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Examination of a Biopsychosocial Model for the Relationship between Posttraumatic Stress Disorder and Chronic Pain

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EXAMINATION OF A BIOPSYCHOSOCIAL MODEL FOR THE RELATIONSHIP BETWEEN POSTTRAUMATIC STRESS DISORDER AND CHRONIC PAIN

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Thesis Advisor: Sandra T. Sigmon, Ph.D.

An Abstract of the Thesis Presented in Partial
Fulfillment of the Requirements for the
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High rates of comorbidity have been reported between PTSD and musculoskeletal pain (e.g., Asmundson & Hadjistavropolous, 2006; Asmundson et al., 1998). Comorbid PTSD and chronic pain have been associated with elevated levels of affective distress, greater perceptions of pain, interference in daily activities, and high rates of disability (Otis et al., 2003; Sherman et al., 2000). Overall, comorbid conditions of PTSD and chronic pain are associated with large personal costs for the individual and economic costs for society.

The triple vulnerability model was originally proposed to account for anxiety symptoms in general, and it was later applied to the specific development of PTSD (Barlow, 2000; Barlow, 2002; Keane & Barlow, 2002). Otis and colleagues (2003) further proposed that the triple vulnerability model may account for the relationship between PTSD and chronic pain. According to the triple vulnerability model, individuals must present with a generalized biological, generalized psychological, and a specific psychological vulnerability for either of these conditions to develop (Keane & Barlow, 2002; Otis et al., 2003).

In the current study, aspects of the triple vulnerability model were examined within the following groups of women: women who have PTSD without chronic pain (*n* = 11), women who have musculoskeletal pain without PTSD (*n* = 10), women with both PTSD and musculoskeletal pain (*n* = 10), and women without PTSD and chronic pain (*n* = 15). Cortisol reactivity and anxious mood were assessed before and after the Trier Social Stress Task (TSST). Participants also completed questionnaires to assess for other potential indicators of the triple vulnerability model.

Results indicate that: 1) the roles of generalized biological, generalized psychological, and specific psychological vulnerabilities toward developing PTSD were supported; 2) limited findings supported the potential role of these vulnerabilities toward developing chronic pain; however, results of these measures were not similar to that of PTSD (e.g., family history of chronic pain); 3) it is not thought that PTSD and chronic pain are associated with the same vulnerabilities; 4) having a diagnosis of PTSD and chronic pain was associated with an increase in symptoms across many measures utilized in the current study.

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TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

Chapter 1

INTRODUCTION

Recently, there has been widespread interest among professionals in the behavioral medicine and medical communities regarding the relationship of chronic pain and anxiety. In particular, high comorbidity rates have been observed among individuals with chronic pain and posttraumatic stress disorder (PTSD). Among individuals who are seeking treatment for chronic pain, approximately 20% to 34% of these individuals are also thought to report high levels of PTSD symptomatology or have an actual PTSD diagnosis (Asmundson, Norton, Allerdings, Norton, & Larsen, 1998; Geisser, Roth, Bachman, & Eckert, 1996). Within the fibromyalgia population in particular, approximately 56% of individuals tend to report symptoms associated with PTSD, and approximately 21% report actual comorbid diagnoses of fibromyalgia and PTSD (Amir et al., 1997; Cohen et al., 2002; Sherman, Turk, & Okifuji, 2000). Thus, high levels of PTSD symptomatology are reported within the chronic pain population.

In studies that assess for the frequency of pain symptomatology within PTSD populations, even higher comorbidity rates have been found (Otis, Pincus, & Keane, 2006). Furthermore, physical symptoms of pain are thought to be the most commonly reported symptoms within the PTSD population, even when different types of traumatic events are taken into account (e.g., military combat, motor vehicle accident, or sexual assault; Asmundson, Coons, Taylor, & Katz, 2002). Among community samples, approximately 20% to 30% of individuals with PTSD are thought to develop different forms of chronic musculoskeletal pain (i.e., general pain conditions that influence muscles, ligaments, tendons, and bones), and such estimates are dramatically increased

within military populations (Asmundson, & Hadjistavropolous, 2006). For example, multiple regression analyses were used to assess for the effects of exposure to war-zone environments on PTSD and health within 109 female Vietnam veterans (Wolfe, Schnurr, Brown, and Furey, 1994). Results indicated that PTSD was the strongest predictor of poor health outcomes (Wolfe et al., 1994). In addition, both PTSD and fibromyalgia have been found to occur more frequently in female populations (Otis, Keane, & Kerns, 2003).

Comorbid conditions of PTSD and chronic pain are also associated with other difficulties (e.g., perceived levels of pain, affective distress, interference in daily activities, higher rates of disability; Otis et al., 2003). Within a sample of fibromyalgia patients who were seeking treatment, those who also reported PTSD symptomatology tended to report more significant amounts of pain, interference in their life, emotional distress, and inactivity. In addition, more than 85% of this subsample with both fibromyalgia and PTSD reported high levels of disability, as opposed to 50% of the subsample that did not report PTSD symptomatology (Sherman et al., 2000). In a female chronic pain population, comorbid reports of childhood sexual abuse were correlated with increased chances of receiving a surgical procedure (Finestone et al., 2000). Overall, comorbid conditions of PTSD and chronic pain are associated with higher pain intensity, psychological distress, disability, and medical utilization.

With such high rates of comorbidity, and subsequent costs to the individual and society, it is imperative to assess for potential mechanisms that account for such relationships. Although several models have been proposed to account for the relationship between chronic pain and PTSD, limited empirical research has been

conducted to provide support for these models. Even though the theoretical underpinnings for these models were based on empirical research to varying degrees, many of the models were originally proposed to account for another population and then later adapted to account for the relationship of PTSD and chronic pain (e.g., triple vulnerability model). Thus, no studies to date have tested these theoretical models in their entirety (Otis et al., 2003). Prior to discussing specific theories that account for the high comorbidity rates between PTSD and chronic pain, the conceptual basis for understanding both PTSD and chronic pain individually is presented.

Posttraumatic Stress Disorder: General Characteristics

Diagnostic Criteria

PTSD was first introduced as a formal psychiatric diagnosis in the third version of the Diagnostic and Statistical Manual in 1980 (DSM-III; American Psychiatric Association [APA], 1980). Since this time, the amount of literature that addresses various aspects of PTSD has increased significantly (Khouzam, Ghafoori, & Hierholzer, 2005). Currently, PTSD is characterized by the experience of a traumatic event, and associated feelings of intense helplessness and fear during the time of the event (DSM-IV-TR; APA, 2000). Traumatic events are characterized by the direct and personal exposure to an extremely distressing event, with subsequent responses involving personal perceptions of extreme fear, helplessness, or horror. In addition, PTSD is characterized by re-experiencing aspects of the traumatic event (e.g., flashbacks, nightmares regarding the traumatic event), avoidance of stimuli that serve as reminders of the traumatic event (e.g., people, places), arousal symptomatology (e.g., hypervigilance), and clinically significant levels of distress and interference. In terms of duration, symptoms must

persist for at least one month (APA, 2000). For many individuals, symptoms associated with PTSD tend to diminish within three months of experiencing the traumatic event; however, individuals who experience symptomatology for longer than three months tend to report fairly stable and chronic symptoms in the absence of any intervention (Resick & Calhoun, 2001).

Prevalence and Demographics

Rates of traumatic events are extremely high in the United States. Within the general population, approximately 90% of individuals will experience a traumatic event (e.g., car accidents, interpersonal violence, natural disaster, or a combat related event) during their lifetime (Breslau et al., 1998). Furthermore, data from the National Comorbidity Study (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995) revealed that approximately 20.4% of females and 8.1% of males go on to develop PTSD following the experience of a traumatic event. These researchers also found that approximately 7.8% of the general population will develop PTSD within their lifetime (Kessler et al., 1995). Within the Primary Care Anxiety Project (Bruce et al., 2001; Weisberg et al., 2002), a multi-site longitudinal study looking at anxiety disorders, approximately 12% of 1500 primary care patients met criteria for PTSD. It is clear that many individuals who experience a traumatic event will not go on to develop PTSD. Thus, the sole experience of a traumatic event is not necessarily adequate to account for the development of PTSD symptomatology. Other vulnerability factors must be present for the disorder to develop (Brewin, Andrews, & Valentine, 2000).

Although prevalence rates of PTSD may vary slightly across different populations, women have consistently been found to be twice as likely to develop PTSD

than men (e.g., Kessler et al., 1995; Khouzam et al, 2005). Many hypotheses have been proposed to account for these differential rates of PTSD. One hypothesis is that women may be more likely to develop PTSD as a result of the nature of their traumatic event (Schnurr, Friedman, & Bernardy, 2002). According to research on this topic, women report experiencing greater frequencies of being raped, sexual molestation, and childhood abuse (Schnurr et al., 2002). Within the National Comorbidity Study, men were more likely to report witnessing someone being badly injured or killed, being involved in a fire, flood, or natural disaster, being physically attacked, combat related experiences, or being threatened with a weapon, held captive, or kidnapped. Conversely, women were more likely to report being raped, sexually molested, neglected by their parents during childhood, and being physically abused during childhood (Kessler et al., 1995). Such qualitative differences in reports of traumatic events by men and women may influence prevalence rates of PTSD.

Although most PTSD research focuses on younger individuals, rates of interpersonal traumatic events are also reported within older populations of women. In a study that surveyed 842 women who were 60 years of age or older, rates of abuse were still highly prevalent (Fisher & Regan, 2006). In this sample, 47% of the women reported experiencing one or more types of abuse since they were 55 years of age. In particular, 45% of the women reported experiencing psychological/emotional abuse, 12% were threatened by somebody, 4% were physically abused and 3% were sexually abused (Fisher & Regan, 2006). These results lend support to the hypothesis that women may be more likely to develop PTSD due to the interpersonal nature of many traumatic events that they experience compared to men.

Conversely, other researchers argue that women still experience higher rates of PTSD after the type of trauma is taken into account (Wong & Yehuda, 2002). In addition, the researchers reported that the higher rates of PTSD in women cannot be accounted for by mere exposure to a traumatic event as men are just as likely to experience a traumatic event as women. Gender differences in PTSD development were found to be significantly greater if an individual's trauma occurred before they were 15 years old (Wong & Yehuda, 2002). Consequently, the age at which an individual experiences a traumatic event may play a large role in PTSD development.

Many other risk factors for PTSD have also been proposed and debated by various researchers (e.g., Khouzam et. al, 2005; Resick & Calhoun, 2001; Tanriverdi, Karaca, Unluhizarci, & Kelestimur, 2007; Wong & Yehuda, 2002). Resick and Calhoun (2001) proposed that women may be at a greater risk in the development of PTSD because of a biological predisposition in addition to the types of traumas that they experience. In particular, the hypothalamic-pituitary-adrenal axis (HPA axis) has gained increasing attention in accounting for PTSD symptomatology. In general, human responses to stress can be largely accounted for by HPA axis functioning (Tanriverdi et al., 2007). Other risk factors may include: lower educational attainment, pre-existing psychopathology, early conduct problems, family history of psychopathology, severity of initial reaction to the trauma, peritraumatic dissociation, poor social support, history of stress or abuse, and early separation from parents (Khouzam et. al, 2005; Wong & Yehuda, 2002). Overall, it is clear that biological, psychological, and social factors may all play a role in PTSD development.

As might be expected, PTSD is associated with a very high cost to society. Not only are individuals with PTSD frequently seen within mental health care settings (Khouzam et al., 2005), they are even more likely to seek medical care (Gillock, Zayfert, Hegel, & Ferguson, 2005). According to a study by Gillock and colleagues (2005), individuals with a diagnosis of PTSD, and those with subsyndromal PTSD symptoms, tend to visit their primary care office more frequently over a 3-month period than individuals without PTSD. In addition, PTSD is associated with more severe physical symptoms and worse physical functioning with regards to bodily pain, role limitations, and general health perceptions (Gillock et al., 2005; Zayfert, Dums, Ferguson, & Hegel, 2002). An increase in medical utilization and health related problems is thought to partially result from biological changes and physical symptoms that develop out of perpetual nervous system activation following the experience of a traumatic event (Butterfield & Becker, 2002; Gillock et al., 2005; McFarlane, 2000; Yehuda, 2002). Furthermore, prolonged stress and nervous system activation have been shown to reduce immune system functioning in both humans and animals (Chiarmonte, 1997; Khouzam et al., 2005). It is clear that the effects of PTSD are costly with regard to physical effects on the body and health care utilization.

Chronic Pain: General Characteristics

Diagnostic Criteria

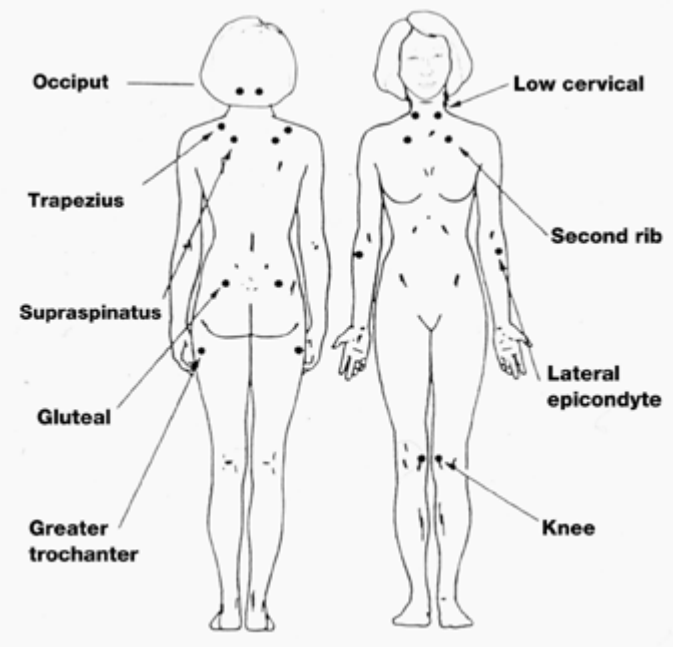
Pain is defined as the experience of unpleasant sensory and emotional symptoms (Merskey, & Bogduk, 1994; Staud, 2004). Pain is typically conceptualized as being either acute or chronic in nature. When pain is defined as acute, it is generally caused by tissue damage (e.g., wound, broken limb) from an injury (Taylor, 2006). Given that the

experience of pain can help alert the body to tissue damage; it is thought to play an adaptive role in day to day functioning. Furthermore, the experience of pain helps to motivate an individual to react in a way that may decrease the risk of potential damage and to also recover from its effects (Wall, 1978). Subsequently, pain from this type of tissue damage tends to abate as the body repairs the damage. However, pain that begins as an acute episode does not always lessen with treatment and time (Taylor, 2006). When pain persists for three or more months and becomes a chronic condition (International Association for the Study of Pain, 1986), it is thought to lose its adaptive functioning, and can create a significant amount of distress and impairment (APA, 2000). Consequently, the experience of pain is often associated with interference, feelings of little control over pain symptoms, depression, and anxiety (Lautenbacher, Spernal, Schreiber, & Krieg, 1999; Maxwell, Gatchel, & Mayer, 1998; Plehn, Peterson, & Williams, 1998). Pain behaviors may also be rewarded when individuals receive special attention from their family or friends (Osterhaus, Lang, Linssen, & Passchier, 1997). It is not surprising that pain is viewed as a very complex experience, with physiological, psychological, social, and behavioral influences (Taylor, 2006).

Musculoskeletal pain entails widespread pain involving the muscles and skeleton. According to the American College of Rheumatology (1990), widespread pain is defined as pain that is experienced above and below the waist, on the right and left sides of the body, or axial skeletal pain. Fibromyalgia, in particular, is characterized as the experience of widespread pain with tenderness in 11 or more of the 18 specified tender points (see Figure 1.1 for a diagram of tender point locations). Tender points are defined

as the experience of severe tenderness upon palpation (American College of Rheumatology, 1990).

Figure 1.1 Fibromyalgia tender point locations (Slavkin, 1997).



Although there are numerous forms of musculoskeletal pain, research has demonstrated that similar mechanisms (to different magnitudes) play a role in both fibromyalgia and other forms of chronic musculoskeletal pain (Bennett, 1998; Carli, Suman, Biasi, Marcolongo, 2002; Granges & Littlejohn, 1993). It has been difficult to detect laboratory or radiographic abnormalities that may be used in the assessment of musculoskeletal pain. Overall, little is known about the etiology of musculoskeletal pain conditions.

Researchers have hypothesized that the HPA axis may play a role in the development of musculoskeletal pain conditions (e.g., fibromyalgia; Crofford et al., 2004). High concentrations of Substance-P, a neurotransmitter associated with pain

levels, have also been found to be associated with fibromyalgia diagnoses (Clauw & Crofford, 2003; Russell et al., 1994). With respect to psychological factors, it has frequently been reported that symptoms of musculoskeletal pain begin or worsen during times of physical or emotional stress (Crofford et al., 2004). In addition, fibromyalgia is frequently associated with childhood and lifetime experiences of physical, sexual, and verbal abuse (e.g., Finestone et al., 2000; Goldberg & Goldstein, 2000). The lengthy and stressful process that individuals may go through to be diagnosed with fibromyalgia often leaves women feeling as though they are being judged as imagining their symptoms (Asbring & Narvanen, 2002; Zavestoski et al., 2004). Thus, there may be a relationship between stress, HPA activity, and symptoms of musculoskeletal pain.

Prevalence and Demographics

Pain is a leading cause of health care utilization in the United States that accounts for more than 70 million (80%) visits to physicians every year (Koch, 1986). According to The National Institute of Health, more than 50 million Americans experience various forms of pain, leading to more than 100 billion dollars in health care costs (as cited in Litcher-Kelly, Martino, Broderick, & Stone, 2007). Of these individuals who experience pain, approximately 17% are experiencing chronic pain (Gureje, 1998). According to epidemiological research, 4.9 million individuals are seeking treatment for their chronic pain condition (Marketdata Enterprises, 1999). Furthermore, approximately 7% of the general population report having experienced chronic pain within the last 12 months (McWilliams, Cox, & Enns, 2003).

Musculoskeletal pain is one of the most frequent forms of chronic pain, and is also one of the primary causes of disability in the United States (Davis, Zautra, & Reich,

2001). Fibromyalgia, one of the common types of musculoskeletal pain, is estimated to occur in approximately 2% of the population, and is about nine times more likely to develop within the female population. Prevalence rates of fibromyalgia have also been found to increase as individuals get older, with approximately 7% of women who are between 60 and 79 years of age reporting symptoms of fibromyalgia (Wolfe, Ross, Anderson, Russell, & Hebert, 1995). On average, individuals with fibromyalgia receive services from an outpatient physician approximately 10 times per year, costing approximately $2,200 for these visits (Wolfe et al., 1997). Furthermore, up to 25% of individuals with fibromyalgia report being on disability (Wolfe, 1996), and symptoms tend to persist with little change over long periods of time (Forseth, Forre, & Gran, 1999; Norregaard, Bulow, Prescott, Jacobsen, & Danneskiold-Samsoe, 1993; Wigers, 1996). Musculoskeletal pain and fibromyalgia, in particular, represent very frequent and debilitating chronic pain conditions within the female population.

Pain Sensitivity

Pain sensitivity is often operationalized as tenderness to pain (e.g., Montoya, Pauli, Batra, Wiedemann, 2005; Petzke Clauw, Ambrose, Khine, & Gracely). A diagnosis of fibromyalgia is based upon the existence of tender points and their sensitivity to touch (American College of Rheumatology, 1990); however, this sensitivity to touch is not necessarily limited to these specific tender points, and pain sensitivity is frequently reported throughout the whole body (Petzke et al., 2003; Scudds, Rollman, Harth, & McCain, 1987; Wolfe et al., 1995).

