

IMPACT OF WRITTEN EMOTIONAL DISCLOSURE OF TRAUMA ON  
LABORATORY INDUCED PAIN

A Dissertation

by

SUZANNAH KATHLEEN CREECH

Submitted to the Office of Graduate Studies of  
Texas A&M University  
in partial fulfillment of the requirements for the degree of

DOCTOR OF PHILOSOPHY

May 2008

Major Subject: Psychology

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## ABSTRACT

Impact of Written Emotional Disclosure of Trauma on Laboratory Induced Pain.

(May 2008)

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This study was undertaken to determine whether written emotional disclosure of trauma impacted capsaicin induced pain immediately after writing and at a one-month follow-up, and the extent to which a lifetime history of trauma alters pain under neutral conditions. Three experiments were conducted to answer these questions. In Experiment 1 participants were randomly assigned to write about either a neutral or a trauma topic, and they concurrently completed the capsaicin test. In Experiment 2, the capsaicin test was administered to trauma history and no trauma history participants and pain ratings and secondary hyperalgesia were recorded under neutral conditions. In Experiment 3, participants wrote for three days and completed the radiant heat test before writing on day 1 and after writing on day 3. They also completed the capsaicin test on either day 4 or at a one-month follow-up (day 30). Taken together, these studies had several important results. First, radiant heat withdrawal latencies, ratings of pain intensity and unpleasantness, and area of secondary hyperalgesia were all significantly increased when participants had a history of traumatic experiences. This is evidence that trauma history

is sufficient to alter pain regulatory mechanisms, and this may be attributable to the chronic negative affective state induced by trauma history and sensitization of shared circuits involved in both pain and emotion. Furthermore, our findings suggest that written emotional disclosure may lead to long-term changes in pain modulatory pathways that regulate central sensitization, without altering systems that regulate spontaneous pain.

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## 1. INTRODUCTION

Prior research has demonstrated that negative emotional states alter pain perception. Although it is generally believed that negative affective states enhance pain, both increases (Haslam, 1966; von Graffenried et al., 1978; Schumacher and Velden, 1984; Weisenberg et al., 1984; Malow et al., 1987; Cornwall and Donderi, 1988; Meagher et al., 2001a; deWied & Verbaten, 2001; Villemure et al. 2003, Wunsch et al., 2003; Rhudy et al., 2005; Godinho et al., 2006) and decreases (Bobey and Davidson, 1970; Willer et al., 1979; Willer et al., 1981; Malow, 1981; Pitman et al., 1990, Janssen and Arntz, 1996, Rhudy et al., 1999; Rhudy and Meagher, 2000, 2001a; 2003a, 2003b, 2004) have been reported in controlled laboratory studies. These divergent findings may be attributable to differences in the intensity of the negative affective state, resulting in different levels of arousal (Meagher et al., 1998, 2001a, b; Sieve et al., 2001; Rhudy & Meagher, 2000; 2001a, b, 2003). Low-to-moderately arousing negative affective states tend to induce heightened pain sensitivity (hyperalgesia), whereas highly arousing negative affective states decrease pain sensitivity (hypoalgesia). Taken together, this research suggests that affective valence interacts with arousal to determine the outcome on pain. Research on affective pain modulation is important because clinical pain syndromes rarely occur without increases in negative affect and stress, which may in turn increase pain, setting up a vicious cycle of physical and emotional pain.

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This dissertation follows the style of the Journal of Pain.

Laboratory studies on the relationship between negative affect and pain have typically used standardized stimuli such as emotive statements (Zelman et al., 1991), evocative pictures (Meagher et al., 2001; deWied & Verbaten, 2001; Wunsch et al., 2003; Rhudy et al., 2005; Godinho et al., 2006), and exposure to aversive stimulation such as electric shock (Rhudy & Meagher 2000), loud noise (Rhudy & Meagher, 2001), or aversive odors (e.g., Villemure et al., 2003). However, recently written emotional disclosure (WED) of trauma has emerged as a method for personally relevant affect induction. In this procedure, participants write for 20 min either about their most traumatic experience (trauma topic) or their plans for the day (neutral topic; Pennebaker & Susman 1988), for 15-20 min 3 days in a row. Functioning is typically measured at baseline, after writing, and at a follow-up one to three months later, however, few studies have applied this method to laboratory pain.

Although results have generally indicated that the writing procedure causes immediate increases in negative emotions, they also support an overall trend toward improved health outcomes at follow-up. For example, after writing, researchers have observed improvements in frequency of visits to the doctor one month later, number of Epstein-Barr virus antibody titers one week later, overall immune function six weeks later, and physical health and mood in individuals with asthma or rheumatoid arthritis four months later (Pennebaker & beall 1986; Greenberg, Wortman & Stone 1996; Esterling et al. 1994; Pennebaker, Kiecolt-Glaser and Glaser 1988; Smyth, Stone, Hurewitz & Kaell 1999; also see Smyth, 1998 and Sloan & Marx, 2004 for a review). A meta-analysis of all written emotional expression studies in healthy populations yielded

a significant Cohen's  $d$  of 0.47, a 23% improvement in the experimental group over the control group (Smyth, 1998). A separate meta-analysis of writing studies conducted in clinical populations yielded a Cohen's  $d$  of 0.19, suggesting a modest but positive and significant improvement in health outcome for clinical populations (Frisina, Borod & Lepore, 2004).

Recent research has shifted from investigating whether the paradigm is effective in laboratory studies to evaluating its utility as a tool in therapy (Lepore & Smyth, 2002; Snyder, Gordon & Baucom, 2004). Researchers have also pushed to uncover specific physiological and cognitive mechanisms that might underlie the model's effectiveness (Kloss & Lissman, 2002; Sloan & Marx, 2004). Our laboratory has become interested in the model both as a personally relevant method of affect induction, as well as for its potential as a therapeutic intervention for co-morbid trauma and chronic pain. To understand how the model might work, it is important to first conceptualize the short and long term impact of stress or trauma on the body.

### *Stress, Trauma and Pain*

Immediately after a traumatic or highly stressful event (for a review see McEwen 2002), the body mobilizes itself for action via sympathetic nervous system arousal and hypothalamic-pituitary-adrenal activation, and the release of norepinephrine and cortisol, respectively. This cascade of cellular and hormonal responses is protective in the short-term but causes damage when repeatedly activated by chronic stressors, resulting in wear and tear on the body and brain (McEwen 2002). The damaging effects

of long-term exposure to stress are relevant to many psychological disorders and especially to post traumatic stress disorder (PTSD).

Current research indicates several long-term autonomic, sensory, and cognitive differences are present in individuals with PTSD, and these differences include exaggerated startle, heightened cardiac, skin conductance and blood pressure responsiveness to trauma reminders, elevated tonic or baseline HR, and HPA axis dysregulation (see Orr, Metzger and Pittman, 2002 for an exhaustive review; Yehuda 2002). Additionally, recent functional neuroimaging studies have demonstrated exaggerated amygdala responses in PTSD patients exposed to trauma-related and other negative affect stimuli (Asmundson et al. 2002, Shin et al. 2004, Armony 2004, Schmahl 2004). In addition to links to onset, recurrence, and exacerbation of psychological disorders such as depression, anxiety and PTSD, the cumulative effects of stress have been implicated in heart disease, hypertension, stroke, diabetes, and other disorders of the immune system (Taylor 1999). Thus, interventions aimed at alleviating stress or reducing continued stress from a traumatic experience are theorized to have a positive impact on both mental and physical health.

A high comorbidity between PTSD and chronic pain has led researchers to propose that the experience of trauma is also linked to alterations in pain systems (Asmundson et al., 2002; Beckham et al., 1997; Drossman et al., 1990; Lampe et al., 2000; Scarinci et al., 1994; Leserman et al., 1996; 2006; Walker et al., 1993; 1999; Walling et al., 1994). For example, a previous study by our laboratory found a significant interaction between lifetime history of trauma and writing topic on pain tolerance (Creech, Grimes &

Meagher, under review). Specifically, participants in the neutral writing/trauma history condition exhibited reduced pain tolerance relative to a neutral writing/no trauma history group, which suggests there may be preexisting differences in pain tolerance due to trauma history.

Several plausible psychobiological explanations for comorbidity between trauma and pain exist. One is that exposure to traumatic life events contributes to the development of depression, anxiety, and affect dysregulation, which in turn amplifies the affective experience of pain (for reviews, see Meagher et al., 2002; Frewen & Lanius, 2006b). An alternative explanation for the high comorbidity between PTSD and chronic pain is that a tonic negative affective state induced by trauma disrupts central and descending pain regulatory mechanisms, leading to increased basal pain sensitivity (Creech, Grimes & Meagher under review).

#### *Mechanisms of Effectiveness*

Several theories have been proposed to account for the effectiveness of written disclosure for trauma. Specifically, the improved health outcomes observed after writing have been attributed to a release of inhibition, cognitive restructuring, or exposure and emotional processing. Pennebaker originally suggested that inhibition of a trauma elicits increased short-term autonomic nervous system activity and leads to constant long-term low-level stress (Pennebaker & Susman, 1988), and that writing about the trauma caused a release of inhibition. In other words, inhibiting emotions, memories, or thoughts associated with a trauma takes a physiological toll, which increases susceptibility to illness; therefore, writing improves health by reducing levels of inhibition. The

physiological costs of inhibition represent a form of “allostatic load,” a more recent concept developed by Bruce McEwen. Allostatic load refers to the cumulative wear and tear or cost to the body when it has had to adapt to stress too often or has developed dysfunctional psychobiological regulation of stress [(disruption in normal adrenal hormone, neurotransmitter, immuno-cytokine release); McEwen 1998, McEwen & Wingfeld 2003; Korte et al 2005].

Although inhibited affect certainly has significant consequences, researchers have since argued that the mechanism behind the observed effects of writing about trauma is more likely to involve the cognitive restructuring of memory, or alternatively, exposure and emotional processing (Littrell, 1998; Sloan & Marx, 2004). The central tenet in cognitive restructuring theories is that the appraisal of the event matters most, not the event itself (Foa & Rothbaum 1998). Thus, the idea is that as participants write about their trauma, its cognitive representation, including the full spectrum of memories and emotions, are reorganized and dealt with in a manner that provides structure, organization and cohesion to the memory (Sloan & Marx 2004, Pennebaker 2007; Smyth, True & Souto 2001). These changes are assumed to decrease stress and therefore improve health. Additionally, a core assumption of this model is that the occurrence of a trauma conflicts with a person’s beliefs and assumptions about the world, thus writing about the experience can help the person to successfully process and reconstruct more accurate beliefs (Janoff-Bulman 1992).

Support for the cognitive restructuring view comes from studies in which positive outcomes are correlated with significant changes in insight and causal words over the

course of writing (Pennebaker, Mayne & Francis 1997) and the use of narrative rather than fragmented sentence structure (Smyth, True & Souto 2001). Writing about trauma has also been shown to cause a decrease in intrusive thoughts (Klein & Boals, 2001; Schoutrop et al 2002), and others have found improvements in cognitive appraisal of the trauma at follow-up for the disclosure group (Park & Blumberg 2002).

Exposure and emotional processing techniques emerged from learning theory as viable explanations for how anxiety might become clinically significant, and have been proposed as one explanation for why writing might be beneficial. From a classical conditioning perspective, a traumatic event is conceptualized as a biologically significant event (UCS) that would normally elicit a response (UR). In addition, the context in which the trauma occurred and its related cues can become paired with the UCS to elicit a conditioned emotional response (CER; Mowrer 1960). Generalization and second-order conditioning then allow stimuli associated with the feared and previously neutral stimuli to elicit the CER (Foa & Rothbaum 1998). Finally, operant learning accounts for any corresponding avoidance behavior because negative reinforcement allows the individual to escape from the CS. The CER is thus maintained because the individual does not learn that the UCS no longer accompanies the CS.

Writing about trauma may help promote a habituation of the anxiety response because participants continually expose themselves to the traumatic memory. Thus, the procedure may improve health because writing about the trauma allows the individual to be exposed to previously avoided aversive stimuli, and that over-time extinction or at least habituation of the CR is elicited, thus reducing basal levels of stress hormones in

the body. Support for this view comes from significant decreases in negative emotions and arousal as the writing procedures continues over time (Sloan & Marx, 2004).

Several studies have used self-report to investigate changes in emotion throughout the course of the writing procedure, and these studies have generally found an immediate change in self-reported unpleasantness and arousal (Smyth 1998; Sloan & Marx 2004, Creech, Grimes & Meagher, under review). Physiological markers for stress or affect have also been examined. For example, Sloan and Marx measured salivary cortisol reactivity before and after each writing session (2004). Results indicated that trauma writing participants showed significantly greater reactivity than control writers; however, neither group differed in reactivity to the remaining two writing sessions, suggesting a decline in emotional reactivity over time (2004a). Furthermore, results from this study showed a significant correlation between physiological response to the first writing session and amount of PTSD and depression symptom severity reduction later on, implying that participants who benefit the most later on show the greatest arousal and reactivity after the first writing session (Sloan & Marx, 2004).

While the specific mechanisms underlying the beneficial effects of writing remain unclear, it is likely that all three of these theories together account for why writing may work. Specifically, cognitive restructuring likely leads to a reduction in inhibition as the event is integrated, while exposure desensitizes the person to the memory of the event.

### *Writing as a Treatment*

Following from the positive changes in health that have been correlated with writing interventions, the literature in this area has begun to address whether writing is a



potentially cost-effective and time-efficient component of treatment in various clinical populations. The most well-known use of writing in an empirically validated treatment is likely found in Cognitive Processing Therapy (Resick & Schnicke 1993). The model uses written disclosure of trauma as one way to facilitate processing and has shown consistently strong treatment outcome results when compared to other types of therapies for trauma.

