THE EFFECT OF POSTTRAUMATIC STRESS AND TRAUMA-FOCUSED DISCLOSURE ON EXPERIMENTAL PAIN SENSITIVITY AMONG TRAUMA-EXPOSED WOMEN

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Digital Object Identifier: https://doi.org/10.13023/etd.2019.176

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Recommended Citation
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THE EFFECT OF POSTTRAUMATIC STRESS AND TRAUMA-FOCUSED DISCLOSURE ON EXPERIMENTAL PAIN SENSITIVITY AMONG TRAUMA-EXPOSED WOMEN

THESIS

A thesis submitted in partial fulfillment of the requirements for the degree of Master of Science in the College of Arts and Sciences at the University of Kentucky

By

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Lexington, Kentucky

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2019

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Previous studies evaluating the impact of trauma history and PTSD on pain sensitivity yield inconsistent findings; the presence of trauma-related negative affective states may account for these discrepancies. Therefore, the proposed study aimed to evaluate the effect of trauma-related negative affect and PTSD symptoms on sensory and affective components of pain sensitivity among trauma-exposed women. Adult women (N = 87) with low and high PTSD symptoms underwent an emotional disclosure paradigm, during which they wrote about a traumatic event or a neutral topic. Participants then completed a pain induction procedure. Compared to women with low PTSD symptoms, women with high PTSD symptoms demonstrated increased time to pain detection (e.g., threshold) and ability to withstand pain (e.g., tolerance), as well as increased pain intensity and when accounting for relevant covariates. Women with high PTSD symptoms who wrote about their worst traumatic experience reported higher pain unpleasantness relative to women with high PTSD symptoms who wrote about the neutral topic and women with low PTSD symptoms who wrote about either topic. Results suggest that PTSD symptoms and trauma-related negative affect may facilitate alterations in pain sensitivity in trauma-exposed women, but this relationship is complex and requires further exploration.

KEYWORDS: Trauma, Posttraumatic Stress, Emotional Disclosure, Pain Sensitivity

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April 26th, 2019
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ACKNOWLEDGMENTS

First and foremost, I would like to thank all of the women who participated in this research project. I would like to acknowledge the fantastic undergraduate research assistants who were instrumental in this project’s success. I would also like to thank the Psi Chi International Honor Society in Psychology and the Center for Clinical and Translational Science at the University of Kentucky for funding this study.

I am especially grateful for my mentor, Dr. Christal Badour, and her never-ending support, excitement, and patience. I would also like to extend a sincere thanks to my colleagues and friends in the Stress, Trauma, and Recovery Research Collaborative for their continuous support and solidarity.

A heartfelt thanks is due to my parents and sisters for their unconditional love and encouragement.
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Chapter One: Introduction

Posttraumatic Stress Disorder (PTSD) is a debilitating psychological disorder that develops in response to the experience of a traumatic event with actual or perceived threat (American Psychiatric Association [APA], 2013). Symptoms of PTSD are comprised of four symptom clusters: 1) re-experiencing (e.g., intrusive memories or flashbacks of the trauma), 2) avoidance (e.g., active avoidance of external reminders or thoughts and feelings associated with the trauma), 3) negative alterations in cognitions and mood (e.g., persistent negative emotional states, negative beliefs about the causes and impact of the trauma), and 4) alterations in arousal and reactivity (e.g., hypervigilance, reckless or destructive behavior; APA, 2013). Many negative mental and physical health outcomes have been associated with PTSD, including depressive and anxiety disorders, substance abuse, cardiovascular disease, and chronic pain (Galatzer-Levy, Nickerson, Litz, & Marmar, 2013; McFarlane, 2010).

Co-occurring PTSD and chronic pain (i.e., pain that is persistent or recurrent and lasts for longer than three months; Treede et al., 2015) appears to be particularly debilitating, as patients with both disorders report poorer quality of life and greater functional impairment than those with either disorder alone (Bryant, Marosszeky, Crooks, Baguley, & Gurka, 1999; Clapp, Beck, Palyo, & Grant, 2008). Additionally, compared to those without PTSD, chronic pain patients with PTSD report more severe pain-related interference, pain intensity, sleep dysfunction, and psychological distress (De Leeuw, Bertoli, Schmidt, & Carlson, 2005; Geisser, Roth, Bachman, & Eckert, 1996; Sherman, Turk, & Okifuji, 2000). Approximately 20-80% of patients being treated for PTSD report experiencing chronic pain; conversely, 10-50% of chronic pain patients
meet criteria for PTSD (Asmundson, Coons, Taylor, & Katz, 2002). Findings from a nationally representative survey suggest that individuals with PTSD are more likely to report a variety of pain conditions (e.g., arthritis/rheumatism, back/neck pain, headaches, and chronic pain) compared to trauma-exposed individuals without PTSD and those without a history of trauma (Sledjeski, Speisman, & Dierker, 2008). Additionally, PTSD is more prevalent among patients diagnosed with fibromyalgia, headaches, migraines, orofacial pain syndromes, accident-related pain, back pain, pelvic pain, mastalgia, and complex regional pain syndrome than among individuals without these pain conditions (see Moeller-Bertram, Keltner, & Strigo, 2012 for a review). PTSD has also been identified as a risk factor for the transition from acute to chronic pain (Kongsted et al., 2008; Shaw et al., 2010). Although research supporting the prevalence of comorbid PTSD and chronic pain conditions in both civilian and military populations has grown over the past several decades (for reviews see Beck & Clapp, 2011; Brennstuhl, Tarquinio, & Montel, 2015), the underlying mechanisms that maintain this relation require further exploration.

**Models of PTSD and chronic pain.** Several theoretical models have been proposed to account for the relationship between PTSD and chronic pain. Asmundson and Katz (2009) proposed the shared vulnerability model, in which individual difference factors may predispose an individual to develop co-occurring PTSD and chronic pain. This model proposes that individuals with psychological vulnerabilities (e.g., anxiety sensitivity, trait negative affect) and lowered alarm reaction thresholds (i.e., increased activity of the sympathetic nervous system) may experience more intense negative emotional reactions to traumatic stressors or injuries, therefore making the individual
more likely to develop co-occurring PTSD and chronic pain. Although empirical
evidence supports the idea that the development of PTSD and chronic pain may be
related to shared etiological factors (Asmundson & Katz, 2009), this model is limited in
that it does not consider preexisting pain or pain that is unrelated to a traumatic event but
is worsened by the experience of trauma and/or by the presence of PTSD symptoms.

Sharp and Harvey (2001) proposed a mutual maintenance model, in which PTSD
is maintained or exacerbated by symptoms of chronic pain, and conversely, chronic pain
is maintained or exacerbated by symptoms of PTSD. Sharp and Harvey outlined seven
underlying factors that maintain the PTSD-chronic pain relationship. The first factor is
attentional biases, where individuals may pay more attention to threatening or painful
stimuli, thus leading to amplified emotional and pain experiences. Second, anxiety
sensitivity (i.e., fear of anxiety-related sensations) is thought to exacerbate the
physiological sensations associated with arousal symptoms of PTSD and pain. Third,
pain sensations may serve as persistent reminders of the trauma, leading to avoidance of
both trauma memories and pain cues. Fourth, avoidant coping style may help to
temporarily relieve trauma-related pain or anxiety, but prolonged avoidance of trauma
cues and pain is thought to maintain arousal symptoms and physical disability. Fifth,
individuals with PTSD and chronic pain may also experience depressive symptoms—
such as lethargy and decreased activity—which may lead to increased trauma-specific
and general avoidance, thus maintaining PTSD and pain-related disability. Sixth, general
anxiety may exacerbate arousal symptoms and pain perception, therefore increasing
disability and distress. Lastly, Sharp & Harvey suggest that individuals with PTSD and
chronic pain have significant cognitive demands, therefore a person’s capacity to cope
with PTSD and pain symptoms by engaging in adaptive strategies is limited. The shared vulnerability and mutual maintenance models present nonexclusive views regarding the etiology and perpetuation of co-occurring PTSD and chronic pain. Yet, these models do not speak to a biological basis for comorbid PTSD and pain experiences.