Many factors have been proposed to alter levels of pain sensitivity within fibromyalgia populations. Stress, in particular, has frequently been shown to perpetuate

levels of pain within individuals with fibromyalgia (Van Houdenhove & Egle, 2004), and people that have a diagnosis of chronic pain tend to report higher levels of environmental stressors, general stress, and psychological distress than individuals without chronic pain (Naidoo & Pillay, 1994). Furthermore, factors associated with stress (i.e., anxiety, depression, irritability) may perpetuate pain related symptoms of fibromyalgia and subsequent levels of disability (Winfield, 1999). Pain sensitivity may also be enhanced as a result of hypervigilance to bodily sensations (Lorenz, Grasedyck, & Bromm, 1996; McDermid, Rollman, & McCain, 1996; Chang, Mayer, Johnson, FitzGerald, & Naliboff, 2000); however, some researchers still argue that no differences in body vigilance reports are demonstrated between individuals with fibromyalgia and individuals without the diagnosis (e.g., Peters, Vlaeyen, & van Drunen, 2000). Higher levels of pain catastrophizing have been found to be associated with lower pain thresholds and tolerance for pain within fibromyalgia populations (Geisser, Casey, Brucksch, Ribbins, Appleton, & Crofford, 2003). Similarly, hypervigilance to pain has been positively correlated with pain intensity, negative affectivity, and catastrophizing about pain conditions (Crombez, Eccleston, Van den Broeck, Goubert, & Van Houdenhove, 2004). Overall, many psychological factors have been found to worsen pain sensitivity within fibromyalgia populations.

Theoretical Models of Comorbid PTSD and Chronic Pain

Although there are high comorbidity rates between PTSD and chronic pain, few factors have been identified to account for these high prevalence rates. To date, three models have been proposed to account for the comorbidity of PTSD and chronic pain: the mutual maintenance model (Sharp & Harvey, 2001), the shared vulnerability model

(Asmundson et al., 2002), and the triple vulnerability model (Otis et al., 2006). Although some aspects of these models have been supported in research, no studies have tested the complete models (Otis et al., 2006). Given that the triple vulnerability model accounts for biological, psychological, and social aspects of PTSD and chronic pain development, the current study will test aspects of this model.

Mutual Maintenance Model

According to the mutual maintenance model (Sharp & Harvey, 2001), there are seven components of both PTSD and chronic pain that may work together to mutually maintain or worsen the symptoms of one another. Furthermore, these components interact to further worsen an individual's distress and disability from each condition. First, attentional biases resulting from a PTSD diagnosis are thought to cue individuals in to potentially threatening or painful stimuli. In both conditions, individuals may have an unrealistically high expectation of confronting or experiencing threatening stimuli (Sharp & Harvey, 2001). Conflicting research has been demonstrated regarding the role of attentional biases in PTSD. When compared to individuals that did not develop PTSD following a traumatic event, Vietnam veterans with PTSD did not take longer to process threat related words (McNally, Amir, & Lipke, 1996). However, many other researchers have demonstrated that individuals with PTSD do demonstrate attentional biases for threat related words (e.g., Bryant & Harvey, 1997; Buckley, Blanchard, & Neill, 2000; Foa, Feske, Murdock, Kozak, & McCarthy, 1991; Harvey, Bryant, & Rapee, 1996). Research has also found that participants with PTSD tend to overestimate the probability of re-experiencing a traumatic event (Warda & Bryant, 1998). Similarly, participants with a chronic pain condition tend to overestimate the likelihood of pain symptomatology

and re-injury (McCracken, Gross, Sorg, & Edmunds, 1993). Consequently, attentional biases towards painful stimuli may serve to amplify pain symptomatology. Research assessing response latencies among individuals with chronic pain have tended to be slightly more conflicting with regard to the existence of attentional biases (Beck, Freeman, Shipherd, Hamblen, & Lackner, 2001). Only one study (Beck et al., 2001) assessed and provided support for the role of attentional biases within a population of individuals with comorbid chronic pain and PTSD. However, the sample was limited to individuals who had developed chronic pain following a motor vehicle accident. Overall, preliminary research tends to support the role of attentional biases in PTSD, although more conflicting results have been demonstrated in the chronic pain population.

Second, anxiety sensitivity is hypothesized to serve as a vulnerability factor for both PTSD and chronic pain conditions (Sharp & Harvey, 2001). Anxiety sensitivity refers to the fear of symptoms of arousal that may develop from misattributions regarding the potential harmfulness of various sensations (Reiss & McNally, 1985). In particular, individuals may be fearful of public displays of anxiety, cognitive dyscontrol, or bodily sensations (Taylor, 2004). High levels of anxiety sensitivity may lead individuals to misinterpret and catastrophize physiological symptoms that are associated with both chronic pain and PTSD (Sharp & Harvey, 2001).

According to a previous hypothesis (Taylor, 2004), anxiety sensitivity may increase following the experience of a traumatic event, and this increased level of anxiety sensitivity may then heighten the experience of PTSD symptoms (Taylor, 2004). Not only has anxiety sensitivity been found to intensify an individual's emotional response to various stimuli, but it has also been found to be associated with PTSD severity (Taylor,

2004). In a study that assessed anxiety sensitivity in children and adolescents (ages 8 to 15) five years after the experience of an earth quake, anxiety sensitivity levels were found to predict PTSD symptomatology (Kilic, Kilic, & Yilmaz, 2008). Overall, research suggests that there may be a relationship between anxiety sensitivity and PTSD symptoms.

Support for the role of anxiety sensitivity in pain perception has generally come from non-clinical undergraduate populations (Stewart & Asmundson, 2006). In a study that assessed for pain experiences within high and low anxiety sensitivity groups, 90 female undergraduate students were administered a cold pressor task. Results demonstrated that no significant differences were reported between both groups with respect to pain threshold, pain recovery, or pain tolerance following a cold pressor task. Conversely, the high anxiety sensitivity group reported greater perceptions of pain and fear of pain. Thus, the relationship between anxiety sensitivity and pain intensity is thought to be mediated by fear (Uman, Stewart, Watt, & Johnson, 2006). Although elevated anticipatory anxiety may not be specific to pain; individuals with high anxiety sensitivity have been found to report greater anticipatory anxiety prior to the experience of a painful stimulus (Conrod, 2006). Finally, a treatment study focusing on high and low anxiety sensitivity groups provided evidence for the role of anxiety sensitivity in pain related anxiety. Following a cognitive-behavioral treatment plan for anxiety sensitivity, pain-related anxiety was found to lessen (Watt, Stewart, Lefaivre, & Uman, 2006).

Research on anxiety sensitivity and attentional biases has indicated that high levels of anxiety sensitivity are associated with color naming interference for physical threat words (Stewart, Conrod, Gignac, & Pihl, 1998). Conversely, other researchers

have reported that neither high nor low levels of anxiety sensitivity are associated with response latencies for physical threat words in the emotional Stroop task. It has been hypothesized that they may have obtained null results because they did not include participants that had experienced panic attacks, whereas Stewart and colleagues (1998) did include participants who had experienced panic attacks (McNally, Hornig, Hoffman, & Han, 1999). In another study, a physical threat subscore on the anxiety sensitivity index was created and was examined for attentional biases within participants with high, moderate, and low physical threat anxiety sensitivity. These researchers also utilized the visual dot probe task instead of the modified Stroop task. Results indicated that high levels of physical threat anxiety sensitivity were associated with attentional biases for anxiety related words (Hunt, Keogh, & French, 2006).

Third, physiological symptoms of pain may serve as a reminder of the traumatic event. One study reported that individuals who have experienced a traumatic event tend to report experiencing increased symptoms of panic, and subsequently fear these panic related symptoms (Falsetti & Resnick, 1997). In particular, they may fear experiencing physiological symptoms such as increased heart rate and numbness and tingling sensations (Falsetti & Resnick, 1997). Arousal responses may lead individuals to further avoid activities that increase pain levels and that remind an individual of his or her trauma (Sharp & Harvey, 2001). PTSD research has demonstrated that various reminders of a traumatic event may initiate further arousal and PTSD symptomatology (Blanchard, Kolb, Gerardi, Ryan, & Pallmeyer, 1986). Furthermore, the experience of a traumatic event has been associated with the onset of chronic pain conditions (e.g., Finestone et al., 2000; Goldberg & Goldstein, 2000).

Fourth, such avoidance may initially lower feared symptomatology; however, prolonged behavioral and cognitive avoidance may lead to additional physical and anxiety symptomatology. Evidence for this claim has been supported in both the PTSD and chronic pain literature (e.g., Harvey & Bryant, 1998; Sharp, 2001). One study assessed for cognitive avoidance (i.e., suppression of trauma related memories) in individuals with acute stress disorder (n = 24) and in a control group (n = 24). Results suggested that attempts to suppress trauma-related thoughts in the acute stress disorder group were associated with a greater increase in ratings of anxiety and more frequent anxious thoughts than the control group (Harvey & Bryant, 1998). In chronic pain populations, behavior avoidance of physical activities has been associated with decreases in physical functioning and increased levels of disability (Waddell et al., 1993). Thus, preliminary evidence has supported the role of behavioral and cognitive avoidance in the perpetuation of PTSD and chronic pain symptomatology.

Fifth, individuals with either PTSD or chronic pain frequently experience symptoms of depression as well. Symptoms of depression have been found to be highly prevalent in samples that have comorbid conditions of PTSD and chronic pain (Roy-Byrne, Smith, Goldberg, Afari, & Buchwald, 2004). It is commonly known that fatigue is a diagnostic symptom for major depressive disorder (APA, 2000). Greater symptoms of fatigue have been associated with decreased participation in physical activities within a fibromyalgia sample (Rutledge, Jones, & Jones, 2007). Thus, symptoms of fatigue and lethargy from depression may result in decreased participation in various activities, which may then help to maintain or worsen PTSD and pain symptomatology.

Sixth, perception of pain may increase due to anxiety associated with PTSD, further limiting participation in activities (i.e., activities involving physical exercise; Sharp & Harvey, 2001). Although researchers have not assessed for increased pain perceptions within PTSD samples, research has indicated that self-perceptions of pain are increased by the experience of anxiety (e.g., Difede, Jaffe, Musngi, Perry, & Yurt, 1997). Seventh, high levels of cognitive demands associated with both conditions (i.e., catastrophic cognitions and recurrent thoughts of trauma) may limit the use of adaptive coping strategies (i.e., cognitive restructuring; Sharp & Harvey, 2001). Further information regarding the use of various coping strategies in PTSD and chronic pain populations will be provided below.

Given that many aspects of the mutual maintenance model are indirectly supported in the PTSD and chronic pain literature, it may hold some promising results as an explanatory model; however, there are some criticisms regarding various aspects of the model. As mentioned earlier, the modified Stroop task has often been used in the PTSD and chronic pain literature as a means of measuring attentional bias, although the validity of the modified Stroop paradigm has been questioned. In particular, it is unclear if this task is actually measuring attentional bias, or if it is measuring another form of information processing (e.g., determining the meaning of words) or motor response (e.g., trying to determine an appropriate response). Although the extant research has generally supported the role of the Stroop paradigm in measuring attentional biases, there is still a possibility that this research may be eliciting a more generalized information processing bias than the intended attentional bias construct (Roelofs, Peters, Zeegers, & Vlaeyen, 2002).

With regard to the role of anxiety sensitivity, research has demonstrated that anxiety sensitivity and pain catastrophizing may independently contribute to the experience of chronic pain (Drahovzal, Stewart, & Sullivan, 2006). As such, the role that anxiety sensitivity plays in the development of pain perceptions and symptomatology may be more complex than originally thought (Stewart & Asmundson, 2006). Finally, the role of biological functioning (e.g., HPA axis) in both the PTSD literature and chronic pain literature has been highly supported (e.g., Crofford et al., 1994; Olff et al., 2006; Rasmusson & Friedman, 2002; Wingenfeld et al., 2008; Yehuda, 2003); however, this model does not take biological or genetic influences (e.g., inherited vulnerabilities) into account. As a result, the mutual maintenance model may not be the best predictor model for the relationship between PTSD and chronic pain.

Shared Vulnerability Model

Asmundson and colleagues (2002) derived the shared vulnerability theory from the mutual maintenance model. In particular, the researchers hypothesized that anxiety sensitivity acts as a shared vulnerability that leads to the development of both PTSD and chronic pain. In particular, they proposed that individuals who report higher levels of anxiety sensitivity may be more likely to develop these conditions given they are experiencing heightened fear levels and avoidance. Furthermore, these individuals with high anxiety sensitivity are thought to demonstrate a greater emotional response (i.e., fear and anxiety) when encountering a traumatic experience or physical injury (Asmundson et al., 2002). If these individuals are demonstrating a stronger emotional reaction, then they may be more likely to develop PTSD (Taylor, 2004). If individuals with chronic pain

subsequently respond to their pain with fear and avoidance, then their symptoms are likely to worsen (Asmundson et al., 2002).

Although anxiety sensitivity may serve as a promising vulnerability factor for PTSD and chronic pain, many criticisms of this model still exist. As mentioned earlier, the role of anxiety sensitivity in PTSD and chronic pain may be mediated by unknown cognitive processes that are not accounted for by pain catastrophizing; thus, the influence of anxiety sensitivity may be more complex than originally thought (Stewart & Asmudson, 2006). Furthermore, a study comparing undergraduates with high and low anxiety sensitivity demonstrated that fear levels in relation to a cold pressor task were found to mediate the relationship between anxiety sensitivity and pain instead of the other way around (Uman et al., 2006). Finally, this model is similar to the mutual maintenance model in that it does not account for many other factors that may also play an important role (e.g., social, biological, coping styles). Support for the role of these additional factors will be provided below. Consequently, it is thought that the triple vulnerability model may better account for biological, psychological, and social variables in the development of PTSD and chronic pain.

Triple Vulnerability Model

The triple vulnerability model was originally proposed by Barlow (2000, 2002) to account for the development of anxiety disorders in general. According to the triple vulnerability model, three separate vulnerabilities must be present to develop anxiety disorders: 1) a generalized biological vulnerability, 2) a generalized psychological vulnerability, and 3) a specific psychological vulnerability. Keane and Barlow (2002) further proposed that the triple vulnerability model may apply to PTSD when an

individual experiences a traumatic event (i.e., the true alarm), which develops into a specific psychological vulnerability (i.e., the learned alarm). However, PTSD cannot develop without an individual having a generalized biological vulnerability (e.g., inherited aspects of personality traits that lead individuals to react to environmental stressors in a defensive manner) and a generalized psychological vulnerability (e.g., sense of uncontrollability and perceived inability to cope) prior to the traumatic experience (Keane & Barlow, 2002). Further information regarding each of these vulnerabilities will be provided below.

Otis and colleagues (2003) proposed that the triple vulnerability model can account for the development of both PTSD and chronic pain. According to this variation, chronic pain may also develop in a similar manner to that of PTSD with respect to the three vulnerabilities. A sense of uncontrollability may be present prior to the development of learned alarms associated with both PTSD and chronic pain. PTSD and chronic pain may serve as a reminder of each other, and may act to maintain and worsen symptomatology of each condition. Thus, increased levels of responsivity (i.e., fear and avoidance) following these learned alarms may develop. Feelings of uncontrollability may then worsen, and disability may develop (Otis et al., 2003).

Generalized Biological Vulnerability

According to Barlow (2000, 2002), individuals who go on to develop anxiety have inherited personality traits that serve as a vulnerability for anxiety disorders. Such traits are thought to consist of being “high-strung,” “nervous,” or “emotional” (Barlow, 2002). Other researchers have also hypothesized that personality traits, such as negative affectivity and neuroticism, could also be inherited vulnerability factors (Clark, Watson,

& Mineka, 1994). Negative affectivity is conceptualized as a general predisposition towards experiencing negative emotions, with subsequent influences on cognitions, self-concept, and world views (Watson & Clark, 1984). Similarly, neuroticism was originally conceptualized as a personality dimension that develops out of a lowered threshold for limbic system activation. Subsequently, neuroticism was defined as a personality tendency to experience "fight or flight emotions" (i.e., fear, anxiety, anger, and distress), which leads to a greater experience of negative moods (Eysenck, 1967, 1981). It is estimated that genetic components of neuroticism may account for about 50% of its expression in individuals (Barlow, 2002; Eysenck, 1967).

Gershuny and Sher (1998) conducted a three year longitudinal study of 466 young adults to determine the correlates of neuroticism and extraversion (e.g., level of sociability; Eaves & Eysenck, 1975) on anxiety and depression scores. Results indicated that low levels of extraversion and high levels of neuroticism predicted higher levels of global anxiety in the third year of the study. Furthermore, their results suggested that there may be similar personality variables (i.e., neuroticism and extraversion) that predict the development of both anxiety and depression (Gershuny & Sher, 1998). Other research has also supported the hypothesis that anxiety and depressive disorders may have a similar genetic vulnerability, with respect to personality, and that environmental stressors may account for specific differences in how the disorders are expressed (Barlow, 2002).

It has been difficult to determine specific genetic contributions for PTSD. For example, it is difficult to differentiate between a vulnerability to being exposed to traumatic events versus a vulnerability to developing PTSD symptomatology. In a study

with World War II veterans with a diagnosis of PTSD, researchers found that 66% of participants had family members with PTSD as well (Davidson, Swartz, Storck, Krishnan, & Hammett, 1985). In another study, individuals who survived the bush fires in Australia were surveyed. Results support the hypothesis that individuals with a family history of PTSD were more likely to develop PTSD than to not develop PTSD following this traumatic event (McFarlane, 1988). However, twin studies have provided conflicting evidence regarding the genetic contributions for PTSD. Many of the studies that have been reviewed also have some significant methodological flaws, and offer only correlational findings instead of demonstrating causation (Keane & Barlow, 2002).

Within the chronic pain population, it has also been hypothesized that musculoskeletal pain conditions (e.g., fibromyalgia) are associated with increased negative affectivity and decreased positive affectivity. In particular, individuals with fibromyalgia were found to report minimal experiences of positive emotions. This finding, however, was not a direct result of decreased social engagements (Zautra et al., 2005). Several researchers have hypothesized that various emotions (e.g., negative affectivity) may serve as a vulnerability and maintenance factor for fibromyalgia (e.g., Davis, Zautra, & Reich, 2001; Geisser et al., 2003; Staud et al., 2003; Zautra et al., 2005). After a prolonged difficulty in maintaining emotional homeostasis during periods of high stress, individuals with fibromyalgia are thought to perceive future events as stressful. This perception of future events as stressful is thought to perpetuate the cycle of poor maintenance of emotional homeostasis (Zautra et al., 2005). Overall, researchers seem to agree that aspects of Barlow's original conception of a generalized biological

vulnerability (i.e., negative affectivity) may apply to musculoskeletal pain populations as well.

Researchers have provided initial support for 10 psychiatric conditions (i.e., attention-deficit hyperactivity disorder, bulimia nervosa, dysthymic disorder, generalized anxiety disorder, major depressive disorder, obsessive-compulsive disorder, PTSD, panic disorder, premenstrual dysphoric disorder, and social phobia) and 4 medical conditions (i.e., fibromyalgia, irritable bowel syndrome, migraine, and cataplexy) to be grouped together as affective spectrum disorders (ASD; Hudson & Pope, 1989, 1990, 1994; Hudson et al., 2004). In particular, these researchers hypothesized that that the psychological disorders and the medical conditions might share a physiological irregularity (i.e., difference in physiological functioning) that plays a role in their etiology. Support for this conception comes from similar responsivity among individuals with the different diagnoses to antidepressant medications (e.g., tricyclics, monoamine oxidase inhibitors, and serotonin reuptake inhibitors). Furthermore, this unknown physiological irregularity is thought to be based in heredity (Hudson & Pope, 1989, 1990, 1994; Hudson et al., 2004). In a study that assessed for heritable pathophysiological features of fibromyalgia, data were collected from 533 relatives of individuals with fibromyalgia and 272 relatives of individuals without fibromyalgia. Results of this study supported the original hypothesis that there are higher familial rates of ASD in relatives of individuals with fibromyalgia as compared to relatives of individuals without fibromyalgia. Subsequently, the idea of a similar genetic heritable component among several conditions (i.e., fibromyalgia and PTSD) was supported. However, the exact nature of this physiological irregularity has not been identified.