However, several researchers have begun to investigate the clinical impact of writing on other types of symptoms. For example, Smyth, Stone, Hurewitz, and Kaell (1999) found a significant improvement in overall disease activity after writing for rheumatoid arthritis patients. Using verbal rather than written disclosure, Kelley, Lumley and Leisen (1997) conducted a study with rheumatoid arthritis patients and found significant improvements in affective disturbance and physical functioning after the first two weeks following the writing phase. Sullivan and Neish (1999) found emotional disclosure is effective in reducing the effects of catastrophizing on pain and may be effective in increasing pain tolerance during a dental procedure. Finally, Gillis et al. (2006) have suggested disclosure may benefit health outcomes in people with fibromyalgia.

#### *The Impact of Writing on Laboratory Pain*

Our laboratory conducted a recent study to determine whether the negative affective state induced by written emotional disclosure of traumatic experiences could alter pain sensitivity and whether this effect interacted with one's history of trauma (Creech, Grimes, and Meagher, under review). Participants were selected based on high or low trauma history, and each wrote for 20 min about a traumatic or neutral topic. Writing

was immediately followed by the radiant heat pain threshold test and the tourniquet pain tolerance test.

As prior work using written emotional disclosure has indicated that writing about trauma induces measurable increases in negative emotion and arousal (Sloan & Marx 2004a; Sloan & Marx 2004 b; Sloan, Marx & Epstein 2005; Creech, Grimes & Meagher in review), we expected that the affective state would be sufficient to modulate pain. Results of this study suggested that written emotional disclosure of trauma produces subjective and physiological increases in arousal immediately after writing and increased pain sensitivity on the radiant heat pain threshold test. In contrast, tourniquet pain tolerance was decreased within the neutral writing/trauma history group, and this effect was reversed by disclosure of trauma, suggesting that there may be preexisting differences in pain sensitivity and pain modulation related to lifetime history of trauma. Alternatively, decreased tourniquet tolerance may also reflect decreasing motivation rather than enhanced pain, in order to resolve this issue, it is necessary to examine pain ratings to a fixed stimulus, such as capsaicin.

Though topical capsaicin takes about 20 min to reach maximum pain, and is removed after 30 min, the inflammation continues even after the substance has been removed. Thus, one benefit of this model is that it eliminates many of the inherent motivational issues that can impact pain threshold and pain tolerance. A second benefit of using a capsaicin pain model to study hyperalgesia is that it provides a means of studying both primary and secondary hyperalgesia, which are triggered by different neural mechanisms.

Primary hyperalgesia is characterized by spontaneous pain and both heat and mechanical hyperalgesia (Raja, Campbell, & Meyer, 1984). In addition, it is likely the result of activation and sensitization of both peripheral and central nociceptors (Raja, Campbell, & Meyer, 1984; Torebjork, Lundberg, & LaMotte, 1992). In contrast, secondary hyperalgesia is characterized by only mechanical (static, dynamic, and punctate) hyperalgesia (Ali, Meyer, & Campbell, 1996; Fuchs, Campbell, & Meyer, 2000; Magerl, Wilk, & Treede, 1998; Raja, Campbell, & Meyer, 1984). Furthermore, secondary hyperalgesia is caused by the sensitization of central nociceptive neurons (Campbell, Khan, Meyer, & Raja, 1988; Torebjork, Lundberg, & LaMotte, 1992). The central mediation of secondary hyperalgesia is supported by the finding that hyperalgesia can be evoked by stimulation of afferent fibers even after peripheral nociceptors have been anesthetized (Torebjork, Lundberg, & LaMotte, 1992).

Capsaicin is an extract from hot chili peppers that causes a neurogenic inflammatory process and sensations of intense burning pain when applied to the skin (Bauman, Simone, Sharin & LaMotte, 1991; Jansco, Jansco-Gabor & Szoclsanyi, 1967). The skin becomes red and inflamed at the site of application resulting in hyperalgesia and allodynia. Importantly, the primary afferents that respond to capsaicin have been shown to initiate and maintain pathological pain states through mechanisms of central sensitization (Simone et al., 1991; Simone, Bauman, Collins, & Lamotte 1989). Thus, capsaicin-induced pain models mimic many of the features of central sensitization that underlie neuropathic and inflammatory clinical pain.

Prior studies on how expressive writing might impact pain have not examined whether these changes in pain are due to alterations in the sensory component of pain versus the affective component. My first experiment will begin to examine this issue by asking subjects to rate sensory intensity and unpleasantness of spontaneous capsaicin-induced pain on separate visual analog scales (VAS). In addition, the use of controlled stimuli in the proposed experiment will allow us to equate the physical stimulus properties of the pain across subjects, whereas the intensity of clinical pain will vary depending on the severity of the patient's disease and their stage in the disease. This is important because chronic pain alone can disrupt the functioning of descending pain pathways and emotional states over time (Ren & Dubner, 2002).

Researchers have suggested that the limbic and brainstem structures that are sensitized by PTSD also modulate pain (Rhudy & Meagher 2001; Rosen & Shulkin 1998; Meagher 2002). Given this, it was hypothesized that even under normal conditions (i.e., no trauma cues are present) these alterations and accompanying symptom constellations are sufficient to induce abnormalities in pain processing. Furthermore, both the clinical symptoms associated with PTSD, and evidence that individuals with PTSD experience increased depression and anxiety, imply that a basal state of negative affect accompanied by low arousal may leave these individuals vulnerable to heightened and easier-elicited negative emotional states. I thus hypothesized that individuals with a history of trauma would demonstrate increased baseline negative affect and increased sensitivity to pain. To test this hypothesis, my second experiment examined whether history of traumatic experience alone is sufficient

to alter spontaneous pain ratings and tactile allodynia testing during topical capsaicin, and whether these differences in pain processing are associated with higher basal levels of negative affect and low-to-moderate levels of arousal. Basal negative affect was tested in several ways prior to pain testing in order to determine whether the trauma group displayed an *a priori* difference in negative affect.

Finally, previous researchers examining the impact of writing on health have conducted longitudinal studies in which health outcomes improve over time. Specifically, most have found an acute increase in stress and negative emotions immediately after writing, and an increase in health at a one-month follow-up (presumably as participants restructure or de-sensitize to their traumatic memories). Based on these findings, I hypothesized that while writing about trauma would elicit negative affect and increased pain sensitivity immediately after writing, pain sensitivity would decline at a one-month follow-up. To test this hypothesis, in experiment 3 participants were asked to write for three days, and multiple pain methods were assessed at baseline, after writing and at a one-month follow-up. At baseline, I expected to observe a pre-existing difference in thermal pain sensitivity between trauma history and no trauma history participants. Specifically, trauma history participants were expected to show reduced thermal pain thresholds compared to no-trauma history participants at baseline.

It was also hypothesized that immediately after the first day of writing self-report and physiological measures will indicate high levels of stress and negative affect for participants in the trauma writing group (experimental group), and these participants

were expected to show increased pain sensitivity relative to controls. I expected this to be reduced after the third day of writing, and even further reduced at the one-month follow-up. However, for high trauma participants, I expected to observe analgesia to capsaicin due to a preexisting autonomic hyperreactivity that is engaged by written emotional disclosure of trauma (and potentially by the pain testing itself).

## 2. GENERAL METHODS

### *Writing Procedure*

Participants were randomly assigned to write about a neutral topic or the most traumatic experience of their life, and they received instructions for the writing procedure in accordance with Pennebaker's previously published procedures (Pennebaker & Susman, 1988). Writing prompts were delivered to participants in envelopes to keep experimenters blind. The writing prompts are shown below.

### *Trauma Writing Prompt*

*Day One. What I would like to have you write about for the next three days is the most traumatic, upsetting experience of your entire life. In your writing, I want you to really let go and explore your very deepest emotions and thoughts. You can write about the same experience on all four days or about different experiences each day. In addition to a traumatic experience, you can also write about major conflicts or problems that you have experienced or are experiencing now. Whatever you choose to write, however, it is critical that you really delve into your deepest emotions and thoughts. Ideally, we would also like you to write about significant experiences or conflicts that you have not discussed in great detail with others. You might tie your personal experiences to other parts of your life. How is it related to your childhood, your parents, people you love, who you are, or who you want to be. Again, in your writing, examine your deepest emotions and thoughts.*

*Day 2. How did yesterday's writing go? Today, I want you to continue writing about the most traumatic experience of your life. It could be the same topic that you wrote about yesterday or it could be something different. But today, I really want you to explore your very deepest emotions and thoughts...*

*Day 3. You have survived the first two days, and today is the last one. In your writing today, I again want you to explore your deepest thoughts and feelings about the most traumatic experience of your life. Remember that this is the last day and so you might want to wrap everything up. For example, how is this experience related to your current life and your future? But feel free to go in any direction you feel*

*most comfortable with and delve into your deepest emotions and thoughts....*

### *Neutral Writing Prompt*

*Day One. What I would like you to write about over the next three days is how you use your time. Each day, I will give you different writing assignments on the way you spend your time. In your writing, I want you to be as objective as possible. I am not interested in your emotions or opinions. Rather I want you to try to be completely objective. Feel free to be as detailed as possible. In today's writing, I want you to describe what you did yesterday from the time you got up until the time you went to bed. For example, you might start when your alarm went off and you got out of bed. You could include the things you ate, where you went, which buildings or objects you passed by as you walked from place to place. The most important thing in your writing, however, is for you to describe your days as accurately and as objectively as possible.*

*Day 2. How did your writing go yesterday? Today, I want you to describe in detail what you will do as soon as the experiment is over until you go to bed tonight. For example, you might start by noting that you will walk out of the door, go down the steps, walk across the campus, and so forth.*

*Day 3. This is the last day of the experiment. In your writing today, I would like you to describe what you will be doing over the next week...*

### *Self-Report Data*

*Manipulation Checks.* Participants rated their reactions to the writing procedure and pain testing using the Self-Assessment Manikin (SAM; Lang, 1980). The SAM is a measure with two pictogram scales indicating various levels of valence (ranging from “happy” to “unhappy”) and arousal (ranging from “excited” to “calm”). Participants are asked to place an “X” on or between any of the figures to indicate their emotional response after writing and after each pain test.

The PANAS-X is comprised of 60 items measuring 11 specific affects in addition to overall mood valence (positive and negative). Participants are asked to rate each



affective descriptor and rate the degree to which they felt that way that day, during and since writing, or during and since the last pain test on a 1 (very slightly) to 5 (extremely) point scale.

*Psychological and Health Symptom Measures.* Participants completed measures of depression, PTSD, and health care utilization. These measures included either the Beck Depression Inventory - II (BDI-II; Beck 1960) or the Center for Epidemiological Study – Depression scale (CES-D, Radloff, 1977), the Trauma Symptom Checklist- 40 (TSC-40), a modified version of Pennebaker’s Childhood Trauma Questionnaire, the Pennebaker Inventory of Limbic Languidness (PILL; Pennebaker, 1982) and a general health status form.

*Depression.* The BDI-II is a twenty-one question multiple-choice self-report inventory that is one of the most widely used instruments for measuring the severity of depression. The questionnaire is designed for adults age 17-80 and is composed of items relating to depression symptoms such as hopelessness and irritability, cognitions such as guilt or feelings of being punished, as well as physical symptoms such as fatigue, weight loss, and lack of interest in sex.

*Emotional Distress.* Because we were interested in the effects of stress on pain reactivity, it was necessary to assess any preexisting emotional distress that may contribute to unwanted group differences. To do so, the CES-D a brief, 20-item questionnaire that taps into depression and anxiety symptoms was filled out prior to the Experiment. Subjects were instructed to read each item and rate the extent to which they felt that way at sometime during the past week.

*Trauma Symptoms.* The TSC-40 is a research measure that evaluates aspects of posttraumatic stress and other symptom clusters found in some traumatized individuals. It is a 40-item self-report instrument consisting of six subscales: Anxiety, Depression, Dissociation, Sexual Abuse Trauma Index (SATI), Sexual Problems, and Sleep Disturbance, as well as a total score. Each symptom item is rated according to its frequency of occurrence over the prior two months, using a four-point scale ranging from 0 ("never") to 3 ("often"). The TSC-40 requires approximately 10-15 min to complete, and can be scored in approximately 5-10 min.

*Trauma History Measures.* Information on history of traumatic events was obtained via a modified version of Pennebaker's Childhood Trauma Questionnaire, a survey of six early traumatic experiences (death, divorce, violence, sexual abuse, illness, or other) and ratings of the degree to which individuals confided the traumas. The measure was modified by this lab to address a problematic rating scale in which participants were asked to rate how "traumatic" the incident was. The rating scale was changed from a 1-7 general Lickert scale, to a 4-point scale with descriptive anchors.

The experience of 5 types of traumas was queried (death of close friend or family member, traumatic sexual experience including rape or molestation, experience of violence, severe illness or injury, major upheaval such as natural disaster, car accident, divorce, loss of job etc.) both within the last 3 years and prior to the age of 17. If the participant indicated "yes" this event did occur, they went on to rate "how bothersome/traumatic" was this experience on a 4 point scale: Did not bother me/Not at all Traumatic, Bothered me for a short time/Slightly traumatic, Bothered me for a

while/Traumatic, and Continues to bother me/Extremely traumatic. They also indicated whether their reaction to the event included fear, helplessness, or horror.

Female undergraduates enrolled in introductory psychology were voluntarily prescreened for traumatic experiences in the Fall 2005, Spring 2006, Fall 2006 and Spring 2007 semesters at Texas A&M University (N = 1244). Following previously used methodology, participants qualified for the no trauma history group if their trauma score was two standard deviations below the population mean of 3.58, ( $SD = 5.15$ ), and for the trauma history group if their trauma score was two standard deviations above the population mean. All qualified participants were contacted by email to let them know they qualified to participate in the study, and participants signed up on a voluntary basis.