More recently, researchers have started focusing on the role of central sensitization in these co-occurring disorders. Central sensitization is characterized by increased excitability of nociceptive circuits (i.e., the pathways by which noxious stimuli are transmitted from the sensory receptors through the spinal cord and into the brain for processing), such that one’s perception of noxious pain stimuli is exaggerated (i.e., hyperalgesia) and normally innocuous stimuli, like light touch, can activate the pain pathway (i.e., allodynia; Woolf, 2011). Central sensitivity can be measured by evaluating a person’s response to experimentally induced activation of nociceptors via exposure to noxious thermal, mechanical, or chemical stimuli. Central sensitization has been demonstrated in several chronic pain conditions (Woolf, 2011), many of which are comorbid with PTSD. Moeller-Bertram and colleagues (2014) proposed that central sensitization may also be relevant for individuals with PTSD since symptoms of hyperarousal signify an exaggerated response to incoming somatosensory stimuli. Preliminary evidence from a study comparing responses to pain induction procedures in chronic pain-free participants with and without PTSD suggests that individuals with PTSD demonstrate heightened central sensitization compared to control participants (Moeller-Bertram et al., 2014). Central sensitization may explain how pain experiences can be exacerbated by PTSD symptoms, even if the pain is not directly related to the traumatic event. For example, hyperarousal symptoms have been shown to mediate the
transition from acute to chronic pain (Liedl et al., 2010), indicating that central sensitization may be relevant in chronic pain etiology for those with PTSD.

Taken together, these theories support the idea that both psychological and physiological components of PTSD and chronic pain may contribute to the development, maintenance, and exacerbation of symptoms of these comorbid disorders. Although these models suggest that negative affective experiences (including both general and trauma-specific negative affect) likely play a role in the PTSD-chronic pain relationship, researchers have yet to establish how trauma-related negative affect may specifically influence pain experiences in individuals with PTSD.

**Pain sensitivity.** Pain is a multifaceted and complex experience and therefore researchers have developed ways to experimentally evaluate perceptions of pain in both clinical and non-clinical populations. Experimental models of pain have been used to evaluate an individual’s sensitivity to painful stimuli. Pain sensitivity is an overarching term that encompasses both sensory and affective domains of pain perception (Fernandez & Turk, 1992). The sensory domain of pain sensitivity includes pain threshold (i.e., lowest stimulus intensity of pain one is able to detect), pain tolerance (i.e., maximum intensity of a stimuli one is able to endure), and the time it takes to recover from exposure to the pain stimuli; the affective domain of pain sensitivity is often measured by evaluating an individual’s pain intensity and unpleasantness ratings.

Evidence from chronic pain populations offers support for the use of pain sensitivity as an experimental model for understanding chronic pain experiences. For example, patients with fibromyalgia (see Gracely, Grant, & Giesecke, 2003 for a review), low back pain (Giesecke et al., 2004), temporomandibular disorders (Maixner, Fillingim,
Booker, & Sigurdsson, 1995; Maixner, Fillingim, Sigurdsson, Kincaid, & Silva, 1998), and irritable bowel syndrome (Stabell, Stubhaug, Flægstad, & Nielsen, 2013) demonstrate increased sensory and affective pain sensitivity. Also, individuals who display increased pain sensitivity before undergoing surgery experience more severe postoperative pain and are more likely to have their postoperative pain become chronic (Granot, 2009). Similarly, a prospective cohort study revealed that healthy volunteers who demonstrated increased pain sensitivity at baseline were more likely to develop temporomandibular disorders during a three-year period (Slade et al., 2007). Results from these studies indicate that increases in pain sensitivity may relate to both the development and maintenance of chronic pain conditions.

Studies that have evaluated the relationship between PTSD and pain sensitivity provide inconsistent results. Some studies show increased pain sensitivity (i.e., hyperalgesia) in people with PTSD, other studies show decreased sensitivity (i.e., hypoalgesia), and a few show no relationship between PTSD and pain sensitivity (see Moeller-Bertram et al., 2012 for a review). Several reasons may explain why previous research on pain sensitivity among individuals with PTSD resulted in inconsistent findings. First, some studies use pain induction procedures that measure superficial heat pain rather than deep and prolonged pain, which may be more relevant in understanding pain experiences among trauma-exposed individuals (Sessle, 1990; Chapman et al., 1985). Second, findings from studies that evaluate pain experiences in both males and females may be confounded, as women typically demonstrate higher pain sensitivity than men (Klatzkin, Mechlin, & Girdler, 2010; Riley, Robinson, Wise, Myers, & Fillingim, 1998). Third, studies that only include combat-exposed military veterans may not be
generalizable to individuals who have experienced other types of trauma related to alterations in pain experiences, such as child sexual abuse (Raphael & Widom, 2011).

Although methodological limitations may partially contribute to the inconsistent findings demonstrated in prior research, it is also possible that individuals with PTSD may exhibit contradictory pain responses. For example, Defrin and colleagues (2008) found that although individuals with PTSD reported higher pain thresholds, they also reported greater pain intensity once pain was detected compared to participants with anxiety and control subjects. Recent evidence further suggests that experiences of trauma-related negative affect may mediate pain sensitivity as measured in the laboratory (Creech, Smith, Grimes, & Meagher, 2011). Creech and colleagues (2011) found that recalling details of a traumatic event may lead to acute decreases in pain threshold (i.e., hyperalgesia) and concurrent increases in pain tolerance (i.e., hypoalgesia). More specifically, the authors found that among trauma-exposed women, writing about details of their worst traumatic event for 20 minutes resulted in reduced heat pain thresholds compared to writing about a neutral event. This reduction was mediated by increased emotional arousal and unpleasantness resulting from the traumatic recall. Compared to those without a history of trauma, trauma-exposed women evidenced lower baseline ischemic pain tolerance (i.e., pain resulting from restriction of blood flow). However, this effect was reversed in the trauma/stressful event writing condition, such that following 20 minutes of writing about their most distressing traumatic event (or stressful life event in the case of the non-trauma-exposed group) trauma-exposed women demonstrated higher ischemic pain tolerance compared to non-trauma-exposed women. Although results of this study were interpreted as evidence that trauma history may influence baseline pain
sensitivity and trauma-related negative affect may result in acute changes in both pain threshold and tolerance, this study did not include a measure of PTSD symptoms and so it is unclear as to how these results may have been influenced by PTSD symptomatology.

Empirical literature has yet to evaluate the combined effect of PTSD symptoms and trauma-related negative affect on pain sensitivity. This is a critical gap in the literature, as PTSD-related alterations in pain sensitivity may be highly influenced by emotional distress associated with frequent traumatic re-experiencing seen among individuals with PTSD. Discrepancies in prior studies of experimental pain sensitivity among individuals with PTSD may be partially accounted for by differences in trauma-related negative affective experiences. As such, the current study aims to evaluate the effect of PTSD symptoms on pain sensitivity in trauma-exposed women. This study will focus on women because PTSD (Kessler et al., 2005) and chronic pain syndromes (Bartley & Fillingim, 2016) disproportionately impact women.

In terms of sensory pain experiences, it was hypothesized that 1a) women with PTSD would demonstrate decreased pain tolerance compared to trauma-exposed women without PTSD (Creech et al., 2011), and 1b) women who wrote about their worst traumatic experience would display lowered pain thresholds compared to women who wrote about an emotionally neutral experience (Creech et al., 2011). Given the mixed findings from past research, exploratory analyses were used to assess the main effects of PTSD on pain threshold and of writing condition on pain tolerance, as well as the interaction of PTSD by writing condition on pain threshold and tolerance. Exploratory analyses were also used to evaluate the effects of PTSD, writing condition, and PTSD by
writing condition on time to recover from the pain stimulus, as support from prior studies regarding these relationships is limited.

In regards to affective pain reports, it was hypothesized that 2a) women with PTSD would demonstrate greater pain intensity and unpleasantness ratings compared to women without PTSD (Gómez-Pérez & López-Martínez, 2013), and 2b) women who wrote about their worst traumatic experience would report greater pain intensity and unpleasantness compared to women who wrote about an emotionally neutral experience (Creech et al., 2011; You et al., 2014). Finally, a PTSD by writing condition interaction was hypothesized (2c), such that women with PTSD who wrote about their worst traumatic experiences would exhibit decreased pain intensity and unpleasantness compared to women without PTSD who wrote about a traumatic experience, as well as women with and without PTSD who wrote about a neutral experience (Mickleborough et al., 2011).
Chapter 2: Method

Participants

Participants in this study included 106 undergraduate women recruited from the University of Kentucky, 87 of whom were included in the final analyses ($M_{age} = 18.82$, $SD = 0.84$; range 18-21 years old). Participants were included in the study if they 1) were between the ages of 18-25 years old, 2) were enrolled as an undergraduate student, 3) reported exposure to at least one stressful or traumatic experience in their lifetime, and 4) scored $< 10$ or $\geq 37$ on the PTSD Checklist for DSM-5 (PCL-5; Weathers et al., 2013). Women who scored $\geq 37$ on the PTSD Checklist for DSM-5 were classified as having probable PTSD (PTSD+), per recommended clinical cut scores on the PCL-5 (Blevins, Weathers, Davis, Witte, & Domino, 2015). Participants with scores on the PCL-5 below 10 were recruited as trauma-exposed controls (PTSD-) based on prior research conducted in our lab that found trauma-exposed women with scores of less than 10 on the PCL-5 were in the lowest quartile. Women who reported contraindications for the pain testing procedure (e.g., a history of cardiovascular disease, fainting, seizures, frostbite, neurological disorders, and Raynaud’s disease), as well as current opioid medication use, were excluded from participation. Reasons for exclusion based on data analytic purposes are further described in the results section. Participants included in the final sample were predominantly White ($n = 74; 85.1\%$), non-Hispanic ($n = 85; 97.7\%$), and had completed their freshman year of college ($n = 63; 72.4\%$) at the time of the study.