With regards to the biological inheritance of personality traits, Barlow (2002) proposed these personality traits (i.e., nervous, emotional, neuroticism, negative affectivity) may lead individuals who develop anxiety disorders to react differently to various changes in their environment. The genetic contribution is viewed as being non-specific and it represents a general tendency to be emotionally unstable. In this sense, an individual is genetically predisposed to respond to environmental changes/stressors in a defensive way with an alarm reaction. Since they are perpetually ready for danger, the threshold needed to initiate the fight or flight response is thought to be lower. However, this biological tendency is only viewed as being vulnerability for the individual if they present with specific psychological variables as well (Barlow, 2002).

Similar to research on PTSD, researchers have begun to look at abnormal brain processing of pain related information (Gracely, Petzke, Wolf, & Clauw; 2002; Yunus, 1992). In particular, it has been hypothesized that musculoskeletal pain conditions (e.g., fibromyalgia), may be initiated and maintained by the hyperexcitability of the central nervous system (Desmeules et al., 2003). Subsequently, hyperexcitability of the central nervous system, and the negative feedback loop of the HPA axis, have been associated with a blunted cortisol reaction following the experience of stressful events (e.g., Crofford et al., 1994; Wingenfeld et al., 2008). Similar to Barlow's (2000, 2002) conception of a generalized biological vulnerability, this HPA responsivity is not thought to be specific to just that of chronic pain (Abeles, Pillinger, Solitar, & Abeles, 2007). Further information regarding the role of HPA axis activation within PTSD and musculoskeletal pain populations will be provided below.

Given that individuals with anxiety disorders and chronic musculoskeletal pain conditions are thought to be in a perpetual state of hyperarousal, it is not surprising that researchers would anticipate that psychophysiological changes at baseline and in response to stressful stimuli within these populations would be different than individuals who do not have this predisposition. Various psychophysiological measures have been used in PTSD and chronic pain research to examine arousal: facial electromyography (EMG; muscle contractions), heart rate (HR; activity associated with cardiac functioning), skin conductance (SC; activity associated with the sweat glands), systolic blood pressure (SBP; blood pressure in the circulatory system with contractions of the heart), and diastolic blood pressure (DBP; blood pressure in the circulatory system when the heart is resting)(e.g., Flor & Turk, 1989; Pole, 2007,). In general, baseline assessments using such measures have tended to show inconsistent results with respect to various psychophysiological measurements within PTSD samples (Pole, 2007). However, inconsistent results may be due to methodological limitations (i.e., differing target muscles for various diagnoses, varying length of adaptation phases, lack of rest between experimental stimuli, failure to counterbalance stressors, poor sampling rates; Flor & Turk, 1989; Pole, 2007).

Frontalis (muscle located on the forehead) or corrugator (muscle located at the end of the eye brow) EMG responsivity is thought to relate to the display of negative emotions, zygomaticus (muscle located along the cheek bone) EMG is thought to reflect positive emotions, and orbicularis oculi (muscle located above the eyelid) EMG is thought to reflect an individual's startle reflex (Blumenthal et al., 2005; Lang, Bradley, & Cuthbert, 1998). The sympathetic nervous system (that prepares the body to react to

emergencies, heightened emotions, or strenuous activity) is thought to regulate SC, and both the sympathetic nervous system and the parasympathetic nervous system (that maintains or helps the body return to normal functioning following reactivity of the sympathetic nervous system) are thought to play a role in HR functioning. Thus, increases in HR might reflect activation of the sympathetic nervous system, a decrease in functioning of the parasympathetic nervous system, or both (Bernston, Cacioppo, & Quigley, 1993; Taylor, 2006). Subsequently, a stronger increase in HR instead of SC might reflect a greater role of the parasympathetic nervous system. Finally, measures of SBP and DBP could provide information regarding reactivity during either contraction or relaxation of the heart (Pole, 2007).

Baseline psychophysiological measurements have been frequently reported within the PTSD literature. In a study of Vietnam veterans, baseline physiological measurements were taken while they were sitting in a reclining position. Results demonstrated that there were no significant differences between the PTSD group and the control group with respect to heart rate (HR), systolic blood pressure (SBP), and diastolic blood pressure (DBP; Orr, Meyerhoff, Edwards, & Pitman, 1998). In a meta-analysis (Prins, Kaloupek, & Keane, 1995), five out of 13 studies focusing on PTSD samples were found to demonstrate higher baseline HR values for the PTSD group than for control groups. Only one study was found to demonstrate significant baseline differences in skin conductance levels (SC); however, this study actually demonstrated that the PTSD group had lower SC levels than the control group. Statistical analyses comparing these studies demonstrated that those with shorter baseline levels (5 minutes) tended to demonstrate more variability within and between groups. It was thought that this increase in

autonomic arousal may be more due to the shorter duration of the baseline period than to group differences (Prins et al., 1995). However, it should also be noted that many of these studies did not report including an adaptation phase in addition to the baseline phase. Conversely, another meta-analysis (Buckley & Kaloupek, 2001) reported that PTSD groups did tend to show higher resting baseline HR and DBP than those without PTSD. It is possible that baseline elevation of HR in PTSD could be mediated by decreased parasympathetic activity instead of increased sympathetic activity (Pole, 2007).

Within the chronic pain literature, elevated baseline psychophysiological measurements have not been demonstrated on a consistent basis in individuals with chronic headaches, chronic back pain, and temporomandibular pain disorders (Flor & Turk, 1989). In contrast, other researchers have found elevated levels. For example, one study assessed baseline levels of HR and SC in 30 patients with fibromyalgia and 30 healthy control patients. Following a 30 minute adaptation phase, baseline psychophysiological measures were collected for 4 minutes while participants were asked to sit quietly and as still as possible with their eyes open. Results demonstrated that the fibromyalgia group had significantly higher HR at baseline than the healthy controls. No significant differences were demonstrated with respect to SCL at baseline (Thieme et al., 2006). Similarly, results from another study indicated significantly higher resting baseline levels of HR, SBP, and DBP in a 10 minute resting baseline period in the low back pain group compared to individuals in the control group; however, this study did not include an adaptation period prior to recording resting baseline measures (Burns, 2006). Similar to findings with PTSD samples, baseline elevation of HR in chronic pain could

also potentially be mediated by decreased parasympathetic activity instead of increased sympathetic activity (Pole, 2007).

In summary, a generalized biological vulnerability is thought to be characterized by a genetic tendency to be emotionally unstable in response to environmental changes and stressors. This emotional instability may be caused by personality traits (i.e., nervousness, emotionality, neuroticism, negative affectivity) that lead individuals to be more responsive to environmental stimuli (Barlow, 2002). Research within the PTSD and chronic pain literature has supported the hypothesis that these personality traits are associated with aspects of both PTSD (e.g., Davidson et al., 1985; Gershuny and Sher, 1998) and chronic pain (e.g., Davis et al., 2001; Geisser et al., 2003; Staud et al., 2003; Zautra et al., 2005). Furthermore, baseline levels of physiological hyperarousal and physiological reactivity to environmental stressors have been demonstrated within both of these populations (e.g. Crofford et al., 1994; Desmeules et al., 2003; Orr et al., 1998; Prins et al., 1995; Wingenfeld et al., 2008). Thus, the current literature supports aspects of the generalized vulnerability hypothesis; however, limited research has assessed for these factors within a comorbid PTSD and chronic pain population.

Generalized Psychological Vulnerability

A generalized psychological vulnerability refers to a general sense of uncontrollability and perceived inability to cope with unpredictable negative life events. In particular, individuals may perceive past attempts at coping with various situations as signs of failure. Conversely, individuals without this vulnerability are thought to develop an "illusion of control," and they are more likely to attribute deficiencies to transient external causes (Barlow, 2002). With respect to chronic pain, individuals with chronic

pain may first view their pain as being uncontrollable and unpredictable. When the pain condition is viewed as being uncontrollable and unpredictable, decreased feelings of self-efficacy and increased negative affect may develop (Otis et al., 2003). According to Barlow (2002), the ability to produce anxiety symptomatology specific to an individual's disorder within laboratory settings is essential to measuring this change in coping strategies and feelings of uncontrollability.

Attributional Style. Attempts to assess for the role of control in humans were initiated by Rotter (1954). He hypothesized that an individual's "locus of control" might be rated along a continuum with internal causality on one end and external causality on the other. Thus, psychometric measures were developed that assessed for "locus of control" and "attributional style" (Barlow, 2002). Seligman (1975) hypothesized that individuals develop a sense of uncontrollability with respect to the environment when they experience numerous negative life events. Consequently, they develop a sense of learned helplessness, and stop attempting to cope with environmental stimuli. Furthermore, the relationship between negative events that individuals experience and the development of learned helplessness is thought to be moderated by an individual's attributional style. In particular, the experience of negative life events is likely to lead to the development of learned helplessness when individuals make internal, global, and stable attributions regarding the negative life events that they experience (Abramson, Seligman, & Teasdale, 1978). Helplessness was then thought to play a role in the development of anxiety, although hopelessness was thought to play a larger role in the development of depression (Abramson, Metalsky, & Alloy, 1989).

Limited research has assessed for the role of learned helplessness in PTSD and within the chronic pain literature. It is thought that similar types of events are experienced that lead to the development of both learned helplessness and PTSD, and that both conditions may be associated with similar behavioral and physiological symptoms (Mineka & Zinbarg, 1996). Animal research has also suggested that similarities may exist (e.g., sense of uncontrollability) in the development and perpetuation of learned helplessness and PTSD (Maier, 2001). With respect to attribution styles within the PTSD literature, helpless attributional styles have been associated with reports of abuse during childhood and the onset of PTSD in adulthood (e.g., Casella & Motta, 1990; Gibb, 2002). Many clinicians who have worked with individuals with PTSD have tended to notice that PTSD is associated with personal feelings of helplessness in which individuals reportedly feel that resolution of additional stressful events after their trauma is uncontrollable (McKeever, McWhirter, & Huff, 2006). Similarly, regression analyses have demonstrated that learned helplessness predicted PTSD symptom severity (McKeever, McWhirter, & Huff, 2006). Only one study was found that assessed for the role of attributional style within the chronic pain literature. This study compared attribution styles within a depressed and chronic pain sample, a chronic pain sample that was not depressed, and a control group. No differences were demonstrated between the groups with regards to attribution styles (Ingram, Atkinson, Slater, Saccuzzo, & Garfin, 1990).

Coping Strategies. Within the PTSD literature, coping strategies have generally been categorized as cognitive or behavioral (De Ridder, 1997; Holahan & Moos, 1987). Cognitive coping strategies involve altering thoughts regarding an event to decrease feelings of distress and to assign meaning to the event. Examples of cognitive coping

strategies include cognitive restructuring, wishful thinking, self-blame, and self-criticism. Conversely, behavioral coping strategies involve observable actions that an individual engages in to help alleviate distress following a traumatic event (e.g., withdrawing from others, seeking social support, and problem-avoidance; Waldrop & Resick, 2004). Research has consistently demonstrated that avoidant coping strategies (e.g., diverting attention) are associated with PTSD severity following the experience of a traumatic event. In a study that assessed coping strategies in a sample of 74 rape victims and 48 physical assault victims, an increased tendency to express emotions, use cognitive restructuring, and seek social support over a three month period of time was positively correlated with diminished PTSD symptomatology. Conversely, increased levels of wishful thinking and social withdrawal were associated with the perpetuation of PTSD symptomatology. Self-criticism was found to be positively associated with PTSD symptomatology at both one and three months following a traumatic event. Thus, some coping strategies have been shown to change over time following a traumatic event (e.g., cognitive restructuring, seeking social support, social withdrawal), while others may remain more stable (e.g., self-criticism; Gutner, Rizvi, Monson, & Resick, 2006).

Since the 1990s, a plethora of research has focused on coping styles in the chronic pain literature (DeGood & Tait, 2001). Research has demonstrated that passive coping styles are positively correlated with increased severity of pain, disability associated with pain conditions, and psychological distress. Conversely, active coping strategies (i.e., seeking social support) have been found to be positively correlated with increased levels of positive affect and higher levels of activity (Snow-Turek, Norris, & Tan, 1996; Zautra et al., 1995). Longitudinal research has demonstrated a similar trend within a

musculoskeletal pain population (DeGood & Tait, 2001; Potter & Jones, 1992). Coping flexibility refers to the number of coping strategies that an individual utilizes on a regular basis to cope with their pain (DeGood & Tait, 2001). In a chronic pain sample that was seeking treatment for their pain; higher levels of coping flexibility were found to predict greater self-perception of control regarding the pain condition, and potentially greater use of effective coping strategies. However, coping flexibility did not predict ratings of pain severity (Haythornthwaite, Menefee, Heinberg, & Clark, 1998). Research that has focused on diverting attention away from the pain condition has been correlated with positive adjustment to pain; however, this relationship has often been found to be moderated by additional variables (e.g., pain intensity; Affleck, Urrows, Tennen, & Higgins, 1992; DeGood & Tait, 2001). Conflicting results have been found regarding the use of reinterpreting pain, coping self-statements, and ignoring pain (DeGood & Tait, 2001). Coping strategies such as praying or hoping have been found to be associated with poor adjustment to pain. Subsequently, it has been hypothesized that this relationship may exist since individuals tend to use these strategies when their pain levels have worsened already (Boothby et al., 1999). Finally, research has also reported that participants with increased symptoms of pain from fibromyalgia tended to experience more difficulty in coping with chronic pain, and that increased levels of illness uncertainty was associated with increased difficulty in coping with pain when participants pain levels were exacerbated; however, a direct relationship between illness uncertainty and coping with chronic pain was not demonstrated (Johnson, Zautra, & Davis, 2006). In general, coping strategies have been found to play an important role in the prediction and maintenance of pain.

Specific Psychological Vulnerability

Specific psychological vulnerabilities refer to factors that lead an individual to view a specific object or events as their primary focus of anxiety. In this sense, individuals are learning what it is that might be threatening to them, and they are focusing their anxious apprehension on stimuli that they view as being dangerous (Barlow, 2002). This perception may occur when a true alarm becomes a learned alarm following the experience of a traumatic event. Symptoms of numbing (i.e., avoidance of feelings) with respect to general responsiveness may reflect avoidance of these learned alarms. Events and the way in which an individual tends to respond to these events are viewed as being unpredictable, which then may perpetuate the experience of anxious apprehension. However, the development of PTSD is thought to be moderated by social support networks and the ability to cope (Keane & Barlow, 2002).

Initial support for the role of learning mechanisms in the development of PTSD began in the 1970s when researchers were treating rape victims and Vietnam veterans (Resick & Calhoun, 2001). Originally, Mowrer's (1947) two-factor theory was proposed to account for the role of classical and operant conditioning in the development of PTSD symptomatology following a traumatic event (e.g., Holmes & St. Lawrence, 1983). Classical conditioning is thought to account for elevated levels of distress and fear that are associated with a traumatic event, and operant conditioning was thought to account for PTSD avoidance and long term maintenance of fear. In particular, traumatic memories are thought to serve as the new conditioned stimuli, whereas fear and anxiety serve as the conditioned emotional responses. Such cues are then avoided, which provides negative reinforcement with the temporary decrease of fear and anxiety (Keane

& Barlow, 2002). Although this model offers some compelling arguments, it is thought that it may not be comprehensive enough to account for all PTSD symptomatology (e.g., recurrent memories, high rates of generalization; Foa, Steketee, & Rothbaum, 1989; Resick & Calhoun, 2001).

Classical and operant conditioning accounts have also been proposed in the chronic pain literature. Researchers have found that fear of pain and re-injury is associated with avoidance of activities that may lead to additional pain; however, this avoidance is associated with even greater physical deconditioning (i.e., deterioration of physical functioning) and maintenance of pain (Asmundson, Norton, & Norton, 1999). Given that anticipatory anxiety regarding potentially pain inducing activities may be reduced through avoidance, such behaviors are then negatively reinforced via operant conditioning with the reduction of anticipatory anxiety (McCracken, Zayfert & Gross, 1993). According to the triple vulnerability model, activities and sensations that are associated with additional pain may serve as the learned alarms, and individuals may try to avoid such activities as a result of their anxious apprehension.

Summary

In summary, research has provided preliminary support for Barlow's (2002) conception of a generalized biological and generalized psychological vulnerability within PTSD and chronic pain populations. These vulnerabilities are thought to be the same for both PTSD and chronic pain (Otis et al., 2003). With respect to a generalized biological vulnerability, both groups have demonstrated baseline levels of hyperarousal, and blunted cortisol responses following experimental stressors (e.g., Crofford et al., 1994; Olff et al., 2006; Rasmusson & Friedman, 2002; Wingenfeld et al., 2008; Yehuda, 2003). Although

preliminary research supports the role of a negative attributional style as a generalized psychological vulnerability for PTSD (e.g., Cassella & Motta, 1990; McKeener et al., 2006; Mineka & Zinbarg, 1996); limited research has been conducted with either condition. Furthermore, the role of negative coping strategies in the maintenance of both conditions individually has been frequently reported (e.g., Gutner et al., 2006; Snow-Turek et al., 1996; Zautra et al., 1995). Finally, evidence of learned alarms has been reported in a number of PTSD and chronic pain related studies (e.g., Asmundson et al., 1999; Holmes & St. Lawrence, 1983; McCracken et al., 1993). However, limited research on all aspects of the triple vulnerability model has been conducted on a comorbid PTSD and chronic pain population.

Stress and Cortisol

Hypothalamic-Pituitary-Adrenal Axis (HPA axis)

As mentioned earlier, HPA axis functioning has received increased attention in efforts to account for PTSD and chronic pain symptomatology development and maintenance. In particular, human responses to stress can be largely accounted for by the HPA axis (Tanriverdi et al., 2007). Prior to discussing the specific relationship of the HPA axis to PTSD and chronic pain, a conceptual basis for how the HPA axis functions and why it is activated in response to stress is presented.

Hypothalamus and Corticotropin-Releasing Hormone. At a general level, the hypothalamus is part of the diencephalon, which is part of a larger structure of the brain known as the forebrain. The size of the hypothalamus is fairly small compared to other areas of the brain, but it contains a significant number of nuclei that play many critical roles. In particular, the hypothalamus has been found to help regulate hunger, thirst,

bodily regulation of temperature, behavior associated with reproductive functioning, and the pituitary gland (Breedlove, Rosenzweig, & Watson, 2007). The hypothalamus subsequently controls the pituitary gland by producing either releasing hormones that activate the anterior pituitary or inhibiting hormones that limit activation of the anterior pituitary. Through its control over the pituitary gland, the hypothalamus subsequently functions as a connector between the nervous system and the endocrine system. The main releasing hormone that the hypothalamus secretes in relation to the HPA axis is corticotrophin-releasing hormone (CRH). CRH is a protein hormone that is released by the hypothalamus via its neurosecretory cells to subsequently increase the amount of cortisol that is secreted (Tortora & Grabowski, 2001).

Anterior Pituitary and Adrenocorticotropic Hormone. The anterior pituitary is located in the front lobe within the pituitary gland, and it has been found to create and release the majority of the hormones within our bodies (Breedlove et al., 2007). As mentioned earlier, stimulation of the anterior pituitary is caused by releasing hormones from the hypothalamus. The pituitary stalk is made up of many axons and blood vessels that allow for the communication of hormones between the hypothalamus and anterior pituitary. The anterior pituitary then secretes adrenocorticotropic hormones (ACTH), a tropic hormone that helps to regulate other endocrine glands. Since ACTH is a protein hormone, it tends to respond to stimulation very quickly, and it subsequently initiates activation of the adrenal cortex (Breedlove et al., 2007).

