervigilence (Criterion D); experience these symptoms for more than one month (Criterion E); and experience social and functional impairment as a result of these symptoms (Criterion F), (DSM-IV, American Psychiatric Association, 2000).

Risk for PTSD. PTSD is the fifth most common of the major psychiatric disorders, with approximately 8% of the United States population having it as a diagnosis (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). The prevalence of the disorder can vary significantly across populations depending on trauma exposure, with military combatants and low-income, highly traumatized populations being at higher risk than

others (Breslau et al., 1998; Kessler et al., 1995; Ogle, Rubin, Berntsen, & Siegler, 2013). Furthermore, women are twice as susceptible to PTSD compared to men (Breslau et al., 1998). While the likelihood of developing PTSD increases the more trauma a person is exposed to (Kessler et al., 1995; Ogle et al., 2013), approximately 60.7% of men and 51.2% of women experience a traumatic event that could qualify as a Criterion A in PTSD diagnosis during their lifetime (Kessler et al., 1995). Nevertheless, only a fraction of these traumatized populations will develop PTSD (Kessler et al., 1995). In contrast, individuals who experience significant trauma, but are resilient to the symptoms of PTSD, show optimism and high social functioning (Feder, Nestler, & Charney, 2009; Tugade & Friedrickson, 2004; Carver, 1997; Ong, Rubin, Berntsen, & Siegler, 2006). For that reason, resilient individuals form a particularly pertinent group for understanding why trauma leads to the selective development of PTSD, what differences exist between the ways in which resilient and affected individuals experience trauma and form a memory of it, and how neurophysiological differences between resilient and affected individuals can account for distinct mental health and behavioral outcomes.

Memory in PTSD

Trauma memories are autobiographical, and PTSD is a disorder of autobiographical memory (Rubin, Feldman, & Beckham, 2004; Rubin, Dennis, & Beckham, 2011). Past research suggests that the devastating symptoms of PTSD can be traced back to how a person encodes the memory of the traumatic event. That is to say, depending on how a person encodes the memory of a trauma, that trauma may or may not lead to long-lasting consequences, including PTSD. The encoding process, in turn, is

influenced by a combination of genetic and environmental factors that modulate the consolidation of fearful cues associated with the traumatic event (Parsons & Ressler, 2013). Some traumatized individuals do not only consolidate fear cues, but also recruit non-associated cues through a process called generalization. Thus, they become sensitized to the traumatic memory with repeated exposure and display the pathology of PTSD. In contrast, individuals who encode and reconsolidate the traumatic memory without associating it with unrelated cues are likely to recover after the trauma by gradually extinguishing, or diminishing, their fear response (Parsons & Ressler, 2013). According to this model, some traumatized individuals recall the traumatic memory in association with the flashbacks, negative thoughts, and hypervigilance specific to PTSD, and others recall it without experiencing the same amount of emotional arousal (Parsons & Ressler, 2013). In this study, only the way in which participants recalled their traumas, and not the way in which they encoded them, was assessed.

The way people incorporate the traumatic event within their life history plays a role into whether they will be resilient or affected by PTSD (Halligan, Clark, & Ehlers, 2002; Berntsen & Rubin, 2006). If the highly negative events in individuals' lives become central, that is, if they form reference points for their personal identity and for the attribution of meaning in future experiences, the outcome is likely to be the pathological behaviors of PTSD: ruminations, worries, flashback memories, and exaggerated attempts to avoid the recurrence of the event. There are two main views regarding the integration of traumatic memories within personal identity. The first view supports the idea that memories for traumatic events are better remembered than other autobiographical events (Berntsen, 2001; Porter & Birt, 2001; Revieve & Bakeman, 2001; Rubin, Feldman &

Beckham, 2004) precisely because they are highly emotional. Consequently, individuals with PTSD form trauma memories that become referential points for the organization of subsequent experiences, while resilient individuals form memories that do not negatively impact the attribution of meaning to new experiences (Porter & Birt, 2001; Berntsen et al., 2003; Robinaugh & McNally, 2011). The second view poses that traumatic events are poorly integrated in individuals' autobiographical memory, and that PTSD symptoms are largely an attempt to make sense of an event that cannot be understood within a life narrative context (Brewin, 1996; Horowitz, 1975; Horowitz & Reidbord, 1992; Nijenhuis & van der Hart, 1999; Van der Kolk & Fisher, 1995). In other words, this model describes PTSD as a memory disorder in which trauma memories are not cognitively integrated, and are thus more fragmented and disintegrated than the memories of resilient individuals (Herman, 1992; Berntsen et al., 2003). In a study trying to reconcile these two views, Berntsen, Willert, and Rubin (2003) found that individuals with PTSD had more vivid and emotional memories of their trauma than trauma controls. This finding is consistent with the idea that trauma memories are central in the life narrative of individuals with PTSD and are, therefore, significantly more emotional than the trauma memories of resilient individuals. It also suggests that cognitive processing is dysfunctional rather than absent in PTSD (Berntsen et al., 2003; Shobe & Kihlstrom, 1997; Porter & Birt, 2001; Rubin, Dennis, & Beckham, 2011).

Trauma narratives and markers of resilience

Autobiographical narratives of trauma are a reliable measure of how individuals remember the adverse events in their lives, and can give insight into how these

individuals process and make sense of their trauma. One of the indicators of resilience is active coping, which is defined as engaging in behaviors meant to relieve the effects of the traumatic event (Feder et al., 2009). Individuals who are able to cope face their trauma-related fears and do not let them interfere significantly with their day-to-day functioning, and show higher levels of optimism and positive attitudes (Alim et al., 2008; Carver, 1997; Ong et al., 2006).

Active coping, or its lack thereof, can be detected in narratives of trauma by analyzing the descriptive language that individuals use (Bohanek, Fivush, & Walker, 2005; Pennebaker, Mehl, & Niederhoffer, 2003). Cognitive words are defined as words that reveal the existence of an underlying cognitive process, that is, one that has as a goal the justification, organizing, and understanding of previous events (Boals & Rubin, 2011). People use cognitive words when they are trying to make sense of events or behaviors; therefore, the use of cognitive words can be a marker of actively coping with trauma (Boals & Rubin, 2011). Examples of cognitive words include “think”, “should”, and “because”, and a more comprehensive list can be found in Appendix B. Furthermore, the use of affective words – words that describe positive and negative emotional states, such as “nice”, “hurt”, and “crying” – is also of interest, as it gives insight into the emotional intensity of the memory (Rubin et al., 2008). Previous research suggests that information about the physical and mental states of individuals penetrates through the language they use (Gottschalk & Gleser, 1969; Rosenberg & Tucker, 1978; Stiles, 1992). Analyzing the structure of autobiographical memories in terms of cognitive and emotional processes could, therefore, be an appropriate source for determining the

existence of active coping, as well as explain the existence of PTSD symptoms or lack thereof (Berntsen & Rubin, 2006).

Neurobiological correlates of PTSD.

Given the significant involvement of the amygdala and prefrontal cortex in the pathology of PTSD, disruptions in the activity of these areas should be correlated to differences in how PTSD and resilient individuals remember traumatic events. In part because people differ in how they encode memories of traumatic events, some seem to be more resilient to the consequences of trauma than others (Feder et al., 2009). The bases for these differences in memory processing have been previously studied through several avenues. Neurochemical mechanisms may contribute to resilience, including differential activity between resilient and PTSD-diagnosed individuals at the level of the hypothalamus-pituitary-adrenal axis and serotonergic, dopaminergic, and noradrenergic systems (Wu et al., 2013; Feder et al., 2009). Another avenue of research involves examining various genes that may play a role in providing resilience (Stevens et al., 2014; Fani et al., 2013; Binder et al., 2008; Boscarino, Erlich, Hoffman, Rukstalis, & Stewart, 2011), and at epigenetic mechanisms, particularly methylation, by which the expression of these genes is either triggered or inhibited (Uddin et al., 2010). While the previously outlined methods provide a better understanding of enduring physiological and genetic characteristics linked to the pathology of PTSD, brain imaging reveals how the brains of those affected by PTSD modulate fear response, and how the activation of key brain areas involved in the processing of fearful memories differs from that of unaffected individuals. Furthermore, brain imaging also offers the chance to look at the

intersection of genetic and environmental risk factors for PTSD, since all of these risk factors operate on the brain.

The brain circuitry most relevant to the study of PTSD includes the hippocampus, amygdala, and prefrontal cortex, all areas associated with memory and cognitive processing. Because PTSD symptomology involves cognitive and emotional deficits, the amygdala – the emotion center of the brain – and the medial prefrontal cortex – the site of various cognitive functions – are of particular interest. The amygdala has been shown to be involved in fear conditioning (Davis and Whalen, 2001; LeDoux, 2000) and assessing threat (Whalen et al., 1998). The medial prefrontal cortex, by virtue of its connections to the amygdala, is involved in regulating emotional arousal to fearful cues (Milad et al., 2009). Patients diagnosed with PTSD show differential activation of both areas compared to trauma-exposed controls and trauma-unexposed controls (Banich et al., 2009). Specifically, individuals with PTSD show more amygdala activation when shown fearful faces or traumatic imagery (Rauch et al., 2000; Simmons et al., 2011; Shin et al., 2005; Armony et al., 2005; Fonzo et al., 2010) and when threatening sounds were played to them (Liberzon et al., 1999). Furthermore, the medial prefrontal cortex of PTSD patients activated less when they heard a traumatic script (Lanius et al., 2001; Bremner et al., 1999), when they viewed fearful faces (Shin et al., 2005; Williams et al., 2006), and when they underwent threat cue trials (Fani et al., 2013). Disruptions in emotion and cognition interactions were evident in the disruption of amygdala-prefrontal connectivity in both women (Stevens et al., 2013) and men (Sripada et al., 2012) with PTSD, as well as in a population including both sexes (Sadeh et al., 2014). Finally, both the medial prefrontal cortex and the amygdala have been linked to processing of emotional

autobiographical memories in PTSD (St Jacques, Botzung, Miles, & Rubin, 2011; Fossati, 2013).

Rationalization for the present study

A parallel analysis of activation in cognitive and emotional processing areas of the brain in response to emotional stimuli and of linguistic features of autobiographical trauma memories could give a multifaceted insight into the cognitive and emotional processes associated with the retrieval of memories of trauma. Since both medial prefrontal cortex activation and the use of cognitive words are markers of cognitive processing, and both amygdala activation and the use of affective words are markers of emotional processing, such parallel analysis could shed light on differences in how the brains of PTSD and resilient individuals process emotional stimuli, and how this difference in cognitive and emotional processing is reflected in autobiographical trauma memories. No previous study that we are aware of has addressed whether the differential brain activation observed in patients with PTSD is reflected in the way they remember their trauma memories.

Consequently, this research will investigate the differences in amygdala and prefrontal cortex activation between PTSD and resilient patients when shown emotionally arousing pictures during the fMRI scan. Furthermore, it will quantify the use of cognitive and emotional words in the autobiographical trauma memories of both groups. Specifically, this research will address whether the increased amygdala activation and decreased prefrontal cortex previously observed in individuals diagnosed with PTSD is

related to the differential use of cognitive and emotional words in these individuals' trauma memories.

Outline and scope of the present research

Previous studies suggest that individuals living in urban communities may be exposed to more trauma, particularly of the assaultive kind, and have a higher risk of developing PTSD than the rest of the population (Kessler et al., 1995; Breslau et al., 1998). Grady Memorial Hospital, a publicly funded facility that primarily serves the low socioeconomic status population of inner-city Atlanta, receives at-risk individuals, 88% of whom experience a major trauma; furthermore, 29% of Grady patients have been victims of childhood maltreatment (Gillespie et al., 2009). From those exposed to trauma, 46% have developed PTSD (Gillespie et al, 2009) at some point in their life. The Grady Trauma Project is an effort to determine the interaction of genetic and trauma-related risk factors in PTSD in this highly traumatized population at Grady.

The participants recruited for this research, which is done under the umbrella of the Grady Trauma Project, were 40 African American female between the ages of 22 and 61. Fifteen were previously diagnosed with PTSD through the administration of the Clinician-Administered PTSD Scale (CAPS), a psychological assessment tool that determines the existence of PTSD based on the DSM-IV-TR criteria (Blake et al., 1995). The other 25 participants were traumatized controls; that is, although they experienced trauma comparable in severity and frequency to the PTSD group, they did not meet enough of the DSM-IV-TR criteria to be diagnosed with PTSD.

First, all 40 participants were interviewed by a clinician to determine trauma history and psychiatric symptoms, with a particular emphasis on the traumatic events they experienced either during childhood or adulthood. The worst childhood and the worst adult traumatic event, which were also used by the clinician to diagnose PTSD, were transcribed from participants' videotaped descriptions of their trauma episodes. The choice to analyze both childhood and adult trauma memories was motivated by the distinct neurobiological influences that suffering trauma as a child or as an adult have on the brain. For example, child trauma may disrupt the regulation of emotional processing supported by the amygdala (Marusak, Martin, Etkin, & Thomason, 2015), while also being a significant risk factor for developing various neuropsychiatric disorders, among which PTSD is included (Kaufman et al., 2000; Birn et al., 2014). The resulting trauma narratives were analyzed with the Linguistic Inquiry and Word Count Software (LIWC), which counts the number of cognitive words and both positive and negative emotional words (Pennebaker, Booth, & Francis, 2007).

Functional imaging data was collected from a subset of 30 participants, 10 of which were diagnosed with PTSD and 20 of which were trauma controls. The BOLD response was recorded during a task that involves visualizing images that are expected to induce either a pleasant reaction (for example, a family smiling and enjoying each other's company), or a negative reaction (injuries, drug use, or violence). The regions of interest are the amygdala (following Amunts et al., 2005) and selected regions in the prefrontal cortex and along the cingulum that have been previously linked to cognitive processing and emotional regulation.

We predicted that pathological amygdala and prefrontal cortex activation would be observed when individuals with PTSD view emotionally arousing negative pictures – that is, PTSD patients were expected to show a hyperactive amygdala response, and a hypoactive prefrontal cortex response in comparison to resilient individuals. For the trauma narrative analysis part of this research, more affective words (and predominantly negative) and less cognitive words were expected to appear in the trauma narratives of individuals suffering from PTSD than in the narratives of resilient individuals, since PTSD manifests with downgraded emotional regulation and less active coping. Moreover, amygdala activation to emotional stimuli was expected to correlate positively with the frequency of affective words, and both amygdala activation and the frequency of affective words were expected to, in turn, correlate positively with the participants' scores on the mPSS. Less prefrontal cortex activation was expected in association with less usage of cognitive processing words, and prefrontal cortex activation and frequency of cognitive words were expected to correlate negatively with mPSS scores.

Methods

Participants

Forty African-American women ages 22 – 61 were recruited as part of an ongoing study of risk factors for Posttraumatic Stress Disorder. Participants were approached in the waiting rooms of general medical clinics of Grady Memorial Hospital, a publicly funded hospital that serves the inner-city population of Atlanta, Georgia. This patient population has high rates of trauma and posttraumatic symptoms, with 88% experiencing a major trauma and 46% being affected by PTSD (Gillespie et al., 2009; Binder et al., 2008). In addition, the hospital population is predominantly African-American, and therefore constitutes an under-represented group in psychiatric and imaging research studies. To enhance data homogeneity, only participants who self-identified as African-American were included in this study. All participants underwent an interview that included self-report measures on demographic characteristics, trauma exposure during childhood and adulthood, and current PTSD and depressive symptoms. Each participant received monetary compensation for taking part in the interview. The Institutional Review Board of Emory University and Research Oversight Committee of Grady Memorial Hospital approved the study procedures, and interviews took place at Grady Memorial Hospital. Clinical and demographic characteristics of the study population are available in Table 1.