Apparatus and Measures

Trauma experiences and PTSD symptoms. Traumatic life experiences were assessed using a modified version of the Trauma History Questionnaire (THQ; Hooper,
Stockton, Krupnick, & Green, 2011). The THQ is a 24-item self-report measure that assesses experiences of potentially traumatic events like natural and manmade disasters, accidents, crime experiences, and physical or sexual assault. For each traumatic experience endorsed, follow up questions assessed the number of times the event occurred, the age at which the event first occurred, and the age at which the most recent event occurred. If a participant indicated experiences of past interpersonal violence, the nature of the relationship with the perpetrator (e.g., spouse or intimate partner, date, friend, acquaintance, etc.) was assessed. Among college students, the THQ demonstrates fair to excellent test-retest reliability over a 2-3 month period, as well as strong construct and cultural validity (Hooper et al., 2011).

Past-month PTSD symptom severity in response to the index (i.e., worst or most distressing) traumatic event identified on the modified THQ was assessed using the PTSD Checklist for DSM-5 (PCL-5; Weathers et al., 2013). Respondents indicated how much they were bothered by 20 symptoms of posttraumatic stress on a five-point Likert-type scale (0 = not at all to 4 = extremely). The total symptom severity score ranges from 0-80. Previous research indicates a cut score of ≥ 37 on the PCL-5 achieves optimal sensitivity (.66), specificity (.97), and efficiency (.93) for predicting probable PTSD in a college sample (Blevins et al., 2015). Therefore, participants with a score at or above 37 were classified as positive for probable PTSD (PTSD+). Women with scores on the PCL-5 of <10 were recruited as trauma-exposed controls (PTSD-). The PCL-5 displayed excellent internal consistency in the present sample (α = .97). This measure also shows evidence of test-retest reliability over one week, as well as convergent and discriminant validity (Blevins et al., 2015).
**Negative affect.** Trait and state negative affect was assessed using a modified version of the Positive and Negative Affect Schedule – Expanded Form (PANAS-X; Watson & Clark, 1999). The 25-item questionnaire was administered at three time points and included four emotion subscales (e.g. fear, sadness, guilt, and hostility). First, trait negative affect was assessed; women were asked to rate the extent to which they generally experience different negative emotions on five-point Likert-type scale ($1 = \text{very slightly or not at all}$ to $5 = \text{extremely}$). Second, the PANAS-X was administered immediately prior to and following the disclosure task to assess changes in state (current) negative affect. Total scores range from 25-125 and higher scores represent higher levels of negative affect. The PANAS-X demonstrates excellent internal consistency in the current study ($\alpha = .93 - .96$), as well as strong convergent and discriminant validity (Watson et al., 1999).

**Chronic pain.** The Chronic Pain Grade Questionnaire (CPGQ; Von Korff, Ormel, Keefe, & Dworkin, 1992) was used to assess two facets of past six-month chronic pain severity: pain intensity and pain-related disability. Subscale scores for pain intensity and disability were combined to calculate a chronic pain grade that enables classification of chronic pain patients into five hierarchical categories (Grade 0 = *no pain* to Grade IV = *high disability-severely limiting pain*). The CPGQ demonstrates good to excellent internal consistency ($\alpha = .85 - .94$ for the pain intensity and disability subscales in the current sample), as well as convergent and construct validity, indicating that it is a reliable and valid measure for evaluating recent chronic pain experiences (Smith et al., 1997).
**Health screening questionnaire.** Several questions were developed to address possible covariates that have been related to alterations in acute pain experiences (Fishbain, Cutler, Rosomoff, & Rosomoff, 2000; Koltyn, 2000; Kowalczyk, Sullivan, Evans, Bisaga, Vosburg, & Comer, 2010; Moore, Keogh, & Eccleston, 2009; Okifuji, & Hare, 2011). Specifically, participants were asked if they were using hormonal birth control, oral or topical steroid medications, or psychotropic medications at the time of the lab visit (Yes/No). Women reported the start date of their last menstrual period, how many minutes they engaged in aerobic and anaerobic exercise in the 24 hours prior to the lab visit, as well as the average number of hours they slept each night in the preceding week. Women also indicated if they consumed caffeine, nicotine, or alcohol in the two hours leading up to the laboratory visit and if they used non-opioid pain medications in the 24 hours prior to the laboratory session.

**Pain induction procedure.** The Cold Pressor Task (CPT; Birnie, Noel, Chambers, von Baeyer, & Fernandez, 2010), a safe and widely used lab-based pain induction task, was employed to measure pain sensitivity (von Baeyer, Piira, Chambers, Trapanotto, & Zeltzer, 2005). Participants first placed their non-dominant hand up to their forearm in a room temperature bath (24 ± 0.5°C) for 120 seconds and then immediately placed the same hand in an ice bath (1.0 ± 0.5°C). Participants were directed to keep their hand submerged in the ice bath for as long as possible but were able to remove their hand at any time if the pain became intolerable. To prevent tissue damage, participants were instructed to remove their hand from the ice bath after 300 seconds. A pump was used to circulate the water in the ice bath to prevent heat build-up around the submerged hand.
Several outcomes related to pain sensitivity were assessed during and after the CPT, including both sensory and affective measures of pain. Sensory measures of pain include pain threshold (i.e., the number of seconds until the participant started to feel pain after the hand was submerged in the ice bath), pain tolerance (i.e., the number of seconds the participant was able to keep their hand submerged in the ice bath after detecting pain), and time to recover (i.e., the number of seconds it took for a participant to stop feeling pain after their hand was removed from the ice bath). Participants also reported affective reports of pain by rating the intensity of their pain and unpleasantness on a scale from 0 (no pain/unpleasantness) to 10 (extreme pain/unpleasantness) at 20 second intervals. Both intensity and unpleasantness were assessed first while the hand was in the ice bath (i.e., during the pain stimulus) and then for 300 seconds after the hand was removed from the ice bath (i.e., during recovery from the pain stimulus).

**Procedure**

Participants were recruited through flyers placed in the campus community, as well as through an online advertisement on the University of Kentucky Psychology subject pool webpage. Those who responded to the advertisement completed a battery of online screening questionnaires, including the THQ, PCL-5, CPGQ, and the general version of the PANAS-X before coming in for a laboratory session. Participants provided written, informed consent prior to completing any laboratory-based procedures. Immediately after obtaining consent, participants completed the health screening questionnaire and the state version of the PANAS-X to provide a rating of their current negative affect at baseline.
Pennebaker’s well-established emotional disclosure paradigm (Pennebaker & Susman, 1988) was used to induce trauma-related negative or neutral affect among participants. Participants were taken into a private laboratory space and were provided with a sealed envelope containing instructions regarding their writing topic, thus ensuring the research personnel were blinded to the participant’s writing condition. Stratified random assignment with permuted blocks was used to assign the PTSD+ and PTSD- participants to write about an emotionally neutral experience (i.e., their day yesterday; “neutral condition”) or the index trauma reported on the THQ (i.e., “trauma condition”). All participants were asked to write for 20 minutes. Participants’ negative affective state was again evaluated using the PANAS-X after the writing task to assess whether trauma-related negative affect was induced in the trauma writing condition relative to the neutral condition.

Following the writing task, participants completed the CPT. The experimenter provided debriefing materials and either assigned course credit or provided $30 in monetary compensation to the participant at the end of the laboratory visit.