Adrenal Glands and Glucocorticoids. Within the adrenal glands, the adrenal cortex has been found to control the metabolism of carbohydrates, sodium, and bodily reactions to inflammation. Conversely, the adrenal medulla is thought to regulate

emotional arousal. Upon stimulation of the adrenal cortex, various steroid hormones are released that are known as adrenocorticoids. Of the adrenocorticoids, glucocorticoids have been found to directly affect the metabolism of carbohydrates and subsequently increase levels of glucose within the body. Cortisol, in particular, is a glucocorticoid hormone that is released in response to stress.

Since hormonal systems are able to both create and evaluate levels of hormones within our bodies, the secretion of these hormones tends to be regulated depending on what the body needs. Therefore, in negative feedback systems, the resulting release of hormones returns to suppress the secretion of additional hormones of that type. Through the process of negative feedback in the HPA axis, glucocorticoids return to the hypothalamus to inhibit the production of releasing hormones and ACTH (Breedlove et al., 2007). In addition, Dehydroepiandrosterone (DHEA) is also released by the adrenal cortex during the experience of stressful situations as a result of changing ACTH levels. DHEA helps to return the body's cortisol levels back to their baseline state. Thus, the production of DHEA may help to protect individuals with PTSD from developing even more detrimental problems with their health (Charney, 2004).

Cortisol. Cortisol, in particular, is one of the most prevalent glucocorticoids, and its production has been found to result in numerous changes within the body. In general, it is known that the negative feedback of cortisol to the hypothalamus subsequently inhibits activation of the HPA axis. Cortisol also activates various energy sources within the body to initiate an automatic fight or flight response (Charney, 2004). This process takes place through the breakdown of proteins, conversion of amino acids and lactic acid to glucose, and through the breakdown of triglycerides (Tortora & Grabowski, 2001).

This activation of energy sources eventually results in fatigue, wear and tear on bodily muscles, and steroid diabetes (Breedlove et al., 2007). Secondly, processes associated with inflammation and immune responses are inhibited (Charney, 2004). Not only does cortisol inhibit the production of white blood cells, it also inhibits the repair of potential wounds and diseases (Breedlove et al., 2007). Next, the inhibition of growth and reproductive systems in response to cortisol may lead to bone degeneration, inability to ovulate, and loss of sexual interest (Breedlove et al., 2007; Charney, 2004). Increased cortisol may also lead to increased arousal, hypervigilance, focused attention, and memory formation associated with emotions. Finally, regulation of the hippocampus, amygdala, and prefrontal cortex are also altered (Charney, 2004; Gold, Drevets, & Charney, 2002). Consistently high levels of cortisol may also lead to the development of hypertension, osteoporosis, immunosuppression conditions, insulin resistance, dyslipidemia (i.e., increased levels of plasma cortisol that can lead to atherosclerosis), dyscoagulation (i.e., change in blood clotting), atherosclerosis, and cardiovascular disease (Charney, 2004; Karlamangla, Singer, McEwen, Rowe, & Seeman).

Fight or Flight Response

Although the fight or flight response is not directly part of HPA axis activation, it is worth mentioning as it is initiated in response to increased cortisol levels within this process. The fight or flight response has been found to be associated with numerous additional changes in body functioning including: 1) dilated pupils, 2) increased heart rate, heart contractions, and blood pressure, 3) dilated airways, 4) constriction of blood vessels that are connected to nonessential organs, 5) greater blood flow to organs associated with exercise or fighting against dangerous stimuli, 6) glycogen and glucose

are broken down by the liver, and triglycerides are broken down by adipose cells, 7) glucose is released by the liver to increase glucose levels in blood vessels, and 8) all other processes involved in nonessential tasks are inhibited (e.g., digestive secretions). Thus, an extensive array of bodily changes occurs during the experience of stressful events (Tortora & Grabowski, 2001).

HPA Axis and the Menstrual Cycle

With respect to women, conflicting results have been found when assessing for cortisol levels across the menstrual cycle. Some researchers have found that there are no differences in cortisol levels across the menstrual cycle, whereas other researchers have found higher levels of cortisol during the luteal phase (i.e., the time period just before menstruation begins; Kajantie & Phillips, 2006; Rasmusson & Friedman, 2002). To date, no studies have found higher levels of cortisol during the follicular phase of the menstrual cycle (i.e., the time period just after menstruation ends). In addition, higher levels of cortisol have been associated with higher levels of estrogen (Lindholm & Schultz-Moller, 1973). Therefore, it may be possible that lower levels of cortisol during the follicular phase of the menstrual cycle may serve as an additional risk factor for the development of PTSD. Conversely, women who experience a traumatic event during the luteal phase of their menstrual cycle may be more likely to develop depressive symptomatology (Rasmusson & Friedman, 2002; Yehuda, 2003).

Only one study was found that assessed cortisol responsivity to stressful laboratory tasks (e.g., the Trier Social Stress Task [TSST]) across phases of the menstrual cycle. In an initial attempt to look at menstrual cycle phase influences, Kirschbaum and colleagues (1999) examined cortisol reactivity to the TSST in men and women. Cortisol

reactivity was compared in a sample of men (*n* = 20), women in the follicular phase of the menstrual cycle (*n* = 19), women in the luteal phase of the menstrual cycle (*n* = 21), and women who were taking oral contraceptives (*n* = 21). Results indicated that women in the follicular phase of the menstrual cycle had similar salivary cortisol level increases following the TSST as women taking oral contraceptives. Conversely, women in the luteal phase of their menstrual cycle had similar cortisol levels to men. Both women in the luteal phase of their menstrual cycle and men demonstrated greater cortisol reactivity to the TSST than women in the follicular phase of their menstrual cycle and women taking oral contraceptives. No significant salivary cortisol level differences were demonstrated at baseline between any of the groups. Within the group as a whole, cortisol levels peaked at 10 minutes following the TSST, and ACTH levels peaked 1 minute following the TSST (Kirschbaum et al., 1999). Thus, women in all phases of the menstrual cycles may demonstrate similar baseline levels of cortisol; however, their cortisol reactivity following the experience of stressful events may vary. Overall, all groups demonstrated significant cortisol responsivity following the TSST; however, women in the luteal phase of their menstrual cycle and men demonstrated larger cortisol increases than women in their follicular phase and women taking oral contraceptives.

HPA Axis and Aging

Research assessing for aging effects on HPA axis functioning has also resulted in conflicting outcomes. Although researchers in one study found that older participants may demonstrate greater cortisol responsivity to stressful situations (Gotthardt et al., 1995), other researchers have found no differences in older versus younger populations in men or women (Kudielka, Schmidt-Reinwald, Hellhammer, & Kirschbaum, 1999, 2000).

In an analysis that compiled data from five independent studies to investigate the role of age and gender on HPA axis functioning and reactivity following the TSST, all age groups (i.e., elderly, adults, young adults, and children) were found to have significant and similar cortisol reactivity to the TSST. Furthermore, post-hoc analyses indicated that there were no age effects in cortisol and ACTH reactivity across the different subgroups of women. Only studies that assessed for cortisol responsivity of premenopausal women in the luteal phase of their menstrual cycle were included to avoid confounding effects of the menstrual cycle. Postmenopausal women were not included if they were receiving hormone replacement therapy. Conversely, younger men were found to have greater ACTH reactivity than older men (Kudielka, Buske-Kirschbaum, Hellhammer, & Kirschbaum, 2004). Overall, age effects in cortisol and ACTH reactivity were not demonstrated when women in the luteal phase of their menstrual cycle were compared to postmenopausal women.

HPA Axis and PTSD

In general, lower levels of cortisol and increased negative feedback inhibition have been associated with hypersensitization of the HPA-axis following the experience of stressful events. Hypersecretion of corticotrophin-releasing factor (CRF) has also been reported, as well as a reduction of the corticotrophin response to CRF (Kaufman et al., 1997; Rasmusson & Friedman, 2002). One hypothesis is that individuals with PTSD have greater numbers of lymphocyte glucocorticoid receptors (which are needed for cortisol to create a strong effect), and that they have greater suppression to cortisol following secretion of dexamethasone (Rasmusson & Friedman, 2002; Stein, Yehuda, & Koverola, 1997; Yehuda et al., 1995). However, conflicting results have been found

regarding the relationship of HPA axis dysregulation and PTSD, and some researchers argue that individuals with PTSD do not have lower levels of cortisol (Rasmusson & Friedman, 2002). In a meta-analysis that cumulated the results of 37 studies assessing for basal cortisol levels among participants with PTSD, inconsistent results were demonstrated with regards to differences in basal cortisol levels between participants with PTSD and those in a control group; however, lower levels of basal cortisol tended to be demonstrated among participants with PTSD that were female and/or those that had experienced physical or sexual abuse, The inability to consistently obtain low cortisol levels in PTSD tends to suggest that other factors may also play a role in the relationship of HPA-axis regulation and PTSD (e.g., chronic pain).

Similarly, cortisol levels have been known to vary due to many factors which may make the assessment of cortisol somewhat complicated. For example, individuals tend to have higher cortisol levels in the morning and late in the evening (Crofford et al., 2004; Kudielka et al., 2004). Levels of cortisol may also vary throughout the menstrual cycle, or with daily stressors (Rasmusson & Friedman, 2002; Yehuda, 2003). In addition, much of the research that has assessed the relationship between PTSD and HPA-axis functioning has been limited in that researchers have tended to focus primarily on male combat veterans (Rasmusson & Friedman, 2002). In a recent review article (Wong &Yehuda, 2002), 20 studies were found that focused on male combat veterans, whereas only 7 focused on other populations (4 of which focused on women specifically). More recently, researchers have been starting to focus more on some of these less researched populations.

Conflicting results have been found with several studies assessing cortisol levels after the experience of a traumatic event. Several studies have found that low levels of cortisol following traumatic events were associated with the development of a PTSD diagnosis within one month of the event (Delahanty, Raimonde, & Spoonster, 2000; Yehuda, Shalev, & McFarlane, 1998). Types of traumatic events in these studies included motor vehicle accidents, natural disasters, and a mining accident. However, one study found that the predictive power of lower cortisol levels immediately following the traumatic event was not predictive of a PTSD diagnosis at a one year follow-up (Anisman, Griffiths, Matheson, Ravindran, & Merali, 2001). For individuals who were recently raped, low cortisol levels following the most recent event were associated with the experience of prior events that were similar in nature (Resnick, Yehuda, Pitman, & Foy, 1995). Research has consistently demonstrated that prior experiences of traumatic events are indicative of greater risk for the development of PTSD (e.g., Yehuda, 2003).

A recent study examined cortisol levels in individuals varying in traumatic event history. In a study looking at PTSD and urinary cortisol levels (Young & Breslau, 2004), 292 participants were assigned to one of three groups: participants that were exposed to a traumatic event, participants with a current diagnosis of PTSD, and a control group. Participants completed a 32 hour sleep study, while urinary cortisol samples were taken every 8 hours (at 8:00 a.m. and 8 p.m.). No main effects were found for the type of trauma that individuals experienced and their levels of cortisol. Similarly, no significant differences were found between individuals who experienced a traumatic event early in their life (prior to the age of 16) versus traumatic events later in life. No group differences were found with respect to urinary cortisol in the PTSD and control groups.

However, women with comorbid PTSD and major depressive disorder (MDD) had higher levels of urinary cortisol than controls and participants with either diagnosis alone. This trend was not demonstrated in male participants (Young & Breslau, 2004).

Another recent study examined cortisol levels and severity of PTSD symptoms. Olff and colleagues (2006) assessed cortisol, DHEA and DHEA sulfate (DHEA-S) levels and severity of PTSD symptoms. Given that many studies have found conflicting results regarding the effects of a comorbid diagnosis of depression, the researchers investigated the potential moderating role of major depressive disorder (MDD). Participants consisted of 39 civilian outpatients with PTSD and 44 healthy controls. Types of traumatic events ranged from sexual abuse, vehicle accidents, to the death of loved ones. Hormone levels were assessed between 8:00 a.m. to 10:00 a.m. via blood samples following an overnight fast. Results indicated that lower cortisol levels were associated with greater severity of PTSD symptomatology. The authors proposed that variability in the cortisol and PTSD literature may be due to severity of PTSD symptoms. The researchers also hypothesized that HPA dysregulation may be more problematic in individuals with more severe levels of PTSD (Olff et al., 2006). Similar to the findings of other researchers (e.g., Oquendo et al., 2003; Yehuda, Halligan, Golier, Grossman, & Bierer, 2004), the current results demonstrated that there were no significant effects in cortisol levels resulting from comorbid MDD (Olff et al., 2006). After accounting for demographic variables (e.g., age, sex), no significant difference was found between ratios of cortisol to DHEA within the different groups. Limitations of the study included the small sample size used in this study, use of blood sampling at only one time point, and levels of insomnia (Olff et al., 2006).

Previous research suggests that there may be a relationship between childhood experiences of sexual abuse and greater body weight (e.g., Lemieuz & Coe, 1995). One study assessed for the relationship between childhood experiences of sexual abuse, obesity in adulthood, and cortisol levels. Participants consisted of 28 women that were placed into three groups: women with PTSD diagnoses that experienced sexual abuse as a child (n = 11), women who did not have PTSD but still experienced sexual abuse as a child (n = 8), and women in the control group with no prior experiences of sexual abuse (n = 9). All participants completed three questionnaires assessing for severity of PTSD symptoms, general health, and premenstrual symptoms. Urinary samples were averaged over a 24-hour time period, and all urine samples were collected during the premenstrual phase of their menstrual cycle (i.e., luteal phase). For participants who were being treated with psychotropic medication, no significant differences were found with respect to cortisol levels. Results indicated that cortisol levels were not significantly associated with weight levels. Cortisol levels were found to be higher in women with PTSD who had experienced sexual abuse as a child than women with PTSD who had not experienced childhood sexual abuse and women in the control group. Interestingly, women in the PTSD group with childhood experiences of abuse tended to be more overweight and reported higher levels of premenstrual symptoms (Lemieux & Coe, 1995). One significant limitation of this study was that urinary cortisol levels were averaged over a 24 hour time period, when research has demonstrated that cortisol levels vary throughout the day (e.g., Crofford et al., 2004). Overall, childhood experiences of sexual abuse were found to be associated with higher levels of cortisol than other forms of PTSD.

HPA Axis Effects Resulting from Age of Trauma. Yehuda and colleagues (2001) have examined cortisol levels in individuals whose parents were Holocaust survivors. Participants were 20 adult males and 31 adult females who were children of Holocaust survivors. Forty-one control participants were also included who were of the same age range. Ten participants were taking psychotropic medications; however, these effects were not included in the analyses as they were not shown to provide significant effects in previous studies (Yehuda et al., 1995; Yehuda et al., 2000). Participants provided urine samples for 24 hours, and they were instructed to obtain these samples on a day in which they were not likely to be stressed out. Thus, many participants remained at home for the day. Results indicated that participants who reported emotional abuse had lower averages of cortisol levels for the 24 hour period than individuals who did not report emotional abuse. Conversely, self-report of childhood sexual abuse was associated with higher averages of urinary cortisol for the 24 hour period. Children of Holocaust survivors tended to report higher levels of childhood trauma than other individuals in their demographic group. Higher levels of abuse among Holocaust survivors tended to be associated with parental diagnoses of PTSD (Yehuda et al., 2001). Results of the current study may be partially limited since many of the participants tended to stay home for the day while others did not. Such variations in schedule could potentially alter cortisol outcomes.

Recent research has also assessed for effects of early adverse experiences and the frequency of stressful events in adulthood (Heim et al., 2002). Researchers wanted to discover if early life experiences of trauma were more predictive of alterations in HPA-axis regulation, or if experiences of stressful events in adulthood accounted for more of

the variance. In this study, 49 females were divided into four groups: a control group with no history of early life stress (*n* = 12), women without a current diagnosis of MDD who were abused as children (*n* = 14), women with a current diagnosis of MDD who were also abused as children (*n* = 13), and women with a current diagnosis of MDD who were not abused as children (*n* = 10). None of the women reported the use of psychotropic medications and medications that could alter their hormone levels. Participants were admitted as inpatients, and were instructed to remain in bed until the experiment began. Participants were then brought to the experimental area via a wheel chair to avoid exercise while walking. All women participated in a stress condition that consisted of a speech and math task. Blood samples were taken prior, during, and after the stress task. When compared to the control group, all three patient groups reported experiencing greater numbers of stressful events in the previous month. Results indicated that a history of abuse during childhood predicted greater ACTH responsivity, even when controlling for stressful events in adulthood. In addition, ACTH responsivity was increased even more when the women experienced traumatic events in adulthood as well. Thus, trauma during adulthood was found to play an additional role in regulating ACTH responsiveness to a stressful event beyond that of just childhood experiences of abuse. Depressive symptoms were found to be associated with the highest levels of ACTH concentrations, which contrasted to the results within the PTSD group without depression (Heim et al., 2002).

HPA Axis Regulation as a Potential Risk Factor. Although many studies have assessed HPA dysregulation following traumatic events, one study attempted to assess the role of low cortisol levels as both a risk factor and/or as a result of maladaptive

responses to stress such as that seen in PTSD in an animal model (Cohen et al., 2006). In this study, adult male Lewis, Fischer 344, and Sprague-Dawley rats were exposed to a stressful event when they were placed on used cat litter for 10 minutes. These rats differ in their sensitivity to stimuli. Control rats were exposed to unused cat litter for 10 minutes as well. Some of the rats were injected with corticosterone (a glucocorticoid in rodents) prior to being placed in the cat litter. Responses to the cat litter were categorized as "extreme behavioral responses" (EBR) and "minimal behavioral responses" (MBR). Rats participated in an elevated plus maze, acoustic startle response task, and the Morris water maze. Blood samples from all rats were collected during times when the rats were experiencing low levels of stress. At baseline, the Lewis rats demonstrated greater anxiety responses (i.e., less time spent in open areas) than the Fischer 344 and Sprague-Dawley rats, although their cortisol levels were not significantly different. Results indicated that rats who demonstrated a blunted HPA response following the stressful task were more likely to demonstrate EBRs than those with regular HPA functioning. Similarly, the rats that were exposed to corticosterone prior to the stressful task were less likely to demonstrate EBRs. Thus, administration of corticosterone to the Lewis rats significantly decreased the amount of EBRs that they demonstrated. Overall, results suggest that susceptibility to PTSD may be decreased by administrating cortisol prior to the experience of a traumatic event by reducing the likelihood of blunted HPA responses to such events (Cohen et al., 2006).

HPA Axis Functioning Following Treatment for PTSD. A recent study investigated cortisol levels in individuals who were treated for PTSD. Participants (n = 21), with varying types of traumatic events (e.g., assault, work accident, loss of loved

ones, car accident, etc.) were included (Olff, de Vries, Güzelcan, Assies, and Gersons, 2007). All participants met criteria for PTSD, and all participants began the study with low baseline levels of cortisol. Participants received a Brief Eclectic Psychotherapy treatment that contained both aspects of cognitive behavior therapy (e.g., psychoeducation, imaginal exposure, writing assignments, and cognitive restructuring) and psychodynamic elements (e.g., "giving meaning" to events and a farewell ritual toward the end of therapy). Without taking depressive symptomatology into account, no changes in cortisol levels were found following treatment. When controlling for severity of depressive symptoms, cortisol levels were found to increase in individuals who were "successfully treated." Cortisol levels decreased further in individuals who were not successfully treated. Since participants with depression tended to have higher baseline levels of cortisol, cortisol levels following the treatment were found to be associated with these baseline levels of depression. Cortisol levels following treatment were also found to be associated with progress that participants made with respect to depressive symptoms.

Various limitations of the study should be taken into consideration when looking at these results. Typically, aspects of cognitive behavior therapy and psychodynamic treatment are not viewed as being compatible with each other. As such, the type of treatment given to participants could potentially be a limitation of the study. Other limitations include the small sample size, no control group for comparison, and cortisol was only assessed between 8:00 a.m. and 10:00 a.m. Treatment results were also only significant when various aspects of treatment for depressive symptoms were taken into

account (Olff et al., 2007). Regardless of the limitations, this study represents a first attempt examining the effects of psychotherapy on HPA dysregulation in PTSD.