*Health Care Utilization.* Participant frequency of visits to the doctor and other health problems was assessed using the PILL. The PILL is a 54-item scale that measures frequencies of various common physical symptoms and sensations such as running nose and headaches.

*Health Status.* In order to participate in pain testing, a brief health status questionnaire was administered to all potential subjects. Anyone with ongoing health conditions such as cardiovascular disease, neurological disorders, chronic pain of any type, and circulatory disorders was ineligible for the study.

#### *Apparatus and Physiological Recording*

*Physiological Recording.* All data collection was computer controlled by LabVIEW software and an AT-MIO-16DL DAQ board (both by National Instruments). Heart rate (HR) and skin conductance level (SCL) sensors were attached to fingers of the non-

dominant hand and sampled at 50 Hz.

*Mechanical- Visual Analogue Scale Pain Ratings.* Participants were asked to rate both their sensory and affective level of discomfort during pain testing using a Mechanical Visual Analogue Scale (M-VAS). The M-VAS is used to assess subjective ratings of the sensory intensity and unpleasantness of the stimulus by using line length to represent the magnitude of the subjective state. The endpoints correspond to numbers and verbal descriptors (e.g., 0= not at all unpleasant, while 10 = most unpleasant imaginable). An M-VAS is a physical instrumentation of a pencil and paper visual analog scale consisting of a 100-mm line. Participants move a sliding lever along the line to indicate their pain ratings. This sends a proportional voltage to the computer indicating threshold has been reached and each time the participant's pain changes.

#### *Pain Testing*

*Capsaicin Test.* In this test, 300  $\mu$ l of a 6.0% capsaicin solution is topically applied to the dominant volar forearm via a 1.5 cm x 1.5 cm gauze pad (Culp, Ochoa, et al., 1989; Simone, Baumann, & LaMotte, 1989). To impede evaporation, the site of application is covered with a dressing (Baron, Wasner, et al., 1999). The pad and dressing is left on the arm for a period of 30 min.

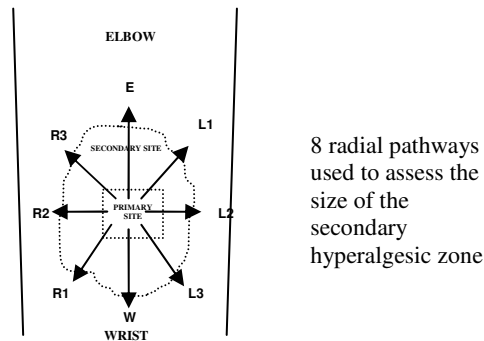


Figure 1. Site of Testing

*Spontaneous Pain Ratings.* To administer this test, during the 30 min capsaicin application subjects are asked to rate their affect using a SAM and a PANAS-X at 5 min intervals. Subjects are also asked to rate their pain at these 5 min intervals using a mechanical VAS, which contains both an “intensity” and an “unpleasantness” component.

*Tactile Allodynia Test.* After the 30 min application, the capsaicin is removed from the forearm. Following capsaicin removal, a second pain test involving application of pressure across the site of inflammation was administered. This test measures allodynia, or sensitivity to mechanical stimuli. In this procedure, a grid with spokes radiating from the center is drawn on the forearm (shown above; Figure 1). Each spoke consists of ten pain application sites. Then beginning at the wrist spoke, all ten sites on each spoke are stimulated using a large diameter von Frey hair (6.65 g), working from the outside in.

A von Frey hair is a flexible nylon filament attached at a right angle to one end of a holder. Each filament has a different thickness, ranging from very fine to thick. The nylon filament is placed on the forearm and deformed by downward pressure. A

measurable and reproducible weight (.065 N) is required to deform the filament, which allows for consistency across experiments and experimenters. Participants make continuous ratings of changes in pain perception after each touch of the von Frey hair on the VAS device.

*Radiant Heat Threshold Test.* Pain thresholds were assessed by measuring the time taken to withdraw the finger from a radiant heat stimulus (temperature = 43.5 degrees centigrade). Participants were asked to withdraw their finger (distal phalanx of the index finger on the left hand) *as soon as* they first feel pain. An overhead projector light provided the radiant heat source, which was focused onto a 2 cm region of the finger. Lateral movements of the finger were detected by a photocell (positioned below the finger embedded within the aluminum finger platform), which records the withdrawal latency and terminates the stimulus. An automatic cut-off of 8 s was used to prevent tissue damage. After a practice trial, 2 pain threshold tests were assessed and averaged using this methodology.

### 3. EXPERIMENT 1 METHOD

The primary goal of Experiment 1 was to determine whether written disclosure of trauma alters capsaicin-induced pain. Given prior evidence that the negative emotional state induced by writing about trauma impacted both radiant heat pain thresholds and tourniquet tolerance, I predicted that writing about the trauma topic would enhance mechanical visual analog scale ratings of the sensory and affective dimensions of capsaicin-induced spontaneous pain.

#### *Participants*

Participants were 28 healthy female students with no history of traumatic experiences. There were also 13 participants who would have qualified as “trauma history” based on previously used methodology from other studies in this lab. These participants qualified as “trauma history” because their scores were two standard deviations above the population mean. As they differed on this variable, they were excluded prior to data analysis and their data will be used elsewhere.

#### *Procedure*

Upon entering the lab, participants were escorted into the experiment chamber for an explanation of procedures and informed consent. Participants were asked to sign the informed consent and then complete the Center for Epidemiological Study-Depression Scale (CES-D; Radloff, 1977). After completing these questionnaires participants received instructions for pain testing, and baseline skin conductance and heart rate were taken. These procedures lasted for 30 min and served to help habituate

participants to their surroundings. A timeline of the experimental procedure used is shown in Figure 2.

Participants were randomly assigned to neutral or trauma writing conditions. After informed consent procedures and directions, capsaicin was applied to the arm and participants were instructed to begin writing. Both subjective (SAM, PANAS) and physiological indicators (skin conductance level, heart rate) were assessed to determine whether writing conditions produced distinct emotional states. During the final 10 min of the study, participants completed spontaneous pain ratings using the M-VAS at 2 min intervals.



#### 4. EXPERIMENT 1 RESULTS

##### *Manipulation Checks – Writing Procedure*

Participants rated both the arousal and valence of their emotional state on the SAM at three time points: at baseline, 10 min into the procedure (in the middle of writing), and at 20 min (the end of writing).

*SAM Valence Ratings.* SAM valence ratings were analyzed using ANOVA with writing topic (neutral or trauma) as a between subjects variable, and valence ratings at the three time points as a repeated measurement. Using this method, a significant main effect of writing topic on SAM valence ratings emerged,  $F(1, 26) = 5.761, p < .05$ , indicating that participants who wrote about the neutral topic rated the valence of their emotional state, on average, as less unpleasant than those who wrote about the trauma topic. All other analyses including SAM completed at baseline were not significant, all  $p$ 's  $> .05$ .

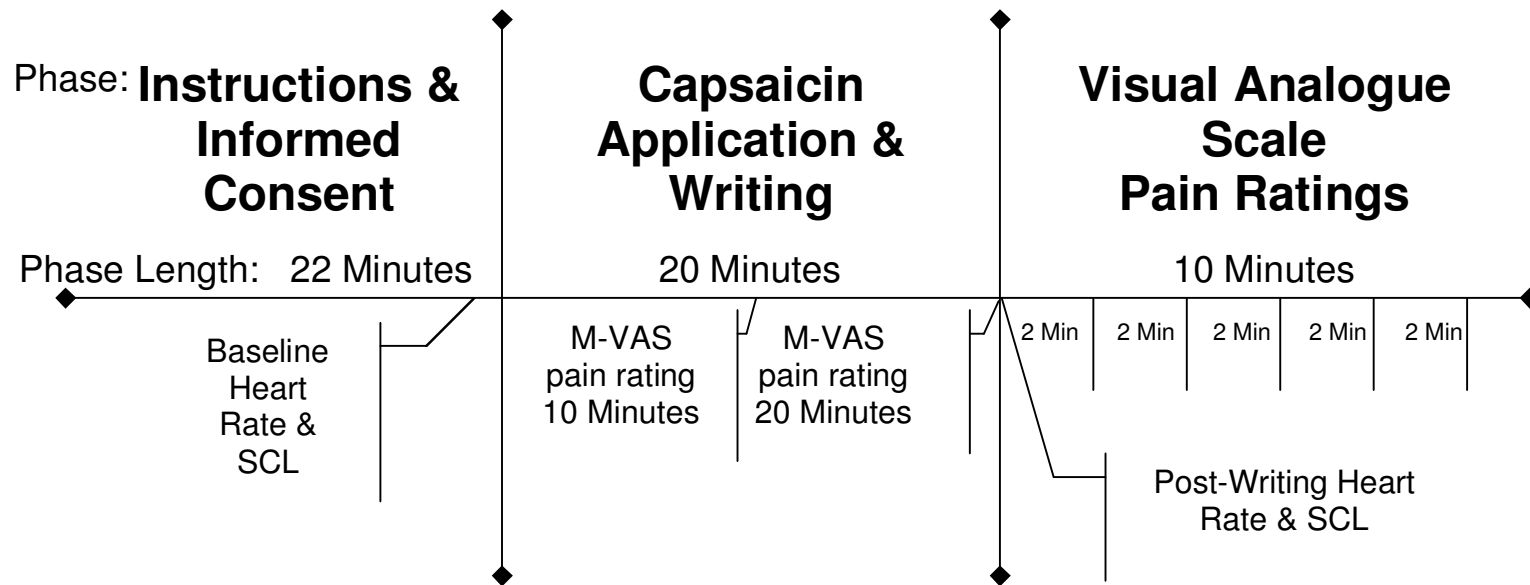


Figure 2: Experiment Timeline

A significant interaction effect between SAM Valence ratings over time and writing topic also emerged,  $F(1, 52) = .0239, p < .05$ . As shown below in Figure 3.a, *post hoc* means comparisons indicated a significant difference in valence ratings between neutral and trauma writers at the 10 min time point,  $F(1, 26) = 15.349, p < .001$ . Participants who wrote about the neutral topic rated their emotional state in the middle of writing as less unpleasant than those who wrote about the trauma topic.

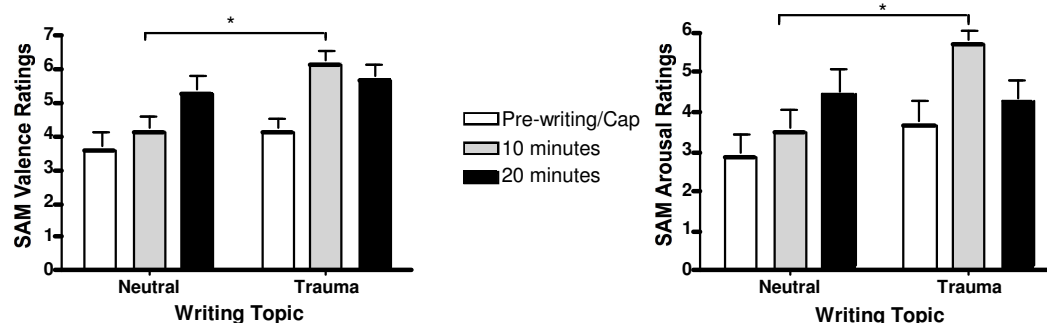


Figure 3. SAM Valence and Arousal Ratings Across 3 Time Points

*SAM Arousal Ratings.* SAM arousal ratings were analyzed using ANOVA with writing topic (neutral or trauma) as a between subjects variable, and valence ratings at the three time points as a repeated measurement. An initial analysis of baseline SAM ratings (prior to writing) was not significant,  $p > .05$ . Using the method above, a main effect of writing topic approached significance,  $F(1, 26) = 3.219, p = .084$ , indicating

that participants who wrote about the neutral topic rated their arousal level as less than those who wrote about the trauma topic.

A significant interaction effect between writing topic and arousal ratings at the three time points also emerged,  $F(1,52) = 4.216, p < .05$ . As shown above in Figure 3.b *post hoc* means comparisons indicated a significant difference in arousal ratings between neutral and trauma writers at the 10 min time point,  $F(1, 26) = 17.023, p < .001$ . Participants who wrote about the neutral topic rated their emotional state in the middle of writing as less aroused than those who wrote about the trauma topic.

#### *Physiological Measurements*

Heart rate and skin conductance levels were collected at baseline, at 10 min, and again at the 20 min post-writing time point. Average heart rate (BPM) and SCL were calculated and change scores were obtained by subtracting recordings the 10 min and 20 min scores from baseline. Although it was predicted that neutral and trauma writing participants would differ physiologically at both 10 min and 20 min, significant differences only emerged for heart rate measurements at the 20 min time point. Specifically, a significant main effect of writing topic on the change in heart rate at 20 min from baseline was found,  $F(1, 25) = 7.76, p < .01$ . As shown below in Figure 4, while neutral writers had an overall decrease in heart rate after writing trauma writers showed an increase in heart rate after writing. All other analyses including SCL were not significant,  $p > .05$ .