For the functional imaging portion of the research, a subset of thirty participants was screened and met the following inclusion criteria: no neurological disorder, psychosis, current psychotropic medication, or metal clips or implants. Ten of the participants were diagnosed with PTSD, while the other twenty were trauma-exposed

controls. Individuals who endorsed a history of bipolar disorder, schizophrenia or any other psychotic disorder were excluded. Because PTSD and depression are highly co-morbid, participants with depression were not excluded from the study. Participants had normal or corrected-to-normal vision. Urine tests for pregnancy and illegal drug use (cocaine, marijuana, opiates, amphetamines, methamphetamines) were conducted 24 hours prior to the MRI scan, and individuals who showed positive results for pregnancy or drugs were excluded.

Psychological Assessment

The Modified PTSD Symptom Scale (PSS; Foa & Tolin, 2000) was used to assess PTSD symptoms, and the Traumatic Events Inventory (TEI) was used to assess types and severity of trauma experience. Childhood trauma was assessed using the Childhood Trauma Questionnaire (CTQ; Bernstein et al., 1994; Scher et al., 2001). Depression symptoms were assessed using the Beck Depression Inventory (BDI). These measures have been used in previous studies with this population (Binder et al., 2008; Fani et al., 2012; Schwartz et al., 2005). Traumatic experiences, depression, and PTSD symptoms were all assessed using the TEI, CTQ, BDI, and mPSS during the recruitment interview.

All participants later returned for a more in-depth interview conducted by a trained clinician. This interview further assessed trauma history and current PTSD symptomology using the Clinician-Administered PTSD Scale (CAPS). The CAPS is a structured interview that assesses the 17 symptoms of PTSD identified in the Diagnostic and Statistical Manual (DSM-IV; American Psychiatric Association, 2000). The CAPS includes standardized questions to determine the frequency and intensity of symptoms, as

well as standardized questions assessing subjective distress, and impairment in social and occupational functioning due to the PTSD symptoms. Symptoms were assessed for frequency and intensity in the preceding month using a 5-point Likert scale (e.g., 0 indicates that the symptom does not occur or does not cause distress and 4 indicates that the symptom occurs nearly every day or causes extreme distress and discomfort). Since the data were collected before the introduction of the DSM-5, PTSD diagnosis was based on DSM-IV-TR criteria (presence of trauma; presence of at least one reexperiencing symptom; presence of at least 3 avoidant/numbing symptoms; presence of at least 2 hyperarousal symptoms; occurrence for at least one month), as assessed by the mPSS. All participants had experienced at least one trauma. The clinician diagnosis was used to form the PTSD and the trauma-exposed control groups.

Comprehensive Trauma History Interviews

Participants were asked to describe in detail their worst childhood and adult traumas. Twenty-seven participants provided both narratives, five participants provided only adult trauma narratives, and eight participants provided only child trauma narratives. Overall, 67 trauma narratives were collected (32 adult trauma narratives and 35 child trauma narratives). Participants frequently described more than two traumas, in which case the clinician selected the worst childhood and worst adult traumas as index traumas for the PTSD diagnosis. Interviews were videotaped with the informed consent of each participant.

Linguistic Inquiry and Word Count (LIWC) Analysis

Two individuals who were blinded to the diagnosis of the narrator manually transcribed the narratives into text format from the videotaped interviews. The narratives of the index traumas were used in the current study. The transcripts were prepared for Linguistic Inquiry and Word Count (LIWC) 2007 (Pennebaker et al., 2007). LIWC is a dictionary-based package that reads textual input and classifies words into various psychologically-relevant categories. Each category is defined as a list of words and word-stems. For the present study, two major psychological categories of LIWC were used: Affective Processes and Cognitive Processes. Appendix A (courtesy of Pennebaker) lists all LIWC categories and gives examples of words in each category. Appendix B focuses on the categories used in the present study, giving more examples of words belonging to the Affective Words and Cognitive Words clusters. As outlined in Appendix A, subcategories of the Affective Word cluster include Positive Emotion, Negative Emotion, Anxiety, Anger, and Sadness. Subcategories of the Cognitive Word cluster include Insight, Causation, Discrepancy, Tentative, Certainty, Inhibition, Inclusive, and Exclusive. Some words belong to more than one subcategory (eg. “awkward” belongs to the Negative Emotion and Anxiety). LIWC has been previously validated (Kahn, Tobin, Massey, & Anderson, 2007; Bantum & Owen, 2009).

Functional Imaging Procedure

Before the experimental session, participants practiced the tasks that they later performed in the scanner to gain familiarity with the timing of the stimulus and the length of the response period. Practice involved viewing examples of scene stimuli and making a like / neutral / dislike judgment following the presentation of each scene.

The scene stimuli used during scanning were static photographic scenes from the International Affective Picture Series (IAPS; Lang, Bradley, & Cuthbert, 2008). Although these images do not differ with regard to visual features such as complexity and color, they do differ in emotional content. To ensure that participants paid attention to each picture stimulus, they were asked to rate their emotional reactions to each picture on a like / neutral / dislike scale using a button box. Thirty-six positive, 36 negative, and 36 neutral full-color scenes were presented in a semi-random order such that no more than two pictures of the same valence preceded another. Each picture was displayed full screen at a resolution of 1024x768 for 1.5 seconds, followed by a screen prompting the participant to make their rating of the picture presented for 1.5 seconds. The rating screen included a black background with "like / neutral / dislike" centered in the middle of the screen in white, 48pt Helvetica font. A white fixation cross centered on a black background followed each trial, comprising a jittered inter-trial interval of 1.5 – 2.5 seconds. The total length of the task was 9 minutes.

MRI acquisition and analysis

Scanning took place on a Siemens Trio 3T MRI scanner with echo-planar imaging (EPI) (Siemens, Malvern, PA). Structural images were acquired with a gradient-echo T1-weighted pulse sequence, with TR =2.30s, TE=0.03s, and 1x1x1mm voxel size. An EPI scout scan was then used to verify whole-brain coverage. EPI functional images were acquired using axial slices collected in an interleaved sequence (TR =2.00s, TE=0.03s, 3x3x3 mm voxel size). ArtRepair software was used to identify data artifacts (Mazaika, Whitfield-Gabrieli, & Reiss, 2007). Slices containing spike or motion artifacts were

identified and replaced using linear interpolation, with no more than 4% of slices repaired per participant (mean=0.02%). Volumes affected by motion were repaired using linear interpolation, with no more than 5% of volumes repaired per participant. Further preprocessing took place using statistical parametric mapping software (SPM8, Wellcome Department of Cognitive Neurology). Volumes were slice-timing corrected to the middle slice in time, and spatially realigned to the first image of the run. A 128Hz high-pass filter was used to remove low-frequency noise (Holmes, Josephs, Buchel, & Friston, 1997). T1s and co-registered functional images were normalized to the Montreal Neurological Institute (MNI) template. To verify that no participant had dropout in any substantial portion of the amygdala or other medial temporal lobe regions, functional images were visually examined for signal dropout. Images were then smoothed with an 8mm Gaussian kernel.

Contrast images reflecting increases in activation for the positive and negative pictures relative to the neutral pictures were created for each individual participant, and individual contrasts then entered group-level random effects analyses. Individual subjects' motion parameters were included as covariates. Activation was measured within *a priori* ROIs for the left and right amygdala, defined anatomically using the Anatomy Toolbox (Eickhoff et al., 2005), and the mPFC, defined using masks of Brodmann's areas 32, 24, and 10. The mean contrast value across voxels (for Negative> Neutral and Positive>Neutral contrasts) was extracted from each ROI, and statistical analyses of group differences and correlations with symptom severity and trauma exposure were conducted in SPSS 21.

Results

Results are presented in six sections, each focused on an individual aspect of the research analysis. The first section outlines the demographic and clinical characteristics of the PTSD and trauma control groups. The second section provides an analysis of the collected trauma narratives and explores the hypotheses that the trauma narratives of participants diagnosed with PTSD contain more emotional language and less cognitive language than the narratives of trauma controls. The third section explores whether the frequency of emotional and cognitive language correlates with childhood and adult trauma history, PTSD symptoms, and depression. The fourth section is focused on the activation observed in the emotional and cognitive centers of the brain – the amygdala and the mPFC, respectively – when PTSD and trauma control participants were presented with emotionally arousing visual stimuli. The fifth section is concerned with correlations between amygdala and mPFC activation and trauma history, PTSD symptoms, and depression. The sixth and last section will focus on a correlational analysis of the frequency of emotional and cognitive words and amygdala and mPFC activation.

Group Characteristics

Table 1 shows demographic and clinical characteristics for the Posttraumatic Stress Disorder (PTSD) and clinical trauma control (TC) groups. The PTSD and TC groups did not differ in age. Relative to trauma controls, PTSD participants had experienced more childhood and more adult trauma, had more symptoms of depression, and had greater PTSD symptoms.

Table 1. PTSD symptoms, childhood and adult trauma history, and depression in trauma controls and PTSD patients.

Demographic Variable	Trauma control (N=25) M (SD)	PTSD (N=15) M (SD)	t
Age	35.5 (10.1)	38.0 (11.0)	.7
PTSD symptoms (PSS)	8.2 (9.02)	22.2 (10.7)	4.4*
Intrusive	1.6 (2.3)	5.5 (3.6)	4.2*
Avoidance/ Numbing	2.5 (3.9)	7.9 (4.8)	3.8*
Hyper-arousal	4.2 (4.2)	8.8 (3.5)	3.6*
# traumas, different types (TEI)	4.0 (2.3)	7.8 (4.0)	3.6*
Childhood trauma (CTQ)	34.1 (12.4)	57.5 (18.8)	4.3*
Depression (BDI)	8.8 (7.0)	20.7 (11.5)	3.6*

* $p < .05$

Description of Narratives

The first hypothesis was that the trauma narratives of those with PTSD would have more affective words and less cognitive words compared to the trauma control participants. A total number of 67 trauma narratives were collected from the 40 participants interviewed. While all participants were asked to provide both a child and an adult memory, trauma narratives for both events were collected only from 27 participants: eight provided only a child memory, and five only an adult memory. Trauma narratives were classified under one of three categories: experienced, witnessed, and

confronted. Most traumas were experienced (n=45), followed by witnessed (n=13), and confronted (n=9). Statistical analysis as a function of trauma type was not conducted because of the limited sample size, but the importance of this categorization will be included in the discussion section. For statistical analysis purposes, trauma narratives were combined regardless of trauma type. All traumas described in the narratives were considered Criterion A index traumas for PTSD diagnosis in the CAPS interview. Appendix C presents examples of childhood and adult traumas experienced by the population recruited for this study. In order to determine whether child and adult trauma narratives differed in the variables of interest paired-samples t-tests were computed on all the LIWC variables. Because there were no differences across these variables in child and adult trauma narratives, subsequent analyses considered all memories irrespective of whether the trauma was experienced in childhood or adulthood.

Differences in the trauma narratives of participants with and without current PTSD

To test the hypothesis that participants diagnosed with PTSD use significantly more affective words than trauma controls and significantly less cognitive words, analyses comparing the percentage of affective and cognitive words between the PTSD and TC groups were conducted. The percentage of emotion and cognitive words used in trauma narratives by group is presented in Table 2. Group comparisons showed that participants diagnosed with PTSD used more affective words in their trauma narratives than trauma controls ($t(65) = 2.472$, $p = .016$). Although PTSD patients did not use significantly more negative emotion words than trauma controls, a trend exists for them to do so ($t(65) = 1.882$, $p = .067$). No significant differences in the use of cognitive

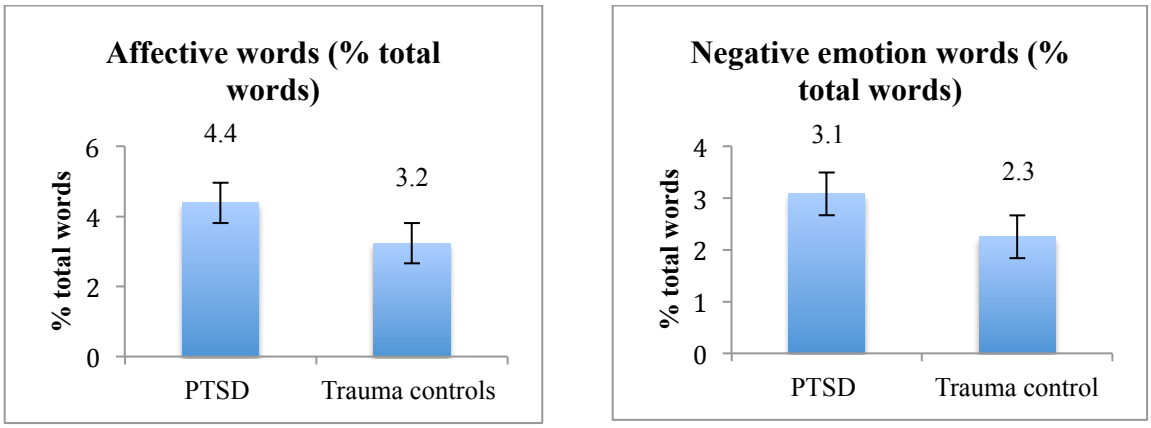
mechanism words were observed between the PTSD and the trauma control group (t(65) = .504, p = .616) (Figure 1).

Table 2. Affective, positive emotion, negative emotion, anxiety, anger, sadness, and cognitive mechanism words as a percentage of total words.

	PTSD	Trauma Control	t
LIWC Categories			
Affective	4.4 (2.2)	3.2 (1.6)	2.47*
Positive Emotion	1.3 (1.2)	1.0 (1.1)	1.08
Negative Emotion	3.1 (1.9)	2.3 (1.4)	1.88
Anxiety	0.3 (0.5)	0.4 (0.5)	-.8
Anger	1.6 (1.4)	1.1 (1.0)	1.69
Sadness	0.6 (1.6)	0.3 (0.4)	.87
Cognitive	17.5 (4.2)	17.0 (2.8)	.50

* $p < .05$

Figure 1. PTSD patients used significantly more affective words than trauma controls and showed a tendency for using more negative emotion words than trauma controls.

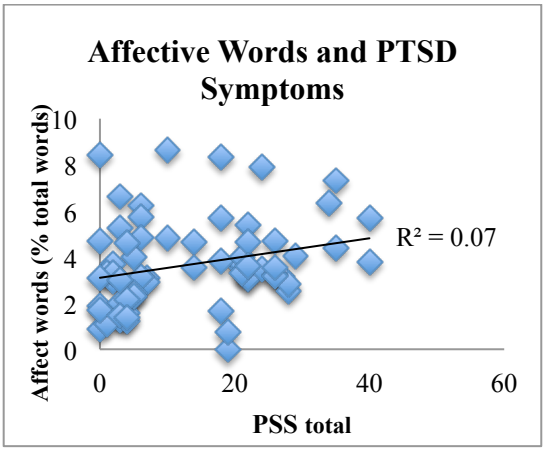


Influence of PTSD symptom severity on the emotional content of narratives

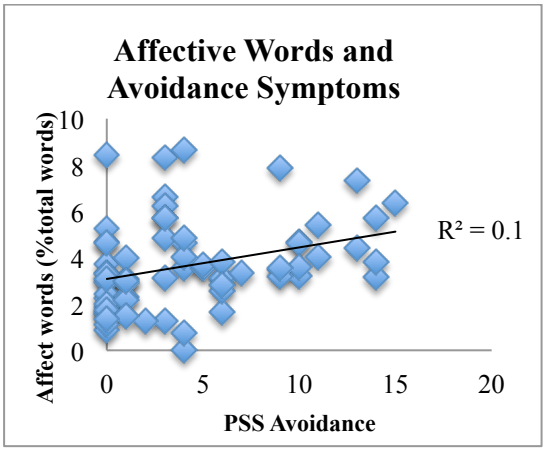
In order to examine relations between clinical groups and trauma narratives more closely, we conducted a series of correlational analyses between scores on the mPSS and narrative word use. There was a significant correlation between the percentage of affective words and PTSD symptoms, as measured by the mPSS ($r(65) = .257$, $p = .036$). Higher PTSD symptomology was associated with using more affective words in narratives of trauma (Figure 2A). Follow-up analysis of specific symptom clusters showed that the use of affective words was positively correlated with avoidance symptoms ($r(65) = .318$, $p = 0.009$), but not re-experiencing or hyper-arousal symptoms ($p > .05$). The proportion of affective words also correlated positively with the amount of childhood trauma ($r(65) = .254$, $p = .038$) and adult trauma ($r(65) = .410$, $p = .001$), as well as depression symptom severity ($r(65) = .254$, $p = .019$), as shown in Figure 2B-E. Therefore, higher avoidance symptoms, more depression, and more childhood and adult

trauma were associated with using more affective words in narratives of adult and child trauma. Regression analysis controlling for the effect of adult and childhood trauma showed that PTSD did not account for significant variance above and beyond the effects of trauma (childhood and adult trauma: $R^2 = .172$, $F(2,64) = 6.654$, $p = .002$; mPSS: $R^2\Delta = .001$, $F(1,63) = .050$, $p = .824$).