HPA Axis and Chronic Pain

Research has consistently associated the increased experience of fibromyalgia symptoms, among other forms of chronic pain, with physical and emotional stress (e.g., Clauw & Chrousos, 1997; Turk, Okifuji, Starz, & Sinclair, 1996; Van Houdenhove & Egle, 2004). Subsequently, several researchers have hypothesized the importance of the role of the HPA axis in chronic pain patients (e.g., Crofford et al., 2004; Okifuji & Turk, 2002); however, few studies have assessed this relationship in chronic musculoskeletal pain samples. Furthermore, no research studies were found that assessed for the relationship between the HPA axis and perceptions of pain.

In a recent study, researchers examined HPA axis functioning in a sample of individuals with chronic pelvic pain and in a sample with fibromyalgia (Wingenfeld et al., 2008). Salivary-free cortisol levels were assessed following the Trier Social Stress Test and in response to pharmacological ACTH stimulation where ACTH was injected into participants. Results indicated that participants with fibromyalgia had lower total cortisol levels following the TSST and ACTH stimulation as compared to controls. Conversely, no significant differences were found between the chronic pelvic pain and control groups (Wingenfeld et al., 2008). Similar results were reported in another study that assessed for HPA functioning in participants with fibromyalgia (n = 12) and a control group (n = 12). Results indicated that the fibromyalgia sample had a lower cortisol response to CRH physiological stimulation. These results provide support for the

hypothesis that HPA functioning is disrupted in individuals with fibromyalgia (Crofford et al., 1994).

The basal circadian rhythm of HPA axis functioning has also been found to be disrupted within a fibromyalgia sample. ACTH levels were found to be lower across a 24-hour time period within the fibromyalgia group when compared to a control group, and cortisol levels were found to be slightly higher within the fibromyalgia group. However, these results were only approaching significance. No experimental stressor was used within this study. These results suggest that fibromyalgia may be associated with decreased resiliency of HPA axis functioning. Thus, it may be more difficult for individuals with fibromyalgia to return to baseline ACTH and cortisol levels following activation of the HPA axis (Crofford et al., 2004). Results from these studies suggest that fibromyalgia may be associated with decreased activation and resiliency of HPA axis functioning.

Summary

From a review of the current literature, it is clear that hypothalamic-pituitary-adrenal axis (HPA) functioning has implications in the experience of PTSD and chronic musculoskeletal pain conditions. What seems less clear is the potential role of HPA axis functioning in the development and maintenance of PTSD and chronic pain, or if alterations in HPA axis functioning play a direct role in the experience of traumatic events and the onset of both conditions. Preliminary results in the study by Cohen and colleagues (2006) seem to suggest that cortisol levels prior to the experience of traumatic experiences may be a risk factor in the development of PTSD. However, Cohen and colleagues (2006) study was conducted with rats, and the generalizability of the study

data to humans is unknown. Unfortunately, it would be extremely time consuming, expensive, and difficult to obtain preventative data with humans because that would require longitudinal research to obtain information both before and after the experience of traumatic events and comorbid diagnoses. A variety of designs will be needed in future research to help clarify the role of HPA axis functioning in both conditions. To date, no research studies have been found that assessed for the role of the HPA axis functioning in a sample of both PTSD and chronic musculoskeletal pain participants.

Overview and Statement of Purpose

Not only have high rates of PTSD and musculoskeletal pain been reported in the literature (e.g., Davis et al., 2001; Geisser et al., 1996; Kessler et al., 1995; Wolfe et al., 1995), but research has also indicated that high comorbidity rates exist between these two conditions (e.g., Asmundson & Hadjistavropolous, 2006; Asmundson et al., 1998). Both conditions occur more frequently within female populations (e.g., Kessler et al., 1995; Khouzam et al, 2005; Wolfe et al., 1995). Comorbid PTSD and chronic pain are also associated with increased perceptions of pain, affective distress, interference in daily activities, and high rates of disability (Otis et al., 2003; Sherman et al., 2000). Thus, comorbid conditions of PTSD and musculoskeletal pain have been associated with a large cost to individuals and to society.

Currently, three models have been proposed to account for the high comorbidity rates between PTSD and chronic pain (i.e., mutual maintenance model, shared vulnerability model, and triple vulnerability model). Given that the triple vulnerability model (Otis et al., 2003) accounts for biological, psychological, and social aspects of PTSD and chronic pain, it is thought to hold the most promise as a comprehensive

theoretical model. This model was derived from Barlow's (2000, 2002) original triple vulnerability model that was developed to account for anxiety symptomatology. According to this model, individuals must have a generalized biological vulnerability (e.g., inherited personality traits that lead individuals to react to environmental stressors in a defensive manner), a generalized psychological vulnerability (e.g., sense of uncontrollability and perceived inability to cope), and a specific psychological vulnerability (e.g., development of learned alarms) before PTSD and chronic pain can develop (Barlow, 2000; Barlow, 2002; Keane & Barlow, 2002; Otis et al., 2003). A learned alarm may elicit both symptoms of chronic pain and PTSD (Otis et al., 2006), and the two diagnoses may work together to maintain or even worsen symptoms of both conditions (Otis et al., 2003).

Human stress responses can be greatly accounted for by the role of the HPA axis (Tanriverdi et al., 2007). Subsequently, the HPA axis has gained increasing attention in accounting for PTSD and musculoskeletal pain symptomatology. Although some research has provided conflicting results, previous research assessing HPA axis functioning in PTSD and musculoskeletal pain populations have generally demonstrated that both conditions tend to be associated with a blunted cortisol response when compared to control groups (e.g., Crofford et al., 1994; Olff et al., 2006; Rasmusson & Friedman, 2002; Wingenfeld et al., 2008; Yehuda, 2003). Thus, assessing for cortisol and psychological reactivity following a stress induction task (i.e., TSST) within PTSD and chronic pain samples may provide beneficial information regarding the generalized biological and psychological vulnerabilities proposed within the triple vulnerability model.

The purpose of the current study was to test aspects of the triple vulnerability model (Barlow, 2000; Barlow, 2002; Otis et al., 2003) in PTSD and chronic musculoskeletal pain populations. Since limited research has tested aspects of this model with a comorbid PTSD and chronic pain population, subsequent study results can contribute to a greater understanding of the relationship between these two diagnoses. These results may have important implications for future research and treatment of both diagnoses.

In the current study, aspects of the triple vulnerability model were examined within the following groups of women: women who have PTSD without chronic pain (*n* = 11), women who have musculoskeletal pain without PTSD (*n* = 10), women with both PTSD and musculoskeletal pain (*n* = 10), and women without PTSD and chronic pain (*n* = 15). Cortisol reactivity and anxious mood were assessed before and after the Trier Social Stress Task (TSST). Participants also completed questionnaires to assess for other potential indicators of the triple vulnerability model.

Hypotheses: Experimental Stressor

1. A general biological vulnerability was predicted to be evident in all three clinical groups (i.e., PTSD and pain group, PTSD group, pain group), although this was not predicted to be demonstrated in the control group. This would be demonstrated by: a) lower baseline cortisol levels prior to the experimental stressor, and b) blunted salivary cortisol levels following the experimental stressor.
2. Anxious mood was hypothesized to exist on a continuum across the measurement occasions of the experimental stressor. Participants in the PTSD and pain group

were anticipated to report the highest levels of anxious mood (both at baseline and in response to the experimental stressor), followed by the PTSD and the pain only groups. Participants in the control group were anticipated to report the lowest levels of anxious mood.

3. Participants in the three clinical groups were predicted to report greater use of negative coping strategies (i.e., denial, behavioral disengagement, self-blame) in response to the experimental stressor, while participants in the control group were predicted to report greater use of positive coping strategies (i.e., active coping, positive reframing, acceptance).

Hypotheses: Questionnaire Data

4. It was hypothesized that depression levels would exist on a continuum. Women in the PTSD and pain group were anticipated to report the highest levels of depression symptoms, followed by women in the PTSD only and the pain only groups. Women in the control group were anticipated to report the lowest level of depression symptoms.

5. A general psychological vulnerability will be evident in all three clinical groups, although this is not hypothesized to be demonstrated in the control group. This will be evident by higher levels of learned helplessness (e.g., greater levels of internal, stable, and global attributions).

6. Support for a general psychological vulnerability will also be evident in all three clinical groups by increased mean impact ratings of negative life events and general levels of self-perceived stress. Subsequently, these participants will also

be more likely to view ambiguous situations as more threatening than participants in the control group.

7. Participants in the three clinical groups were predicted to report greater use of negative coping strategies (i.e., denial, behavioral disengagement, self-blame) in general, while participants in the control group were predicted to report greater use of positive coping strategies (i.e., active coping, positive reframing, acceptance) in general.

8. Differential learned alarms will be evident for the three clinical groups. Due to the nature of PTSD, it is expected that both the PTSD group and the PTSD and pain group will demonstrate a learned alarm with respect to PTSD (self-report). It is hypothesized that participants in the pain only group and PTSD and pain group will report greater anxiety associated with pain than participants in the PTSD only group or control group. All three clinical groups are hypothesized to report greater levels of anxiety sensitivity than the control group.

Chapter 2

METHOD

Participants

Participants were 46 females who were 18 years of age or older. All participants were recruited from the local community and undergraduate psychology courses at the University of Maine. Based on initial screening criteria (see below), participants were categorized into the following four groups: (1) women who have PTSD without chronic pain (hereafter referred to as the PTSD group; $n = 11$), (2) women who have musculoskeletal pain without PTSD (hereafter referred to as the pain group; $n = 10$), (3) women with both PTSD and musculoskeletal pain (hereafter referred to as the PTSD and pain group; $n = 10$), and (4) women without PTSD and chronic pain (hereafter referred to as the control group; $n = 15$). Power analyses were conducted using the GPower 3.0 program (Faul, Erdfelder, Lang, & Buchner, 2007). With an alpha level of .05, power analyses revealed that a sample size of 48 would result in a power of .97 for cortisol analyses. This calculation was based on a 4 (Group: PTSD, pain, PTSD and pain, control) X 5 (Measurement collection point: baseline, 10 minute post-stress induction, 20 minute post-stress induction, 40 minute post-stress induction, and 60 minute post-stress induction) mixed between-within-subjects ANOVA. Previous research has indicated that there is a correlation of .76 between cortisol samples that are taken by the same individual within 20 minutes of each other, with variance of .042 and an effect size of .20 (Hruschka, Kohrt, & Worthman, 2005).

Participants from the local community received $20 for participation in the study, and participants from undergraduate psychology courses at the University of Maine

received 4 research credits. Participants were also offered enrollment in a free treatment study. All participants read and signed an informed consent form (Appendix A) prior to their participation in the study. Individuals were notified that they could discontinue their participation in the study at any time, and that they could choose to not answer any questions that they did not feel comfortable answering. Once participation was completed, all participants were debriefed regarding the study protocol.

Study Criteria

Participants were enrolled in the PTSD group if they: 1) met DSM-IV-TR research criteria for PTSD, and 2) did not report experiencing chronic pain. Participants in the PTSD and Pain group: 1) met diagnostic criteria for PTSD, and 2) had self-reported symptoms of chronic musculoskeletal pain. The Pain group had: 1) self-reported symptoms of chronic musculoskeletal pain, and 2) they did not meet criteria for PTSD. In order for the musculoskeletal pain to be defined as chronic pain, it must have lasted for at least the last three months (International Association for the Study of Pain, 1986). For the purposes of this study, self-report of chronic pain symptoms was utilized to categorize participants. To qualify for the control group: 1) participants did not meet criteria for PTSD, and 2) they did not report experiencing any chronic pain conditions. Participants in all groups were excluded if they reported having: 1) cardiovascular disorder, 2) current alcohol or substance abuse disorders, 3) a history of psychotic disorder, 4) a history of bipolar disorder, 5) daily use of opioid medications, or 6) were receiving hormone replacement therapy.

Recruitment

Participants were recruited from the local community, websites, and undergraduate psychology courses at the University of Maine. Flyers were hung up within approving local establishments and doctors' offices. All potential participants who contacted the principal investigator were given a brief description of the study and completed a phone screening (Appendix B). All participants who met the initial screening criteria were invited to participate in the study.

Experimenters

A fifth-year graduate student was the principal investigator and oversaw all aspects of the study. Other graduate students in clinical psychology who were trained to administer diagnostic interviews helped to interview participants. A graduate student who was blind to the participants' group status also reviewed each audiotape recording of the SCID-I interview to establish interrater reliability for diagnostic status. Undergraduate research assistants were trained on study procedures to assist in the experimental task and with data entry. All personnel read the American Psychological Association ethics code regarding research, were familiar with IRB procedures, and completed the University of Maine IRB website training for human subjects.

Measures

Structured Clinical Interview for DSM-IV-TR Axis I Disorders, Research Version, Patient Edition (SCID- I/P). The SCID-I/P was used to establish PTSD diagnoses, establish comorbid diagnoses, and to rule out the existence of exclusionary diagnoses. The SCID-I/P is a semi-structured interview designed to assess for both current and lifetime Axis I diagnoses (First, Spitzer, Gibbon, & Williams, 2002). During

the interview, individuals are asked questions regarding potential symptoms that they may experience with regard to Axis I diagnoses such as depression (e.g., In the past month, has there been a period of time when you were feeling depressed or down most of the day nearly every day?), and anxiety (e.g., In the past six months, have you felt particularly nervous or anxious?).

The SCID has been used extensively in studies to establish anxiety disorder diagnoses, and to rule out the presence of other psychiatric disorders (e.g., Horsch, McManus, Kennedy, & Edge, 2007; Mehner & Koch, 2007; Orr et al., 1998). Studies that have assessed for the reliability of the SCID-I have demonstrated good interrater reliability with kappa values of .57-1.00, and test-retest reliability with kappa values of .35 -.78 (Zanarini & Frankenburg, 2001; Zanarini et al., 2000). The SCID-I has also demonstrated superior validity when compared to routine clinical interviews (Basco et al., 2000; Shear et al., 2000).

Short Screening Scale for PTSD. The Short Screening Scale for PTSD is a brief clinician-administered interview to assess for a PTSD diagnosis (Appendix B; Breslau, Peterson, Kessler, & Schultz, 1999). The Short Screening Scale for PTSD was used as a prescreening measure to be administered over the phone since it can be administered in 3 minutes (Orsillo, Batten, & Hammond, 2001). The measure consists of 7 yes/no questions to assess for various aspects of a DSM-IV diagnosis of PTSD (e.g. Did you avoid being reminded of this experience by staying away from certain places, people, or activities?). A score of 4 or higher has been shown to have the greatest sensitivity (.80 - .85), specificity (.84 - .97), and predictive value (.71 positive predictive value, and .98 negative predictive value) when considering a DSM-IV diagnosis for PTSD (Breslau et

al., 1999; Kimerling et al., 2006). Furthermore, the measure has good test-retest reliability with a Spearman's rank correlation of .84 (Kimerling et al., 2006).

McGill Pain Questionnaire (MPQ). The MPQ is a self-report questionnaire, designed to assess for pain descriptors, the spatial distribution of pain, temporal properties of pain, and an overall pain intensity rating (Melzack, 1975; Appendix C). Pain descriptor words are grouped into four major subgroups: Sensory (e.g., pulsing, pricking, pinching), Affective (e.g., tiring, sickening, blinding), Evaluative (e.g., annoying, troublesome), and Miscellaneous (e.g., tight, cold, nagging). Drawings of the human body are provided to mark the location of both internal and external pain. Finally, pain intensity levels are rated on a 6 point Likert scale (0 = "No Pain" to 5 = "Excruciating"). The MPQ is a widely used questionnaire, and has been administered in over 350 studies assessing various pain populations. For the purposes of this study, the MPQ was used to confirm symptoms of musculoskeletal pain (i.e., location of pain), and to determine the number of tender points reported by participants (Melzack & Katz, 2001).

The MPQ has demonstrated good test-retest reliability within a chronic low back pain sample (Love, Leboeuf, & Crisp, 1989). Factor analyses have remained inconsistent, with the factor loadings possibly being dependent of the type of pain that is being assessed. The measure has been shown to be sensitive to treatment effects in reducing pain levels, and has demonstrated discriminative ability between various forms of chronic pain (Melzack & Katz, 2001).

Expanded Attributional Style Questionnaire (Expanded ASQ). The Expanded ASQ is a 24-item self-report scale, designed to assess for attributional styles associated

with the learned helplessness model of depression. In particular, the measure assesses for three subscales: *Internality*, *Stability*, and *Globality* (Appendix D; Peterson & Villanova, 1988). Each of the 24 items involves reactions to a range of hypothetical negative events. Individuals were asked to imagine themselves in each situation, and then to rate three 7-point Likert scales with regards to internality (1 = "totally due to others," and 7 = "totally due to me"), stability (1 = the cause of the situation will be "never present" in the future, and 7 = the cause of the situation will be "always present" in the future), and globality (1 = the cause is associated with "just this situation," and 7 = the cause is associated with "all situations").

The Expanded ASQ was derived from the original Attributional Style Questionnaire (ASQ; Peterson et al., 1982) to improve upon the measure's reliability (Peterson & Villanova, 1988). The Expanded ASQ demonstrated good reliability with Cronbach's coefficient alpha scores of .66 for the *Internality* subscale, .85 for the *Stability* subscale, and .88 for the *Globality* subscale. The expanded version also demonstrated good predictive validity, and greater internal consistency than the original ASQ (Peterson & Villanova, 1988).

Life Experiences Survey (LES). The LES is a 57-item self-report measure designed to assess individuals' experiences with a wide range of situations in the past 6 and 12 months (Appendix E; Sarason, Johnson, & Siegel, 1978). Three additional blank spaces were also included in the LES in the event that an individual experienced a situation that was not listed in the questionnaire. Items were chosen to represent a wide range of situations that are frequently experienced by the general population (e.g., marriage, new job, death of a close family member, serious illness or injury). Each item

that an individual has experienced within the past year is rated on a 7-point Likert scale from -3 (“extremely negative”) to +3 (“extremely positive”). The LES yields a positive change score (sum of the positive impact ratings), a negative change score (sum of the negative impact ratings), and a total change score (sum of all impact ratings). Research indicates that the negative change scores have demonstrated the greatest test-retest reliability across a 5-6 week time period (.56 - .88, $p < .001$), followed by the total change scores (.63 - .64, $p < .001$) and positive change scores (.19 - .53, $p < .001$). When compared to the other change scores, the negative change score was found to be most predictive of health status. In addition, negative change scores demonstrated a positive correlation with state (.29, $p < .01$) and trait (.46, $p < .01$) anxiety (Sarason et al., 1978). Since the negative change score has demonstrated the greatest test-retest reliability, is most predictive of health, and is positively correlated with trait anxiety symptomatology, only the negative change score will be used for the purposes of this study. The LES was also slightly modified to assess for experiences that each individual has experienced within the past 12 months, and within their lifetime.

Brief Cope. The Brief Cope is a 28-item self-report scale, designed to assess for the use of a variety of coping strategies. Fourteen subscale scores are obtained for individual coping strategies including: *Active Coping*, *Planning*, *Positive Reframing*, *Acceptance*, *Humor*, *Religion*, *Using Emotional Support*, *Using Instrumental Support*, *Self-Distraction*, *Denial*, *Venting*, *Substance Use*, *Behavioral Disengagement*, and *Self-Blame* (Appendix F; Carver, 1997). The Brief Cope was derived from the longer original version of the COPE, which consisted of 60-items (Carver, Scheier, & Weintraub, 1989). The Brief Cope was designed to obtain similar reliability and validity ratings in a shorter

amount of time (Carver, 1997). Each individual item ranges from 1 ("I haven't been doing this at all") to 4 ("I've been doing this a lot"). Reliability analyses have yielded acceptable coefficient alpha scores for the individual subscales, ranging from .50 to .90 (with most exceeding .60; Carver, 1997). For the purposes of this study, participants completed the Brief Cope with regards to how they cope in general and in response to the experimental stress task.