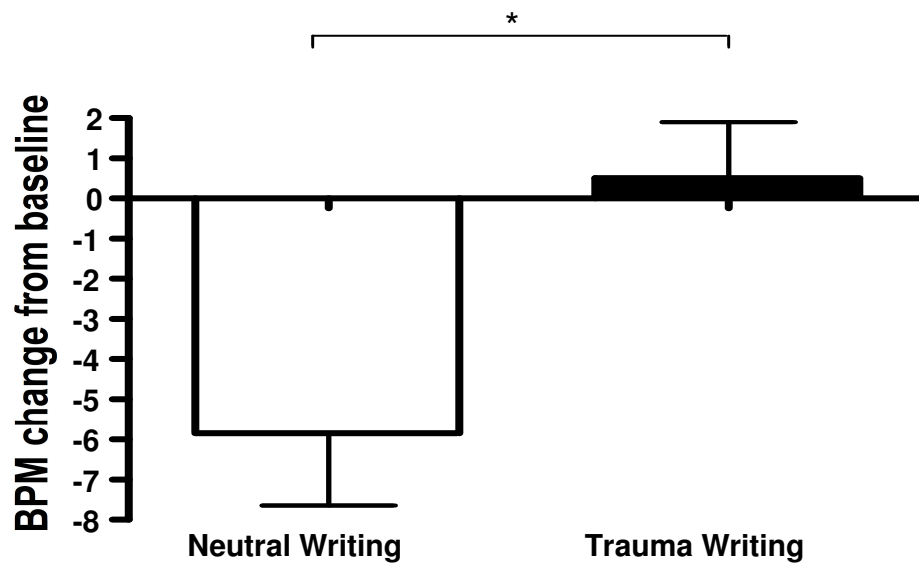


Figure 4. BPM Change from Baseline by Writing Topic

#### *M-VAS Pain Intensity Ratings*

Pain intensity ratings for the last ten min of the capsaicin test were analyzed using ANOVA with day one writing topic (trauma or neutral) and VAS Intensity ratings over time as a repeated measurement. As depicted in Figure 5 below, a significant main effect of writing topic on VAS Intensity ratings was observed,  $F(1, 26) = 4.517, p < .05$ , indicating that participants who wrote about the trauma topic rated their pain as significantly less intense than those who wrote about the neutral topic. All other analyses were not significant, all  $p$ 's  $> .05$ .

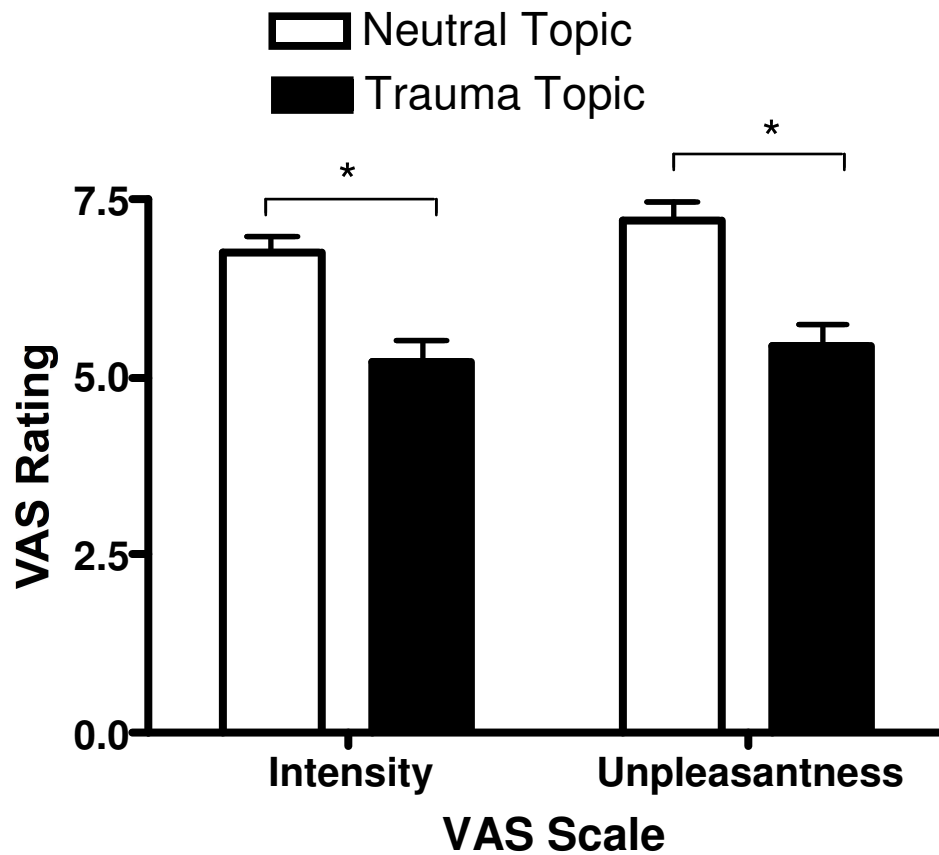


Figure 5. M-VAS Pain Ratings to Capsaicin

#### *M-VAS Pain Unpleasantness Ratings*

Pain unpleasantness ratings for the last 10 min of the capsaicin test were collapsed and analyzed using ANOVA with day one writing topic (trauma or neutral) and VAS unpleasantness ratings over time as a repeated measurement. A main effect of writing topic was observed,  $F(1, 26) = 4.096$ ,  $p < .05$ . Again, participants who wrote about the neutral topic rated their pain unpleasantness as, on average, more unpleasant than those who wrote about the trauma topic. This is depicted above in Figure 5.

Figure 6 (below) depicts the interaction effect of writing topic and M-VAS ratings of pain unpleasantness over time to the capsaicin test  $F(1, 104) = 4.106, p < .01$ . *Post hoc* mean comparisons indicated significant between groups differences in unpleasantness ratings at both the 28 min ( $F[1, 26] = 8.014, p < .01$ ) and 30 min ( $F[1, 26] = 7.931, p < .01$ ) time points. Specifically, participants who wrote about the neutral topic rated their pain unpleasantness as significantly greater at 28 and 30 min than did trauma writers.

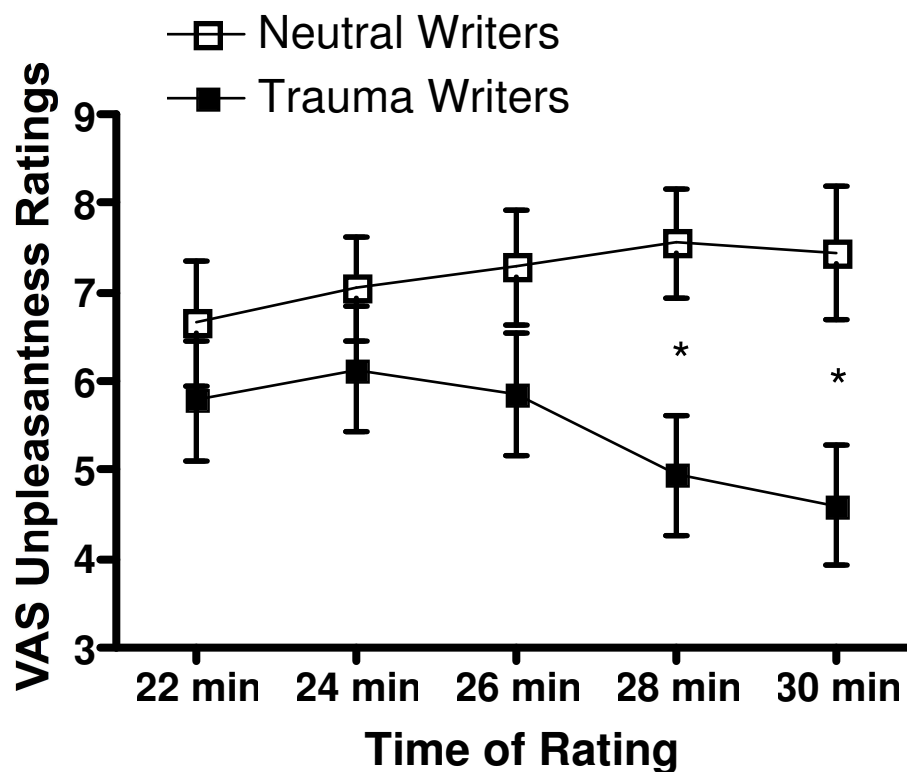


Figure 6. VAS Unpleasantness Ratings to Capsaicin

## 5. EXPERIMENT 1 DISCUSSION

The goal of experiment 1 was to determine whether written emotional disclosure impacted capsaicin-induced spontaneous pain by creating a personally relevant negative affective state. Although I had previously shown that written emotional disclosure impacted radiant heat withdrawal latencies, the capsaicin model more closely mimics pathological pain caused by inflammation and central sensitization. It also eliminates many of the inherent motivational issues that can impact pain threshold and pain tolerance measures, thus it was unclear whether the emotional state induced by writing would impact this type of pain.

It was hypothesized that participants who wrote about the trauma topic would rate their mood as more negative and more unpleasant than those who wrote about a neutral topic. It was also hypothesized that participants writing about the trauma topic would report increased pain intensity and unpleasantness when compared to the neutral group. As predicted, the trauma writing group reported increased unpleasantness and arousal, and physiological indicators showed significantly increased heart rate after writing about trauma. However, contrary to previous findings (Creech, Grimes & Meagher under review), we were unable to detect a change in SCL after writing in the trauma writing condition. It is possible that the stress induced by the capsaicin test alone, which is concurrent with writing, may be masking the effect of trauma writing on SCL. Taken together, both self-report and physiological data indicate that written emotional disclosure elicited a negative affective state.



Based on the prediction that this affective state would be negative, but not highly arousing because the participants had no history of trauma, I predicted increased pain in the trauma-writing group. However, trauma writers rated their pain intensity and unpleasantness as significantly less than those writing about the neutral topic. Although these results were not in the predicted direction, this finding is consistent with previous work indicating decreases in pain after negative affect induction (Bobey and Davidson, 1970; Willer et al., 1979; Willer et al., 1981; Malow, 1981; Pitman et al., 1990, Janssen and Arntz, 1996, Rhudy et al., 1999; Rhudy and Meagher, 2000, 2001a; 2003a, 2003b, 2004).

Rhudy and Meagher (2000) have previously suggested the level of arousal may determine whether a negative affective state decreases or increases pain. More specifically, they have proposed that highly arousing negative affect may cause hypoalgesia in humans, whereas low-to-moderately arousing negative affect results in hyperalgesia (Rhudy and Meagher, 2000, 2001c; Meagher et al., 2001a,b). In this study, capsaicin was applied to the arm at the same time that participants were instructed to begin writing, thus I posit that this overlap increased the arousal level of trauma-writing participants causing a stress-induced hypoalgesia. This is in contrast to our prior studies in which writing and pain testing occurred during discrete intervals and there was no overlap, thus hyperalgesia was elicited because the affective state was only moderately arousing (Creech, Grimes & Meagher, in review). This stress-induced hypoalgesia may be due to the activation of the mu-opioid receptor system by endogenous opioid peptides resulting in reductions in sensory and affective ratings of pain (Zubieta et al., 2001,

2003). The significant increase in heart rate as well as self-reported arousal at the 20 min time point in the trauma writing group also supports this view because arousal was high while pain intensity and unpleasantness ratings were low.

## 6. EXPERIMENT 2 METHOD

The goal of experiment 2 was to further examine the impact of trauma history on laboratory pain outside of any affect induction procedures. This is important as results obtained after affect induction may be influenced by prior history of trauma, and a prior study by this laboratory found decreased tourniquet tolerance in trauma history females under neutral conditions. It was thus hypothesized that trauma and no trauma history individuals would differ in pain ratings and area of secondary hyperalgesia to the capsaicin test. Specifically, I predicted that the trauma history group would show greater basal pain sensitivity and greater secondary hyperalgesia when compared to a no trauma group.

### *Participants*

Thirty-three healthy female students were included as trauma history or no trauma history participants based on trauma history scores obtained during departmentally organized prescreening sessions. Participants qualified for the trauma history condition if their summed trauma history score was two standard deviations above the population mean. Means and standard deviation scores for both groups are shown in Table 1 along with data from the PANAS negative affect scale, CES-D scores and the PILL.

Table 1

*Number of participants per group and trauma history scores*

Trauma Group	Trauma History Score			PANAS NA score		CES-D		PILL	
	M	SD	N	M	SD	M	SD	M	SD
No Trauma	3.45	3.05	16	10.750	6.598	7.938	7.573	13.875	6.612
Trauma	15.82*	6.05	17	10.529	9.618	17.294*	6.191	20.00*	10.886

*Procedure*

Upon entering the laboratory participants were escorted into the experiment chamber for an explanation of procedures and informed consent. Participants were asked to sign the informed consent and then completed the PILL, the CES-D and the PANAS (scores shown in Table 1). After completing these questionnaires participants received instructions for pain testing, and baseline skin conductance and heart rate were taken. These procedures lasted for 30 min and served to help habituate participants to their surroundings. Following acclimation to the experiment room, participants completed the capsaicin test. No emotion induction was used in this experiment as it was designed to determine the impact of trauma history on pain under affectively neutral environmental conditions. However, pre-existing differences in trait affect related to trauma history were assessed using the CES-D (scores shown in Table 1).