**Data Analytic Approach**

**Descriptive statistics.** Independent samples t-tests, chi-squared tests of independence, and zero-order correlations were used to identify potential covariates. Pooled within cell correlations were used to assess the relations among pain outcome variables.

**Manipulation check.** Writing responses were evaluated to ensure that participants wrote about the topic (trauma vs. neutral) they were assigned during randomization. Further evaluation of the writing responses in the trauma condition
ensured that women in this group wrote about the index trauma they reported on the THQ.

A 2-way analysis of variance (ANOVA) was used to assess whether the trauma writing condition led to increased state negative affect relative to the neutral condition. A negative affect gain score (i.e., post-disclosure negative affect - pre-disclosure negative affect) was included as the dependent variable (Fitzmaurice, Laird, & Ware, 2004). The \( F \)-test of significance was used to assess the main effects of writing condition (trauma vs. neutral) and PTSD group (PTSD+ vs. PTSD-), as well as the interaction of writing condition by PTSD group on the PANAS-X gain score. The assumptions of normality, linearity, homogeneity of variance were tested.

**Pain sensitivity.** Factorial multivariate analysis of covariance (MANCOVA) models were performed to test the primary hypotheses. Unadjusted models were run first, followed by models that included covariates that were significantly related to the dependent variables (Miller & Chapman, 2001). To test the specificity of PTSD symptoms on sensory and affective pain outcomes, additional models were run that included covariates that differed by PTSD group (i.e., time since index trauma, trait negative affect, chronic pain grade; Zinbarg, Suzuki, Uliaszek, & Lewis, 2010). In all tests, PTSD group (PTSD+ vs. PTSD-), writing condition (trauma vs. neutral), and the interaction of writing condition by PTSD group were included as independent variables. Sensory (threshold, tolerance, and time to recover) and affective (intensity and unpleasantness during and following the pain stimulus) pain reports were entered as dependent variables in separate MANCOVA models. Following a significant global effect, post-hoc univariate comparisons were performed by using 2-way analyses of
covariance (ANCOVA) tests to examine the influence of writing condition, PTSD group, and their interaction on each dependent variable. The assumptions of factorial MANCOVA (e.g., normality, linearity, and homogeneity of variance-covariance) were assessed.

**Power.** An a priori power analysis was conducted using G*Power 3.1 (Faul, Erdfelder, Buchner, & Lang, 2009). Limited research is available on the interaction between PTSD symptoms and writing condition on sensory and affective pain sensitivity and therefore Cohen’s (1988) guidelines for medium to large effect sizes were used as estimates to determine power. For the multivariate interactive effect of PTSD group by writing condition on sensory pain outcomes (e.g., threshold, tolerance, and time to recover), a sample size of 92 participants was needed to detect a medium effect and 44 was needed to detect a large effect ($\alpha = .05$) at power of .80. For the multivariate interactive effect of PTSD group by writing condition on affective pain reports (e.g., intensity and unpleasantness), a sample size of 120 participants was needed to detect a medium effect and 52 was needed to detect a large effect ($\alpha = .05$) at power of .80. Thus, the size of the final samples included in the models testing the multivariate interactive effect on sensory pain ($N = 87$) and affective pain during recovery from the stimulus ($N = 84$), but not affective pain during the pain stimulus ($N = 53$), were adequately powered for detecting a large effect but underpowered for detecting a medium effect. Past research suggests a medium to large main effect of PTSD on sensory pain sensitivity (Moeller-Bertram et al., 2014). Thus, a sample size of 92 participants would be required for 80% power to detect a medium effect and 44 participants would detect a large effect ($\alpha = .05$). For affective pain sensitivity, past research also suggests a medium to large main effect
of writing condition. A sample size of 120 participants would be required for 80% power to detect a medium effect and 52 participants would detect a large effect ($\alpha = .05$).
<table>
<thead>
<tr>
<th>Trauma Type</th>
<th>Total N = 87</th>
<th>PTSD- n = 43</th>
<th>PTSD+ n = 44</th>
</tr>
</thead>
<tbody>
<tr>
<td>Actual or attempted break-in or robbery</td>
<td>2 (2.3%)</td>
<td>2 (4.7%)</td>
<td>0 (0.0%)</td>
</tr>
<tr>
<td>Natural or man-made disasters</td>
<td>3 (3.4%)</td>
<td>3 (7.0%)</td>
<td>0 (0.0%)</td>
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<td>Physical assault</td>
<td>5 (5.7%)</td>
<td>2 (4.7%)</td>
<td>3 (6.8%)</td>
</tr>
<tr>
<td>Seeing or handling dead bodies</td>
<td>2 (2.3%)</td>
<td>2 (4.7%)</td>
<td>0 (0.0%)</td>
</tr>
<tr>
<td>Seeing someone seriously injured or killed</td>
<td>1 (1.1%)</td>
<td>0 (0.0%)</td>
<td>1 (2.3%)</td>
</tr>
<tr>
<td>Serious accident</td>
<td>9 (10.3%)</td>
<td>5 (11.6%)</td>
<td>4 (9.1%)</td>
</tr>
<tr>
<td>Serious illness, injury, or fear of death (self)</td>
<td>3 (3.4%)</td>
<td>1 (2.3%)</td>
<td>2 (4.5%)</td>
</tr>
<tr>
<td>Serious illness, injury, or unexpected death (others)</td>
<td>26 (29.9%)</td>
<td>16 (37.2%)</td>
<td>10 (22.7%)</td>
</tr>
<tr>
<td>Unwanted sexual experience</td>
<td>24 (27.6%)</td>
<td>5 (11.6%)</td>
<td>19 (43.2%)</td>
</tr>
<tr>
<td>Other or not listed</td>
<td>12 (13.8%)</td>
<td>7 (16.3%)</td>
<td>5 (11.4%)</td>
</tr>
</tbody>
</table>
Chapter 3: Results

Of the 106 participants enrolled in the study, six women were ineligible due to CPT contraindications (e.g., history of fainting or cardiovascular/neurological conditions), six women were excluded from analyses due to missing pain outcome data (i.e., pain threshold, tolerance, or time to recover was not recorded), two women did not complete the CPT as instructed, and one woman was given the incorrect writing topic. Prior to testing hypotheses, the assumptions of multivariate analyses (normality, linearity, homogeneity of variance-covariance) were tested and met. Three cases had multivariate outliers with a Mahalanobis distance of greater than 16.27 and were, therefore, excluded from analyses. Little’s MCAR test was used to assess patterns in missing questionnaire data (Little, 1988). For data missing at random or completely at random, expectation maximization was used to impute missing data points (Tabachnick & Fidell, 2013). Listwise deletion was used for missing data on single-item measures ($n = 1$), leaving a sample of 87 women for the primary analyses.

Descriptive Statistics

Ethnic/racial identity did not differ by PTSD diagnostic status (white vs. non-white; $\chi^2 = 0.12, p = .73$). The prevalence of participants’ index (i.e., worst or most distressing) lifetime traumas are depicted in Table 1, with the most commonly reported traumatic experiences being serious illness, injury, or unexpected death of a loved one (29.9%), unwanted sexual experiences (27.6%), and “other” or not listed (13.8%).

Independent samples $t$-tests, zero-order correlations, and chi-squared tests of independence were used to evaluate relations between possible covariates (i.e., number of days since the start of the last menstrual cycle; hormonal birth control, oral/topical
steroid, and psychotropic medication use; minutes of aerobic/anaerobic exercise and non-opioid medication use in prior 24 hours; average number of hours slept each night in the prior week; caffeine, nicotine, and alcohol consumption in the preceding two hours) and primary outcome variables (i.e., tolerance, threshold, time to recover, intensity and unpleasantness), as well as classification variables (e.g., PTSD group; writing condition). Participants on hormonal birth control demonstrated significantly longer time to recover from the pain stimulus compared to women not taking hormonal birth control (No: \( M = 94.54, SD = 59.61 \) vs. Yes: \( M = 128.89, SD = 75.62 \); \( t = -2.22, p = .03 \)). Participants also demonstrated a longer time to recover if they consumed caffeine in the two hours leading up to the lab visit (No: \( M = 109.55, SD = 66.52 \) vs. Yes: \( M = 170.56, SD = 93.70 \); \( t = -2.49, p = .02 \)) and, therefore, these covariates were included in the MANCOVA evaluating sensory measures of pain. Minutes of anaerobic exercise in the 24 hours prior to the study session was negatively correlated with average intensity ratings during the pain stimulus (\( r = -.33, p = .02 \)) and average pain unpleasantness during the recovery period from the pain stimulus (\( r = -.22, p = .04 \)), and were included in the MANCOVAs assessing affective reports of pain. None of the other variables evaluated as potential covariates were significantly related to sensory or affective pain outcomes. Rates of hormonal birth control use, as well as the number of days since the start of participants’ last menstrual cycle, did not differ by PTSD group or writing condition. Similarly, caffeine intake and minutes of anaerobic exercise prior to the lab visit did not differ by PTSD group or writing condition.