Impact of Event Scale – Revised (IES – Revised). The IES – Revised is a self-report measure to assess PTSD symptomatology based upon DSM-IV diagnostic criteria (Appendix G; Weiss & Marmar, 1997). The IES – Revised consists of 22 items, with each item ranging from 0 ("Not at all") to 4 ("Extremely"). The measure is broken down into three subscales: *Intrusion*, *Avoidance*, and *Hyperarousal* (Weiss & Marmar, 1997). The measure was derived from the original Impact of Event Scale (IES; Horowitz, Wilner, & Alvarez, 1979), with some changes to add the Hyperarousal subscale and update the diagnostic criteria from DSM-III criteria to DSM-IV criteria (Weiss & Marmar, 1997). The IES – Revised has demonstrated high internal consistency within various studies for the Intrusion subscale (coefficient alpha scores ranging from .87 - .92), Avoidance subscale (alpha scores ranging from .84 - .84), and the Hyperarousal subscale (alpha scores ranging from .79 - .90). Test-retest reliability was also assessed in two samples, demonstrating acceptable reliability for all of the subscales (Weiss & Marmar, 1997).

Pain Anxiety Symptom Scale-20 (PASS-20). The PASS-20 is a self-report measure designed to assess anxiety and fear that is associated with pain conditions (Appendix H; McCracken & Dhingra, 2002). The PASS-20 was derived from the

original PASS (McCracken, et al., 1993) to decrease the amount of time and effort needed to administer the questionnaire within clinical and research populations (McCracken & Dhingra, 2002). The PASS-20 consists of 20 items that are rated on a 6-point Likert scale, ranging from 0 ("Never") to 5 ("Always"). Responses on the PASS-20 are used to compute 4 subscales: *Cognitive*, *Escape/Avoidance*, *Fear*, and *Physiological Anxiety*. A total score may also be computed by adding all items of the questionnaire (McCracken & Dhingra, 2002).

The PASS-20 has demonstrated good reliability and validity within a chronic pain sample. Subscale scores on the PASS-20 are highly correlated with the original subscale scores of the PASS with 40 items (ranging from $r = 0.93$ to $r = 0.97$). The PASS-20 demonstrated good internal consistency (ranging from $\alpha = 0.75$ to $\alpha = 0.91$). Results also supported the predictive and construct validity of the PASS-20 (McCracken & Dhingra, 2002). Research with fibromyalgia and chronic low back pain samples also supported the one and four-factor structures of the PASS-20 (Roelofs et al., 2004).

Anxiety Sensitivity Index (ASI). The ASI (Reiss, Peterson, Gursky, & McNally (1986); Appendix I) is a 16 item self-report questionnaire designed to measure fear of symptoms related to anxiety. In particular, anxiety sensitivity is a functional diagnostic dimension in which high levels may serve as a risk factor for anxiety disorders (Reiss, 1991). High levels of anxiety sensitivity are associated with catastrophic views regarding consequences of anxiety related symptoms (Peterson & Reiss, 1992). Each item in the ASI is rated using a 5-point Likert scale ranging from 0 ("very little") to 4 ("very much"). The total score is determined by adding up the scores for all 16 items.

Numerous studies have been conducted looking at internal reliability scores within the ASI, with Cronbach's alpha coefficient scores ranging from .82 to .91 (Peterson & Heilbronner, 1987; Taylor et al., 1991; Taylor, Koch, McNally, & Crocket, 1992; Telch et al., 1989). The two-week test-retest reliability of the ASI, as computed by Pearson product-moment correlations, was 0.74 for the women (Reiss et al., 1986). Similarly, the ASI was found to have a three year test-retest reliability of .71 (Maller & Reiss, 1992). There is also strong evidence supporting the construct validity of the ASI (Peterson & Reiss, 1992).

Intolerance of Uncertainty Scale – Short Version (IUS-12). The IUS-12 is a self-report measure designed to assess individual reactions to uncertainty, ambiguous situations, and to the future (Appendix J; Carleton, Norton, & Asmundson, 2007). In particular, intolerance to uncertainty may mean that an individual views that there is no way to predict negative events, to the point that all ambiguous information is viewed as being threatening (Heydayati, Dugas, Buhr, & Francis, 2003). The IUS-12 was derived from the original IUS to decrease the number of questions and boost the measure's factor structure (Carleton et al., 2007). The IUS-12 consists of 12 items that are rated on a 5-point Likert scale ranging from 1 ("not at all characteristic of me") to 5 ("entirely characteristic of me"). The sum of all items creates a total score for a general intolerance of uncertainty. The IUS-12 has demonstrated very good internal consistency ($\alpha = .91$), and is highly correlated with the original 27-item IUS ($r = .96$). The IUS-12 also demonstrated convergent validity with the Generalized Anxiety Disorder Questionnaire-IV (GADQ; $r = .61$), the Penn State Worry Questionnaire (PSWQ; $r = .54$), and the Beck Anxiety Inventory (BAI; $r = .57$; Carleton et al., 2007).

Profile of Mood States (POMS). The POMS is a self-report measure of transitory mood states (e.g., nervous, irritated, bored, etc.; McNaire, Lorr, & Droppleman, 1971; Appendix K). The POMS consists of six subscales that may be used individually or in combination with each other as one measure. Items are rated on a 5-point Likert scale, ranging from 0 ("not al all") to 4 ("extremely"). For the purposes of this study, the *anxiety* (POMS-A) subscale was administered prior to and following the experimental task. This assisted in measuring participants' anxiety levels before and after the stress task. Research has demonstrated that the POMS has good reliability and validity, with coefficient alpha levels ranging from .90 or greater (McNair et al., 1971).

Visual Analog Stress Scale (VASS). A 7-point Likert scale (Appendix L) was constructed to assess for the level of stress that participants experienced during the experimental task. Items on the scale range from 0 ("Not at all stressful") to 7 ("Extremely stressful"). Since the Trier Social Stress Task is expected to induce moderate levels of psychological and physiological stress (Kirschbaum, Pirke, & Hellhammer, 1993), it is important to measure self perceptions of stress as a manipulation check.

Perceived Stress Scale (PSS). The PSS is a 14-item self-report measure designed to assess general self-perceptions of stress over the previous month (Cohen, Kamarck, & Mermelstein, 1983; Appendix M). This is a measure of stress in general, and not a measure of cumulative stressful events. Items are rated on a 5-point Likert scale ranging from 0 ("never") to 4 ("very often"). Research indicates that the PSS has good internal consistency (r = 0.84 - 0.86) and test-retest reliability (r = 0.85; Cohen et al., 1983). In

addition, scores on the PSS have been found to be positively correlated with negative health outcomes (e.g., Cohen, Tyrell, & Smith, 1993).

Beck Depression Inventory-II (BDI-II). The BDI-II (Beck, Steer, & Brown, 1996; Appendix N) is a 21-item self-report measure used to assess the severity of depressive symptomatology. Each item response ranges from 0 to 3 possible points (with 0 representing that the participant does not experience the individual symptom and 3 representing the most severe degree that the symptoms may be experienced). Total scores may range from 0 to 63 points. For the purposes of this study, the BDI was used to assess for comorbid depressive symptoms.

In a meta-analysis of studies that assessed for the psychometric properties of the initial BDI (Beck, Steer, & Carbin, 1988), the average internal consistency found for the measure demonstrated a Cronbach's alpha coefficient of .86 for clinical samples and .81 for nonclinical samples. The BDI-II has demonstrated high levels of internal consistency within a sample of clinical outpatients ($\alpha = 0.92$; Beck et al., 1996). Statistical analyses have demonstrated that the BDI-II has stronger factorial validity than the initial version of the BDI (Dozois, Dobson, & Ahnberg, 1998). In a study that assessed psychometric properties of the BDI-II within an older adult population, results supported the internal reliability of the measure ($\alpha = .86$). The convergent and discriminant validity of the BDI-II was also supported within this population (Segal, Coolidge, Cahill, & O'Riley, 2008).

Experimental Task

Trier Social Stress Test (TSST). The TSST (Kirschbaum et al., 1993) has been used within both the PTSD and chronic pain literature to induce moderate physiological and psychological stress (e.g., Dorn et al., 2003; Jones, Rollman, & Brooke, 1997;

McRae et al., 2006; Simeon et al., 2007; Wingenfeld et al., 2008). The TSST was established to elicit HPA axis activation in a large proportion of participants, and has been found to double or even quadruple salivary cortisol levels within numerous samples. Within the TSST, participants are first given a ten minute anticipation time period, during which they are instructed to prepare a five minute speech regarding why they are the perfect applicant for a particular job. Participants are told that they will be giving the speech in front of two "managers" that are trained to monitor nonverbal behavior (these individuals are really research assistants trained in study protocol). Following the ten minute anticipation period, participants are told that their speech is also being videotaped (which is not true). Participants then begin their 5 minute speech. If their speech does not last for the full five minute period, they are prompted to continue with a series of previously prepared questions. Following the five minute speech, the participant is instructed to serially subtract 13 from 1,022 as quickly and accurately as possible for a five minute period. If a mathematical error is made during this time period, then they are asked to start over from the beginning (Kirschbaum et al., 1993).

Psychophysiological Recording. Salivary cortisol samples were collected using a Salimetrics drool tube. Participants were instructed to drool a small amount of saliva into a plastic tube and then replace the lid on the tube. This method of collecting salivary cortisol levels has been found to have better volume recovery of saliva than the Salivette® (Jennifer Jewell, personal communication, November, 2008). Research has demonstrated high correlations between salivary cortisol levels and blood-based samples of cortisol ($r > 0.90$ in most reports; e.g., Kirschbaum & Hellhammer, 1989). Salivary cortisol samples were utilized for this study because they are less invasive and could be

sent for analysis through the mail. In an attempt to increase reliability and validity of cortisol samples, participants were instructed to not eat anything, drink anything (other than water), not exercise, or smoke cigarettes for up to 1.5 hours before coming in for their research visit. Menstrual cycle phase was also controlled. All of these variables have been found to alter salivary cortisol levels (Kirschbaum & Hellhammer, 1989).

Procedure

Participants who met screening criteria (see above) were invited to participate in the study. Participants who expressed an interest in participating were scheduled to come in to the laboratory in the Psychology Department for their research visit. Premenopausal women were scheduled during the luteal phase of their menstrual cycle (1 to 7 days before menstruation begins). All participants were scheduled between 2:00 to 6:00 p.m. Previous research has demonstrated that there are diurnal rhythms to cortisol levels, subsequently, similar time restrictions have been used in previous research that assessed cortisol levels (e.g., Kirschbaum, Klauer, Filipp, & Hellhammer, 1995; Kirschbaum et al., 1999; Rohleder, Wolf, Piel, & Kirschbaum, 2003).

Upon arriving for their scheduled appointment, participants were asked to read and sign an informed consent form (Appendix A) before participating in the study. The experimenter also reviewed the content of the consent form with each participant, and answered any questions that participants had. Each participant completed the MPQ to confirm self-report of chronic musculoskeletal pain. Participants were then interviewed using the SCID-I/P to confirm diagnoses or rule out the specified exclusionary diagnoses. Participants who met exclusionary criteria following administration of the MPQ and SCID-I/P were debriefed regarding the study and received $5 for their participation.

Participants who met criteria for any of the experimental groups (PTSD group, pain group, or PTSD and pain group) or the control group were placed in a sound-attenuated room to help minimize the effects of external stimuli during the study protocol. Prior to receiving experimental instructions for the TSST, participants were asked to sit quietly for a 10-minute baseline period while they adjusted to the laboratory setting. Following the adaptation period, participants were asked to complete the POMS, and a baseline salivary cortisol sample was collected. After baseline assessments, participants began the experimental task using the TSST study protocol described above. Following the completion of the TSST, participants were asked to remain in their chair and rest quietly for 60 minutes. Ten minutes after the completion of the TSST, participants completed measures assessing changes in their mood state (POMS) following the experimental task. Participants also completed a Brief Cope as a measure of coping strategies utilized during the experimental task, and the VASS as a measure of self-perceived stress during the TSST. A second salivary cortisol sample was collected at this time, and at 20 minutes following the TSST, to assess for peak salivary cortisol levels. Research has demonstrated that cortisol levels are at their highest during these time points (e.g., Kirschbaum et al., 1993). The POMS was completed with each saliva sample to measure changes in mood following the experimental task. A fourth cortisol sample and POMS was collected at 40 minutes after the completion of the TSST to measure changes in cortisol as the participant begins to relax again. To ensure that stress levels have been lowered before leaving the lab, participants then listened to a 20 minute relaxation exercise before leaving the lab. Following the relaxation exercise, participants completed their fifth salivary cortisol sample and POMS.

Participants were then debriefed regarding study procedures (Appendix P), and were given instructions for completion of their counterbalanced packet of questionnaires including: Expanded ASQ, LES, Brief Cope, IES-Revised, PASS-20, ASI, IUS-12, PSS, BDI-II, and the POMS. Included in this packet of questionnaires was a demographics information form (Appendix O) which included the date, age, race, education (in years), current household income, current medications, weight, height, and menstrual cycle status. Finally, participants were compensated for their participation.

Chapter 3

RESULTS

The current study represents a quasi-experimental, four groups (PTSD, pain, PTSD and pain, control), design. Data analyses were completed using PASW version 18.0 software for Windows. Participant characteristics and results for each hypothesis are presented in this section. When possible, power and effect size (eta^2) were calculated. Cohen (1988) suggested guidelines with respect to interpreting effect size in which .10 is small, .25 is medium, and .40 is large. Marginal results were reported with alphas <.10 for omnibus tests. Chi-square analyses were used for frequency data. When the test of sphericity was significant in a mixed design, degrees of freedom were adjusted using the Greenhouse-Geisser correction (Brace, Kemp, & Snelgar, 2009). In general, post-hoc comparisons were conducted using Tukey's HSD test with alpha set at .05. Given specific predictions in the hypotheses of this study, a priori planned comparisons were completed when appropriate with independent samples t-tests by combining group scores that were hypothesized to be similar. This was completed as part of the ANOVA calculations.

Participant Characteristics

The total sample consisted of 46 females. Participants ranged in age from 18 to 66 (see Table 3.1.for group averages). The four groups of participants (PTSD and pain, PTSD only, pain only, controls) differed significantly in age [$F(3,43) = 6.15$, $p<.01$]. Post-hoc analyses revealed that the pain only group was significantly older than the PTSD only and control groups. Similarly, women in the PTSD and pain group were significantly older than controls. There were no significant differences in age between

the PTSD and pain and pain only groups, between the PTSD and pain and PTSD only groups, or between the PTSD only and control groups. The four groups of participants also differed significantly in body mass index; BMI [$F(3,41) = 3.86$, $p = .02$]. Post-hoc analyses revealed that the pain only group had a significantly higher BMI rating than the PTSD only and control groups.

Table 3.1. Group Averages in Age and BMI

	PTSD and pain (n = 10)	PTSD only (n = 11)	Pain only (n = 10)	Control (n = 15)
Mean Age	42.80_{ab} (15.39)	27.44_{bc} (15.90)	45.90_{a} (15.62)	25.27_{c} (11.05)
BMI	28.19_{ab} (7.09)	23.75_{b} (3.89)	31.73_{a} (6.29)	24.59_{b} (5.29)

Note. Standard deviations are in parentheses. Means with different subscripts differ significantly at $p < .05$.

Chi-square analyses indicated that there were significant group differences in diagnoses of depression [χ^2 (3, N = 45) = 17.22, p = .001]. In particular, the PTSD and pain group had the highest prevalence of depression, followed by the PTSD only, control, and pain only groups respectively. No significant group differences were found for social phobia [χ^2 (3, N = 45) = 0.14, *ns*], specific phobia [χ^2 (3, N = 45) = 0.43, *ns*], obsessive-compulsive disorder [χ^2 (3, N = 45) = 7.33, *ns*], panic disorder [χ^2 (3, N = 45) = 3.15, *ns*], agoraphobia [χ^2 (3, N = 45) = 1.07, *ns*], or generalized anxiety disorder [χ^2 (3, N = 45) = .17, *ns*]. See Table 3.2 for specific percentages of these diagnoses.

Table 3.2. DSM-IV Diagnoses

	PTSD & Pain	PTSD Only	Pain Only	Control
Depression	70%	40%	0%	7%
Social Phobia	30%	10%	10%	0%
Specific Phobia	40%	10%	30%	20%
OCD	20%	0%	0%	0%
Panic Disorder	20%	20%	0%	7%
Agoraphobia	0%	10%	10%	7%
Generalized Anxiety Disorder	40%	20%	10%	7%

Advanced clinical graduate students reviewed audiotapes of the SCID-IV interviews and rated whether or not the participants met criteria for PTSD. These ratings were compared to diagnoses given to the participants during their laboratory session. Unfortunately, inter-rater reliability could only be assessed for 17 out of 46 interviews (37%). Many of the interviews could not be reviewed due to equipment failure during the recording process. Several of the interviews were also missing from the laboratory tapes. Of the 17 interviews that were rated, the principal investigator and the advanced clinical graduate students agreed 100% that women in the PTSD and pain and PTSD only groups met DSM-IV criteria for PTSD, and that women in the pain only and control groups did not meet criteria for PTSD.

Participant demographics are found in Table 3.3. Chi-square analyses indicated that there were no significant group differences in race [χ^2 (3, *N*=43) = 1.91, *ns*], income [χ^2 (24, *N*=41) = 23.32, *ns*], or education [χ^2 (21, *N*=44) = 24.05, *ns*]. However, there

were significant differences between groups regarding relationship status [χ^2 (9, N=44) = 22.60, $p < .007$]. Women in the PTSD and pain and pain only groups were more likely to be married or divorced, whereas women in the PTSD only and control groups were more likely to be single.

Table 3.3. Participant demographics

	PTSD & Pain	PTSD Only	Pain Only	Control
Race				
Caucasian	100%	100%	90%	93%
Asian	0%	0%	0%	7%
Unknown	0%	0%	10%	0%
Education				
< High school	10%	0%	0%	0%
High school diploma	0%	33%	20%	33%
Associate's degree	30%	0%	10%	7%
Partial undergraduate	20%	56%	40%	40%
Bachelor's degree	30%	0%	20%	0%
Partial graduate	0%	11%	0%	7%
Master's degree	10%	0%	10%	7%
Doctorate	0%	0%	0%	7%
Family Income				
< 30,000	60%	44%	20%	47%
30,000 – 39,999	0%	22%	30%	0%
40,000 – 49,999	20%	11%	0%	13%
≥50,000	20%	11%	40%	33%
Unknown	0%	12%	10%	7%
Relationship Status				
Single	0%	78%	20%	73%
Living with partner	10%	0%	20%	7%
Married	60%	11%	50%	20%
Divorced	30%	11%	10%	0%

There were no significant group differences regarding the use of birth control pills [χ^2 (3, N=44) = 5.38, *ns*]. Significant group differences in menstrual status were reported [χ^2 (3, N=44) = 8.15, p = .04]. Women in the PTSD only and control groups were more likely to be premenstrual than women in the other two groups. Women in the PTSD and

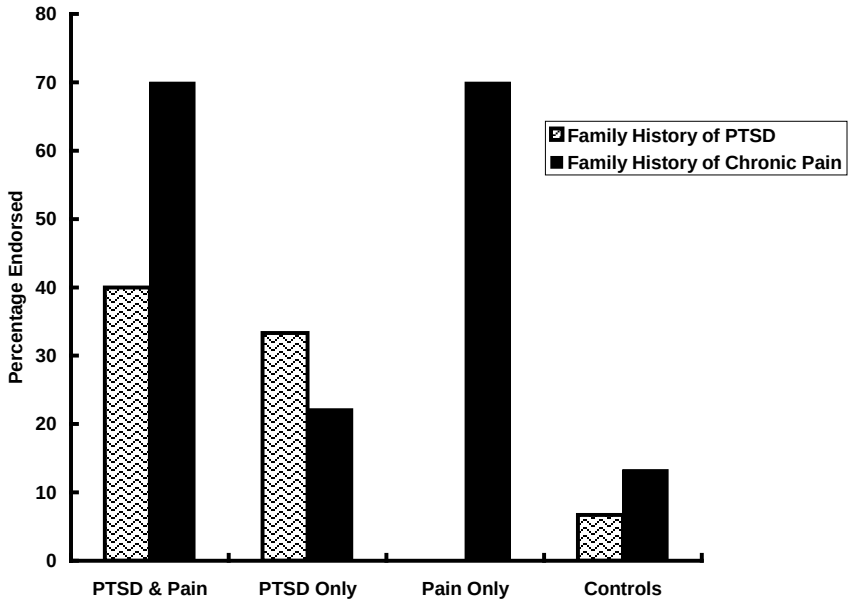
pain and pain only groups reported more postmenopausal status than women in the other two groups (see Table 3.4. for specific percentages).