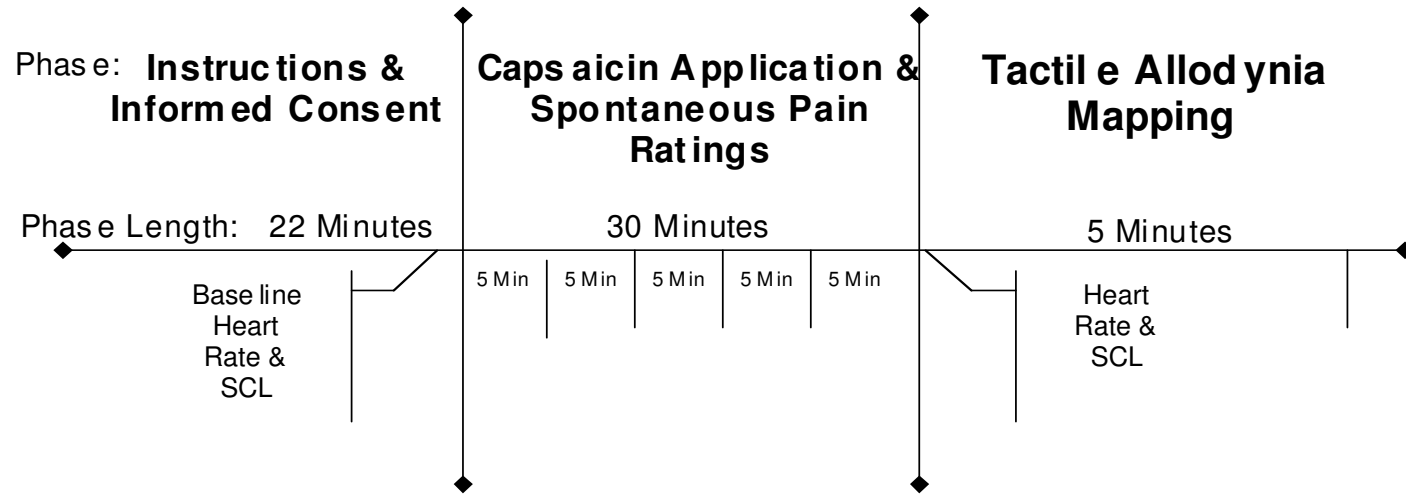


Figure 7. Experiment 2 Timeline

## 7. EXPERIMENT 2 RESULTS

### *Physiological Measurements*

Heart rate and skin conductance levels were collected prior to capsaicin application, during capsaicin testing, and at the 30 min time point. Although it was predicted that trauma and no trauma history participants might differ in physiological measurement at baseline, there were no significant between or within group differences in SCL and BPM at baseline or in change from baseline after capsaicin testing, all  $p$ 's  $> .05$ . As predicted, the trauma group did display a higher number of BPM at baseline ( $M = 78$ ,  $SEM = 2.163$ ) than the no trauma group ( $M = 70.93$ ,  $SEM = 4.8$ ), however this was not significant,  $F(1,31) = 1.862$ ,  $p = .18$ . While these results were in contrast to our predictions, they are consistent with our prior findings in which there was no baseline GSR or HR difference between trauma history and no trauma history groups (Creech, Grimes & Meagher under review).

### *Baseline Affect and Health Care Utilization*

In order to determine whether a preexisting between groups difference in negative affect existed, participants each completed the PANAS with instructions to rate how they had felt that day. The trauma and no trauma groups did not differ on this test,  $p > .05$  (means shown in Table 1). However, the two groups did significantly differ in baseline levels of distress on the CES-D,  $F(1,31) = 14.988$ ,  $p < .001$ , indicating that the trauma history group showed significantly higher emotional distress than the no trauma group at baseline. The two groups approached a significant difference in self-reported health care

utilization on the PILL,  $F(1, 31) = 3.757$ ,  $p = .06$ . This suggests that on average, trauma history participants reported increased number of visits to the doctor, illnesses, and overall poorer health in the last semester than no trauma history participants (means shown in table 1). Its important to note that this measure does not assess for catastrophizing or other interpersonal variables that could impact perception of health, therefore it is not possible to conclude that the measure accurately reflects health status.

#### *Self-Assessment Manikin Ratings*

Participants made SAM ratings of the valence and arousal of their emotional state every 5 min for the first 30 min of the capsaicin test. There were no significant differences between the trauma ( $M$  Valence = 5.012, SEM = .261;  $M$  Arousal = 3.965, SEM = .294) and no trauma groups ( $M$  Valence = 4.775, SEM = .191;  $M$  Arousal = 3.925, SEM = .243) for either the valence or arousal of their emotional state at any time point, all  $p$ 's > .05.

#### *M-VAS Pain Intensity Ratings*

Pain intensity ratings were collected every 5 min for the first 30 min of capsaicin application. Ratings were analyzed using ANOVA with trauma group (no trauma history or trauma history) as between subject variables and VAS unpleasantness ratings over time as a repeated measurement serving as both within and between subjects variable. As can be seen in panel A of Figure 8, a significant interaction emerged between trauma group and intensity ratings over time  $F(4, 124) = 3.422$ ,  $p < .01$ . *Post hoc* means comparisons were conducted to determine which time points were driving the significant interaction, however only the difference in intensity ratings between the no

trauma and trauma group at time 1 approached significance,  $F(1,31) = 2.391$ ,  $p = .13$ . Although not significant, intensity ratings at time 1 (5 min after capsaicin application) were lower in the no trauma group than they were in the trauma group. However, by time 5 (25 min into capsaicin procedure) this effect was reversed. When change in intensity rating scores from time 1 to time 5 were computed, a significant main effect of trauma level emerged. As shown in panel B of Figure 8 the no trauma group increased their intensity rating while the trauma group decreased their rating  $F(1,31) = 5.723$ ,  $p = .0230$ . It should be noted, however, that this analysis reflects a change in pain intensity from time 1 and not from baseline.

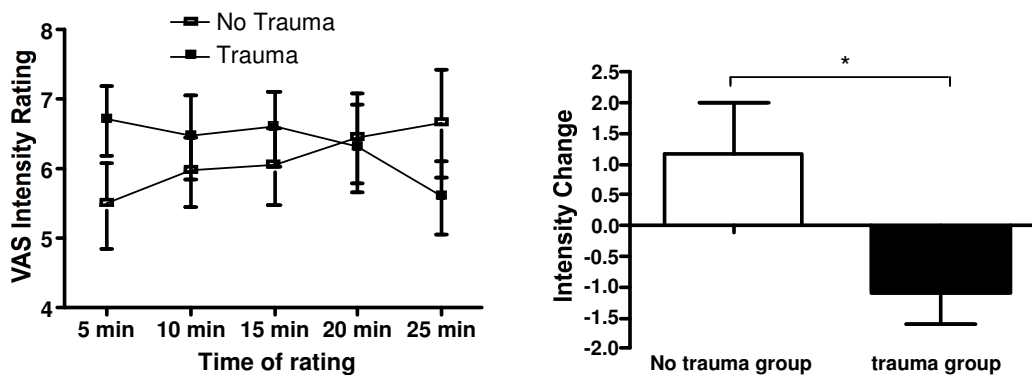


Figure 8. VAS Intensity Ratings Over Time



*M-VAS Pain Unpleasantness Ratings*

Pain unpleasantness ratings were collected every 5 min for the first 30 min of capsaicin application. Ratings were analyzed using ANOVA with trauma group (no trauma history or trauma history) as between subjects variables and VAS unpleasantness ratings over time as a repeated measurement. Although the two groups did not differ significantly when their average unpleasantness rating was analyzed,  $F(1,31) = .822, p = .37$ , a significant interaction emerged between trauma group and unpleasantness ratings over time,  $F(4, 120) = 6.165, p < .001$ . As shown in panel A of Figure 9, means comparisons indicated that the unpleasantness ratings at time 1 (5 min after capsaicin application) were significantly lower in the no trauma than in the trauma group,  $F(1,31) = 4.402, p < .05$ . However, by time 5 (25 min into capsaicin procedure) this effect was reversed,  $F(1,30) = 3.038, p = .09$ . When change in unpleasantness rating scores from time 1 to time 5 were computed, a significant main effect of trauma level emerged. As shown in panel B of Figure 9 the no trauma group had an increase in unpleasantness between time 1 and time 5, while the trauma group decreased their ratings  $F(1,31) = 10.974, p < .002$ .

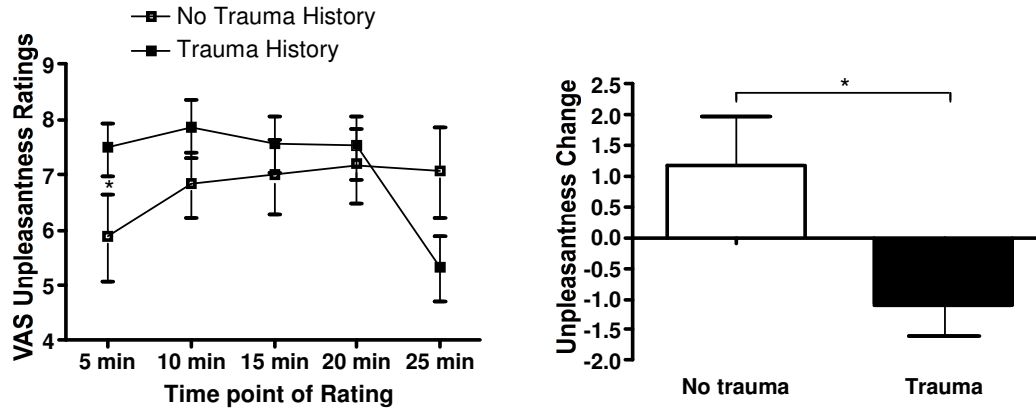


Figure 9. VAS Unpleasantness Ratings Over Time

*Area of Secondary Hyperalgesia and Pain Ratings*

To examine the impact of trauma history on area of secondary hyperalgesia an ANOVA was used with trauma history entered as a between-subject variable. As is depicted in Figure 10, the area of secondary hyperalgesia was significantly smaller in the no trauma group than it was in the trauma history group, [ $F(1, 31) = 6.448, p < 0.02$ ].

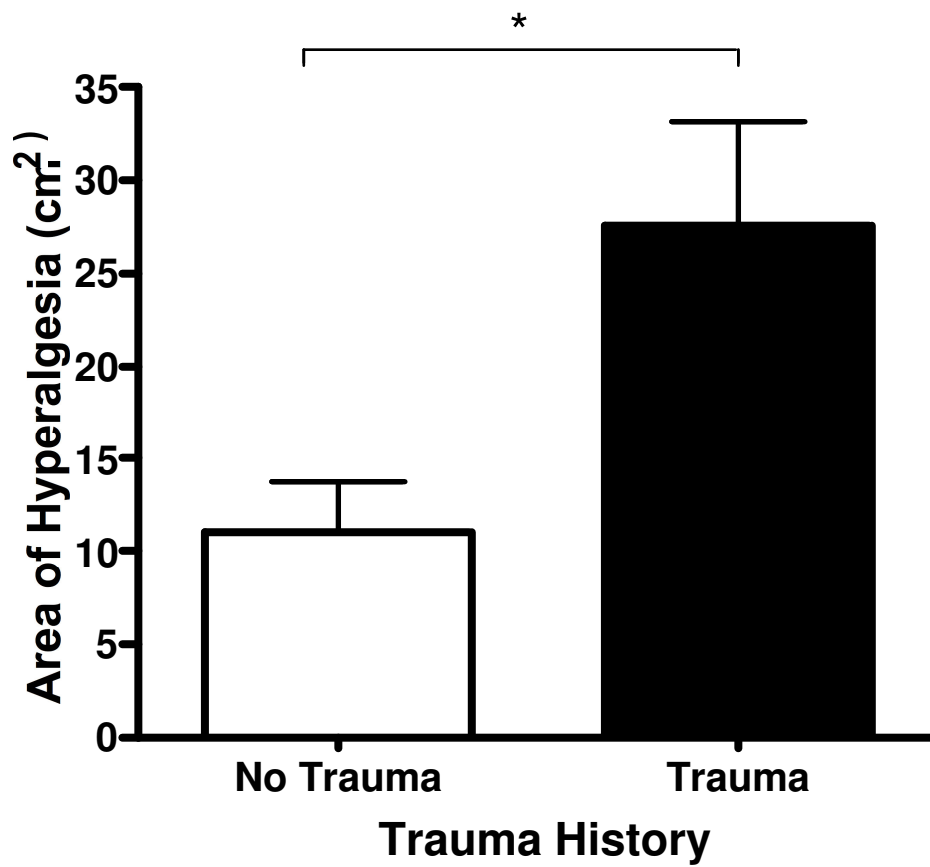


Figure 10. Area of Secondary Hyperalgesia by Trauma History Group

## 8. EXPERIMENT 2 DISCUSSION

The purpose of experiment 2 was to determine whether trauma history impacted capsaicin pain under neutral conditions. Exposure to traumatic life events contributes to the development of depression, anxiety, and affect dysregulation, which in turn may amplify the affective experience of pain, even under basal conditions (for reviews, see Meagher 2002, Frewen and Lanius, 2006a). It was thus hypothesized that trauma history women would show increased baseline negative affect, and that they would also show increased reports of pain intensity, unpleasantness and increased area of secondary hyperalgesia, even under neutral conditions. In addition, PTSD is associated with increased elevated tonic or baseline HR, and HPA axis dysregulation. Given this, I hypothesized that no trauma and trauma history groups (see Orr, Metzger and Pittman, 2002) would show baseline differences in heart rate and SCL.

CES-D scores indicated the trauma history group reported significantly higher negative affect at baseline. No between-groups baseline differences in physiological reactivity were detected, and this was consistent with findings from our previous study and not entirely unexpected given that our participants did not actually have PTSD (Creech, Grimes & Meagher, under review).

Participants did differ in VAS ratings of their pain experience. Specifically, while the no trauma group showed a normal progression in VAS intensity and unpleasantness ratings to capsaicin (starting low and ending high as the capsaicin takes effect), the trauma history group did the opposite (starting high and ending low). Change scores for

intensity and unpleasantness ratings at time 1 and time 5 showed a significant difference between no trauma women (an overall increase in pain) and trauma women (an overall decrease in pain). Finally, the trauma group showed a significantly increased area of secondary hyperalgesia when compared to the no trauma group. Thus, women with trauma history initially experience greater levels of capsaicin-induced spontaneous pain which declines over time to be equivalent to that in no trauma controls, at the same that they show increased secondary hyperalgesia.