Participants in the PTSD+ group reported more recent index traumas (PTSD-: \( M = 5.86 \) years, \( SD = 5.08 \) vs. PTSD+: \( M = 3.48 \) years, \( SD = 3.82 \); \( t = 2.47, p = .02 \)), higher
chronic pain grades (PTSD-: $M = 0.79$, $SD = 0.51$ vs. PTSD+: $M = 1.52$, $SD = 0.95$; $t = -4.48$, $p < .001$), and greater trait negative affect (PTSD-: $M = 38.26$, $SD = 16.20$ vs. PTSD+: $M = 69.34$, $SD = 22.58$; $t = -7.39$, $p < .001$) compared to the participants in the PTSD- group. Participants assigned to write about their index trauma also reported higher trait negative affect compared to participants assigned to write about the neutral topic (neutral: $M = 48.70$, $SD = 21.11$ vs. trauma: $M = 59.90$, $SD = 27.93$; $t = -2.13$, $p = .04$), indicating a failure of randomization. Moreover, time since index trauma was inversely associated with pain intensity during the pain stimulus ($r = -.31$, $p = .02$), indicating more recent exposure to a traumatic event was related to higher pain intensity ratings. Chronic pain grade scores were inversely associated with intensity and unpleasantness ratings during recovery from the pain stimulus ($r = -.25$, $p = .02$; $r = -.24$, $p = .03$, respectively); more intense and disabling past six-month chronic pain experiences were associated with lower intensity and unpleasantness ratings after the pain stimulus was removed. Trait levels of negative affect were not related to sensory or affective pain outcomes ($rs = -.02$ to -.15, $ps > .18$). Thus, years since index trauma, chronic pain grade, and trait negative affect were included as additional covariates in secondary MANCOVA models testing sensory and affective measures of pain (Miller & Chapman, 2001; Zinbarg et al., 2010).

Pooled within cell correlations ranged from .24 to .62 for pain threshold, tolerance, and time to recover ($ps < .03$). Positive associations were demonstrated between intensity and unpleasantness ratings both during the pain stimulus ($r = .64$, $p < .001$) and during recovery from the pain stimulus ($r = .70$, $p < .001$).
Manipulation Check

A factorial ANOVA was used to assess differences in baseline state negative affect by PTSD group and writing condition. As expected, a significant main effect of PTSD group emerged, $F(1,83) = 34.16, p < .001, \eta^2 = .29$. Participants in the PTSD+ group reported greater state negative affect at baseline compared to participants in the PTSD- group (PTSD-: $M = 28.40, SD = 3.87$ vs. PTSD+: $M = 40.50, SD = 13.51$).

However, the main effect of writing condition, $F(1,83) = 3.64, p = .06, \eta^2 = .04$, and the interaction of PTSD group by writing condition, $F(1,83) = 2.40, p = .13, \eta^2 = .03$, were non-significant, indicating although participants in the trauma writing condition were higher in trait negative affect, the randomization procedure was sufficient with regards to group equivalence in baseline state negative affect across writing conditions.

Results of the factorial ANOVA assessing change in negative affect following the writing task revealed a significant main effect of writing condition, $F(1,83) = 43.61, p < .001, \eta^2 = .34$, such that participants who wrote about their index trauma demonstrated a significantly greater increase in negative affect compared to participants who wrote about the neutral topic (neutral topic: $M_{\Delta} = -0.83, SD = 4.77$ vs. trauma topic: $M_{\Delta} = 13.67, SD = 14.01$). Neither the main effect of PTSD group, $F(1,83) = 1.83, p = .18, \eta^2 = .02$, nor the interaction of PTSD group by writing condition, $F(1,83) = 0.91, p = .34, \eta^2 = .01$, were related to change in negative affect following the writing task.

Primary Hypotheses

Sensory Pain. Results of the MANCOVA models testing sensory pain measures (e.g., threshold, tolerance, and time to recover) are displayed in Table 2. The unadjusted results are presented in Models 1a and 1b; the main and interactive effects of PTSD
group and writing condition were not related to sensory pain outcomes. The multivariate effects of PTSD group, writing condition, and the interaction of PTSD group and writing condition were not significantly related to evaluations of sensory pain in the unadjusted models (Models 1a and 1b) or when including hormonal birth control status and caffeine consumption prior to the CPT as covariates (Models 2a and 2b).

A third model was conducted including time since index trauma, trait negative affect, and chronic pain grade as additional covariates (see Table 2, Models 3a and 3b). A significant multivariate main effect emerged for PTSD group but the multivariate effects for writing condition and the PTSD by writing condition interaction remained non-significant. As displayed in Table 3, post hoc ANCOVAs revealed a significant univariate effect of PTSD group on pain threshold and pain tolerance, but not time to recover. Compared to participants in the PTSD- group, those in the PTSD+ symptom group demonstrated a higher pain threshold (i.e., longer time to detect pain; PTSD-: estimated marginal $M = 5.69, SE = 0.87$; PTSD+: estimated marginal $M = 9.09, SE = 0.86$) and greater ability to tolerate pain stimuli (PTSD-: estimated marginal $M = 23.86, SE = 10.57$; PTSD+: estimated marginal $M = 65.56, SE = 10.50$).

**Affective Pain.** The results of the MANCOVA models testing affective pain measures (e.g., intensity and unpleasantness) during and immediately following the pain stimulus (during recovery) are displayed in Tables 4 and 5, respectively. The unadjusted main and interactive effects of PTSD group and writing condition were not related to affective reports of pain during (Table 4, Models 1a and 1b) or after removal of (Table 5, Models 1a and 1b) the pain stimulus. When covarying for anaerobic exercise, the multivariate effect of affective pain reports (e.g., intensity and unpleasantness) during the
pain stimulus (Table 4, Models 2a and 2b) and during the recovery period (Table 5, Models 2a and 2b) did not differ by PTSD group, writing condition, or the interaction of PTSD group by writing condition.

Time since trauma, trait negative affect, and chronic pain grade were entered as additional covariates in the MANCOVA models in order to evaluate whether PTSD group, writing condition, and their interaction significantly predicted affective pain reports above and beyond these factors. Affective reports of pain during the pain stimulus did not differ by PTSD group, writing condition, or the interaction of PTSD group by writing condition (Table 4, Models 3a and 3b). Although affective pain reports during recovery also did not differ by writing condition, there was a significant main effect of PTSD group, as well as a significant PTSD group by writing condition interaction (Table 5, Models 3a and 3b). Post hoc ANCOVAs revealed a significant univariate PTSD group by writing condition interaction for pain unpleasantness but not intensity (Table 6). As displayed in Figure 1, women in the PTSD+ group who wrote about their trauma reported significantly higher pain unpleasantness ratings during the recovery period compared to 1) women in the PTSD+ group who wrote about the neutral topic and 2) women in the PTSD- group in either writing condition. There was also a significant univariate main effect of PTSD group on pain intensity in addition to pain unpleasantness (Table 6). Compared to participants in the PTSD- group, those in the PTSD+ group reported higher pain intensity during the recovery period from the pain stimulus (PTSD-: estimated marginal $M = 3.34, SE = 0.26$ vs. PTSD+: estimated marginal $M = 4.36, SE = 0.27$).
Table 2
Multivariate Analysis of Covariance of Sensory Pain Measures

<table>
<thead>
<tr>
<th>Source of Variance</th>
<th>Model 1a</th>
<th>Model 2a</th>
<th>Model 3a</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Wilk’s Λ</td>
<td>df1,2</td>
<td>F</td>
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<tr>
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<td>3,82</td>
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<td>Writing condition</td>
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<td>Birth control status</td>
<td>.91</td>
<td>3,80</td>
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<tr>
<td>Caffeine intake</td>
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<td>3,80</td>
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<td>Chronic pain grade</td>
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<td>Trait negative affect</td>
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<tr>
<td>Years since trauma</td>
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<td>PTSD*writing</td>
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</tr>
<tr>
<td>Years since trauma</td>
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</table>
Note: Models 1a, 2a, and 3a reflect the main effects of PTSD group and writing condition on sensory pain outcomes. Models 1b, 2b, and 3b include both the main and interactive effects. Models 1a and 1b reflect unadjusted models. Models 2a and 2b include covariates that relate to the sensory pain outcomes. Models 3a and 3b include both covariates that relate to sensory pain outcomes and covariates that differ by classification. PTSD = posttraumatic stress disorder. * p < .05. ** p < .01. *** p < .001.
Table 3
*Post Hoc Univariate Analysis of Covariance Tests of Sensory Pain Measures*