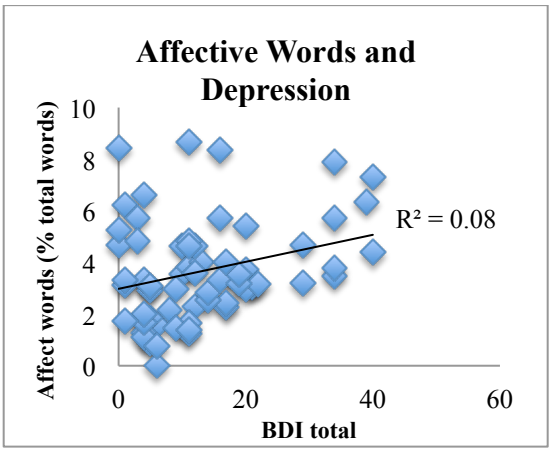
Figure 2. Significant correlations with frequency of affect words in trauma narratives



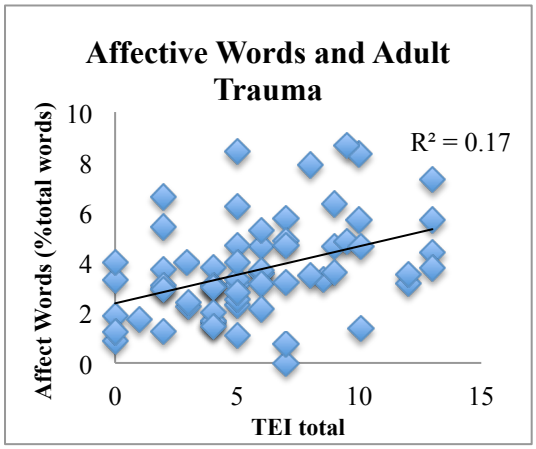
A)



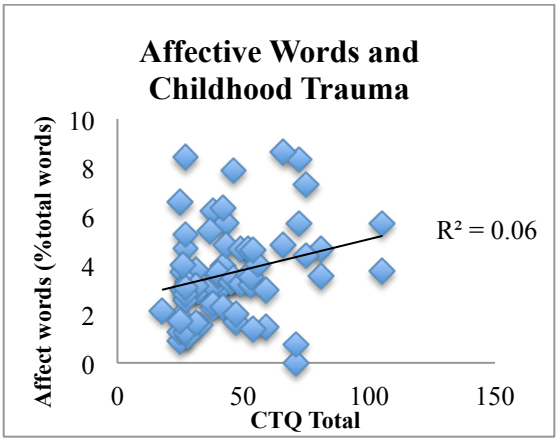
B)



C)



D)

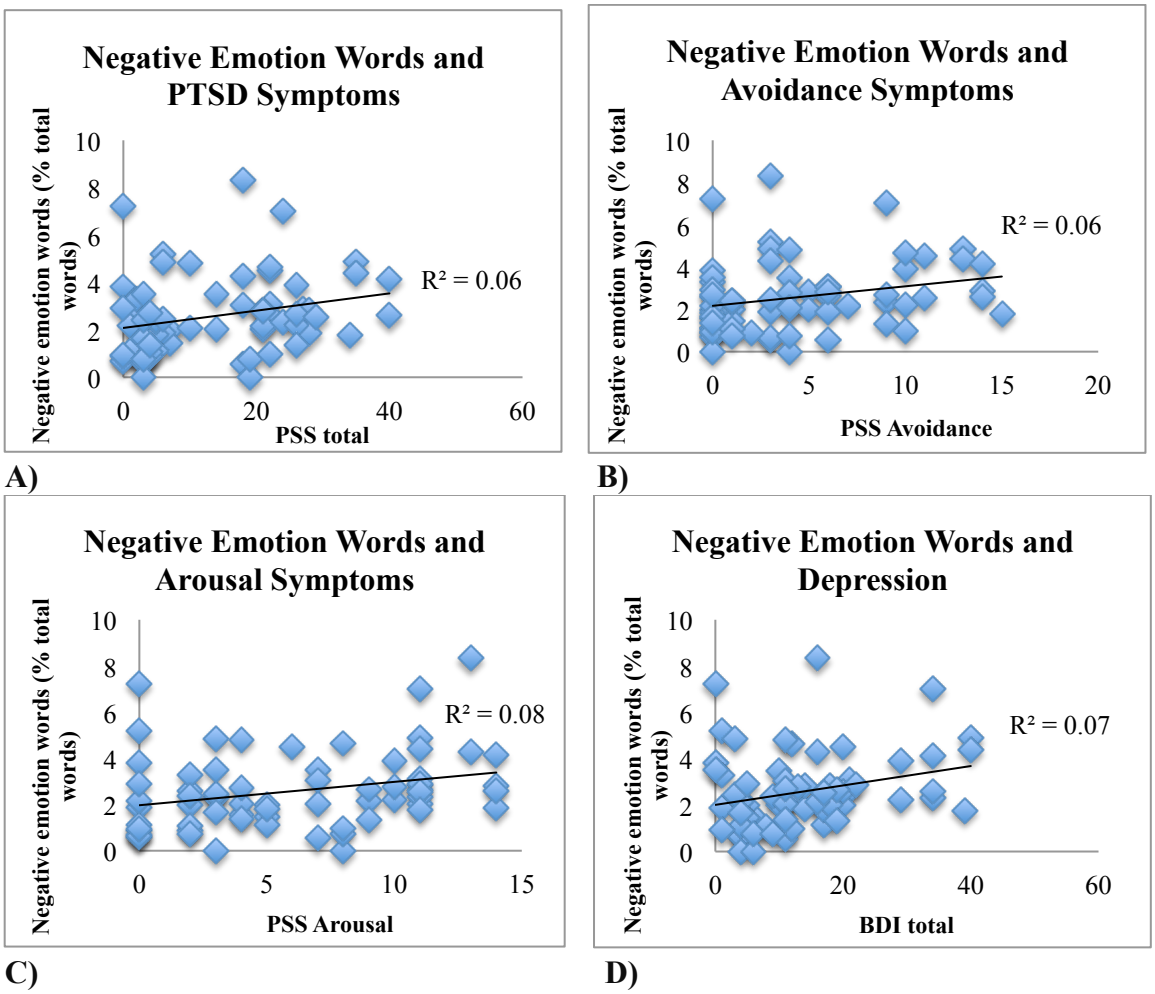


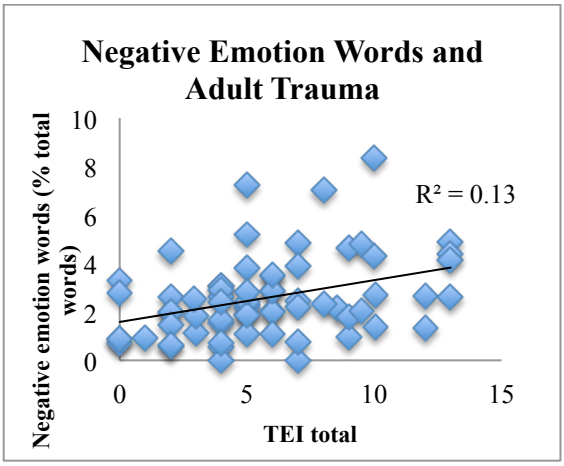
E)

To explore this finding in more detail, analyses were conducted on positive and negative words separately. Although both positive and negative emotion words belong to the LIWC Affective cluster, only the use of negative emotional words was significantly correlated with PTSD symptom severity, as measured by the mPSS ($r(65) = .253$, $p = 0.039$; positive emotion words: $r(65) = .050$, $p = .685$) (Figure 3A). When examining symptom clusters, use of negative emotion words was positively correlated with avoidance symptoms ($r(65) = .249$, $p = .043$) and arousal symptoms ($r(65) = .275$, $p = .024$), but not re-experiencing symptoms ($p > .05$). Negative emotion words were also correlated with depression ($r(65) = .264$, $p = .031$), and amount of adult trauma ($r (65) = .240$, $p = .051$) as shown in Figure 3B-E . There was a positive trend for the association of negative emotion words and childhood trauma ($r(65) = .240$, $p = 0.051$ (Figure 3F). The frequency of positive emotion words did not significantly correlate with levels of childhood trauma ($r(65) = .058$, $p = .642$), adult trauma ($r(65) = .158$, $p = .203$) and depression ($r(65) = .086$, $r = .491$). Therefore, higher avoidance symptoms, higher arousal symptoms, more depression, and more childhood and adult trauma were associated with using more negative emotion words in narratives of adult and child

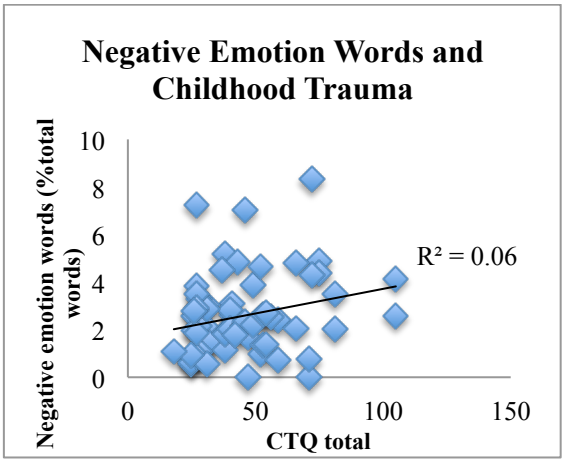
trauma. Regression analysis controlling for the effect of adult and childhood trauma showed that PTSD did not account for significant variance above and beyond the effects of trauma (childhood and adult trauma: $R^2 = .130$, $F(2,64) = 4.770$, $p = .012$; mPSS: $R^2\Delta = .003$, $F(1,63) = .197$, $p = .658$).

Figure 3. Significant correlations with the frequency of negative emotion words in trauma narratives





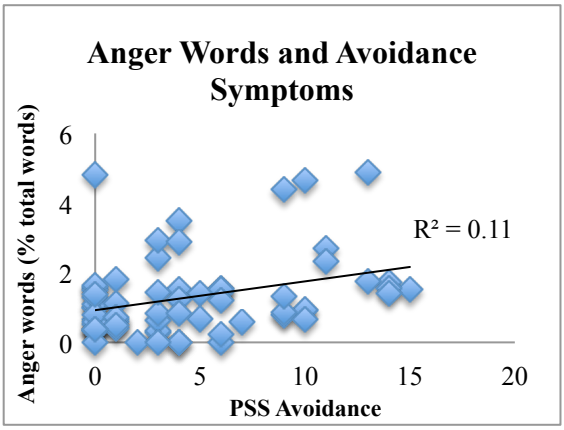
E)



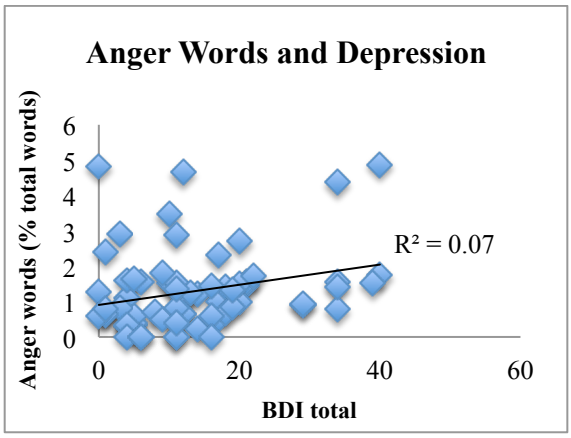
F)

Two specific categories of negative emotion were significantly related to PTSD symptoms: anger and anxiety. The use of anger words was positively correlated with symptoms of avoidance and numbing ($r(65) = .325$, $p = 0.007$), but not with total PTSD symptom severity, re-experiencing, or hyper-arousal symptoms (ps > .05). The use of anger words was positively correlated with depression ($r(65) = .263$, $p = 0.032$), and adult trauma ($r(65) = .277$, $p = 0.023$) as seen in Figure 4, but not childhood trauma ($p > .05$). The more anger words participants used in their trauma narratives, the more avoidance and numbing, and depression symptoms they showed, and the more extensive a trauma history they had. In addition, participants with greater hyper-arousal symptoms used more anxiety words ($r(65) = .245$, $p = .046$, Figure 5). Anxiety words were not significantly correlated with total PTSD symptom severity, adult or child trauma, or depression (ps > .05). The use of sadness words, also a subcategory of negative emotion words, did not correlate with total PTSD symptom severity, childhood or adult trauma, or depression (ps > .05).

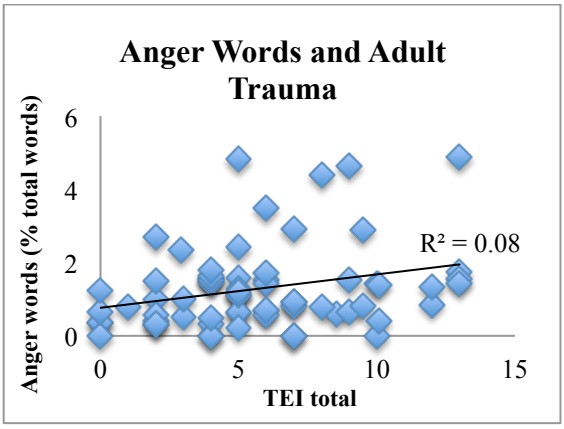
Figure 4. Significant correlations with frequency of anger words in trauma narratives



A)

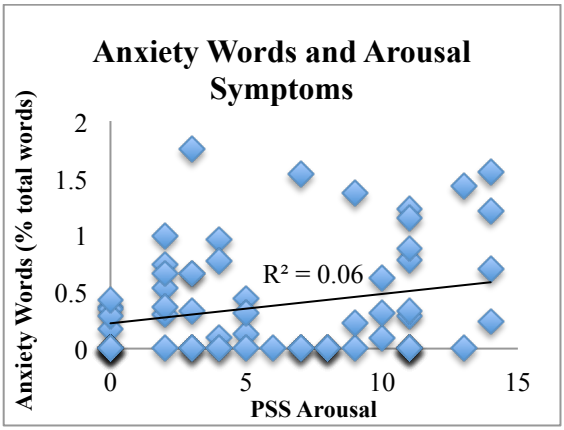


B)



C)

Figure 5. Significant correlation between the frequency of anxiety words and arousal symptoms



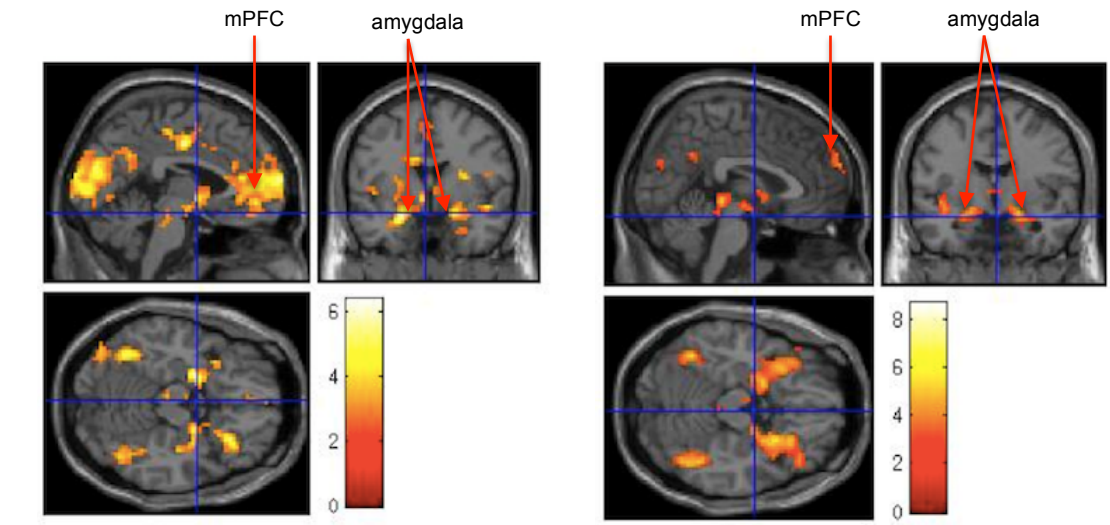
Influence of PTSD symptom severity on the cognitive content of narratives

Cognitive word use was not significantly correlated with PTSD or depression symptom severity, or adult or child trauma load (ps > .05).