Table 3.4. Menstrual Cycle Demographics

	PTSD & Pain	PTSD Only	Pain Only	Control
Birth Control	20%	56%	20%	53%
Pre-menopausal	60%	89%	50%	93%
Post-menopausal	40%	11%	50%	7%

There were significant group differences regarding family history of PTSD [χ^2 (3, N=44) =8.15, p = .04]. Women with PTSD and chronic pain and PTSD only were more likely to report a family history of PTSD than those without a diagnosis of PTSD ([pain only and control groups). There were also significant group differences in family history of chronic pain [χ^2 (3, N=44) =13.02, p = .005]. Women who reported experiencing chronic pain (PTSD and pain and pain only groups) were more likely to report a family history of chronic pain than those without chronic pain (PTSD only and control groups (see Figure 3.1 for specific percentages).

Figure 3.1. Family History of PTSD and Chronic Pain



Hypothesis Results: Experimental Stressor

Hypothesis One

According to hypothesis one, participants in the clinical group (for combination hypotheses, the three clinical groups combined are referred to as the clinical group) were predicted to demonstrate: a) lower baseline cortisol levels prior to the experimental stressor, and b) blunted salivary cortisol levels following the experimental stressor as compared to controls. Two participants were considered outliers (at least 2 standard deviations above the mean) and were removed from statistical analyses for cortisol related hypotheses. First, comparisons of baseline cortisol levels were assessed using a one-way ANOVA with incorporated planned comparisons. Planned comparisons in baseline cortisol levels between the clinical group and control group were not significant (t = -.78, df = 38, *ns*). Post-hoc power analyses were completed for t-test results on

baseline levels of cortisol between the clinical and control groups. Given the observed effect size and variability within the current study, power analyses revealed that a sample size of 40 would result in a power of .18. In addition, a one-way ANOVA did not reveal a significant difference between the groups on baseline cortisol levels [$F(3,41) = .36$, *ns*].

Planned comparisons in cortisol levels across occasions for the clinical group and control group were completed within a 2 (Group: clinical group, control) X 5 (Occasion: baseline, 10 minutes post-stress induction, 20 minutes post-stress induction, 40 minutes post-stress induction, and 60 minutes post-stress induction) repeated measures ANOVA. Given that the test of sphericity was found to be significant, degrees of freedom were adjusted using the Greenhouse-Geisser correction. The Group x Occasion interaction effect was not significant [$F(2.50, 99.91) = .66$, *ns*]. The main effect for Group [$F(1, 40) = .67$, *ns*] was not significant. The main effect for Occasion was significant [$F(2.50, 99.91) = 5.89$, $p < .01$, partial $\eta^2 = .13$, power = .92].

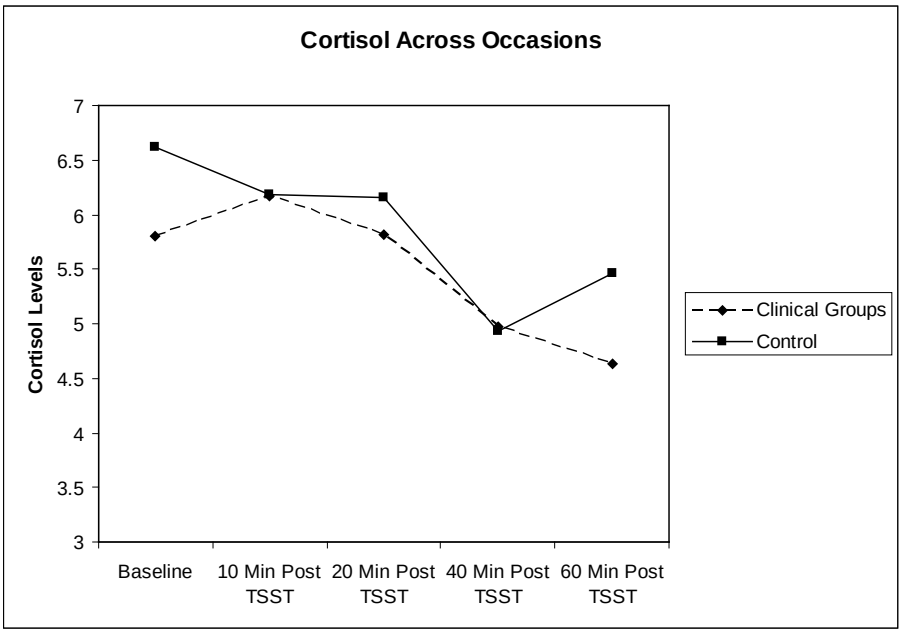
Paired samples t-tests revealed that baseline levels of cortisol were higher than those at 40 [$t = 2.56$, $df = 41$, $p = .01$] and 60 min [$t = 2.96$, $df = 41$, $p = .01$]. Cortisol levels at 10 minutes following the TSST were greater than those at 40 minutes [$t = 3.70$, $df = 41$, $p = .01$] and at 60 minutes [$t = 3.62$, $df = 41$, $p = .01$]. Finally, cortisol levels at 20 minutes were higher than those at 40 minutes [$t = 3.82$, $df = 41$, $p = .01$] and at 60 minutes [$t = 3.38$, $df = 51$, $p = .01$]. All other paired samples t-tests did not reveal any significant differences in cortisol levels. Group averages are presented in Table 3.5 and Figure 3.2.

Table 3.5. Cortisol Means (nmol/L)Across Time Periods

Time Period	Control	Clinical Group	Total Sample
Baseline	6.62_a (3.94)	5.80_a (2.73)	6.09 (3.19)
10 Min Post TSST	6.19_a (3.16)	6.17_a (3.78)	6.18 (3.54)
20 Min Post TSST	6.16_a (3.04)	5.82_a (3.70)	5.94 (3.45)
40 Min Post TSST	4.93_a (2.88)	4.97_a (3.04)	4.96 (2.95)
60 Min Post TSST	5.46_a (3.08)	4.63_a (2.44)	4.93 (2.68)
Total Sample	5.87 (.74)	5.48 (.55)	

Note. Standard deviations are in parentheses. Means with different subscripts differ significantly at $p < 05$.

Figure 3.2 Cortisol Levels across Occasions



To further assess for any group differences, a 4 (Group: PTSD, pain, PTSD and pain, control) X 5 (Occasion: baseline, 10 minutes post-stress induction, 20 minutes post-stress induction, 40 minutes post-stress induction, and 60 minutes post-stress induction) repeated measures ANOVA was completed. Given that the test of sphericity was found to be significant, degrees of freedom were adjusted using the Greenhouse-Geisser correction. The Group x Occasion interaction effect was not significant [$F(7.41, 93.84) = .45$, *ns*]. The main effect for Group [$F(1, 38) = .63$, *ns*] was not significant. The main effect for Occasion was significant [$F(2.47,93.84) = 5.97$, $p = .002$, partial $\eta^2 = .14$. Please refer to planned comparisons for paired samples t-test analyses (see Figure 3.3 and refer to table 3.6 for individual group averages). A logarithmic transformation of the cortisol values yielded the same results. The interaction effect for Group x Occasion was not significant when age was included as a covariate [$F(7.47, 87.12) = .84$, *ns*]. Similarly, the interaction effect for Group x Occasion was not significant when BMI was used as a covariate [$F(7.47, 84.58) = .87$, *ns*].

Figure 3.3. Cortisol levels across occasions: Individual Groups

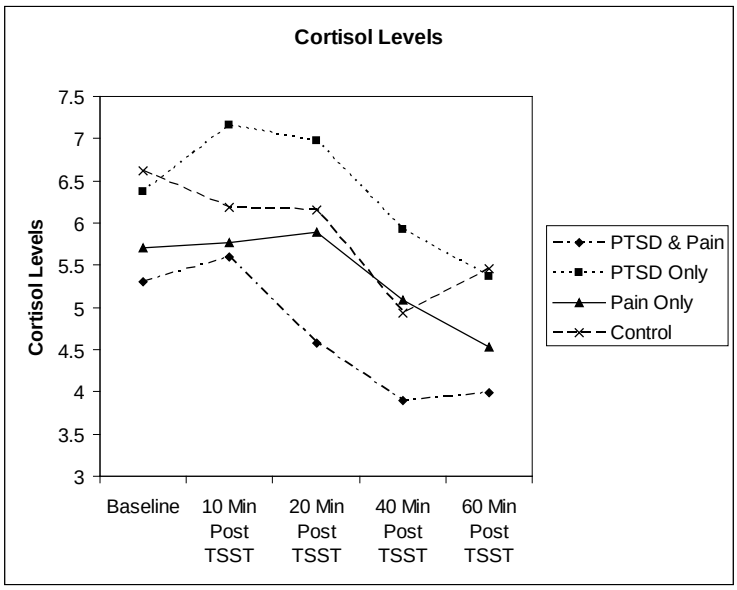


Table 3.6. Cortisol Means (nmol/L) Across Time Periods: Individual Groups

Time Period	PTSD & Pain	PTSD Only	Pain Only	Control	Total Sample
Baseline	5.31_a (2.07)	6.37_a (3.26)	5.70_a (2.94)	6.62_a (3.94)	6.09 (3.19)
10 Min Post TSST	5.60_a (2.82)	7.16_a (4.85)	5.77_a (3.63)	6.19_a (3.16)	6.18 (3.53)
20 Min Post TSST	4.58_a (2.14)	6.98_a (4.13)	5.89_a (4.44)	6.16_a (3.04)	5.94 (3.45)
40 Min Post TSST	3.89_a (1.79)	5.93_a (3.55)	5.09_a (3.42)	4.93_a (2.88)	4.96 (2.94)
60 Min Post TSST	3.99_a (1.80)	5.37_a (2.80)	4.53_a (2.69)	5.46_a (3.08)	4.93 (2.68)
Total Sample	4.68 (.96)	6.36 (.96)	5.40 (.96)	5.87 (.74)	

Note. Standard deviations are in parentheses. Means with different subscripts differ significantly at $p < 05$.

Ancillary Findings

Cortisol Levels within Groups. Although not hypothesized, analyses were conducted to determine within group differences in cortisol levels across the stress task. As the test of sphericity was found to be significant across occasions in the PTSD and pain group, degrees of freedom were adjusted using the Greenhouse-Geisser correction. There was a significant Occasion effect for women in the PTSD and pain group [$F(4, 32) = 2.87$, $p = .04$, partial $\eta^2 = .26$]. Paired samples t-tests revealed that cortisol levels at 10 min were greater than those at 40 min [$t = 2.65$, $df = 8$, $p = .03$], 60 min [$t = 2.27$, $df = 8$, $p = .05$]. Cortisol levels at 20 min were greater than those at 40 min [$t = 2.39$, $df = 8$, $p =$

.05] for women in the PTSD and pain group. There were no significant Occasion effects for women in the PTSD only [$F(4,32) = 1.89$, *ns*] pain only [$F(4,32) = 1.19$, *ns*], and control groups [$F(4,32) = 1.19$, *ns*].

Hypothesis Two

It was predicted that group means for anxious mood would exist on a continuum across the measurement occasions of the experimental task (POMS anxiety subscale). Participants in the PTSD and pain group were anticipated to report the highest levels of anxious mood, followed by the PTSD and the pain only groups. Participants in the control group were anticipated to report the lowest levels of anxious mood. Two participants considered to be outliers for cortisol results were excluded from experimental session analyses. To test this hypothesis, the PTSD only and pain only were combined to form a PTSD only plus pain only group.

As the test of sphericity was found to be significant in the 2 (Groups: PTSD and pain, PTSD only plus pain only) X 5 (Occasions) mixed-factor ANOVA, degrees of freedom were adjusted using the Greenhouse-Geisser correction. The main effect for Occasion [$F(1.92,49.95) = 21.24$, $p < .0001$, partial $\eta^2 = .45$] and Group [$F(1,26) = 13.18$, $p < .001$, partial $\eta^2 = .34$] were significant. The test of the Group x Occasion interaction effect was marginally significant [$F(1.92, 49.95) = 2.65$, $p = .08$, partial $\eta^2 = .09$]. The PTSD and pain group reported significantly higher levels of anxious mood than the PTSD only and pain only group. Refer to Table 3.7 for group averages.

Paired samples t-tests indicated that baseline levels of anxious mood were significantly higher than levels of anxious mood at 10 minutes [$t = -.2.58$, $df = 27$, $p = .02$]. Levels of anxious mood were significantly lower from baseline at 20 minutes [$t =$

2.95, *df* = 27, *p* = .007], 40 minutes [*t* = 4.00, *df* = 27, *p* = .001], and at 60 minutes [*t* = 4.82, *df* = 27, *p* = .0001]. Levels of anxious mood were significantly higher at 10 minutes following the TSST than at 20 [*t* = 4.98, *df* = 27, *p* = .0001], 40 [*t* = 5.29, *df* = 27, *p* = .0001], and 60 [*t* = 5.79, *df* = 27, *p* = .0001] minutes. Levels of anxious mood at 20 minutes were significantly higher than anxious mood at 60 minutes [*t* = 3.75, *df* = 27, *p* = .001]. Finally, levels of anxious mood at 40 minutes were significantly higher than those at 60 minutes [*t* = 3.06, *df* = 27, *p* = .005].

As the test of sphericity was found to be significant in the 2 (Groups: PTSD only plus pain only, control) X 5 (Occasions) mixed-factor ANOVA, degrees of freedom were adjusted using the Greenhouse-Geisser correction. The main effect for Occasion [*F*(1.64, 52.59) = 16.98, *p* <.0001, partial η^2 = .35] was significant. The Group [*F*(1,32) = .96, *ns*] and Group x Occasion interaction [*F*(1.64, 52.59) = 1.16, *ns*] were not significant.

Paired samples t-tests indicated that baseline levels of anxious mood were significantly higher than levels of anxious mood at 10 minutes [*t* = -.3.81, *df* = 33, *p* = .001]. Levels of anxious mood were significantly lower from baseline at 20 minutes [*t* = 2.79, *df* = 33, *p* = .009], 40 minutes [*t* = 2.50, *df* = 33, *p* = .02], and at 60 minutes [*t* = 3.54, *df* = 33, *p* = .001]. Levels of anxious mood were significantly higher at 10 minutes following the TSST than at 20 [*t* = 5.14, *df* = 33, *p* = .0001], 40 [*t* = 4.50, *df* = 33, *p* = .0001], and 60 [*t* = 4.84, *df* = 33, *p* = .0001] minutes. Finally, levels of anxious mood at 40 minutes were significantly higher than those at 60 minutes [*t* = 2.03, *df* = 33, *p* = .05]. All other paired samples t-tests were not significant. Refer to Table 3.7 for group averages.

Table 3.7. POMS Anxiety Levels Across Time Points

Occasion	PTSD & Pain	PTSD Only & Pain Only	Control	Total Sample
Baseline	13.33_a (4.80)	6.26_a (2.83)	5.67_a (3.39)	7.53 (4.56)
10 Min Post TSST	13.11_a (5.65)	10.26_a (6.45)	7.93_a (5.68)	10.05 (6.19)
20 Min Post TSST	8.67_a (3.50)	5.16_a (2.52)	4.07_a (1.28)	5.51 (2.93)
40 Min Post TSST	7.56_a (3.28)	4.58_a (1.77)	4.73_a (1.53)	5.26 (2.37)
60 Min Post TSST	6.11_a (2.15)	4.00_a (0.75)	4.27_a (1.03)	4.53 (1.47)
Total Sample	9.76 (.77)	6.05 (.53)	5.33 (.60)	

Note. Standard deviations are in parentheses. Means with different subscripts differ significantly at $p < .05$

As the test of sphericity was found to be significant in the 4 (Groups) X 5 (Occasions) mixed-factor ANOVA, degrees of freedom were adjusted using the Greenhouse-Geisser correction. The main effect for Occasion [$F(1.90, 74.09) = 28.42$, $p = .0001$, partial $\eta^2 = .42$] and Group [$F(3,39) = 7.82$, $p = .0001$, partial $\eta^2 = .38$] were significant. The test of the Group x Occasion interaction effect was also significant [$F(5.70, 74.09) = 2.32$, $p = .05$, partial $\eta^2 = .15$]. Refer to Table 3.8 and Figure 3.4 for group averages.

F tests for the significant Group x Occasion interaction effect revealed the following: baseline [$F(3, 42) = 10.47$, $p < .0001$, partial $\eta^2 = .45$, power = .99], 10 min [$F(3, 42) = 2.41$, *ns*], 20 min [$F(3, 42) = 6.81$, $p < .001$, partial $\eta^2 = .34$, power = .96], 40

min[$F(3, 42) = 4.48$, $p = .009$, partial $\eta^2 = .26$, power = .85], and 60 min [$F(3, 42) = 6.07$, $p < .002$, partial $\eta^2 = .32$, power = .94].

Tukey's HSD indicated that participants in the PTSD and pain group reported significantly higher baseline levels of anxious mood than the PTSD only (Mean Difference = 6.56, *SE* = 1.66, $p = .002$), pain only (Mean Difference = 7.53, *SE* = 1.62, $p < .001$), and control groups (Mean Difference = 7.67, *SE* = 1.49, $p < .001$). No significant group differences were demonstrated at 10 minutes following the TSST. The PTSD and pain group reported significantly higher levels of anxious mood at 20 minutes following the TSST than the PTSD only (Mean Difference = 3.57, *SE* = 1.16, $p = .02$), Pain only (Mean Difference = 3.47, *SE* = 1.13, $p = .02$), and control groups (Mean Difference = 4.60, *SE* = 1.04, $p < .001$). The PTSD and pain group reported significantly higher levels of anxious mood at 40 minutes following the TSST than the PTSD only (Mean Difference = 2.89, *SE* = 1.00, $p = .04$), pain only (Mean Difference = 3.06, *SE* = .98, $p = .02$) and control groups (Mean Difference = 2.82, *SE* = .90, $p = .02$). Finally, the PTSD and pain group reported significantly higher levels of anxious mood at 60 minutes following the TSST than the PTSD only (Mean Difference = 2.11, *SE* = .59, $p = .01$) and pain only (Mean Difference = 2.11, *SE* = .58, $p = .01$) groups (Group averages are presented in Table 3.6). Overall, the PTSD and pain group reported higher levels of anxious mood across all time points except at 10 minutes following the TSST (see Figure 3.3).

Paired samples t-tests indicated that baseline levels of anxious mood were significantly lower than levels of anxious mood at 10 minutes [$t = -3.28$, $df = 42$, $p = .002$], and significantly greater than at 20 minutes [$t = 3.63$, $df = 42$, $p = .001$], 40 minutes

[t = 3.75, df = 42, p = .001], and 60 minutes [t = 4.72, df = 42, p = .0001]. Levels of anxious mood were significantly higher at 10 minutes following the TSST than at 20 [t = 5.74, df = 42, p = .001], 40 [t = 5.53, df = 42, p = .0001], and 60 [t = 5.60, df = 42, p = .0001] minutes. Levels of anxious mood at 20 minutes were significantly higher than anxious mood at 60 minutes [t = 2.90, df = 42, p = .006]. Finally, levels of anxious mood at 40 minutes were significantly higher than those at 60 minutes [t = 2.30, df = 42, p = .005].