<table>
<thead>
<tr>
<th>Source of Variance</th>
<th>Model 1</th>
<th></th>
<th>Partial $\eta^2$</th>
<th>Model 2</th>
<th></th>
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</tr>
</thead>
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<td></td>
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<td>$p$</td>
<td></td>
<td>$F$</td>
<td>$p$</td>
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<td>.02</td>
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<td>0.73</td>
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<td>.01</td>
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*Note:* Models 1 reflects the main effects of PTSD group and writing condition on sensory pain outcomes. Model 2 includes both the main and interactive effects. PTSD = posttraumatic stress disorder.
Table 4
Multivariate Analysis of Covariance of Affective Pain Measures During the Pain Stimulus

<table>
<thead>
<tr>
<th>Source of Variance</th>
<th>Model 1a</th>
<th></th>
<th>Model 2a</th>
<th></th>
<th>Model 3a</th>
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<td>df$_{1,2}$</td>
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<td>Wilk’s $\Lambda$</td>
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<td>0.42</td>
<td>.02</td>
<td>.97</td>
<td>2,48</td>
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<td>0.91</td>
<td>.04</td>
<td>.98</td>
<td>2,48</td>
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<td>4.72*</td>
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<table>
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<th>Model 2b</th>
<th></th>
<th>Model 3b</th>
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<td>F</td>
<td>Partial $\eta^2$</td>
<td>Wilk’s $\Lambda$</td>
<td>df$_{1,2}$</td>
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<td>.98</td>
<td>2,47</td>
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<tr>
<td>Anaerobic exercise</td>
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<td>.12</td>
<td>.83</td>
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</tr>
<tr>
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<td>.97</td>
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</tr>
<tr>
<td>Years since trauma</td>
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<td>2,44</td>
<td>4.62*</td>
<td>.17</td>
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Note: Models 1a, 2a, and 3a reflect the main effects of PTSD group and writing condition on affective pain outcomes. Models 1b, 2b, and 3b include both the main and interactive effects. Models 1a and 1b reflect unadjusted models. Models 2a and 2b include covariates that relate to the affective pain outcomes. Models 3a and 3b include both covariates that relate to affective pain outcomes and covariates that differ by classification. PTSD = posttraumatic stress disorder. * $p < .05$. ** $p < .01$. *** $p < .001$. 
Table 5
Multivariate Analysis of Covariance of Affective Pain Measures During Recovery from the Pain Stimulus

<table>
<thead>
<tr>
<th>Source of Variance</th>
<th>Model 1a</th>
<th>Model 2a</th>
<th>Model 3a</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Wilk’s Λ</td>
<td>df, F, Partial η²</td>
<td>Wilk’s Λ</td>
</tr>
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<td>.02</td>
<td>.96, 2.79, 1.52</td>
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<tr>
<td>Writing condition</td>
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<td>.03</td>
<td>.97, 2.79, 1.34</td>
</tr>
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<td>Anaerobic exercise</td>
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<td>.07</td>
<td>.90, 2.76, 4.21*</td>
</tr>
<tr>
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<td>.18</td>
<td>.99, 2.76, 0.08</td>
</tr>
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<td>.002</td>
<td>.96, 2.76, 1.50</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Source of Variance</th>
<th>Model 1b</th>
<th>Model 2b</th>
<th>Model 3b</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Wilk’s Λ</td>
<td>df, F, Partial η²</td>
<td>Wilk’s Λ</td>
</tr>
<tr>
<td>PTSD group</td>
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<td>.03</td>
<td>.96, 2.78, 1.75</td>
</tr>
<tr>
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<td>.03</td>
<td>.97, 2.78, 1.33</td>
</tr>
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<td>.05</td>
<td>.94, 2.78, 2.41</td>
</tr>
<tr>
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<td>.08</td>
<td>.88, 2.75, 4.94**</td>
</tr>
<tr>
<td>Chronic pain grade</td>
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<td>.20</td>
<td>.99, 2.75, 0.08</td>
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<tr>
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<td>.002</td>
<td>.95, 2.75, 1.82</td>
</tr>
<tr>
<td>Years since trauma</td>
<td>.99, 2.75, 0.08</td>
<td>.002</td>
<td>.95, 2.75, 1.82</td>
</tr>
</tbody>
</table>

Note: Models 1a, 2a, and 3a reflect the main effects of PTSD group and writing condition on affective pain outcomes. Models 1b, 2b, and 3b include both the main and interactive effects. Models 1a and 1b reflect unadjusted models. Models 2a and 2b include covariates that relate to the affective pain outcomes. Models 3a and 3b include both covariates that relate to affective pain outcomes and covariates that differ by classification. PTSD = posttraumatic stress disorder. * p < .05. ** p < .01. *** p < .001.
<table>
<thead>
<tr>
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</thead>
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<tr>
<td></td>
<td>$F$</td>
<td>$p$</td>
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<td><strong>Intensity</strong></td>
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<td>Writing condition</td>
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<td>.41</td>
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<td>Anaerobic exercise</td>
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<td>Chronic pain grade</td>
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<tr>
<td>Trait negative affect</td>
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<td>.73</td>
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<td>Years since trauma</td>
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<td>.09</td>
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<td><strong>Unpleasantness</strong></td>
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<td>PTSD group</td>
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<tr>
<td>Writing condition</td>
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<td>Anaerobic exercise</td>
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<td>Chronic pain grade</td>
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<td>.96</td>
</tr>
<tr>
<td>Years since trauma</td>
<td>1.79</td>
<td>.19</td>
</tr>
</tbody>
</table>

*Note:* Models 1 reflects the main effects of PTSD group and writing condition on affective pain outcomes. Model 2 includes both the main and interactive effects. PTSD = posttraumatic stress disorder.
Figure 1. *Interaction between PTSD Group and Writing Condition Predicting Pain Unpleasantness during Recovery from the Pain Stimulus.* Covariates include Anaerobic Exercise, Past 6-month Pain Intensity/Disability, Trait Negative Affect, and Years Since Index Trauma.

Note: PTSD = posttraumatic stress disorder.

* $p < .05$. ** $p < .01$. *** $p < .001$. 

Chapter 4: Discussion

Although the comorbidity of PTSD and chronic pain is well documented in the literature, this phenomenon is complex and not well understood. Prior experimental research evaluating pain experiences among trauma-exposed individuals with and without PTSD provides inconsistent findings, likely due to methodological differences between studies as well as contradictory sensory and affective pain responses among individuals with PTSD (Moeller-Bertram et al., 2012). Additionally, the role of trauma-related negative affect on pain sensitivity has been largely neglected in previous research with the exception of work by Creech et al. (2011). As such, the current project utilized an emotional disclosure paradigm and experimental pain sensitivity task to assess the effects of trauma-related negative affect on sensory and affective pain sensitivity among trauma-exposed women with and without PTSD.

Elicitation of Trauma-Related Negative Affect

As expected, women who wrote about their worst or most distressing (i.e., index) trauma demonstrated a greater change in state negative affect compared to women who wrote about a neutral topic (Creech et al., 2011; You et al., 2014). However, women in the high and low PTSD symptom groups reported similar increases in negative affect following the writing task. Contrary to our hypothesis, women in the high PTSD symptom group who wrote about their index trauma did not demonstrate a significant change in negative affect relative to 1) women in the low PTSD group who wrote about their index trauma and 2) women in either PTSD group who wrote about the neutral topic. These results may be due, in part, to the failure of randomization to yield equivalent groups based on trait negative affect to emotional disclosure conditions.
Regardless of PTSD symptoms, women assigned to the trauma writing condition reported higher levels of trait negative affect compared to women assigned to the neutral condition, and therefore, may have been more likely to report increases in negative affect after writing about their worst traumatic experience during the emotional disclosure paradigm. Alternatively, these results may reflect ceiling effects, such that writing about a traumatic event in the context of the emotional disclosure paradigm may be sufficient to evoke high levels of negative affect regardless of PTSD status. This task may not be the most sensitive procedure for discriminating higher trauma-related negative affect among women with PTSD compared to women without PTSD. Script-driven imagery is another laboratory paradigm in which participants are assigned to write a description of their index trauma or a neutral topic and are then asked to listen to a brief recording of this description (i.e., “script”; Pitman, Orr, Forgue, de Jong, & Claiborn, 1987). Script-driven imagery has been consistently shown to elicit greater physiologic and emotional arousal in trauma-exposed individuals with PTSD compared to trauma-exposed individuals without PTSD (Orr, Metzger, & Pitman, 2002). Such an approach may be more effective in discriminating between PTSD-specific and general trauma-related negative affect in future studies.