While some experiments have shown decreased pain sensitivity in PTSD participants under experimental conditions (Geuze 2007; Van der kolk et al. 1999), prevalence estimates point towards increased clinical complaints (Klossika 2006; Smith et al. 2002, Asmundson et al. 2002; Beckham et al. 1997). One explanation for this difference may be that in individuals with a significant trauma history, descending pain inhibitory mechanisms are dysregulated. Considerable evidence indicates that brainstem mechanisms tonically inhibit incoming nociceptive signals at the level of the spinal cord (see Ren & Dubner 1999), through the release of endogenous opioids, norepinephrine, and serotonin. For example, Anderson et al. (2002) has provided evidence that endogenous opioids normally inhibit capsaicin-induced pain. Specifically, they showed that administration of an opioid receptor antagonist, naltrexone, increases capsaicin pain when compared to a placebo group. This suggests that acute pain is actively suppressed by endogenous opioid receptor activation. Thus, it is tempting to speculate that the heightened M-VAS pain ratings observed in trauma history participants relative to no-trauma controls on M-VAS early in the session may reflect that this descending

inhibitory system is not working normally in the trauma history subjects. The descending inhibitory system does not appear to engage until much later, resulting in more intense initial pain transmission, followed by inhibition 25 minutes later.

Taken together, these data indicate that a lifetime history of trauma is sufficient to alter pain regulation, and that this occurs even under neutral environmental conditions in which there are no trauma reminders. However, the results also indicate that opposing pain processes may be at work, given that the trauma group showed increased secondary hyperalgesia but decreasing spontaneous pain ratings over time. The opposing results in this study may be due in part to the different processes underlying spontaneous pain ratings and secondary hyperalgesia and thus the mechanisms mediating and modulating spontaneous pain/primary hyperalgesia inhibition and those regulating allodynia/secondary hyperalgesia could be distinct. For example, spontaneous pain likely reflects pain from the site of capsaicin application; therefore it could reflect both primary and secondary hyperalgesic processes. In contrast, the region of allodynia is exclusively secondary hyperalgesia and centrally mediated (Ziegler et al. 1999; Klede et al. 2003).. Thus, the diminishing pain ratings at the primary site may occur due to the higher state of arousal seen in the trauma history subjects, but at the same time that tactile sensitivity (secondary hyperalgesia) is enhanced to promote protection of the site and recuperation. The former may be related to stress-induced analgesia at the primary site mediated by endogenous opioids (Anderson et al., 2002), while the latter may be related to stress-induced increases in circulating levels of norepinephrine (Drummond 2001).

## 9. EXPERIMENT 3 METHOD

The goal of experiment 3 was to determine whether written emotional disclosure of trauma on pain would differentially affect pain tested on day 4 (day after writing) versus at a one-month follow-up. I hypothesized that while writing about trauma would elicit negative affect and increased pain sensitivity immediately after writing, pain sensitivity would decline at a one-month follow-up. To test this hypothesis, participants were asked to write for three days, and multiple pain methods were assessed at baseline, after writing and at a one-month follow-up. At baseline, I expected to observe a pre-existing difference in thermal pain sensitivity between trauma history and no trauma history participants. Specifically, trauma history participants were expected to show reduced thermal pain thresholds compared to no-trauma history participants at baseline.

It was also hypothesized that immediately after the first day of writing self-report and physiological measures would indicate high levels of stress and negative affect for participants in the trauma writing group (experimental group), and these participants were expected to show increased pain sensitivity relative to controls. I expected this to be reduced after the third day of writing, and even further reduced at the one-month follow-up. However, for high trauma participants, I expected to observe analgesia to capsaicin due to a preexisting autonomic hyperreactivity that is engaged by written emotional disclosure of trauma (and potentially by the pain testing itself).

### *Procedure*

The study was conducted in one-month modules consisting of three consecutive days of writing, a day 4 lab visit, and a one-month follow-up (see Figure 11 below for experiment timeline). To assess possible long-term changes in pain sensitivity, both acute and long-term effects of writing on pain, depression, and PTSD symptoms were measured. Participants wrote for 20 min, three days in a row about a traumatic or neutral topic (randomly assigned but consistent across all three days). Some participants also completed radiant heat pain tests immediately before writing on day one, on day 4, and at the one-month follow-up; although I intended to administer this test to all participants, we were unable to do so due to technical problems.

In addition, participants were randomly assigned to either receive the topical capsaicin test after writing on day four or at the one-month follow-up. The topical capsaicin test was included as a between subjects variable based on previous research conducted by our laboratory that indicates repeated measures using a suprathreshold model of pain like capsaicin may interfere with emotion induction by creating a conditioned contextual fear. Thus the day 4 and one-month conditions were designed so that capsaicin testing could take place at the end of writing and one-month later, and both groups could be tested under equivalent conditions.

Both subjective (Self-Assessment Manikin - SAM, Positive Affect Negative Affect Schedule – PANAS, Visual Analogue Scale - VAS) and physiological (heart rate, galvanic skin response) indicators of affect, stress and arousal were collected before and after each writing session and throughout each pain testing session.



		Day 1	Days 1, 2 & 3	Day 3	Day 3 or Follow-up		
Prescreening	Trauma History	Baseline Measures 1. Self Report 2. Thermal Pain	Trauma Writing	Thermal Pain Testing	Capsaicin Spontaneous Pain Ratings	Capsaicin Tactile Allodynia Test	Follow-up Self-Report measures
			Neutral Writing				
	No Trauma History		Trauma Writing				
			Neutral Writing				

Figure 11. Experiment 3 Timeline

### *Participants*

In order to obtain satisfactory power of 0.80 for  $\alpha = .05$  based on the effect size of 0.40 and degrees of freedom of four (based on data from experiment 1), Pearson-Hartley power charts estimates indicated 15-20 participants will be needed per cell, for a total of 120 participants.

Table 2

### *Number of participants needed per group*

Condition	<i>Capsaicin</i>	<i>Trauma Writing</i>	<i>Neutral writing</i>
Trauma History	Acute	15	15
	Follow-up	15	15
No Trauma History	Acute	15	15
	Follow-up	15	15

Following the previously described methodology, study participants were obtained through departmentally organized prescreening sessions. Participants were eligible for the study if they were in good health, not taking any psychotropic medications, and they had a trauma history score that two standard deviations above the population mean on the CTQ (for the trauma group), or a trauma history score that is at zero (for the no trauma control group).

## 10. EXPERIMENT 3 RESULTS

### *Self-Report Ratings for Writing Procedure*

In order to determine whether writing impacted self-reported negative affect and arousal, SAM and PANAS ratings were collected before and after writing on days 1-3 of the study. SAM and PANAS ratings both before and after writing were analyzed using ANOVA, with trauma group (No trauma history or Trauma history) and writing topic (Neutral or trauma) as between subjects variables and SAM or PANAS ratings after writing on day 1, day 2 and day 3 as a within subject variable. As expected a significant main effect of writing topic emerged for all three self-report measures, indicating that overall, participants who wrote about the trauma topic rated their mood after writing as more negative (PANAS)  $F(1, 158) = 7.586, p < .001$ , more unpleasant (SAM - Valence)  $F(1, 166) = , p < .001$  and more aroused (SAM - Arousal)  $F(1, 166) = 8.094, p < .01$ . These effects are shown below in Figure 12.a for the SAM data and Figure 12.b for the PANAS data. All other analyses were not significant,  $p > .05$ .

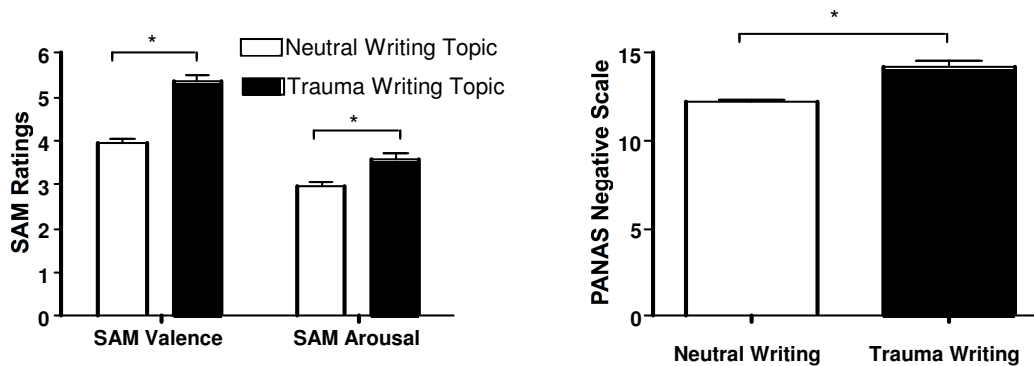


Figure 12. SAM Arousal, SAM Valence and PANAS Ratings for Writing

#### *Physiological Changes Before and After Writing*

Heart rate (BPM) and skin conductance levels were collected before and after writing on days 1-3 of the Experiment and after the 20 min habituation period on days 4 and 5. The two groups significantly differed in baseline heart rate,  $F(1, 58) = 4.02$ ,  $p < .05$ , with the trauma group recording significantly fewer baseline BPM compared to the no trauma group (shown in panel a of Figure 13). The two groups also approached a significant difference in baseline SCL,  $F(1, 42) = 2.935$ ,  $p = .09$  (depicted in panel b, Figure 13). In this case, the trauma group showed higher baseline SCL, however, this difference did not achieve significance and thus is an inconclusive finding.

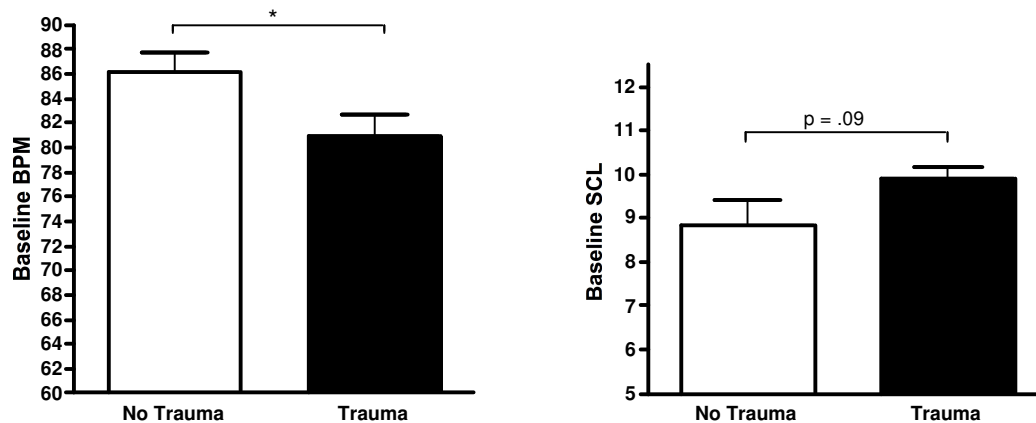


Figure 13. Baseline BPM and SCL by Trauma group

In order to examine the physiological effects of writing and whether this changed over the three days of writing, each participant's BPM or average SCL score was subtracted from their day one baseline and a change score was obtained and analyzed. Previous studies have already demonstrated the impact of writing is greatest on day 1 and dissipates on day 2 and 3, presumably as subjects habituate to the procedure (Sloan 2004, 2005). Our results were consistent with these findings, and a significant main effect of writing topic on BPM was only obtained for day 1. Results indicated a significant difference in change in BPM from baseline between the neutral and trauma writing groups,  $F(1, 67) = 27.808, p < .001$ . As shown below in Figure 14, individuals who wrote about the trauma topic had an increase in BPM after writing, while neutral writers had a decrease in BPM after writing. All other analyses were not significant,  $p > .05$ .

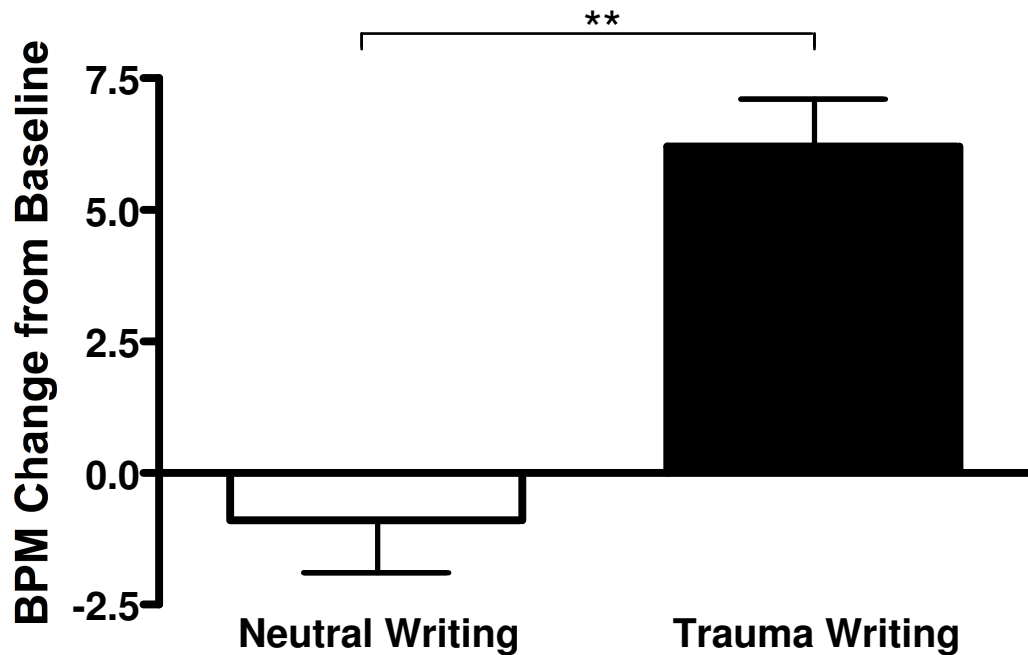


Figure 14. BPM Change from Baseline after Writing

### *Radiant Heat Testing*

Following previously used methodology, each participant completed one practice radiant heat test, and the latencies from the next two tests were averaged. To determine whether there was a baseline difference in radiant heat withdrawal latencies between no trauma history and trauma history individuals, average withdrawal latencies on day one of the study (baseline; prior to writing), were entered into ANOVA with trauma history as a between subject variable. Using this method, a significant main effect of trauma history emerged,  $F(1, 39) = 4.868, p < .05$ . As shown in Figure 15.a, individuals in the

trauma history group had significantly shorter withdrawal latencies than those without a trauma history. This finding is consistent with data from prior studies in which we found preexisting differences in tourniquet tolerance based on trauma history, however it should be noted that we did not find a preexisting difference on radiant heat latencies in that study (Creech, Grimes & Meagher under review).