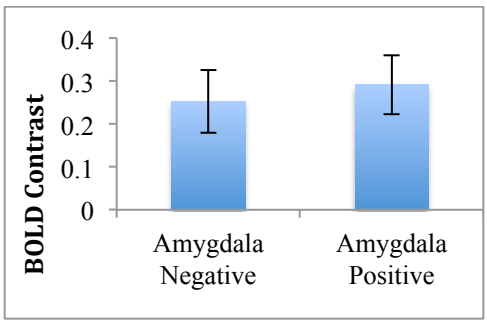
FMRI activation in response to emotional stimuli

Participants in the PTSD group showed bilateral activation within the amygdala and medial prefrontal cortex in response to both positive relative to neutral images, and negative relative to neutral images, $p < .05$, corrected for multiple comparisons. Region-of-interest (ROI) analyses showed that amygdala activation in response to positive emotional stimuli (positive > neutral) was not significantly different than amygdala activation in response to negative stimuli (negative > neutral contrast), as shown in Figure 6 ($t(29) = .522$, $p > .05$). However, mPFC activation in response to positive emotional stimuli (positive > neutral) was greater than from mPFC activation in response to negative emotional stimuli (negative > neutral) ($t(29) = 2.224$, $p = .034$).

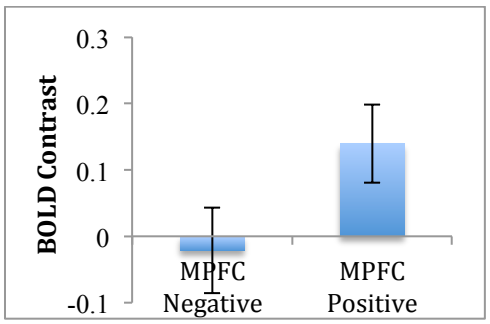
Figure 6. Brain responses to positive and negative emotional stimuli in the IAPS task. A) Whole-brain analysis of regions that showed a significant response to positive stimuli (Pos > Neut), $p<.05$, corrected for multiple comparisons. The red-yellow color scale indicates t-scores representing the difference between the Positive and Neutral emotion conditions. Images are displayed in neurological orientation. B) Whole-brain analysis of regions that showed a significant response to negative stimuli (Neg >Neut), $p<.05$, corrected for multiple comparisons. The red-yellow color scale indicates t-scores representing the difference between the Negative and Neutral emotion conditions. C) Amygdala activation in response to negative stimuli was not significantly different than amygdala activation in response to positive stimuli. D) More medial prefrontal activation was observed in response to positive stimuli than negative stimuli.



A) B)



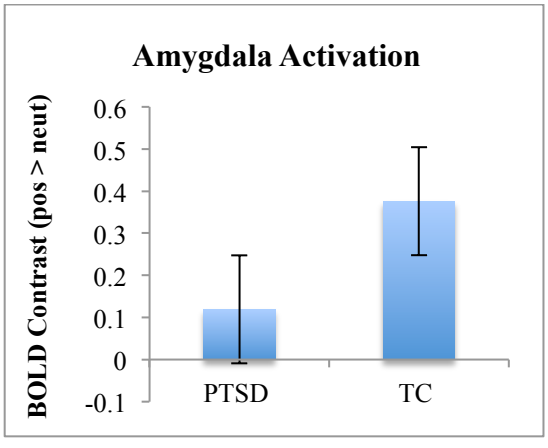
C)



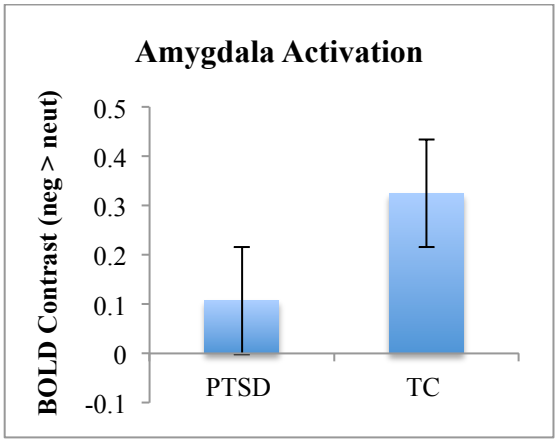
D)

Independent samples t-tests were conducted to verify whether the PTSD and trauma control groups showed differential amygdala and mPFC activation in response to either positive versus neutral or negative versus neutral emotional stimuli. Results showed that there was no significant difference between any of these variables (ps > 0.05). Figure 7 shows side-by-side comparisons of activation in all four conditions.

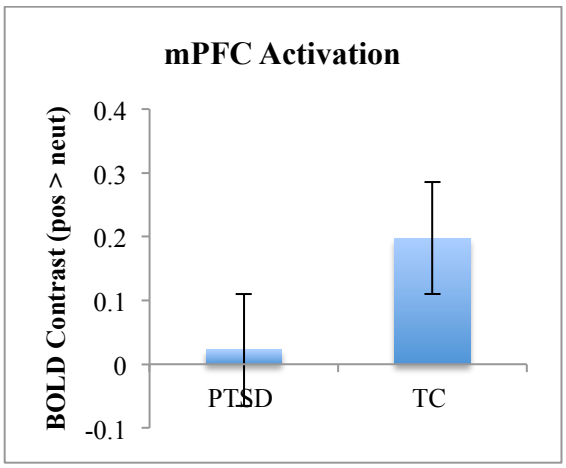
Figure 7. Amygdala activation to A) positive versus neutral emotional stimuli and B) negative versus neutral stimuli for the PTSD patients and for trauma controls (ps >0.05). Medial prefrontal cortex activation to C) positive versus neutral emotional stimuli and D) negative versus neutral stimuli for PTSD patients and for trauma controls (p>0.05).



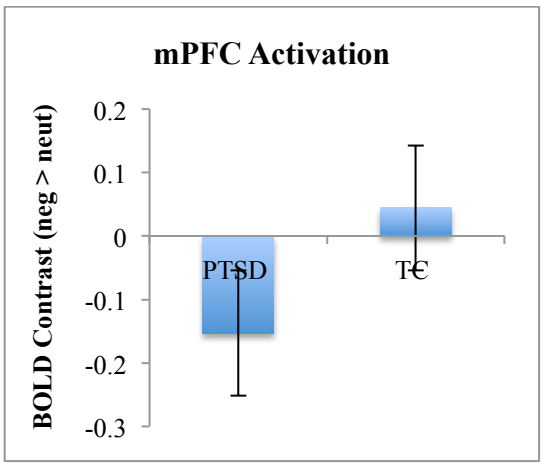
A)



B)



C)



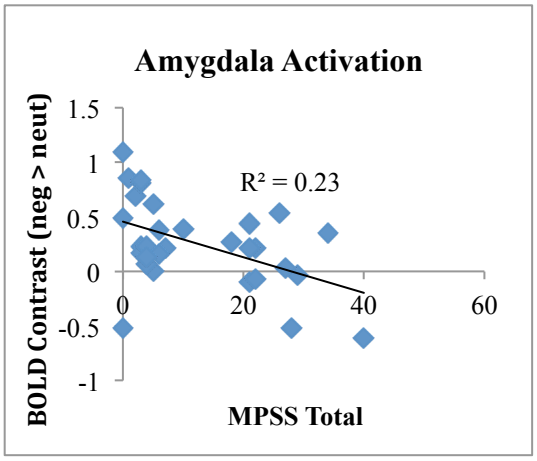
D)

Relations between PTSD symptom severity and amygdala and mPFC activation

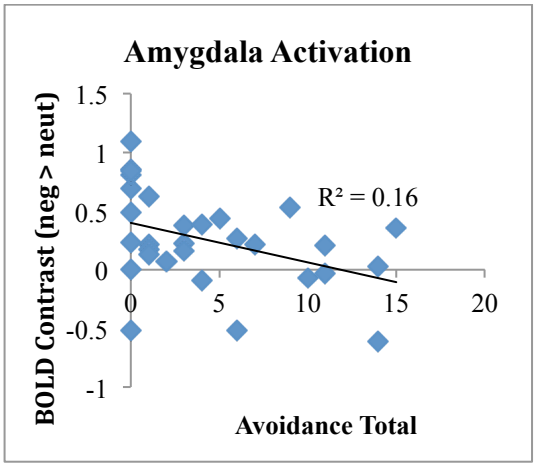
A hypothesis made in this research was that amygdala activation to negative stimuli is positively correlated with PTSD symptoms and depression. Findings were contrary to the initial hypothesis. Correlational analysis showed that amygdala activation to negative versus neutral stimuli was negatively correlated with PTSD symptoms, such that the more amygdala activation, the lower the score on the mPSS ($r(29) = -.481$, $p = .007$). Figure 8 shows the correlations between amygdala activation and mPSS total score, as well as amygdala activation to negative versus neutral emotional stimuli and scores on the avoidance ($r(29) = -.406$, $p = .026$), re-experiencing ($r(29) = -.447$, $p = .013$), and hyperarousal ($r(29) = -.498$, $p = .005$) subscales. There was no significant correlation between amygdala activation to positive stimuli and PTSD symptoms and depression ($ps > .05$). In addition, no significant correlation was observed between amygdala activation to either positive or negative emotional stimuli and number and types of adult trauma (TEI) ($ps > .05$). However, amygdala activation to negative emotional stimuli was negatively correlated to depressive symptoms ($r(29) = -.388$, $p =$

.034). While amygdala activation to negative versus neutral stimuli was not correlated to childhood trauma (CTQ) (p >.05), amygdala activation to positive versus neutral stimuli did correlate positively with childhood trauma (r(29) = -.361, p = .050).

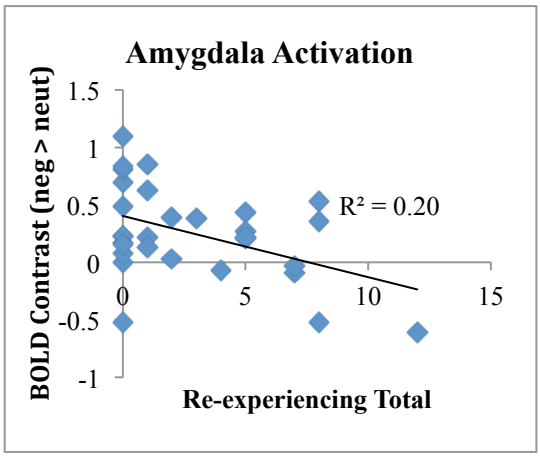
Figure 8. Amygdala activation and PTSD symptoms



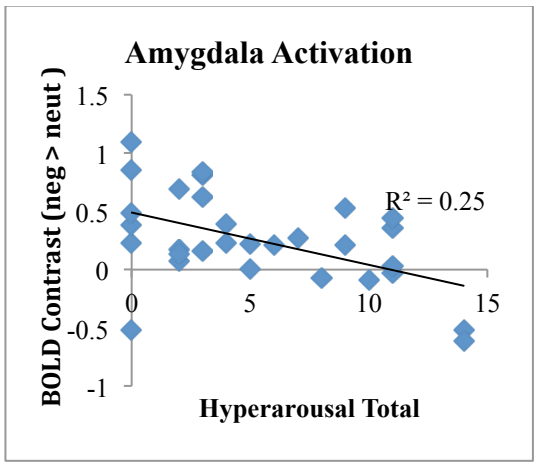
A)



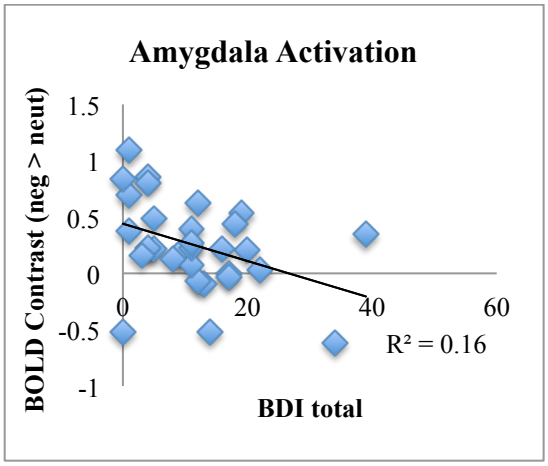
B)



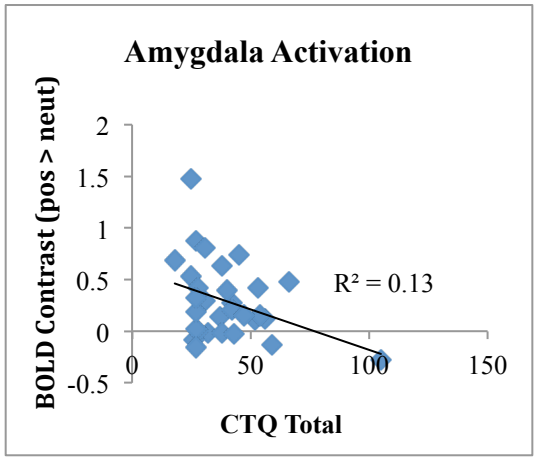
C)



D)



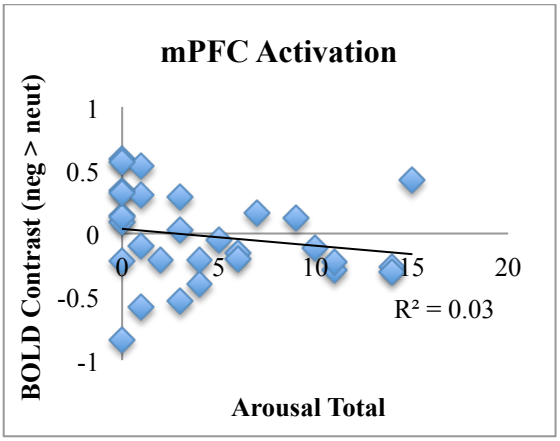
E)



F)

Another hypothesis we tested was and that mPFC activation is negatively correlated with PTSD symptoms and depression. Medial prefrontal cortex activation to negative versus neutral stimuli was negatively correlated with arousal symptoms, such that the more mPFC activation, the lower the score on the arousal subscale of the mPSS ($r(29) = -.361$, $p = .050$). Correlational analysis suggested no outstanding associations between mPFC activation to positive versus neutral or negative versus neutral emotional stimuli and other PTSD symptom clusters, depression (BDI), number and types of adult trauma (TEI), and childhood trauma (CTQ) (ps > .05).