**Sensory and Affective Pain Sensitivity**

Initial unadjusted models and models that only included covariates that were related to sensory pain outcome measures (i.e., use of hormonal birth control, caffeine consumption prior to the lab visit) failed to detect significant effects for PTSD group, writing condition, or the interaction of PTSD group by writing condition. However, when trait negative affect, chronic pain grade, and time since index trauma were added as
covariates, women in the high PTSD symptom group demonstrated a longer time to pain
detection (i.e., increased threshold) and greater ability to the withstand the pain stimuli
(i.e., increased tolerance). The results for pain tolerance contradicted our hypothesis that
women in the high PTSD symptom group would demonstrate decreased ability to
withstand the pain stimuli (Hypothesis 1a), which was formulated based on findings from
work by Creech and colleagues (2011). Although the study by Creech et al. (2011) and
the present project are methodologically similar, a notable difference exists in that we
recruited trauma-exposed women with low and high PTSD symptoms whereas Creech
and colleagues recruited women with and without trauma histories but neglected to assess
for PTSD. Thus, our findings not only expand upon prior work by accounting for the
influence of PTSD symptoms on sensory pain in trauma-exposed women but also suggest
that women with high PTSD symptoms may experience a hypoalgesic response to
sensory pain. Hypoalgesic responses to sensory pain among individuals with PTSD have
been found in prior work, where individuals with PTSD demonstrate higher pain
thresholds compared to those with other anxiety disorders (Defrin et al., 2008) and
trauma-exposed controls (Kraus et al., 2009). Both Defrin et al. (2008) and Kraus et al.
(2009) suggest that the hypoalgesic responses demonstrated among individuals with
PTSD may be reflective of altered neurobiological systems (e.g., increased activity in the
hypothalamic pituitary adrenal axis, changes in the endogenous opioid system). Despite
women in the high PTSD symptom group demonstrating increased pain threshold and
tolerance, participants reported similar recovery times following removal of the pain
stimulus regardless of PTSD status. These findings are consistent with those of Gómez-
Pérez et al. (2013), in which trauma-exposed women with high and low PTSD symptoms demonstrated similar recovery times following the CPT.

Similar to findings for sensory pain outcomes, the unadjusted models and the models that only included anaerobic exercise as a covariate (due to its association with affective outcome measures during the pain stimulus and recovery period) failed to detect significant effects for PTSD group, writing condition, or the PTSD group by writing condition interaction on affective ratings of pain intensity and unpleasantness. When adding trait negative affect, chronic pain grade, and time since index trauma as covariates, results remained non-significant for affective outcomes during the pain stimulus. However, a significant PTSD group by writing condition interaction emerged for pain unpleasantness ratings during the recovery period. This finding was counter to our expectation (Hypothesis 2c; Mickleborough et al., 2011), such that women with high PTSD symptoms who wrote about their index trauma reported increased unpleasantness ratings relative to women with high PTSD symptoms who wrote about a neutral event, as well as women with low PTSD symptoms who wrote about either topic. Our results contradict the argument put forth by Mickleborough and colleagues (2011) that trauma reminders may produce a stress-induced analgesic effect among individuals with PTSD (i.e., decreased intensity/unpleasantness ratings), although methodological differences between studies may account for the difference in findings. For example, Mickleborough and colleagues included both men and women in their study and participants listened to a trauma or neutral script while receiving warm/hot thermal stimuli during an fMRI scan. One possible interpretation of the PTSD group by writing condition interaction on pain unpleasantness ratings during the recovery period is that the combination of writing about
one’s worst trauma and then undergoing a pain induction procedure (i.e., compounding stressors) may have had a carry-over effect on pain unpleasantness into the recovery period, but for only women with high PTSD symptoms. This finding highlights the importance of understanding the interaction between PTSD symptoms and exposure to trauma cues on alterations in pain experiences among trauma-exposed women with PTSD. However, this interaction should be interpreted with caution since the emotional disclosure paradigm did not elicit different levels of trauma-related negative among women with low and high PTSD symptoms. Again, this may have been due to ceiling effects, as well as a lack of sensitivity in the writing task to differentiate PTSD-specific emotional reactivity from trauma-reactivity generally. It is also possible that for women high in PTSD symptoms, writing about their most distressing traumatic event may have led to higher unpleasantness ratings during the recovery via an alternative mechanism not measured in this study. Continued work investigating the influence of trauma-related negative affective states on pain experiences among trauma-exposed women is necessary.

Consistent with findings from Gómez-Pérez et al. (2013), women in the high PTSD symptom group reported increased pain intensity and unpleasantness ratings during the recovery period relative to women in the low PTSD symptom group (Hypothesis 2a). It should be noted that women with high PTSD symptoms reported increased pain intensity and unpleasantness ratings after the pain stimulus was removed, but not during the pain stimulus. Although these findings may be reflective of higher affective pain sensitivity related to PTSD symptomatology (consistent with the findings of Gómez-Pérez et al., 2013), these results could also be a function of the increased pain tolerance displayed by women with high PTSD symptoms who kept their hand in the ice
water longer than women with low PTSD symptoms. Though there were no differences between the high and low PTSD symptom groups in terms of time to recover following removal from the pain stimulus, it is possible that higher intensity and unpleasantness ratings during this period may have been impacted by unique features of this pain induction procedure (i.e., the CPT) that precluded separation of pain tolerance and affective indices. Relatedly, 34 women withdrew their hand from the ice water within the first 20 seconds of the task—before the first affective reports of pain were recorded during the stimulus—meaning that intensity and unpleasantness ratings during the pain stimulus were only available for 53 participants (60.9% of the sample). Given that the length of contact with the painful stimulus (i.e., tolerance) differed by PTSD group, the small effect of PTSD symptoms (Partial \( \eta^2 = .05 \)) on affective reports during the pain stimulus may be significantly biased. These null findings should be interpreted cautiously due both to these methodological limitations as well as low power in these models.

Additionally, the difference in unpleasantness ratings among women with high and low PTSD symptoms should be interpreted within the context of the significant PTSD group by writing condition interaction.

The lack of effect of writing condition (trauma vs. neutral) on sensory and affective pain outcomes was in contrast to our prediction that women who wrote about their index trauma would display lowered pain thresholds (Hypothesis 1b) and increased pain intensity and unpleasantness (Hypothesis 2b) compared to women who wrote about the neutral topic (Creech et al., 2011; You et al., 2014). These hypotheses were informed by prior studies that also used Pennebaker’s emotional disclosure paradigm to induce trauma-related negative affect (Creech et al., 2011; You et al., 2014). As mentioned
previously, our methodology deviated from prior work in that our study only included trauma-exposed women (versus women with and without a history of trauma) in order to isolate the unique impact of PTSD symptoms and trauma-related negative affect on pain outcomes. Because these prior studies failed to assess for or consider the impact of PTSD symptoms, it is possible that the effects of their emotional disclosure task on sensory and affective pain outcomes may have been confounded by the unmeasured PTSD symptoms present in the group of trauma-exposed women.