In order to determine whether writing had an effect on day 4 radiant heat withdrawal latencies, these were entered into an ANOVA with trauma history and writing topic as between subjects variables, and day 1 and day 4 withdrawal latencies as a repeated measurement. As shown below in Figure 15.b, a significant interaction effect between day of testing (1 or 4) and trauma history emerged,  $F(1, 36) = 4.011, p = .05$ . *Post hoc* means comparisons indicated that within the no trauma history group, withdrawal latencies were significantly shorter on day four than they were on day one,  $F(1,17) = 12.38, p < .01$ . Thus, within the no trauma history group, regardless of writing topic, day four latencies were significantly shorter than they were on day one, which suggests a possible conditioned contextual fear. A second means comparison between day 1 and day 4 latencies within the trauma history group approached significance, indicating day four withdrawal latencies were shorter than day one latencies,  $F(1, 19) = 3.86, p = .06$ .

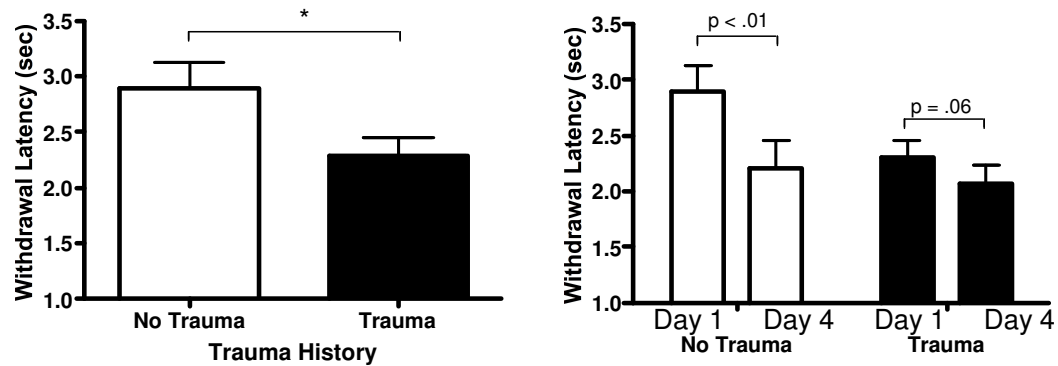


Figure 15. Baseline and Repeated Measures Radiant Heat Latencies

#### *M-VAS Pain Intensity Ratings*

Pain intensity ratings were collected every 5 min for the first 30 min of capsaicin application. Ratings were analyzed using ANOVA with trauma group (no trauma history or trauma history), writing topic (neutral or trauma), and capsaicin test day (day 4 or one month later) as between subjects variables and VAS intensity ratings over time as a repeated measurement. Although there were no main effects of capsaicin test day, writing topic, or trauma group (all  $p$ 's  $> .05$ ), a significant interaction between intensity ratings over time and trauma group emerged,  $F(4,312) = 4.248$ ,  $p < .01$  (shown below in Figure 16, panel a). *Post hoc* mean comparisons indicated that the trauma history group rated their pain as significantly more intense than the no trauma group,  $p < .05$ . All other comparisons were not significant,  $p > .05$ .



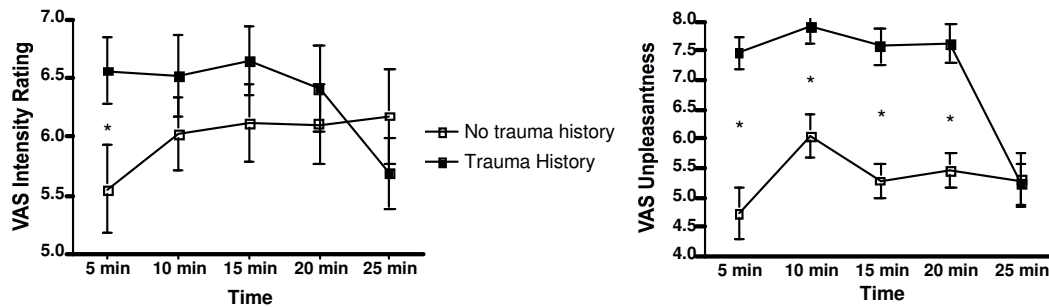


Figure 16. VAS Intensity and Unpleasantness Ratings Over Time

#### *M-VAS Pain Unpleasantness Ratings*

Pain unpleasantness ratings were also collected every 5 min for the first 30 min of capsaicin application. Ratings were analyzed using ANOVA with trauma group (no trauma history or trauma history), writing topic (neutral or trauma), and capsaicin test day (day 4 or one month later) as between subjects variables and VAS unpleasantness ratings over time as a repeated measurement. Using this method, a main effect of trauma group emerged  $F(1, 76) = 18.76$ ,  $p < .001$ , indicating that participants in the trauma history group ( $M = 7.118$ ,  $SEM = .150$ ) rated their unpleasantness as, on average, significantly higher than those in the no trauma history group ( $M = 5.327$ ,  $SEM = .167$ ). Similar to the intensity ratings, a significant interaction between unpleasantness ratings over time and trauma group emerged,  $F(4, 304) = 11.925$ ,  $p < .001$ . As depicted above in Figure 16.b, *post hoc* means comparisons indicated significant differences in unpleasantness ratings at each of the 5, 10, 15 and 20 min time points, all  $p$ 's  $< .001$ ,

indicating the trauma group rated their pain unpleasantness as significantly higher than the no trauma group at every time point except for the final 25 minute rating ( $p = .9$ ).

#### *Short Term Study Pain Testing Results*

To examine the impact of our independent variables on area of secondary hyperalgesia, an ANOVA was used with trauma history and writing topic (neutral or trauma), as between-subject variables. As is depicted in Figure 17, the area of secondary hyperalgesia was significantly smaller in the no trauma group than it was in the trauma history group. This observation was confirmed by a significant main effect of trauma history, [ $F(1, 34) = 8.329, p < .01$ ]. This effect is similar to the significant finding of increased area of secondary hyperalgesia in the trauma group from Experiment 2. There were no main effects of writing topic, all  $p$ 's  $> .05$ . Although the interaction between trauma history and writing topic was not significant,  $F(1,34) = .615, p = .43$ , post hoc testing revealed a significant difference between trauma and no trauma history participants who wrote about the trauma topic,  $p \leq .05$ . Taken together, these results suggest that although trauma writing did not significantly amplify central sensitization in the short term, trauma history clearly impacted central sensitization.

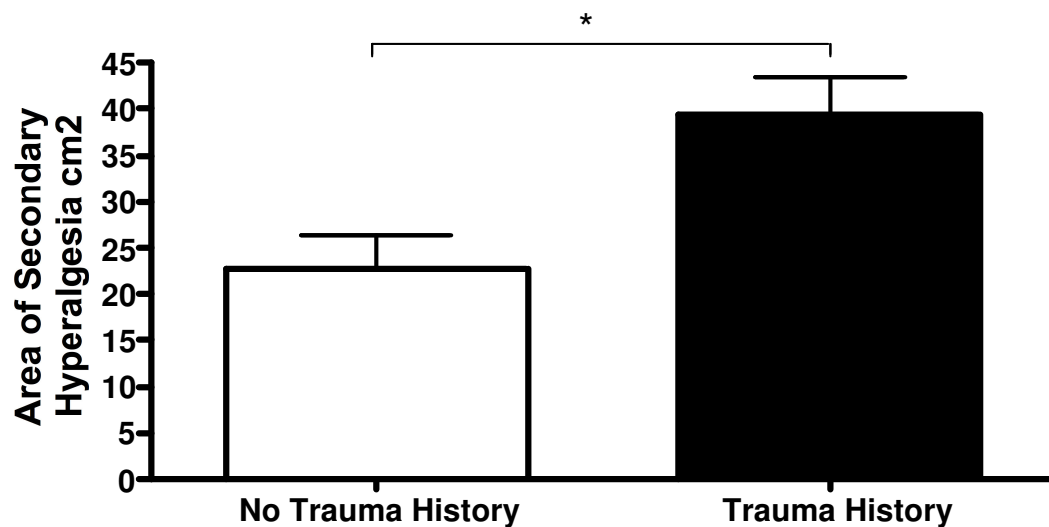


Figure 17. Area of Secondary Hyperalgesia by Trauma History

#### *Long Term Study Pain Testing Results*

As depicted below in Figure 18, area was again analyzed using trauma history and writing topic (neutral or trauma), as between-subject variables. Results indicated that by day 5 the main effect of trauma history was no longer significant,  $F(1, 43) = .443$ ,  $p > .05$ . Importantly, the interaction between trauma group and writing topic approached significance,  $F(1, 43) = 3.124$ ,  $p = .07$ . *Post hoc* means comparisons indicated that the interaction was driven by a significant difference between trauma and no trauma history participants within the neutral writing group,  $p = .05$ . Specifically, individuals with a trauma history who wrote about the neutral topic showed a significantly larger area of secondary hyperalgesia than the no trauma history group, which is similar to the short-term pattern of trauma-induced sensitization observed at day 4. Importantly, the impact of trauma history was no longer apparent for trauma writers, suggesting that writing

about trauma reversed the impact of trauma history detected in the short-term analysis, while neutral writing did not (see Figure 18).

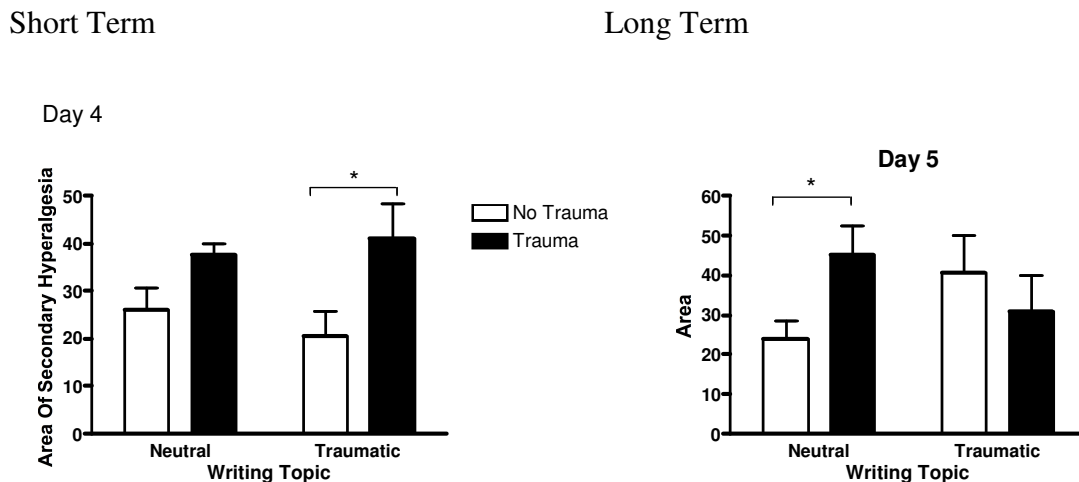


Figure 18. Area of Secondary Hyperalgesia Interactions

To further analyze these data, we conducted an ANOVA entering capsaicin test day, trauma group, and writing topic as between group variables. There was an interaction between capsaicin test day, trauma group, and writing topic which approached significance,  $F(1, 77) = 3.323$ ,  $p = .07$ . *Post hoc* means comparisons indicated the same pattern of group mean differences observed in the separate ANOVAs conducted on day 4 and day 5 area. In addition, the no-trauma history participants who wrote about trauma on day 5 showed an increased area when compared to no-trauma history participants who wrote about trauma on day 4,  $p < .03$ . No other differences were significant.

To determine whether there was a between-groups difference in pain intensity within the area of hyperalgesia, each participant's pain intensity ratings from the site of injury to the boundary of the area of secondary hyperalgesia was calculated and averaged. Although the trauma group seemed to display, on average, lower pain ratings, these differences were not significant [ $F(1,31) = .33, p = .57$ ].

## 11. EXPERIMENT 3 DISCUSSION

Experiment 3 had several goals. Our previous studies had all used one day of writing followed by pain testing in order to determine whether writing was a sufficient affect induction procedure. Most research using writing has participants write over 3-4 days followed by measurement of health outcomes about one month later. Our first goal was thus to model these studies by extending the writing period to 3 days and using laboratory pain as an outcome measure. Using a between subjects design, we tested capsaicin pain on day 4 to analyze the short-term impact of writing about trauma, and we also tested capsaicin pain one month later in another group to examine the long-term impact of trauma writing. The second goal of the study was to replicate previous findings in which increased thermal pain was observed immediately after writing about trauma.

Self-reported affect and arousal were collected before and after writing on all 3 days, and as expected, results indicated that participants who wrote about the trauma topic rated their mood after writing as more negative (PANAS), more unpleasant (SAM) and more aroused (SAM) than the neutral writing group. Physiological data confirmed an increase in heart rate after writing about trauma on day one, but not on days 2 or 3, which is consistent with data from other studies showing the impact of writing about trauma declines over time (Sloan & Marx, 2004a).