Figure 9. Medial prefrontal cortex activation and arousal symptoms

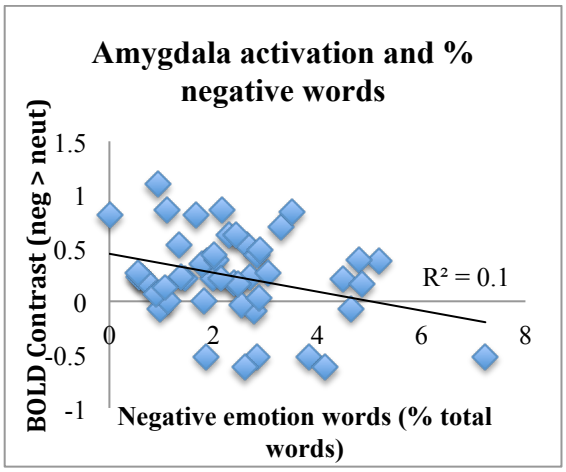


Relations between amygdala and mPFC activation and frequency of emotional and cognitive words

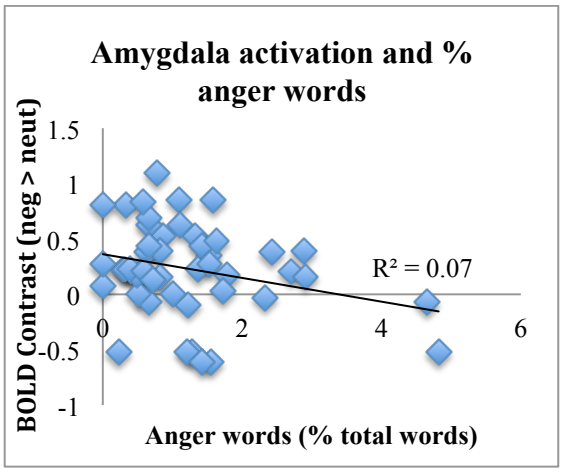
A last set of analyses explored potential correlations between the frequencies of affect words (including positive and negative emotion, anger, and anxiety) and cognitive words and amygdala and medial prefrontal cortex activation to positive and negative emotional stimuli. Although we hypothesized that amygdala activation to negative stimuli will correlate positively with the use of negative emotion words, a negative correlation was found instead ($r(65) = -.313$, $p = .030$) (Figure 10A). Amygdala activation to negative versus neutral stimuli did not correlate significantly with the frequency of affect words, anxiety, and anger words (ps > .05). However, a negative trend was observed when correlating amygdala activation to negative versus neutral stimuli with anger words ($r(65) = -.274$, $p = .060$) (Figure 10B). We also hypothesized that medial prefrontal cortex would correlate positively with the frequency of cognitive words. While that correlation was not significant ($p > .05$), mPFC activation to positive versus neutral emotional stimuli did correlate negatively with the frequency of affect ($r(65) = -.382$, $p = .007$) and negative emotion words ($r(65) = -.285$, $p = .049$) (Figure

10C, D). Medial prefrontal cortex activation to negative versus neutral stimuli did not correlate significantly with any LIWC categories (ps > .05).

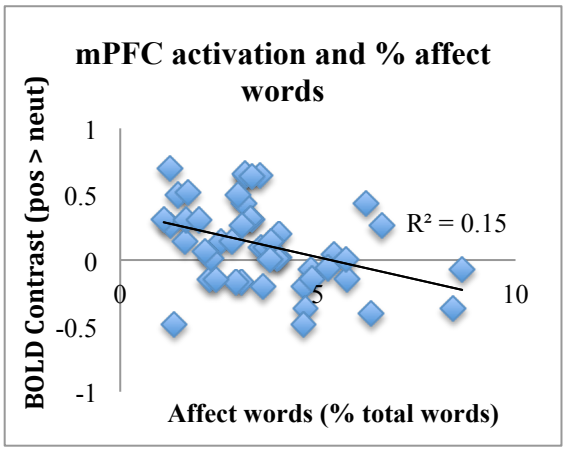
Figure 10. Amygdala activation to negative versus neutral emotional stimuli and A) Negative emotion words and B) Anger words**.** Medial prefrontal cortex activation to positive versus neutral emotional stimuli and C) Affect words and D) Negative emotion words



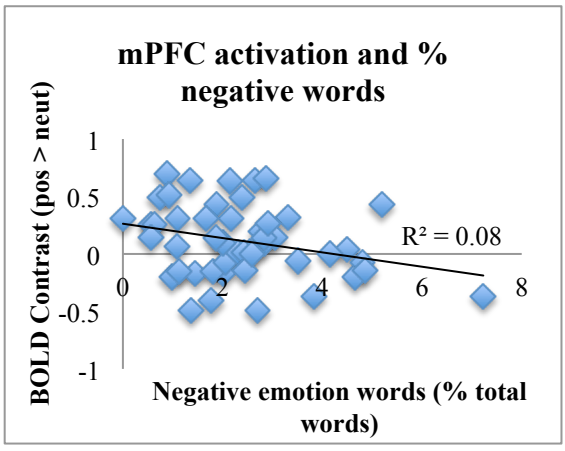
A)



B)



C)



D)

Discussion

The present study investigated the way in which Posttraumatic Stress Disorder affects the emotional and cognitive content of women's autobiographical memories of traumatic events as well as the processing of emotional stimuli in a functional imaging task. Consistent with the hypotheses, this research found that women diagnosed with PTSD used more affect words, and specifically more negative emotion words, when remembering the most traumatic childhood and adult events of their lives than women with comparable trauma histories but without PTSD. As predicted, the use of affect words was positively correlated with the severity of PTSD symptoms in the entire sample of traumatized participants. Participants with PTSD and trauma controls did show differential amygdala activation during the IAPS task. However, PTSD participants showed less activation to emotional images rather than more activation compared to trauma controls as we predicted. While we hypothesized that amygdala activation would be positively correlated with symptom severity across participants and with the use of affect words in narratives of trauma, we instead found a negative correlation in both instances. Contrary to our hypotheses, the use of cognitive words was not significantly different between PTSD participants and traumatized controls, did not correlate with PTSD symptom severity, and was not associated with activation in the mPFC during processing of emotional stimuli. Consistent with our prediction, PTSD participants activated the mPFC less than trauma controls when viewing emotionally arousing images. This activation seen in the mPFC correlated negatively with the frequency of affect and particularly negative emotion words in trauma narratives across all

participants. The discussion of these main findings, as well as several secondary findings, is organized in five distinct sections following the same organization used to present the results. The first section will consider the trauma narrative findings, and discuss any correlations found between specific symptom clusters and affect and cognitive language categories of the Linguistic Inquiry and Word Count word bank. The second section will explore the differential activation in the amygdala and mPFC in PTSD and trauma controls as well as correlations with PTSD symptoms. The third section will explore the correlations found between markers of emotion and cognitive language in the trauma narratives and amygdala and mPFC activation when processing emotional visual stimuli. The fourth section will consider both the strengths and the weaknesses of the present study, and propose future studies to tackle these weaknesses. Finally, the fifth section will be a conclusion summarizing the main findings in this study.

Trauma Narratives

Emotion Words. Consistent with our hypothesis, women diagnosed with PTSD used significantly more words of affect in their narratives of trauma compared to trauma controls. This finding is consistent with previous literature (Berntsen, Willert, & Rubin, 2003; Hellawell & Brewin, 2004; Jaeger, Lindblom, Parker-Guilbert, & Zoellner, 2014; Jones, Harvey, & Brewin, 2007; Kennardy, Smith, Spence, Lilley, Newcombe, Dob, & Robinson, 2007; Negrao, Bonanno, Noll, Putnam, & Trickett, 2005), and reinforces the idea that PTSD patients have more emotional memories of their personal traumas compared to trauma controls (Brewin et al., 1996; Ehlers & Clark, 2000), and that this higher emotional content is reflected in the language they use in their narratives. A study

by Holmes et al. (2007) found that when describing partner violence, women's use of emotional words was positively correlated with their subjective experience of physical pain (Holmes et al., 2007). They attributed this finding to a higher degree of immersion in the trauma memory (Holmes et al., 2007). Since the comorbidity of PTSD and chronic pain is well documented (Asmundson, Norton, Allerdings, Norton, & Larsen, 1998; Palyo & Beck, 2005; Mostoufi et al., 2014), it may be that the psychological and physical distress associated with PTSD is exteriorized through the use of emotional language. This finding provides support for Berntsen, Willert, and Rubin's (2003) model of emotional processing in trauma, which poses that traumatic events in PTSD are central to the individual's identity, and are therefore remembered more vividly and allocated more emotional significance (Berntsen et al., 2003).

When investigating differences in the use of specific categories of emotional words such as positive and negative emotion, the PTSD group did not differ from the resilient group in the present study. Nevertheless, there was a trend for PTSD participants to use more negative emotion words, and that is consistent with previous findings in the literature (Kahn, Tobey, Massey, & Anderson, 2007; Pennebaker, Mehl, & Niederhoffer, 2003). Taken together, these findings suggest that in PTSD the significant emotional impact attached to the memory of a traumatic event seems to surface through the content – affect words – of the trauma narrative in question. Interestingly, no difference was seen in the use of positive emotion words between the two groups, although a previous study by Fredrickson, Tugade, Waugh, and Larkin (2003) found that positive emotion is associated with resilience (Fredrickson, Tugade, Waugh, & Larkin, 2003). In fact, the PTSD participants used more positive emotion words than the trauma control group,

although they did not do so at a significance level. Although more studies are needed to confirm whether this finding is important for understanding emotional memory in PTSD, it may be that emotion pervades the autobiographical memories of those impacted. In this model, the emotional content of trauma memories becomes evident in their content, regardless of its valence.

Jaeger et al. (2014) and others also showed that the content of individuals' trauma narratives is more potently associated with PTSD symptoms and trauma-related reactions (eg. depression) than ways of narrative production such as fragmentation and dissociation are (Jaeger et al., 2014). Similarly, the present study revealed that higher PTSD and depressive symptoms were associated with using more affect words in narratives of trauma. This finding is consistent with previous research (Zoellner et al., 2002; Watkins, Vache, Verney, Muller, & Andrews, 1996). Other studies, however, found that a higher use of positive and negative emotion words in narratives of trauma was associated with lower PTSD symptoms (Jaeger et al., 2014; Friedrickson et al., 2003), thus supporting the idea that individuals who are better able to express their emotions when talking about their trauma have more successfully processed the emotional content of the memory of the traumatic event (Foa & Kozak, 1986; Foa & Riggs, 1993; Rauch & Foa, 2006). When examinining specific symptom cluster, a higher percentage of affect words was associated with avoidance symptoms, but not with hyper-arousal and re-experiencing symptoms. More research should address this finding, but a possible explanation may be that individuals who develop PTSD after suffering a trauma and actively block the re-emergence of the trauma memory for a significant period of time have less control over their emotional expressivity when they are finally prompted to recall it.

The more childhood and adult trauma participants had, the more words of affect they used in their trauma narratives. Since PTSD is a disorder characterized by problems with emotional memory, and individuals with PTSD use more affect language than traumatized controls, this finding is supported by previous research showing that the likelihood of developing PTSD increases the more trauma a person is exposed to (Kessler et al., 1995; Ogle et al., 2013). Future research should directly investigate the relationship between trauma exposure and emotional word use in trauma narratives.

It was of interest to explore whether all affect words or only certain categories of affect words were associated with PTSD symptoms. Indeed, only negative emotion words, and not positive emotion words were found to correlate with PTSD symptoms, adult trauma, and depression. This finding is consistent with the definition of traumas as highly negative life events, and replicates findings by Kahn, Tobey, Massey, and Anderson (2007) showing that written narratives about negative events contain more negative emotion words than other autobiographical narratives (Kahn, Tobey, Massey, & Anderson, 2007).

Upon further dissecting LIWC categories of affect words, we found that the more anger words used, the more avoidance, depression, and adult trauma participants showed. Furthermore, the more anxiety words participants used, the more PTSD hyperarousal symptoms they had. Both these findings are consistent with the literature, as anger and depression are often associated (Fava, Kellner, Munari, Pavan, & Pesarin, 1982; Riley, Treiber, & Woods, 1989), and highly traumatized individuals show more anger than controls. In addition, previous studies have shown that the expression of anger may trigger the reliving of trauma memories, therefore causing arousal symptoms such as

palpitations (Clark, 1999; Hinton, Rasmussen, Nou, Pollack, & Mary-Jo, 2009). While in the present study participants were mostly calm when they were instructed to recall their traumatic memory, it is possible that the intimate and affective quality of the act of recalling itself led to more anger, which in turn facilitated the reliving of the event. A possibility is that once participants acknowledged their reliving experience, they further tried to avoid it.

Cognitive Words. We did not find any difference in cognitive word use between the PTSD and trauma control group, and no correlation between the amount of cognitive words and PTSD symptomology as we predicted. The research literature on cognitive processing in PTSD is split. In a study by Boals and Klein (2005), participants who recently broke up with their partner were instructed to describe their pre-break up, break up, and post break-up situations. More cognitive words were present in the break up and post-break up narratives than the pre-break up, suggesting that participants were actively trying to organize and understand the recent negative event after it happened (Boals & Klein, 2005). For trauma narratives in particular, however, Rubin (2011) found that individuals used less cognitive words, particularly cause and insight words, in trauma narratives than narratives about other less emotional events (Rubin, 2011). Similarly, Alvarez-Conrad, Zoellner, and Foa (2001) showed that the use of cognitive words by trauma survivors led to better PTSD post-treatment functioning (Alvarez-Conrad, Zoellner, & Foa, 2001; Pennebaker, Mayne, & Francis, 2007). Rubin's (2005) study, however, also suggested that cognitive processing is evident in both individuals diagnosed with PTSD and in those who are resilient, which would be consistent with the current findings (Rubin, 2011).

A different view is the disintegration view. Studies that support it report less cognitive processing in PTSD (Brewin, 1996; Horowitz & Reidbord, 1992; Nijenhuis & van der Hart, 1999; Van der Kolk & Fisher, 1995; Halligan, Clark, & Ehlers, 2003), and use that to suggest that trauma memories in PTSD are fragmented and poorly integrated into an individual's life story. According to this view, participants with PTSD would show less cognitive word use when describing their trauma than those who are resilient. In addition, the lower-than-average IQ and level of education of the study population undermines participants' ability to process their trauma memories, since low intellectual ability has been associated with an increased risk of developing PTSD (McNally & Shin, 1995; Ehlers & Clark, 2000; Breslau, Chen, & Luo, 2013). However, a particularly interesting finding in this study is that both PTSD and trauma control participants used significant amounts of cognitive words, and, in fact, more than traumatized individuals in similar studies (17-18 % in the present study compared to 6-8% in a study by Bohanek, Fivush, and Walker (2005), 7-9% in a study by Boals and Klein (2005), and 7-10% in a study by Jelinek et al. (2010). Therefore, the findings suggest that all traumatized participants were able to process their trauma, and that it is not their inability to integrate their traumas in their overall life story that lead to the development of PTSD symptoms.

The current findings may suggest that, in accordance with the view expressed by Berntsen et al. (2003), PTSD is characterized by a dysfunctional integration of traumatic memories within one's life narrative, rather than a lack of such integration (Berntsen et al., 2003). This dysfunctional integration would not be apparent in the number of cognitive words used in the trauma narrative, but instead in relations between the trauma

narrative and life story, as measured by the Centrality of Event Scale (CES) developed by Berntsen and Rubin (2006).