**Impact of Trait Negative Affect**

By including covariates that differed between trauma-exposed women with low and high PTSD symptoms, we were able to test the specificity of PTSD symptoms above and beyond differences attributable to general trait negative affect, pain intensity/disability, and time since trauma in predicting sensory and affective components of pain sensitivity (Zinbarg et al., 2010). Of the variables that differed as a function of PTSD group, trait negative affect was the only covariate that differed by classification (e.g., PTSD group and writing condition) but did not relate to sensory or affective pain outcomes and, therefore, may be acting as a suppressor variable to mask the association between PTSD and pain sensitivity (MacKinnon, Krull, & Lockwood, 2000; Tzelgov & Henik, 1991). When trait negative affect was included in the multivariate models evaluating pain outcomes (in addition to the other covariates), the size of the main effect of PTSD group increased from small to medium for both sensory and affective pain responses (.03 to .10, .04 to .10, respectively). It is not surprising that women with high PTSD symptoms would report greater trait negative affect compared to women with low PTSD symptoms, especially given that persistent negative emotional states of fear,
horror, anger, guilt, or shame are a symptom of PTSD (APA, 2013). Thus, covarying for trait negative affect allowed for a test of the unique association between PTSD symptoms and alterations in pain sensitivity above and beyond differences attributable to trait negative affect. In line with the shared vulnerability model (Asmundson et al., 2002), it is possible that higher levels of trait negative affect may serve as a psychological vulnerability that can contribute to the development of both PTSD and chronic pain experiences following a traumatic event. However, trait negative affect may also serve to mutually maintain PTSD and chronic pain symptoms after both conditions have developed (Sharp & Harvey, 2001). General levels of negative affect among trauma-exposed individuals may have clouded the results of prior laboratory-based studies focused on evaluating the relationship between PTSD symptoms and pain sensitivity and, thus, should be assessed in future research. Additionally, longitudinal studies are necessary in order to establish the role of preexisting differences in trait negative affect on the etiology and maintenance of PTSD and chronic pain conditions following trauma.

Evidence of Contradictory Sensory and Affective Responses in PTSD?

Our contradictory results for sensory and affective pain responses among individuals with PTSD appear to be in line with those of Defrin and colleagues (2008) who found that compared to participants with anxiety disorders and healthy controls, individuals with PTSD evidenced increased pain thresholds to warm and heat-pain sensations (indicative of a hypoalgesic sensory response), as well as increased intensity ratings during exposure to the pain stimulus (indicative of a hyperalgesic affective response). Since more individuals in the PTSD group reported pre-existing chronic pain conditions compared to the anxiety and healthy control groups in the study by Defrin and
colleagues (2008), the authors concluded that the presence of chronic pain in individuals with PTSD may contribute to a dampened awareness of incoming noxious stimuli due to altered sensory pain processing (i.e., continuous induction of the descending pain inhibition pathway) leading to increased pain detection thresholds. However, once the noxious or painful stimuli was detected, the authors argued that these individuals may have responded with greater negative affect to the presence of pain due to psychological predispositions characteristic of the PTSD/chronic pain comorbidity (e.g., anxiety sensitivity, attentional biases). Notable methodological differences exist between procedures used in the present study and those used by Defrin and colleagues (e.g., all female vs. mixed-sex sample; college-aged women vs. veterans; trauma-exposed women with and without PTSD vs. individuals with PTSD, anxiety, and healthy controls), yet similarities exist in the pattern of findings for sensory and affective pain responses. Given that the statistical models in the present study included both past 6-month chronic pain intensity/disability and trait negative affect as covariates, our findings do not corroborate the explanation that alterations in pain sensitivity are related to activation of the descending pain inhibition pathway and psychological characteristics in women with PTSD and preexisting pain experiences. The alterations in sensory and affective pain sensitivity demonstrated in women with high PTSD symptoms may be mediated by an alternative mechanism that has yet to be identified.

Although speculative, another possibility for the contradictory sensory and affective pain responses demonstrate in women with high PTSD symptoms may be related to the interaction between negative emotional experiences and the degree of physiological arousal (Rhudy and Williams, 2005). Prior research indicates that exposure
to highly threatening stimuli elicits intense negative affect and high arousal, producing a hypoalgesic response to pain (Rhudy et al., 2008). However, stimuli associated with low levels of threat lead to moderately intense negative affect, moderate levels of arousal, and subsequently heightened levels of pain sensitivity. Among individuals with PTSD, previously neutral cues associated with a traumatic event (e.g., sights, smells, sounds) acquire the ability to trigger intense negative emotional responses following a trauma, even in the absence of actual or perceived threat of harm. These intense negative emotional reactions are sustained via avoidance of conditioned trauma-related stimuli, or reminders of the trauma that may trigger re-experiencing of the traumatic event. In the present study, women with high PTSD symptoms may have demonstrated a hypoalgesic sensory pain response, as well as a hyperalgesic affective pain response, because they may be more prone to experiences of intense negative, highly-arousing emotions than women with low PTSD. Measures of physiological and neuroendocrine reactivity during the emotional disclosure paradigm and the pain induction procedure may help to elucidate the role of arousal on alterations in sensory and affective pain sensitivity.

Alternatively, there is some evidence to suggest that sensory and affective components of pain sensitivity may actually be tapping into different constructs that should not necessarily demonstrate concordance (Edwards & Fillingim, 2007). These authors found that among healthy participants, increased self-report ratings of affective pain were associated with higher levels of anxiety but not pain threshold or tolerance. It is unclear whether the findings for sensory and affective pain responses in the present study are representative of a true discrepancy in pain sensitivity among individuals with PTSD.
or if these contradictory results are related to the methodological limitations in measuring experimentally induced pain.

Limitations

These results must be considered within the context of the study’s limitations. First, the PTSD classification was based on scores from a self-report questionnaire and therefore women in the high PTSD group may not have met criteria for a PTSD diagnosis according to the DSM-5 (APA, 2013). Second, the sample was comprised of college-aged women in accordance with prior research (Creech et al., 2011; Gómez-Pérez & López-Martínez, 2013; You et al., 2014); however, results may not be generalizable to non-college aged adults or males. Although both PTSD and chronic pain are more common in women than men (Bartley & Fillingim, 2016; Kessler et al., 2005), these conditions are not unique to women and, therefore, future research is needed to investigate the possible sex differences in the relation between trauma-related symptomology and pain experiences that may account for the discrepant findings in past studies (Moeller-Bertram et al., 2012). Third, the study is limited in that only sensory and affective components of pain sensitivity were assessed. Studies examining the physiological and neuroendocrine responses to pain stimuli following emotional disclosure in conjunction with sensory and affective reports are needed as alterations in physiological/neuroendocrine activity have been demonstrated in both PTSD and chronic pain populations (McBeth et al., 2005; Santa Ana et al., 2006). Fourth, reports of unwanted sexual experiences were more common in the high PTSD symptom group compared to the low PTSD symptom group (11.6% vs 43.2%). This finding is unsurprising, given that sexual victimization is the trauma type most likely to result in the
development of PTSD (Kilpatrick, Badour, & Resnick, 2017). Researchers should consider comparing women with and without PTSD who report exposure to sexual trauma to women with and without PTSD who report other types of traumatic events in order to establish the unique contributions of sexual trauma and PTSD symptoms on alterations in pain sensitivity. Finally, as outlined previously, our null results may be a function of the methodological procedures used to induce trauma-related negative affect, as well as to assess pain sensitivity. Although our study utilized a validated pain induction procedure that generates deep and prolonged pain responses (Chapman et al., 1985; Sessle, 1990), researchers may consider utilizing an experimental pain paradigm that is adjusted for individual participants’ baseline pain threshold and tolerance (e.g., quantitate sensory testing; Rolke et al., 2006). An advantage of using experimental pain models is that these procedures provide the opportunity to evaluate underlying mechanisms that contribute to alterations in pain experiences in highly controlled laboratory settings, which may serve to inform prevention and intervention targets for chronic pain conditions. Preliminary evidence suggests that alterations in pain sensitivity may predict the transition from acute to chronic pain (Granot, 2009; Slade et al., 2007), yet the specific contribution of alterations in pain sensitivity to the etiology of comorbid PTSD and chronic pain conditions remains unclear.

Conclusions

Despite its limitations, this study expands upon prior research investigating the influence of PTSD symptoms on alterations in pain sensitivity. Including a sample of trauma-exposed women with high and low levels of PTSD symptoms allowed for a more thorough evaluation of the direct contribution of PTSD symptoms (rather than just
trauma exposure) on alterations in pain sensitivity, and 2) avoided confounds due sex differences in pain sensitivity. Additionally, this is the first study to investigate the contribution of both trauma-related negative affect and PTSD symptoms on alterations in pain experiences among trauma-exposed women. Finally, this study is unique in its inclusion of multiple aspects of both sensory and affective components of pain sensitivity. Findings suggest that PTSD symptoms may be particularly relevant for understanding the nuances in both sensory and affective components of pain experiences, but that the impact is complicated by elevations in both trait and state negative affect. Additional research is needed to better understand the influence of trauma-related negative affect on pain experiences for women with and without PTSD.
References


Okifuji, A., & Hare, B. D. (2011). Do sleep disorders contribute to pain sensitivity?. Current rheumatology reports, 13(6), 528.


the cold pressor task among individuals with child vs. adult trauma.


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