Changes in acute pain were tested using the radiant heat device, and participants completed this test before writing on day 1 to obtain baseline pain thresholds and on day

4 to determine whether the writing procedure impacted pain thresholds. Importantly, results showed a baseline difference between no trauma history and trauma history women; women who were positive for a lifetime history of trauma showed significantly shorter baseline pain thresholds than no trauma history women. This is consistent with data obtained in study 2 in which trauma history women showed a significantly increased area of secondary hyperalgesia. However, in a previous study there was no baseline difference in radiant heat latencies due to trauma history (Creech, Grimes & Meagher under review). This difference may be due to use of a more specific measure of trauma history used in this study.

On day 4, pain thresholds were significantly shorter than day one thresholds in the no trauma history group, suggesting that repeated testing resulted in a sensitization effect. This could be attributed to anxiety regarding repeated testing which increased pain reactivity, and is similar to accounts of the impact of anxiety on radiant heat latencies from previous studies (Rhudy & Meagher 2000). A similar trend was also observed in the trauma history group. Taken together, this suggests that regardless of writing topic, repeated pain testing sensitized the participants to radiant heat. However it should be noted that changes in latencies are difficult to detect due to an overall floor effect with this test.

We looked at the short-term and long-term effects of writing on spontaneous pain ratings and area of secondary hyperalgesia using a between subjects design. The short-term group was administered capsaicin the day after writing, while the long-term group was administered capsaicin one-month after writing. Spontaneous pain intensity and

unpleasantness ratings during the capsaicin test followed the same pattern observed in Experiment 2, and no main effect of writing topic or difference between the short and long-term groups emerged. Trauma history participants rated their pain intensity as significantly more intense at the 5-min time point, and tended to show a decline in intensity over time, while no trauma history women showed increasing intensity over time. The trauma history group also rated their pain unpleasantness as significantly higher than the no trauma group at the 5, 10, 15 and 20 min time points. These results suggest that preexisting history of trauma is an important variable in influencing spontaneous pain, and that it overpowers the impact of written emotional disclosure. We propose that endogenous pain inhibitory mechanisms are not functioning properly in trauma history individuals.

Analyses of area of secondary hyperalgesia in the short-term group revealed a significant main effect of trauma history, as well as a significant difference between no trauma and trauma history participants who wrote about the trauma topic. These findings supported our hypothesis that trauma history would increase pain in the short-term study. This effect was reversed for the trauma history participants who wrote about the trauma topic in the long-term study, but remained significant for neutral writers in the long-term study. This indicates that writing about trauma reversed the effects of trauma history on secondary hyperalgesia when tested one month after writing.



## 12. GENERAL DISCUSSION AND SUMMARY

The present series of experiments examined the effects of written emotional disclosure on capsaicin-induced pain. Results indicated radiant heat withdrawal latencies, spontaneous pain ratings, and area of secondary hyperalgesia were all significantly increased when participants had a history of traumatic experiences. This is evidence that trauma history is sufficient to alter pain regulatory mechanisms, and this may be attributable to the chronic negative affective state induced by trauma history and sensitization of shared circuits involved in both pain and emotion. CES-D data collected in study support this notion as the trauma history group entered the study with significantly higher emotional distress.

One pathway through which emotions impact pain is a circuit linking the amygdala (AMG) with the periaqueductal grey (PAG) and rostral ventral medulla (RVM) regions of the brain with the dorsal horn region of the spinal cord (Crown et al., 2000; McLemore et al., 1999; Rhudy & Meagher 2001, Rosen & Shulkin, 1998, Fields and Basbaum 1994). In addition to transmitting pain signals, this ascending pain pathway can also directly activate structures involved in emotion (Rhudy & Meagher 2001). This AMG-PAG-RVM circuit is also involved in descending pain modulation, through which emotion could influence pain processing at the level of the spinal cord (Fields and Basbaum 1999).

Imaging and other studies have provided evidence for overlap between structures involved in pain and structures involved in emotion and cognition (Ranville, 2002). For

example, Damasio and colleagues have shown activation in many areas (brainstem nuclei, amygdala, and hypothalamus) involved in pain during subjective experience of fear, sadness, anger, and happiness (2000). Others have shown activation in the basal forebrain, extended amygdala, nucleus accumbens, and PAG in response to tonic heat pain (Becerra et al., 2001). Thus considering the shared biology of pain and emotion, we have proposed that the relationship between PTSD and pain is bi-directional.

Exposure to trauma may change the affective experience of pain through the development of depression, anxiety, and affect dysregulation after the traumatic event – even if these do not reach clinically significant levels (see Meagher et al., 2002; Frewen & Lanius, 2006a). For example, McEwen's concept of allostatic load predicts both physiological and psychological consequences when the body has had to adapt to stress too often or has developed dysfunctional psychobiological regulation of stress (McEwen 1998, McEwen & Wingfeld 2003; Korte et al 2005).

If this model is extended to consider the impact of repeated stressors that are likely to occur, it is easy to see how the emotional and physiological consequences of stress might be exacerbated over time. Support for this view comes from clinical studies linking trauma history to chronic pain (e.g., Asmundson et al., 2002; Beckman et al., 1997; Drossman et al., 1990; Lampe et al., 2000; Scarinci et al., 1994; Leserman et al., 1996; 2006; Walker et al., 1993; 1999; Walling et al., 1994) and our results are consistent with these prior accounts of increased chronic pain in trauma history groups. We propose that the tonic negative affective state induced by trauma disrupts central and descending pain regulatory mechanisms, which may lead to increased basal pain sensitivity. We propose

that pain may be enhanced due to a stress-related increase in pro-inflammatory cytokines, and this increase is likely exacerbated by alterations in the hormonal system designed to inhibit further release of cytokines. Thus, if this effect is a lasting change and occurs in response to repeated stressors it is easy to see how stress might magnify pain, and vice versa.

Empirical evidence exists to support the notion that alterations in these systems would impact pain. Current research indicates glial activation and a corresponding release of proinflammatory cytokines is involved in pain modulation and hyperalgesia (Frank, Maier & Watkins, 2005; Watkins & Maier 1998). Specifically, researchers have suggested that a stress-induced increase in cytokine levels would amplify pain messages sent from the spinal cord to the brain by decreasing the threshold for pain pathway activation by a noxious stimulus. (Frank, Maier & Watkins, 2005; Watkins & Maier 1998).

Research has also indicated that PTSD and negative affective states such as depression have been linked to increased levels of cytokines (Irwin, 2002). A recent meta-analysis of 293 studies on stress and immunity indicated that both acute stress challenges and naturalistic stress challenges overall led to increases in proinflammatory cytokines in humans (Segerstrom & Miller, 2004). Further synthesis and release of proinflammatory cytokines is normally inhibited by elevated glucocorticoids (Bertini, et al 1988, Butler et al., 1989, Parant et. al, 1991, Johnson et al. 2002). However, HPA axis dysregulation is an accepted psychobiological consequence of PTSD. To the extent that stressful life events interfere with this chain of events by altering effectiveness of

this feedback system, further release of cytokines may not be inhibited, thus interfering with normal pain processing.

Although the current study did not examine the immunological variables discussed above, results do suggest that preexisting history of trauma is an important variable in influencing spontaneous pain, and that it overpowers the impact of written emotional disclosure. In experiment 3, both spontaneous pain ratings to capsaicin and area of secondary hyperalgesia were tested in two groups (short term and long-term). Results indicated that trauma history participants rated their pain intensity as significantly more intense than the no trauma group at the 5-min time point, and tended to show a decline in intensity over time. The trauma history group also rated their pain unpleasantness as significantly higher than the no trauma group at the 5, 10, 15 and 20 min time points.

Results for secondary hyperalgesia indicated trauma history increased the area of secondary hyperalgesia in the short-term group. However, when the long-term group was tested one-month later, written emotional disclosure reversed this process, and the effect of trauma history was only apparent in participants who wrote about the neutral topic. This suggests that while written emotional disclosure may not have countered the impact of trauma history on spontaneous pain ratings, it can reverse the trauma-induced sensitization of allodynia, which is mediated by central sensitization.

There have been relatively few experimental studies investigating the impact of trauma on pain, but most have observed analgesia after exposure to trauma cues (Geuze et al., 2007; Nishith et al., 2002; Van der kolk et al. 1999, Pitman et al., 1990). Pitman studied the impact of exposure to a combat video on pain in veterans with and without

PTSD (1990). In addition to increased negative affect and arousal, the veterans with PTSD showed a naloxone-reversible decrease in pain intensity ratings after viewing a combat video, whereas the veterans without PTSD showed a trend toward increased pain. Nishith and colleagues (2002) also reported that cold pain distress ratings were negatively correlated with the level of PTSD symptoms and hyperarousal in battered women. It is likely that stress-induced hypoalgesia (analgesia) was not apparent in our trauma-history participants because of the delay between the end of writing and post-writing pain testing (24 hours). However, we intentionally added a delay in the present study in order to compare the short-term and long-term effects of written emotional disclosure under equivalent test conditions. To examine whether written emotional disclosure induces an acute stress-induced hypoalgesia, future studies would need to test the effects of writing immediately following capsaicin application.

Other studies have also found improvements after writing in clinical populations; (Smyth et al., 1999, Kelley et al., 1997), and two studies have also found improvements after emotional disclosure in patients with fibromyalgia both 10 weeks (Wetherell et al 2005) and 4 months (Broderick et al. 2005) after writing. Although our participants had no preexisting health problems, it is possible that the effects observed in this study would have been further amplified in a chronic pain group. For example, overtime chronic pain causes changes in descending pain regulation and CNS mechanisms, so interventions that alter affect regulation may have more of an impact against a background of chronic pain. However, written emotional disclosure's ability to reverse the effects of trauma history on central sensitization is sufficient to warrant further

testing of writing and other emotion-processing techniques as interventions for chronic pain.

The present findings suggest that exposure to trauma dysregulates endogenous pain modulatory systems, resulting in enhanced spontaneous pain and central sensitization. Furthermore, our findings suggest that written emotional disclosure may lead to long-term changes in pain modulatory pathways that regulate central sensitization, without altering systems that regulate spontaneous pain. It is possible that repeated exposure to traumatic stimuli during written emotional disclosure is resulting in a desensitization of the amygdala and other limbic structures that are known to regulate both pain and emotion. To test this hypothesis, future fMRI studies should examine whether the long-term decreases in trauma related central sensitization are accompanied by corresponding changes in the pattern of activation of these structures.

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## APPENDIX

*Figure Captions*

Figure 1. Diagram of the capsaicin application site and grid drawn on forearm.

Figure 2. Timeline of procedures and method of experiment 1.

Figure 3a. SAM valence ratings taken before writing, at 10 min and at 20 min for neutral and trauma writers. Participant valence ratings were significantly different at the ten-minute time point between neutral and trauma writers.

Figure 3b. SAM arousal ratings taken before writing, at 10 min and at 20 min for neutral and trauma writers. Participant arousal ratings were significantly different at the ten-minute time point between neutral and trauma writers.

Figure 4. Change in BPM from baseline after 20 minutes of writing. Neutral writers displayed a decrease in heart rate while trauma writers had an increase in heart rate.

Figure 5. The main effect of writing topic on M-Vas ratings of pain intensity and unpleasantness. Participants who wrote about the trauma topic rated their pain as significantly less intense and less unpleasant than neutral writers.

Figure 6. The interaction effect of writing topic and pain unpleasantness VAS ratings over time during the capsaicin test. Trauma writers rated their pain as significantly less unpleasant at the 28 min and 30 min time points.

Figure 7. Timeline of procedures and method of experiment 2.

Figure 8a. VAS pain intensity ratings taken every 5 min. during the capsaicin test for both no-trauma and trauma history participants.

Figure 8b. Change scores for VAS pain intensity ratings between the 5 min and 25 min time points. The trauma history group had an overall decrease in pain intensity while the no-trauma history group had an increase.

Figure 9a. VAS unpleasantness ratings taken every 5 min. during the capsaicin test for both no-trauma and trauma history participants.

Figure 9b. Change scores for VAS pain unpleasantness ratings between the 5 min and 25 min time points. The trauma history group had an overall decrease in pain unpleasantness while the no-trauma history group had an increase.

Figure 10. The area of secondary hyperalgesia by trauma history group. Area was significantly smaller in the no trauma history group.

Figure11. Timeline of procedures and method of experiment 3.

Figure12a. Participant ratings of valence and arousal on the SAM after writing. Participants who wrote about the trauma topic rated their arousal and valence levels as significantly higher than participants who wrote about the neutral topic.

Figure12b. Participant mood ratings on the PANAS after writing. Ratings were significantly more negative for the trauma writing group.

Figure13a. BPM taken at baseline for both the no trauma and the trauma-history groups.

Figure13b. SCL taken at baseline for both the no trauma and the trauma-history groups.

Figure14. Change in BPM between baseline and after writing. BPM increased significantly for trauma writers.

Figure15a.. Baseline radiant heat withdrawal latencies in seconds taken on day 1. Latencies were significantly shorter for the trauma history group.

Figure15b.. Baseline radiant heat withdrawal latencies in seconds taken on day 1 compared to withdrawal latencies on day 4.

Figure16a. VAS pain intensity ratings taken every 5 min. during the capsaicin test for both no-trauma and trauma history participants.

Figure16b. VAS pain unpleasantness ratings taken every 5 min. during the capsaicin test for both no-trauma and trauma history participants.

Figure17. . Area of secondary hyperalgesia on day 4 of the short-term study. The area was significantly greater in the trauma history group.

Figure18. The area secondary hyperalgesia on day 4 of the short-term study by trauma history and writing topic, and the area secondary hyperalgesia on day 5 of the long-term study by trauma history and writing topic.

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