Amygdala and medial prefrontal cortex activation to emotional stimuli

Amygdala. Participants with PTSD did not show significantly different amygdala activation to visual emotional stimuli compared to trauma controls, although there was a trend for PTSD participants to show less activation to both positive and negative emotional stimuli. This finding is not consistent with our predictions, which posed that the PTSD group will show more activation than controls in both conditions and particularly in the negative stimuli condition as previously shown by St Jacques, Botzung, Miles, and Rubin (2011). However, it is notable that both groups of participants showed more activation to both positive and negative images compared to neutral images, a finding replicated in the literature (Sabatinelli et al., 2011). No difference was found between amygdala response to positive versus neutral stimuli and negative versus neutral stimuli, which further supports the idea that the amygdala seems to process both positive and negative emotions (Garavan, Pendergrass, Ross, Stein, & Risinger, 2001; Hamann & Mao, 2001). A possible interpretation might be that individuals showing avoidance symptoms may also show less amygdala response to negative stimuli. Consistent with this idea, we observed a negative correlation between amygdala activation and avoidance symptoms. However, we also found similar negative correlations for the other symptom clusters (re-experiencing and hyperarousal), even though this finding may be due to co-linearity across symptom clusters. Future studies investigating the connection between amygdala activity and emotional numbing are

needed to provide a clear idea. Additionally, the more depressed individuals were, the less amygdala activation they showed to negative stimuli. Both emotional numbing and depression have been linked to PTSD severity (Feeny, Zoellner, Fitzgibbons, & Foa, 2000), and avoidance coping is in turn associated with depressive symptoms (Holahan et al., 2005). Therefore, this sample of highly traumatized women may be avoiding the reliving of a traumatic episode through numbing when shown negative emotional stimuli that may trigger such response.

An interesting result was that the less childhood trauma participants had, the more amygdala activation they showed to positive images. This finding may suggest that individuals who experience more childhood trauma have a diminished emotional response to positive stimuli. Indeed, a recent study by Broekhof et al. (2015) found that children with a history of emotional maltreatment show a less positive affect and lower levels of dispositional optimism (Broekhof et al., 2015). This finding is also consistent with research by McTeague et al. (2009), who reported a reduction in physiological responses to emotional stimuli as a result of multiple traumas (McTeague et al., 2009). A potential follow-up study would use, in addition to the CTQ, measures of optimism and positive outlook on life such as the Life Orientation Test Revised (LOT-R) (Broekhof et al., 2015) to investigate whether amygdala activation to positive emotional stimuli in participants with childhood trauma correlates with optimism levels.

Medial prefrontal cortex. No difference in medial prefrontal cortex activation was seen between the PTSD and trauma control group. Previous studies have shown less mPFC activation in PTSD (Shin et al., 2006; Bremner et al., 1999; Koenigs & Grafman, 2009). When specific symptom clusters were examined, mPFC activation to negative

stimuli was negatively correlated with arousal symptoms, which is consistent with the idea that hyperarousal is associated with less prefrontally-mediated emotion regulation.

Exploring associations between affect and cognitive word use and amygdala and mPFC activation

The current study is the first one to date to look at the correlation between the processing of emotional memories of trauma, as made evident in the use of emotion words in trauma narratives, and the processing of visual emotional stimuli, as shown by amygdala activation. The more amygdala activation participants showed to negative visual stimuli, the less negative emotion and anger words they used in their trauma narratives. More studies are needed to both validate and explain the present results. While this finding is not consistent with the initial prediction, it could nevertheless be explained in terms of an intentional blocking of the expression of negative emotion when recalling a traumatic event, potentially as a method of coping. It is not that traumatized individuals do not become aroused when they are presented with emotional stimuli, because the amygdala activation that they show in response to both positive and negative emotional stimuli suggests that they are. One possibility could be that when prompted to recall a memory that they find to be highly emotional arousing, participants become emotionally aroused upon processing the clinician's instructions, and either consciously or unconsciously avoid using negative emotion language as a coping strategy. It may also be that other factors influence the expression of emotion when relating autobiographical memories, and that the amygdala is not the sole brain region orchestrating the linguistic expression of trauma memories. A future study could be designed similarly to those

conducted by Lanius et al. (2001, 2004), who performed functional magnetic resonance imaging on PTSD participants and trauma controls shown a script designed to provoke PTSD symptoms (Lanius et al., 2001; Lanius et al., 2004). In a similar paradigm, participants with PTSD and trauma controls could be instructed to narrate their worst trauma memories inside the brain scanner. Then, both narrative measures and brain activation could be measured in response to remembering a traumatic and thus highly emotional event.

We found no correlation between medial prefrontal cortex activation and cognitive word use. This finding is consistent with the previous components of this study, since we did not find any significant differences in mPFC activation and cognitive word use between the PTSD group and trauma controls. Although we expected a greater mPFC response and a higher frequency of cognitive words to track a resilient profile, we did not find any evidence to support that hypothesis. However, the more mPFC activation that participants showed to positive stimuli, the less affect and, specifically, negative emotion words they used in their narratives. Since the mPFC is thought to inhibit pathological emotional responses by acting on the amygdala (Quirk, Likhtik, Pelletier, & Paré, 2003), it would be expected to see less emotional word use given more mPFC-regulated amygdala inhibition.

Strengths, Limitations, and Future Directions

A strength of the current study is that both childhood and adult index traumas were queried. These events were the self-selected most impactful negative life events in the lives of our participants, and were mostly of an interpersonal nature (eg. domestic

abuse, abuse between caregivers, sexual abuse, violence with weapons, etc.). However, child and adult traumas were not significantly different in their content, which raises interesting questions about the processing of traumatic events at different points in life. For example, research by Ogle, Rubin, Berntsen, and Siegler (2013) found that severe childhood trauma, in particular of interpersonal nature, can have long term effects on the social and emotional functioning of individuals (Ogle, Rubin, Berntsen, & Siegler, 2013). Furthermore, their research also suggests that childhood trauma, more so than adult trauma, causes pathological processing of memories (Ogle et al., 2013). The present population sample had mostly experienced trauma – 47 out of the 67 traumas recorded were experienced, 13 were witnessed, and 9 were confronted – and most of the experienced traumas were interpersonal. Yet, no differences were found between child and adult traumas, which is a finding worthwhile pursuing in further research. One potential way to do so would be by controlling for the amount of time that has passed since the traumatic event, a step that was not taken into account the current study. Previous research on camp survivors from World War II suggests that trauma memories older than four decades do not decay in accuracy and amount of detail (Schelach & Nachson, 2001; Wagenar & Groeneweg, 1990); however, Boals, Hayslip, and Banks (2014) showed that older individuals modify their recollection of negative events over time to better incorporate them within their life narrative (Boals, Hayslip, & Banks, 2014). More research should target whether the amount of time that has passed since the trauma affects the emotional content of traumatic memories, and whether it is a predictor of PTSD symptom severity.

A limitation of this study was that several clinicians interviewed and diagnosed the participants, so the format of the interview varied slightly depending on the interviewer. For instance, the number of interviewer prompts about both the event itself and the interviewee's emotional reaction to the event varied between none and twenty, and some interviewers emphasized open-ended questions over close-ended questions or vice versa. While it does not appear that the participants were less honest in their answers, future studies should use a standardized interviewing methodology to ensure accuracy and completeness. One potential approach would be to ask patients one single open-ended question and then allow them to narrate their traumas freely, without interruption, and as clearly and detailed as possible, similarly to what Halligan, Michael, Clark, and Ehlers (2003) did in their study of assault victims (Halligan, Michael, Clark, & Ehlers, 2003).

The Linguistic Inquiry and Word Count program, while widely used to analyze language in autobiographical narratives, has a limited range of functions. Because it counts words regardless of context, sentence structure, or the narrative structure in which they appear, LIWC often misses humor, sarcasm, or cultural nuances. For example, if a person says "I was not calm", LIWC will count "calm" as a positive emotion word and disregard the fact that the sentence actually refers to a negative experience. Similarly, the word "mad" would be categorized under "Anger" words even if the context in which it appears is "I was mad about her" (Tausczik & Pennebaker, 2010). To address this problem, further studies should analyze emotional and cognitive language through manual coding done by at least two trained coders abiding by a pre-specified set of rules. Another option is to use a computerized program that counts groups of words as well as

individual words, such as n-grams, thus taking context into consideration (Oberlander & Gill, 2006).

Only women participated in this study. While the study population was indeed special in that it constitutes a civilian, rather than military, sample of individuals at a very high-risk of developing PTSD, the findings cannot be generalized to men. Furthermore, previous studies suggest that women are twice more likely to develop PTSD than men are (Breslau et al., 1998). Future studies should investigate the language used in narratives of trauma of a sample of traumatized civilian men.

Adding vantage point as an independent variable may also produce a relevant future study. It has been shown that trauma can be remembered from either a field or an observer perspective, and that the qualities of the recalled memories are different in the two scenarios: the field recall is more emotional in nature, while the observant recall is more avoidant (McIsaac & Eich, 2004). In future studies, participants could be instructed to recall their trauma memories from only one of the two perspectives, and the linguistic differences between the resulting memories may be analyzed to determine whether one of the two vantage points predicts PTSD symptomology. A hypothesis might be that people who remember their trauma from an observant perspective are more avoidant, in which case therapy encouraging the adoption of the field perspective may be beneficial.

Conclusions

In summary, we found that individuals diagnosed with PTSD used more affect language, and specifically more negative emotion language, when narrating their worst child and adult traumas. The use of affect language was correlated with PTSD symptoms

and was associated with emotional responses in the medial prefrontal cortex. PTSD resilience was not associated with greater cognitive processing of emotional memories, as measured by the frequency of cognitive words in trauma memories, or by greater medial prefrontal cortex activation in response to positive or negative emotional stimuli. This study furthers the current research on the processing of emotional stimuli by individuals with PTSD, and could be potentially used for implementing new therapeutic methodologies for treating PTSD. Future research should investigate emotional memory in PTSD by co-analyzing the narrative structure and content of trauma memories narrated within a scanner and the activation of specific brain regions when individuals do so.

References

Alim T.N., Feder A., Graves R.E., Wang Y., Weaver J., Westphal M., Alonso A., Aigbogyn N.Y., Smith B.W., Doucette J.T., Mellman T.A., Lawson W.B., & Charney D.S. (2008). Trauma, resilience, and recovery in a high-risk African-American population. American Journal of Psychiatry, 165(12), 1566-1575.

Alvarez-Conrad J., Zoellner L.A., & Foa E.B. (2001) Linguistic Predictors of Trauma Pathology and Physical Health. Applied Cognitive Psychology, 15, S159-S170.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders*, (4th ed.). Washington, DC: American Psychiatric Publishing.

American Psychiatric Association. (2013) *Diagnostic and statistical manual of mental disorders*, (5th ed.). Arlington, VA: American Psychiatric Publishing.

Amunts K., Kedo O., Kindler M., Pieperhoff P., Mohlberg H., Shah N.J., Habel U., Schneider F., & Zilles K. (2005). Cytoarchitectonic mapping of the human amygdala, hypocampal region, and entorhinal cortex: intersubject variability and probability maps. Anatomy and Embryology, 210, 343-352.

Armony J.L., Corbo V., Clément M.H., & Brunet A. (2005) Amygdala response in patients with acute PTSD to masked and unmasked emotional facial expressions. American Journal of Psychiatry, 162(10), 1961-3.

Asmundson G.J., Norton G.R., Allerdings M.D., Norton P.J., Larsen D.K. (1998). Posttraumatic stress disorder and work-related injury. Journal of Anxiety Disorders, 12, 57-69.

Banich, M.T., Mackiewicz, K.L., Depue, B.E., Whitmer, A.J., Miller, G.A., & Heller,

W., 2009. Cognitive control mechanisms, emotion and memory: a neural perspective with implications for psychopathology. *Neuroscience and Biobehavioral Reviews* 33 (5), 613–630.

Bantum E.O., & Owen J.E. (2009). Evaluating the validity of computerized content analysis programs for identification of emotional expression in cancer narratives. Psychological Assessment, 21, 79-88.

Berntsen D. (2001). Involuntary memories of emotional events. Do memories of traumas and extremely happy event differ? Applied Cognitive Psychology, 15, 135-158.

Berntsen, D., Willert, M., & Rubin, D. C. (2003). Splintered memories or vivid landmarks? Qualities and organization of traumatic memories with and without PTSD. Applied Cognitive Psychology, 17, 675– 693.

Berntsen D., & Rubin D.C. (2006) The centrality of event scale: a measure of integrating a trauma into one's identity and its relation to post-traumatic stress disorder symptoms. Behaviour Research and Therapy, 44(2), 219-231.

Bernstein D.P., Fink L., Handelsman L., Foote J., Lovejoy M., Wenzel K., Sapareto E., & Ruggiero J. (1994). Initial reliability and validity of a new retrospective measure of child abuse and neglect. American Journal of Psychiatry, 151(8), 1132-1136.

Binder E.B., Bradley R.G., Liu W., Epstein M.P., Deveau T.C., Mercer K.B., Tang Y., Gillespie C.F., Heim C.M., Nemeroff C.B., Schwartz A.C., Cubells J.F., & Ressler K.J. (2008). Association of *FKBP5* polymorphisms and childhood abuse with risk of post-traumatic stress disorder symptoms in adults. The Journal of the American Medical Association, 299(11), 1291–1305.

Birn R.M., Patriat R., Phillips M.L., Germain A., & Herringa R.J. (2014). Childhood

maltreatment and combat posttraumatic stress differentially predict fear-related fronto-subcortical connectivity. Depression and Anxiety, 31(10), 880-892.

Blake D.D., Weathers F.W., Nagy L.M., Kaloupek D.G., Charney D.S., & Keane T.M. (1995). The development of a clinician-administred PTSD scale. Journal of Traumatic Stress, 8, 75-90.

Boals A., & Rubin D.C. (2011). The Integration of Emotions in Memories: Cognitive-Emotional Distinctiveness and Posttraumatic Stress Disorder. Applied Cognitive Psychology, 25(5), 811-816.

Boals, A., & Klein, K. (2005). Word use in emotional narratives about failed romantic relationships and subsequent mental health. Journal of Language and Social Psychology, 24, 252-268

Boals A., Hayslip B. Jr., & Banks J.B. (2014) Age differences in autobiographical memories of negative events. International Journal of Aging and Human Development, 78(1), 47-65.

Bohanek J.G., Fivush R., & Walker E. (2005). Memories of Positive and Negative Emotional Events. Applied Cognitive Psychology, 19, 51-66.

Bonanno G.A. (2004) Loss, trauma, and human resilience: Have we underestimated the human capacity to thrive after extremely aversive events? American Psychologist, 59, 20-28.

Boscarino JA, Erlich PM, Hoffman SN, Rukstalis M, & Stewart WF (2011) Association of *FKBP5*, *COMT* and *CHRNA5* polymorphisms with PTSD among outpatients at risk for PTSD. Psychiatry Research, 188(1), 173–174.

Bremner J.D., Narayan M., Staib L.H., Southwick S.M., McGlashan T., & Charney D.S.

(1999). Neural correlates of memories of childhood sexual abuse in women with and without posttraumatic stress disorder. American Journal of Psychiatry 156(11), 1787-1795.

Breslau N., Kessler R.C., Chilcoat H.D., Schultz L.R., Davis G.C., & Andreski P. (1998). Trauma and posttraumatic stress disorder in the community: the 1996 Detroit Area Survey of Trauma. Archives of General Pscyhiatry, 55(7), 626-632.

Breslau N., Chen Q., & Luo Z. (2013). The role of intelligence in posttraumatic stress disorder: does it vary by trauma severity? PLOS One, 8(6), e65391.

Brewin, C. R. (2001). Memory processes in post-traumatic stress disorder. International Review of Psychiatry, 13, 159–163.

Broekhof R., Rius-Ottenheim N., Spinhoven P., van der Mast R.C., Penninx B.W., Zitman F.G., Gilltay E.J. (2015). Long-lasting effects of affective disorders and childhood trauma on dispositional optimism. Journal of Affective Disorders, 175, 351-358.

Bruner, J. (1987). Life as narrative. Social Research, 54, 11-32.

Carver C.S. (1997). You want to measure coping but your protocol's too long: consider the brief COPE. International Journal of Behavioral Medicine, 4(1), 92-100.

Clark D.M. (1999). Anxiety Disorders: Why they persist and how to treat them. Behaviour Research and Therapy, 37, S5-S27.

Cohn M.A., Mehl M.R., & Pennebaker J.W. (2004) Linguistic Markers of Psychological Change Surrounding September 11, 2001. Psychological Science, 15(10), 687-693.

Davis M. & Whalen P.J. (2001). The amygdala: vigilance and emotion. Molecular

Psychiatry, 6(1), 13-34.

Ehlers A., & Clark D.M. (2000). A cognitive model of posttraumatic stress disorder. Behaviour Research and Therapy, 38(4), 319-345.

Eickhoff S.B., Stephan K.E., Mohlberg H., Grefkes C., Fink G.R., Amunts K., & Zilles K. (2005). A new SPM toolbox for combining probabilistic cytoarchitectonic maps and functional imaging data. Neuroimage, 25(4), 1325-1335.

Engelhard I.M., van den Hout M.A., Kindt M., Arntz A., & Schouten E. (2003). Peritraumatic dissociation and posttraumatic stress after pregnancy loss: a prospective study. Behaviour Research and Therapy, 41(1), 67-78.

Fani N., Gutman D., Tone E.B., Almli L., Mercer K.B., Davis J., Glover E., Jovanovic T., Bradley B., Dinov I.D., Zamanyan A., Toga A.W., Binder E.B., & Ressler K.J. (2013) FKBP5 and Attentioan Bias for Threat: Associations With Hippocampal Function and Shape. JAMA Psychiatry, 70(4), 392-400.

Fani N., King T.Z., Jovanovic T., Glover E.M., Bradley B., Choi K., Ely T., Gutman D.A., & Ressler K.J. (2012). White matter integrity in highly traumatized adults with and without post-traumatic stress disorder. Neuropsychpharmacology 37(12): 2740-2746.

Fava G.A., Kellner R., Munari F., Pavan L., Pesarin F. (1982). Losses, hostility, and depression. Journal of Nervous Mental Disorders, 170, 474-478.

Feder A., Nestler E.J., & Charney D.S. (2009). Psychobiology and molecular genetics of resilience. Nature Reviews Neuroscienc, 10, 446-457.

Feeny N.C., Zoellner L.A., Fitzgibbons L.A., Foa E.B. (2000). Exploring the roles of emotional numbing, depression, and dissociation in PTSD. Journal of Trauma and

Stress, 13(3), 489-498.

Foa E.B. & Tolin D. F. (2000). Comparison of the PTSD Symptom Scale-Interview Version and the Clinician-Administered PTSD scale. Journal of Trauma and Stress, 13(2), 181-191.

Fonzo G.A., Simmons A.N., Thorp S.R., Norman S.B., Paulus M.P., & Stein M.B. (2010). Exaggerated and disconnected insular-amygdalar blood oxygenation level-dependent response to threat-related emotional faces in women with intimate-partner violence posttraumatic stress disorder. Biological Psychiatry, 68(5): 433-441.

Fossati P. (2013). Imaging autobiographical memory. Dialogues in Clinical Neuroscience,15(4): 487-490.

Fredrickson B.L., Tugade M.M., Waugh C.E., & Larkin G.R. (2003). What Good Are Positive Emotions in Crises? A Prospective Study of Resilience and Emotions Following the Terrorist Attacks on the United States on September 11th, 2001. Journal of Personality and Social Psychology, 84(2), 365-376.

Garavan H., Pendergrass J.C., Ross T.J., Stein E.A., Risinger R.C. (2001) Amygdala response to both positively and negatively valenced stimuli. Neuroreport, 12(12), 2779-2783.

Gillespie, C. F., Bradley, B., Mercer, K., Smith, A. K., Conneely, K., Gapen, M. & Ressler, K. J. (2009). Trauma exposure and stress-related disorders in inner city primary care patients. General hospital psychiatry, *31*(6), 505-514.

Gottschalk, L.A., & Gleser G.C. (1969). *The measurement of psychological states through the content analysis of verbal behavior*. Berkeley: University of

California Press.

Halligan S.L., Clark D.M., & Ehlers A. (2002). Cognitive processing, memory, and the development of PTSD symptoms: two experimental analogue studies. Journal of Behavior Therapy and Experimental Psychiatry, 33(2), 73-89.

Halligan S.L., Michael T., Clark D.M., & Ehlers A. (2003). Posttraumatic Stress Disorder Following Assault: The Role of Cognitive Processing, Trauma Memory, and Appraisals. Journal of Consulting and Clinical Psychology, 71(3), 419-431.

Hamann S., Mao H. (2001). Positive and negative emotional verbal stimuli elicit activity in the left amygdala. NeuroReport, 13(1), 15-19.

Herman J.L. (1992) *Trauma and Memory*. Basic Books: New York.

Hellawell, S. J., & Brewin, C. R. (2004). A comparison of flashbacks and ordinary autobiographical memories of trauma: Content and language. Behaviour Research and Therapy, 42, 1–12.

Hinton D., Rasmussen A., Nou L., Pollack M., Mary-Jo G. (2009). Anger, PTSD, and the Nuclear Family: A Study of Cambodian Refugees. Social Science and Medicine, 69(9), 1387-1394.

Holahan C.J., Moos R.H., Holahan C.K., Brennan P.L., Schutte K.K. (2005). Stress Generation, Avoidance Coping, and Depressive Symptoms: A 10-Year Model. Journal of Consulting and Clinical Psychology, 73(4), 658-666.

Holmes D., Alpers G.W., Ismailji T., Classen C., Wales T., Cheasty V., Milller A., & Koopman C. (2007). Cognitive and emotional processing in narratives of women abused by intimate partners. Violence Against Women, 13(11), 1192-1205.

Holmes A.P., Josephs O., Büchel C., Friston K.J. (1997). Statistical modeling of low

frequency confounds in fMRI. NeuroImage 5:S480.

Horowitz, M. J. (1975). Intrusive and repetitive thought after experimental stress. Archives of General Psychiatry, 32, 1457–1463.

Horowitz, M. J., & Reidbord, S. P. (1992). Memory, emotion, and response to trauma. In S.A. Christianson (Ed.), The handbook of emotion and memory. Research and theory (pp. 343–357). Hillsdale, New Jersey: Lawrence Erlbaum.

Jaeger , J., Lindblom, K.M., Parker-Guilbert, K., & Zoellner, L.A. (2014). Trauma Narratives: It's What You Say, Not How You Say It. Psychological Trauma, 6(5), 473-481.

Jelinek L., Stockbauer C., Randjbar S., Kellner M. Ehring T., & Moritz S. (2010). Characteristics and organization of the worst moment of trauma memories in posttraumatic stress disorder. Behaviour Research and Therapy, 48(7), 680-685.

Jones, C., Harvey, A. G., & Brewin, C. R. (2007). The organisation and content of trauma memories in survivors of road traffic accidents. Behaviour Research and Therapy, 45, 151–162.

Kahn J.H., Tobin R.M., Massey A.E., & Anderson J.A. (2007). Measuring emotional expression with the Linguistic Inquiry and Word Count. The American Journal of Psychology, 120(2), 263-286.

Kaufman J., Plotsky P.M., Nemeroff C.B., Charney D.S. (2000). Effects of early adverse experiences on brain structure and function: clinical implications. Biological Psychiatry, 48(8), 778-790.

Kenardy, J., Smith, A., Spence, S. H., Lilley, P.R., Newcombe, P., Dob, R., & Robinson, S. (2007). Dissociation in children's trauma narratives: An exploratory

investigation. Journal of Anxiety Disorders, 21, 456 – 466.

Kessler R.C., Sonnega A., Bromet E., Hughes M., & Nelson C.B. (2005). Posstraumatic stress disorder in the National Comorbidity Survey. Archives of General Psychiatry, 52(12), 1048-1060.

Koenigs M., & Grafman J. (2009). Post-traumatic stress disorder: The role of medial prefrontal cortex and amygdala. Neuroscientist, 15(5), 540-548.

Lang, P.J., Bradley M.M., & Cuthbert, B.N. (2008). International affective picture system (IAPS): Affective ratings of pictures and instruction manual. *Technical Report A-8*. University of Florida: The Center for research in Psychophysiology.

Lanius R.A., Williamson P.C., Densmore M., Bokman K., Gupta M.A., Neufeld R.W., Gati J.S., & Menon R.S. (2001). Neural correlates of traumatic memories in posttraumatic stress disorder: a functional MRI investigation. American Journal of Psychiatry, 158(11), 1920-1922.

Lanius R.A., Williamson P.C., Densmore M., Boksman K., Neufeld R.W., Gati J.S., Menon R.S. (2004). The Nature of Traumatic Memories: A 4-T fMRI Functional Connectivity Analysis. American Journal of Psychiatry, 160, 1-9.

LeDoux J.E. (2000). Emotion Circuits in the Brain. Annual Review of Neuroscience, 23, 155-184.

Liberzon I., Taylor S.F., Amdur R., Jung T.D., Chamberlain K.R., Minoshima S., Koeppe R.A., & Fig L.M. (1999.) Brain activation in PTSD in response to trauma-related stimuli. Biological Psychiatry, 45(7), 817-26.

Macklin M.L., Metzger L.J., Litz B.T., McNally R.J., Lasko N.B., Orr S.P., & Pitman R.K. (1998). Journal of Counseling and Clinical Psychology, 66, 323-326.

Marusak H.A., Martin K.R., Etkin A., & Thomason M.E. (2015). Childhood Trauma Exposure Disrupts the Automatic Regulation of Emotional Processing. Neuropsychopharmacology, 40, 1250-1258.

Mazaika, P., Whitfield-Gabrieli, S., Reiss A.L. (2007). "Artifact repair for fMRI data from high motion clinical subjects," in Paper Presented at the Annual Meeting of the Organization for Human Brain Mapping.

McIsaac H.K., & Eich E. (2004).Vantage point in traumatic memory. Psychological Science, 15(4), 248-253.

McNally R.J., & Shin L.M. (1995). Association of Intelligence with severity of posttraumatic stress disorder symptoms in Vietnam Combat veterans. American Journal of Psychiatry, 152(6), 936-938.

McTeague L.M., Lang P.J., Laplante M.C., Cuthbert B.N., Shumen J.R., & Bradley M.M. (2010) Aversive imagery in posttraumatic stress disorder: trauma recurrence, comorbidity, and physiological reactivity. Biological Psychiatry, 67(4), 346-356.

Milad M.R., Pitman R.K., Ellis C.B., Gold A.L., Shin L.M., Lasko N.B., Zeidan M.A., Handwerger K., Orr S.P., & Rauch S.L. (2009). Neurobiological Basis of Failure to Recall Exctinction Memory in Posttraumatic Stress Disorder. Biological Psychiatry, 66(12), 1075-1082.

Mostoufi S., Godfrey K.M., Ahumada S.M., Hossain N., Song T., Wright L.J., Lohr J.B., & Afari N. (2014). Pain sensitivity in posttraumatic stress disorder and other anxiety disorders: a preliminary case control study. Annals of General Psychiatry, 13(31).

Negrao, C., Bonanno, G. A., Noll, J. G., Putnam, F. W., & Trickett, P. K. (2005). Shame, humiliation, and childhood sexual abuse: Distinct contributions and emotional coherence. Child Maltreatment, 10, 350 –363.

Nijenhuis, E. R. S., & van der Hart, O. (1999). Forgetting and reexperiencing trauma. In J. Goodwin, & R. Cittias (Eds.), Splintered reflections: Images of the body in the trauma (pp. 39–65). New York: Basic Books.

Oberlander, J., & Gill, A.J. (2006). Language with character: A Stratified Corpus Comparison of individual differences in e-mail communication. Discourse Processes, 42(3), 239-270.

Ogle C.M., Rubin D.C., Berntsen D., & Siegler I.C. (2013). The Frequency and Impact of Exposure to Potentially Traumatic Events Over the Life Course. Clinical Psychological Science, 1(4), 426-434.

Ong A.D., Bergeman C.S., Bisconti T.L., Wallace K.A. (2006). Psychological resilience, positive emotions, and successful adaptation to stress in later life. Journal of Personality and Social Psychology, 91(4), 730-749.

Quirk G.J., Likhtik E., Pelletier J.G., & Paré D. (2003). Stimulation of medial prefrontal cortex decreases the responsiveness of central amygdala output neurons. Journal of Neuroscience, 23(25), 8800-8807.

Palyo S.A., & Beck J.G. (2005). Post-traumatic stress disorder symptoms, pain, and perceived life control: Associations with psychosocial and physical functioning. Pain, 117(1-2), 121-127.

Parsons R.G., Ressler K.J. (2013). Implications of memory modulation for post-traumatic stress and fear disorders. Nature Neuroscienc, 16(2), 146-153.

Pennebaker J.W., Booth R.E., & Francis M.E. (2007). Linguistic inquiry and word count: LIWC – operator's manual.

Pennebaker, J., Francis, M., & Booth, R. J. (2001). Linguistic inquiry and word count (LIWC): LIWC2001. Mahwah, NJ: Erlbaum.

Pennebaker, J. W., Mehl, M. R., & Niederhoffer, K. G. (2003). Psychological aspects of natural language use: Our words, our selves. Annual Review of Psychology, 54, 547–577.

Porter S., Birt A.R. (2001). Is traumatic memory special? A comparison of traumatic memory characteristics with memories for other life experiences. Applied Cognitive Psychology, 15, 101-117.

Rauch S.L., Whalen P.J., Shin L.M., McInerney S.C., Macklin M.L., & Lasko N.B. (2000). Exaggerated amygdala response to masked facial stimuli in posttraumatic stress disorder: a functional MRI study. Biological Psychiatry, 47(9), 769-776.

Rampp C., Binder E.B., & Provençal N. (2014). Chapter Two – Epigenetics in Posttraumatic Stress Disorder. Progress in Molecular Biology and Translational Science, 128, 29-50.

Reviere S.L., & Bakeman R. (2001) The effects of early trauma on autobiographical memory and schematic self-representation. Applied Cognitive Psychology, 15, 89-100.

Riley W.T., Treiber F.A., Woods M.G. (1989). Anger and hostility in depression. Journal of Nervous Mental Disorders, 177, 668-674.

Robinaugh D.J., & McNally R.J. (2011). Trauma centrality and PTSD symptom severity in adult survivors of childhood sexual abuse. Journal of Trauma and Stress, 24(4),

483-486.

Rosenberg, S.D. & Tucker, G.J. (1978). Verbal behavior and schizophrenia: The semantic dimension. Archives of General Psychiatry, 36, 1331-1337.

Rothbaum B.O., Foa E.B., Riggs D.S., Murdock T., Walsh W. (1992). A prospective examination of post-traumatic stress disorder in rape victims. Journal of Traumatic Stress, 5(3), 455-475.

Rubin D.C, Feldman M.E., & Beckham J.C. (2004). Reliving, emotions and fragmentation in the autobiographical memories of veterans diagnosed with PTSD. Applied Cognitive Psychology, 18, 17-35.

Rubin D.C., Berntsen D., & Boals A. (2008). Memory in Posttraumatic Stress Disorder: Properties of Voluntary and Involuntary, Traumatic and Nontraumatic Autobiographical Memories in People With and Without Posttraumatic Stress Disorder Symptoms. Journal of Experimental Psychology, 137, 591-614.

Rubin D.C., Dennis M.F., & Beckham J.C. (2011). Autobiographical Memory for Stressful Events: The Role of Autobiographical Memory in Posttraumatic Stress Disorder. Consciousness and Cognition, 20(3), 840-856.

Rubin D.C. (2011). The coherence of memories for trauma: Evidence from posttraumatic stress disorder. Consciousness and Cognition, 20(3), 857-865.

Sabatinelli D., Furtune E.E., Li Q., Siddiqui A., Krafft C., Oliver W.T., Beck S., Jeffries J. (2011). Emotional perception: Meta-analyses of face and natural scene processing. NeuroImage, 54(3), 2524-2533.

Sadeh N., Spielberg J.M., Warren S.L., Miller G.A., & Heller W. (2014). Aberrant Neural Connectivity during Emotional Processing Associated with Posttraumatic

Stress. Clinical Psychological Science, 2(6), 748-755.

Schelach, L., & Nachson, I. (2001). Memory of Auschwitz survivors. Applied Cognitive Psychology, 15, 119–132.

Scher C.D., Stein M.B., Asmundson G.J.G., McCreary D.R., & Forde D.R. (2001). The Childhood Trauma Questionnaire in a community sample: Psychometric properties and normative data. Journal of Trauma and Stress 14(4): 843-857.

Schwartz A.C., Bradley R.L., Sexton M., Sherry A., & Ressler K.J. (2005). Posttraumatic stress disorder among African Americans in an inner city mental health clinic. Psychiatric Services, 56(2), 212-215.

Shin L.M., Rauch S.L., & Pitman R.K. (2006). Amygdala, Medial Prefrontal Cortex, and Hippocampal Function in PTSD. Annals of the New York Academy of Sciences, 1071, 67-79.

Shin L.M., Wright C.I., Cannistraro P.A., Wedig M.M., McMullin M.M., Maris B., Macklin M.L., Lasko N.B., Cavanagh S.R., Krangel T.S., Orr S.P., Pitman R.K., Whalen P.J., & Rauch S.L. (2005). A functional magnetic resonance imaging study of amygdala and medial prefrontal cortex responses to overtly presented fearful faces in posttraumatic stress disorder. Archives of General Psychiatry, 62(3), 273-281.

Shobe K.K., & Kihlstrom J.F. (1997). Is traumatic memory special? Current Directions in Psychological Science, 6, 70-74.

Simmons A.N., Matthews S.C., Strigo I.A., Baker D.G., Donovan H.K., Motezadi A., Stein M.B., & Paulus M.P. (2011). Altered amygdala activation during face processing in Iraqi and Afghanistani war veterans. Biology of Mood and Anxiety

Disorders, 1(1), 6.

Sripada R.K., King A.P., Garfinkel S.N., Wang X., Sripada C.S., Welsh R.C., & Liberzon I. (2012). Altered resting-state amygdala functional connectivity in men with posttraumatic stress disorder. Journal of Psychiatry and Neuroscience, 37(4), 241-249.

Stevens S.J., Jovanovic T., Fani N., Ely T.D., Glover E.M., Bradley B., & Ressler K.J. (2013) Disrupted amygdala prefrontal functional connectivity in civilian women with posttraumatic stress disorder. Journal of Psychiatric Research, 47(10), 1469-1478.

Stevens J.S., Almli L.M., Fani N., Gutman D.A., Bradley B., Norrholm S.D., Reiser E., Ely T.D., Dhanani R., Glover E.M., Jovanovic T., Ressler K.J. (2014). PACAP receptor gene polymorphism impacts fear responses in the amygdala and hippocampus. Proceeding of the National Academy of Science, 111(8), 3158-3163.

Stiles, W.B. (1992). Describing talk: A taxonomy of verbal response modes. Newbury Park, CA: Sage.

St Jacques P.L., Botzung A., Miles A., & Rubin D.C. (2011). Functional neuroimaging of emotionally intense autobiographical memories in post-traumatic stress disorder. Journal of Psychiatric Research, 45, 630-637.

St. Jacques P.L., Kragel P.A., & Rubin D.C. (2013). Neural Networks supporting autobiographical memory retrieval in posttraumatic stress disorder. Cognitive, Affective, & Behavioral Neuroscience, 13(3), 554-566.

Tausczik Y.R. & Pennebaker J.W. (2010). The Psychological Meaning of Words: LIWC

and Computerized Text Analysis Methods. Journal of Language and Social Psychology, 29(1), 24-54.

Tugade M.M. & Friedrickson B.L. (2004). Resilient Individuals Use Positive Emotions to Bounce Back From Negative Emotional Experiences. Journal of Personality and Social Psychology, 86(2), 320-333.

Uddin M., Aiello A.E., Wildman D.E., Koenen K.C., Pawelec G., de Los Santos R., Goldmann E., Galea S. (2010). Epigenetic and immune function profiles associated with posstraumatic stress disorder. Proceedings of the National Academy of Science, 107(20), 9470-9475.

Van der Kolk, B. A., & Fisler, R. (1995). Dissociation and the fragmentary nature of traumatic memories: overview and exploratory study. Journal of Traumatic Stress, 8, 505–525.

Wagenaar, W. A., & Groeneweg, J. (1990). The memory of concentration camp survivors. Applied Cognitive Psychology, 4, 77–87.

Watkins P.C., Vache K., Verney S.P., Muller S., Matthews A. (1996). Unconscious mood-congruent memory bias in depression. Journal of Abnormal Psychology, 105(1), 34-41.

Whalen P.J., Rauch S.L., Etcoff N.L., McInerney S.C., Lee M.C., & Jenike M.A. (1998). Masked Presentations of Emotional Facial Expressions Modulate Amygdala Activity without Explicit Knowledge. The Journal of Neuroscience, 18(1), 411-418.

Williams L.M., Kemp A.H., Felmingham K., Barton M., Olivieri G., Peduto A., Gordon E., & Bryant R.A. (2006). Trauma modulates amygdala and medial prefrontal

responses to consciously attended fear. Neuroimage, 29(2), 347-57.

Wu G., Feder A., Cohen H., Kim J.J., Calderon S., Charney D.S., & Mathé A.A. (2013). Understanding Resilience. Frontiers of Behavioral Neuroscience, 7, 10.

Zoellner, L. A., Alvarez-Conrad, J. A., & Foa, E. B. (2002). Peritraumatic dissociative experiences, trauma narratives, and trauma pathology. Journal of Traumatic Stress, 15(1), 49-57.

Appendix A. LiWC 2007 Dimensions and Sample Words

DIMENSION	EXAMPLES	# WORDS
I. STANDARD LINGUISTIC DIMENSIONS		
Total function words		464
Total pronouns	I, them, itself	116
Personal pronouns	I, them, her	70
1st person singular . . . ,	I, me, mine	12
1st person plural	we, our, us	12
2nd person	you, your, thou	20
3rd person singular	she, her, him	17
3rd person plural	they, their, they'd	10
Impersonal pronouns	It, its's those	46
Articles	a, an, the	3
Verbs	walk, went, see	383
Auxiliary verbs	Am, will,have	144
Past tense	walked, were, had	145
Present tense	Is, does, hear	169
Future tense	will, gonna	48
Adverbs	very, really, qucikly	69
Prepositions	with, above	60
Conjonctions	but, whereas	28
Negations	no, never, not	57
Quantifiers	few, many, much	89
Numbers	one, thirty, million	34
Swear words	damn, fuck, piss	53
II. PSYCHOLOGICAL PROCESSES		
Social Processes	talk, us, friend	455
Friends	pal, buddy, coworker	37
Family	mom, brother, cousin	64
Humans	boy, woman, group	61
Affective Processes	happy, ugly, bitter	915
Positive Emotions	happy, pretty, good	405
Negative Emotions	hate, worthless, enemy	499
Anxiety	nervous, afraid, tense	91
Anger	hate, kill, pissed	184
Sadness	grief, cry, sad	101
Cognitive Processes	cause, know, ought	730
Insight	think, know, consider	195
Causation	because, effect, hence	108

Discrepancy	should, would, could	76
Tentative	maybe, perhaps, guess	155
Certainty	always, never	83
Inhibition	block, constrain	111
Inclusive	with, and, include	18
Exclusive	but, except, without	17
Perceptual Processes	see, touch, listen	273
Seeing	view, saw, look	72
Hearing	heard, listen, sound	51
Feeling	touch, hold, felt	75
Biological Processes	eat, blood, pain	567
Body	ache, heart, cough	180
Health	clinic, flu, pill	236
Sexuality	horny, love, incest	96
Ingestion	eat, swallow, taste	111
Relativity	area, bend, exit, stop	638
Motion	walk, move, go	168
Space	Down, in, thin	220
Time	hour, day, oclock	239
III. PERSONAL CONCERNS		
Work	work, class, boss	327
Achievement	try, goal, win	186
Leisure	house, TV, music	229
Home	house, kitchen, lawn	93
Money	audit, cash, owe	173
Religion	altar, church, mosque	159
Death	bury, coffin, kill	62
IV. SPOKEN CATEGORIES		
Assent	agree, OK, yes	30
Nonfluencies	uh, rr*	8
Fillers	blah, you know, I mean	9

Appendix B. Affect and Cognitive Word Categories and Subcategories.

AFFECT WORDS				
Positive Emotions	**Negative Emotions**	**Anxiety**	**Anger**	**Sadness**
acceptance, affection, agree, appreciation, attract, beautiful, best, calm, comedy, comfort, cool, cute, easy, hug, improve, joke, like, LOL, love, nice, okay, promise, party, play, respect, rich, safe, sure, thanks, true, wow	abuse, afraid, alone, anger, asshole, attack, awful, awkward, bitch, boring, crazy, cry, danger, disaster, dislike, emotional, enemy, fail, fake, fear, fuck, gossip, gross, hate, helpless, insult, lazy, loss, mad, pain, pity, rape, sad, serious, stress, suck, terror, trauma, weapon	afraid, anxious, avoid, embarrassing, fear, guilt, scary, shy, stress, suspicion, tremble, upset, worried	agitated, angry, attack, contempt, cruel, danger, disgust, envy, fury, hate, hell, idiot, offense, punish, rage, rude, stupid, torture, violate, weapon	abandon, cry, defeat, despair, discourage, dull, fail, heartbreak, hopeless, lonely, lost, low, miss, pity, remorse, suffer, tears, whine, worthless

COGNITIVE WORDS							
Insight	**Causation**	**Discrepancy**	**Tentative**	**Certainty**	**Inhibition**	**Inclusive**	**Exclusive**
accept, admit, aware, become, believe, choose, confess, consider, discover, effect, feel, idea, information, insight, know, knowing, learn, lesson, mean, notice, prove, question, refer, remember, secret, seem, solve, think, understand, wonder	affect, allow, because, change, control, depend, effect, hence, how, make, obey, origin, purpose, react, reason, since, therefore, thus, use, why	besides, could, hope, if, must, need, ought, should, undo, want, wish, would	almost, anything, anywhere, barely, depend, doubt, fuzz, guess, if, kind, luck, may, might, most, often, probably, quite, shaky, someone, something, sometime, suppose, unclear, wonder	absolute, all, clear, complete, correct, definitely, everything, exact, indeed, must, never, perfect, proof, sure, true,	abandon, avoid, block, careful, defense, duty, forbid, ignore, keep, limit, neglect, protect, restrict, safe, stop, tight, wait, yield	add, around, both, came, each, inside, open, plus, we, with	but, either, except, if, just, not, or, sometime, versus, whether, without

Appendix C. Examples of trauma narratives

Childhood (sexual abuse, experienced):

Participant: *I was not a very popular kid. And there was this little boy named Eddy. He had a tent. And all the kids got to go to the tent, except for me. So, um, because I was little, I, and I wanted to be a part of the tent, so one day, my mom was busy and my brother was out doing something, And he tells me if you want to be in my tent, you have to perform oral sex on me. Of course he didn't say oral sex, not oral sex, suck my dick. So, um, I was like ok, whatever. So he takes me to this little patch of our apartments, and he made me give him head. So this continued until it became like, abusive. Like, if you don't do that, I'll beat you up. So, that was that.*
Interviewer: *And how old was he?*
P: *He had to be about my brother's age. So I'm gonna say he had to be between 9 and 11.*
I: *Ok. And you were 4 years old?*
P: *Mhm.*
I: *And you said that sort of eventually it became this way? Like how long did it, was this going on?*
P: *Almost a year.*

Childhood (domestic abuse, experienced):

P: *I'll answer. I was abused as a child, that's how I ended up in foster care. My mom, my mom abused me. Mhm.*
I: *And was there a specific time that you remember that was particularly bad? That , you know…*
P: *I can remember one time. I shared with the young man, too. Um, I was in foster care, they put me in foster care 9 years old, and they released me back to my mom at 13. I wasn't with her but one week before she snapped again. And she hit me in my mouth, with the broomstick, and the mark still there. I just never forgot that. Cause when she beat me, it was because my little brother had lied on me, which he was a troubled young man anyway. And she just took that lie and just snapped on me. So they took me immediately out of her house again. And I ended up back in foster care. And they arrested her and whatever, I don't know. But I remember that, it was like, wow. All the beatings she gave me, that particular one I remember.*

Childhood Trauma (violence, confronted):

I: *So you mentioned that a couple of different things have happened to you when you were younger, um. One was that, um, your cousin was murdered?*
P: *Yeah.*
I: *Can you tell me a little more about that?*
S: *Yeah, my cousin was murdered. Um, it was in Louisiana, and he was murdered, and they brought him back to my aunt's house, the one that I'm very close to, my mother's older sister. And they hung him in the tree. And we got the call that something had happened. Um, we drove to their house. And he was still hanging in the tree when we got there. So we saw his body hanging from the tree. And my first cousin, got a sheet and, you know, threw it over him of course, and then they got something and cut him down. So I was like, that just did a number on me, because I was like, I never, never seen that before. You know, I've never seen that before. And then, in February, when they have black history month, you think of, they go over, you know, slavery, and the hangings, and all this kind of staff so that's the first thing that pop in your mind. Oh my God. You know, of course, we found out that it was drug-related and all this kind of stuff so I guess*

however did it was sending a sign, a message to however, however, I don't know. But yeah, but that was, I was in my mid-teens when that happened.
I: *So about 15?*
P: *Mhm.*
I: *And, you know, is that still something that still affects you today, or maybe it comes back to you in nightmares, or intrusive memories, where it's really hard to get out of your head?*
P: *Sometimes, sometimes, because is like, when you hear different scenarios that happened to people, like when the Travyon Martin case happened, that brought that back to my memory because it was a white person who did this to my cousin.*

Adult Trauma (armed violence, witnessed):

P: *I was 22. Like this guy, who went to school with us, he was arguing with somebody, I don't remember who he was arguing with, but like, drew the guy in the corner, and the guy like, shot him. Shot him like 4 times or something, and he ended up dying right there. So like…*
I: *Did you see him get shot?*
P: *Mhm. But they caught the guy who did it, and he got like life in prison now, so… But like, just being there, and like, see him. You see him displaying the gun, but you don't expect for them to actually shot each other. Because, it's like, I guess, um, I don't know how to say this, that's not supposed to be a part of life.*
I: *Yeah, no, I know what you mean exactly.*
P: *But like, when he shot him, it was like, it sounded like a broken wing. And then just, how he like, I see, like I can see how he was laying, like he was just like, bottled up in like a fetal position. And then like, his sister was there, so his sister saw it. Like his sister was holding him and stuff.*

Adult Trauma (domestic violence, experienced):

P: *He was my first husband. Um, he was playing Russian Roulette. With me. And a gun.*
I: *Why? What was the reason?*
P: *He didn't want me to leave.*
I: *Ok. Like leave him?*
P: *No, like go out.*
I: *Ok. And did he pull the trigger?*
P: *I mean, several times, but the bullet wasn't in those specific holes.*
I: *Wow.*
P: *So, what made him stop?*
I: *Mhm?*
P: *I told him to go ahead and do it.*