There was a significant within-group effect for women in the PTSD and pain group [F(2.04, 16.32) = 9.97, p = .001, partial η^2 = .56, power = .96], PTSD only group [F(1.59, 12.69) = 11.1, p = .003, partial η^2 = .58, power = .94], pain only group [F(1.46, 13.11) = 4.58, $p < .05$, partial η^2 = .34, power = .59], and control group [F(1.64, 22.97) = 4.78, $p < .05$, partial η^2 = .25, power = .68].

For the simple main effect for Group, Tukey's HSD indicated that women in the PTSD and pain group reported significantly higher baseline levels of anxious mood than at 60 minutes (Mean Difference = 7.22, SE = 1.71, p = .03) following the TSST. Anxious mood at 10 minutes (Mean Difference = 7.00, SE = 1.84, p = .05) was also significantly higher than levels of anxious mood at 60 minutes following the TSST. The PTSD only group reported a significantly higher levels of anxious mood at 10 minutes following the TSST than at 20 minutes (Mean Difference = 7.56, SE = 1.83, p = .03), 40 minutes (Mean Difference = 8.00, SE = 2.11, p = .05), and 60 minutes (Mean Difference = 8.67, SE = 2.29, p = .05). No significant differences across occasions were demonstrated for the pain only group. Finally, the control group reported a significant decrease in anxious mood from 10 to 20 minutes follow-up (Mean Difference = 3.87, SE = 1.29, $p < .05$).

Ancillary Analyses

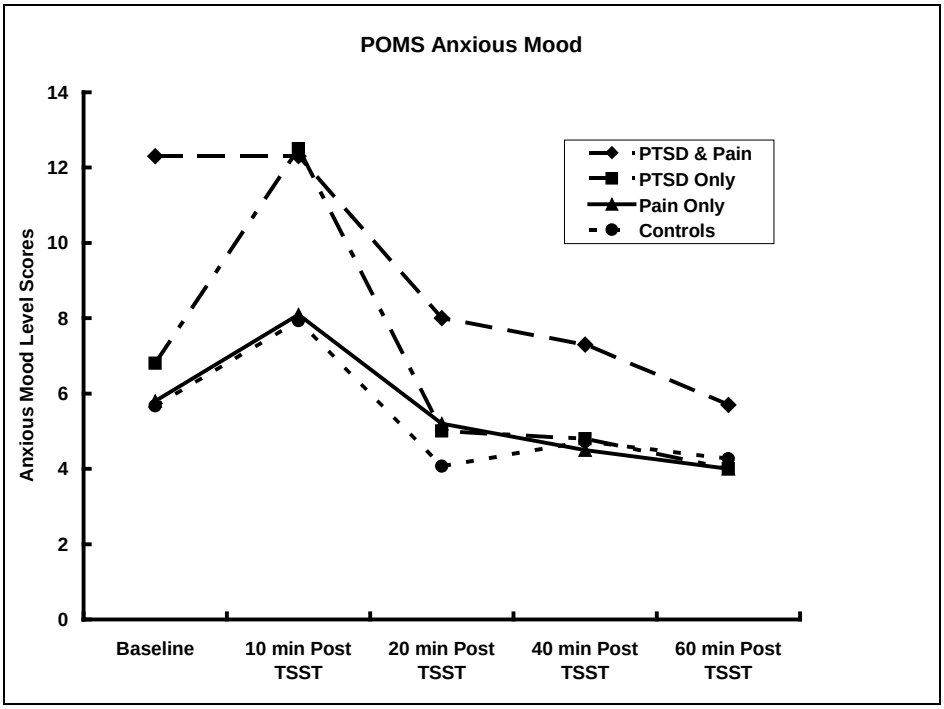
Since the PTSD and pain group reported significantly higher levels of anxious mood at baseline than the remaining three groups [$F(3,44) = 14.16$, $p < .0001$], a repeated measures ANOVA with baseline levels of anxious mood entered as a covariate was conducted on the remaining 4 occasions. The main effects for Occasion [$F(1.40, 54.18) = .61$, *ns*] and Group [$F(3, 38) = .55$, *ns*] were not significant. The Group x Occasion interaction effect was significant [$F(4.20, 54.18) = 2.96$, $p = .03$, partial $\eta^2 = .19$, power = .77]. A breakdown of the interaction revealed no significance differences between groups regarding anxious mood. Results continue to indicate that women in the PTSD and pain group reported the highest levels of anxious mood across most time points.

Table 3.8. POMS Anxiety Levels Across Time Points: Individual Groups

Occasion	PTSD & Pain	PTSD Only	Pain Only	Control	Total Sample
Baseline	13.33_a (4.80)	6.78_b (3.11)	5.80_b (2.62)	5.67_b (3.39)	7.53 (4.56)
10 Min Post TSST	13.11_a (5.65)	12.67_a (6.91)	8.10_a (5.47)	7.93_a (5.68)	10.05 (6.19)
20 Min Post TSST	8.67_a (3.50)	5.11_b (1.83)	5.20_b (3.12)	4.07_b (1.28)	5.51 (2.92)
40 Min Post TSST	7.56_a (3.28)	4.67_b (1.50)	4.50_b (2.07)	4.73_b (1.53)	5.26 (2.37)
60 Min Post TSST	6.11_a (2.15)	4.00_b (0.50)	4.00_b (.94)	4.27_b (1.03)	4.53 (1.47)
Total Sample	9.76 (.77)	6.64 (.77)	5.52 (.73)	5.33 (.60)	

Note. Standard deviations are in parentheses. Means with different subscripts differ significantly at $p < .05$

Figure 3.4. Anxious Mood across Experimental Task: Individual Groups



Visual Analog Stress Scale

Although not originally incorporated into the study hypotheses, participants completed the Visual Analog Stress scale immediately after completing the experimental stressor as a means of measuring their self-perceived stress levels during this task. A one way ANOVA demonstrated that no significant group differences were reported in self-reported stress levels in response to the TSST [$F(3,40) = .93$, *ns*]. In general, all groups reported experiencing moderate levels of stress during the TSST. Group averages are presented in Table 3.9.

Table 3.9. Visual Analog Stress Scale

	PTSD and pain	PTSD only	Pain only	Control
Mean VASS Score	4.89_a (1.36)	4.78_a (2.22)	4.50_a (1.41)	4.00_a (.76)

Note. Standard deviations are in parentheses. Means with different subscripts are significantly at $p < 05$.

Hypothesis Three

Participants in the three clinical groups were predicted to report greater use of negative coping strategies (i.e., denial, behavioral disengagement, self-blame), while participants in the control group were predicted to report greater use of positive coping strategies (i.e., active coping, positive reframing, acceptance). This trend was hypothesized to occur in both general use of coping strategies and coping strategies utilized in response to the TSST (general use of coping strategies will be discussed with questionnaire data).

Planned comparisons did not indicate any significant group differences between the clinical group and the control group for levels of denial [$t = .21$, df = 38, *ns*], behavioral disengagement [$t = .62$, df = 38, *ns*], self-blame [$t = 1.57$, df = 38, *ns*], active coping [$t = 1.33$, df = 38, *ns*], positive reframing [$t = .45$, df = 38, *ns*], and acceptance [$t = .27$, df = 38, *ns*]. Refer to Table 3.10 for group averages.

Table 3.10. Brief COPE Scores in Response to TSST

Subscales	Control	Clinical Group
Active Coping	2.47_a (.58)	2.81_a (.91)
Positive Reframing	2.47_a (.95)	2.59_a (.81)
Acceptance	2.67_a (.98)	2.74_a (.74)
Denial	1.37_a (.61)	1.41_a (.61)
Behavioral Disengagement	1.30_a (.49)	1.41_a (.56)
Self-Blame	1.77_a (.62)	2.15_a (.86)

Note. Standard deviations are in parentheses. Means with different subscripts differ significantly at $p < .05$.

A series of one-way ANOVAs were completed to assess for additional group differences. These analyses indicated a near significant group difference in self-blame [$F(3,41) = 2.65$, $p = .06$] in response to the TSST. No significant group differences were demonstrated for denial [$F(3,41) = .85$, *ns*], behavioral disengagement [$F(3,41) = .80$, *ns*], active coping [$F(3,41) = 1.28$, *ns*], positive reframing [$F(3,41) = .32$, *ns*], or acceptance [$F(3,41) = .14$, *ns*]. Individual group averages are presented in Table 3.11.

Table 3.11. Individual Group Brief COPE Scores in Response to TSST

Subscales	PTSD & Pain	PTSD Only	Pain Only	Control
Active Coping	2.50_a (.75)	2.94_a (.77)	3.00_a (1.17)	2.47_a (.58)
Positive Reframing	2.67_a (1.00)	2.72_a (.91)	2.39_a (.49)	2.47_a (.95)
Acceptance	2.61_a (.86)	2.83_a (.66)	2.78_a (.75)	2.67_a (.98)
Denial	1.67_a (.83)	1.28_a (.36)	1.28_a (.51)	1.37_a (.61)
Behavioral Disengagement	1.61_a (.65)	1.28_a (.44)	1.33_a (.56)	1.30_a (.49)
Self-Blame	2.17_a (.61)	2.56_a (.85)	1.72_a (.97)	1.77_a (.62)

Note. Standard deviations are in parentheses. Means with different subscripts differ significantly at $p < .05$.

Hypothesis Results: Questionnaire Data

Hypothesis Four

It was predicted that depression levels would exist on a continuum. Women in the PTSD and pain group were anticipated to report the highest levels of depression symptoms, followed by women in the PTSD only and the pain only groups. Women in the control group were anticipated to report the lowest level of depression symptoms. Planned comparisons between the PTSD and pain group and the combined PTSD only and pain only groups did not indicate any significant group differences in levels of depression [$t = 1.82$, df = 39, *ns*]. Similarly, planned comparisons between the combined PTSD only and pain only groups and the control group did not demonstrate any

significant group differences [t = 1.74, df = 39, *ns*]. Please refer to table 3.12 for group averages.

Table 3.12. Depression Severity Scores

	PTSD & Pain	PTSD Only & Pain Only	Control
BDI-II	19.78_a (3.63)	13.58_a (11.08)	8.43_a (5.81)

Note. Standard deviations are in parentheses. Means with different subscripts differ significantly at $p < .05$

To further assess for individual group differences, a one-way ANOVA indicated significant group differences in levels of depression on the BDI-II [$F(3,41) = 7.19$, $p < .001$]. Post-hoc analyses revealed that the PTSD and pain and PTSD only groups reported significantly higher levels of depression than the pain only and control groups. BDI-II scores reported by the PTSD and pain and PTSD only groups were indicative of mild to moderate levels of depression, whereas scores reported by the pain only and control groups were in line with minimal levels of depression. Group averages are presented in Table 3.13.

Table 3.13. Depression Severity Scores: Individual Groups

	PTSD and pain	PTSD only	Pain only	Control
BDI-II	19.78_a (3.63)	19.22_a (12.31)	8.5_b (7.10)	8.43_b (5.81)

Note. Standard deviations are in parentheses. Means with different subscripts differ significantly at $p < .05$.

Hypothesis Five

It was predicted that all three clinical groups would report higher levels of learned helplessness than the control group. Planned comparisons did not reveal significant

group differences between the three clinical groups and the control group for Internality (t = 1.26, df = 39, *ns*), Stability (t = 1.03, df = 39, *ns*), or Globality (t = .43, df = 39, *ns*) subscales on the Expanded Attributional Style Questionnaire. Group averages are presented in Table 3.14.

Table 3.14. Expanded Attributional Style Questionnaire Subscales

ASQ Subscales	Control	Clinical Group
Internality	4.28_a (.94)	4.58_a (.62)
Stability	4.28_a (1.21)	4.58_a (.68)
Globality	4.09_a (1.10)	4.19_a (.63)

Note. Standard deviations are in parentheses. Means with different subscripts differ significantly at $p < .05$.

Individual group differences in learned helplessness were completed using a 4 (Group: PTSD, pain, PTSD and pain, control) X 3 MANOVA with the 3 ASQ subscales (Internality, Stability, Globality) as criterion variables. Results did not indicate a significant effect for Group on the combined dependent construct of learned helplessness [F(9, 90.20) = .77, *ns*; Wilks' Lambda = .86; partial η^2 = .05]. Analysis of individual dependent variables indicated no significant Group effects for Internality [F(3, 39) = .72, *ns*, partial η^2 = .05]; Stability [F(3, 39) = .89, *ns*, partial η^2 = .06]; or Globality [F(3, 39) = 1.13, *ns*, partial η^2 = .08]. Individual group averages are presented in Table 3.15.

Table 3.15. Expanded Attributional Style Questionnaire Subscales: Individual Groups

ASQ Subscales	PTSD & Pain	PTSD Only	Pain Only	Control
Internality	4.74_a (.73)	4.53_a (.25)	4.49_a (.53)	4.28_a (.94)
Stability	4.89_a (.68)	4.45_a (.30)	4.40_a (.52)	4.28_a (1.21)
Globality	4.55_a (.61)	4.17_a (.27)	3.88_a (.63)	4.09_a (1.10)

Note. Standard deviations are in parentheses. Means with different subscripts differ significantly at $p < .05$.

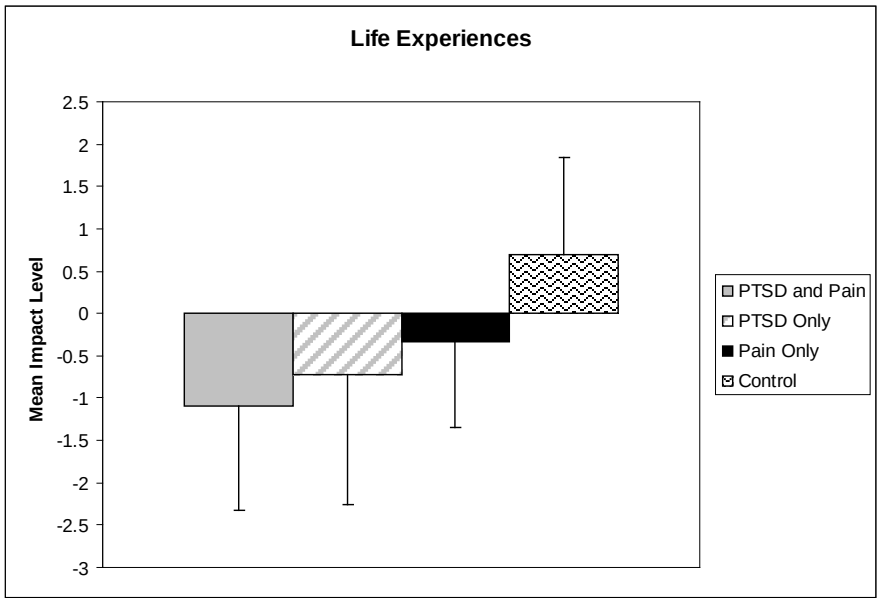
Hypothesis Six

Participants in all three clinical groups were predicted to report greater levels of negative life events and general stress than the control group. Participants in the three clinical groups were also anticipated to report viewing ambiguous situations as being more threatening. To test these hypotheses group differences on self-reported measures (LES, IUS-12, PSS) were assessed in a series of one-way ANOVAs.

Life Experiences Survey. Planned comparisons revealed significant group differences between the three clinical groups and the control group for the mean impact of life experiences in the previous year [t = -3.41, df = 35, $p < .01$]. Overall, the three clinical groups combined reported significantly more negative mean impacts of life events than the control group. Refer to Table 3.16 for planned comparison group averages. Similarly, a one-way ANOVA indicated that there was a significant effect for mean impact levels of life experiences in the previous year [$F(3, 38) = 4.22$, $p = .01$, partial $\eta^2 = .27$]. Post-hoc analyses revealed that the PTSD and pain and PTSD only groups reported significantly more negative impact levels of life experiences in the past

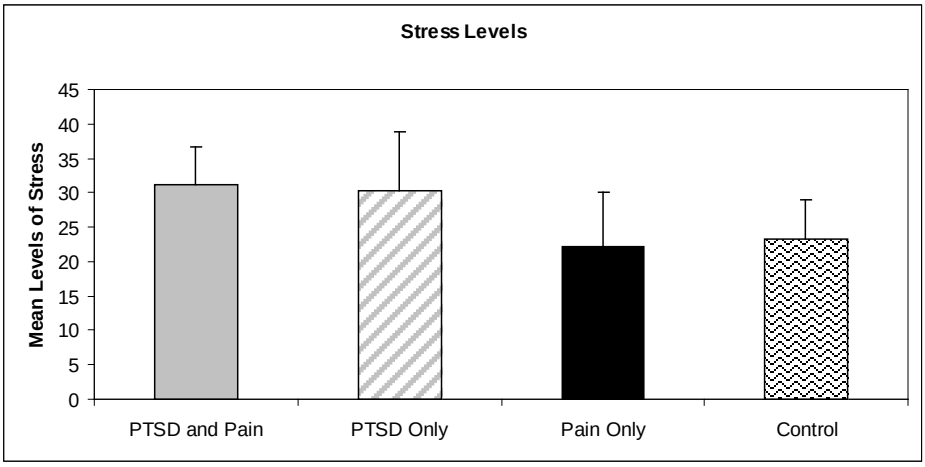
year than the control group (see Figure 3.5). Refer to Table 3.17 for individual group averages.

Figure 3.5. Mean Impact of Life Experiences in past Year



Perceived Stress Scale. Planned comparisons indicated significant group differences between the three clinical groups and the control group on general levels of perceived stress in the past month [$t = 2.15$, df = 39, $p < .05$]. Refer to Table 3.16 for planned comparison group averages. A one-way ANOVA also indicated that there was a significant effect for overall ratings of stress [$F(3, 42) = 4.79$, $p < .01$, partial $\eta^2 = .23$]. Post-hoc analyses demonstrated that the PTSD and pain group reported significantly higher levels of stress than the pain only and control groups in the past month (see Figure 3.6). Refer to Table 3.17 for individual group averages.

Figure 3.6. General Levels of Stress



Intolerance of Uncertainty Scale – Short Version. Planned comparisons between the three clinical groups and the control group did not reveal significant group differences for intolerance of uncertainty [t = .94, df = 39, *ns*]. Refer to Table 3.16 for planned comparison group averages. Similarly, a one-way ANOVA did not indicate significant group differences in levels of intolerance of uncertainty [$F(3, 42) = 1.43$, *ns*, partial η^2 = .03]. Refer to Table 3.17 for individual group averages.

Table 3.16. Stress Levels and Negative Life Experiences

	Control	Combined Clinical Groups
LES: Mean Impact	$.69_a$ (1.15)	$-.69_b$ (1.26)
PSS	23.20_a (5.70)	27.71_b (8.31)
IUS-12	29.47_a (8.39)	32.39_a (11.29)

Note. Standard deviations are in parentheses. Means with different subscripts differ significantly at $p < .05$.

Table 3.17. Stress Levels and Negative Life Experiences: Individual Groups

	PTSD & Pain	PTSD Only	Pain Only	Control
LES: Mean Impact	-1.09_{a} (1.23)	$-.72_{a}$ (1.53)	$-.34_{ab}$ (1.01)	$.69_{b}$ (1.15)
PSS	31.22_{a} (5.48)	30.22_{ab} (8.58)	22.20_{b} (7.86)	23.20_{b} (5.70)
IUS-12	37.33_{a} (12.93)	31.55_{a} (11.39)	28.70_{a} (8.83)	29.47_{a} (8.39)

Note. Standard deviations are in parentheses. Means with different subscripts differ significantly at $p < .05$.

Ancillary Findings

Frequency of Life Events. Although not originally included in hypotheses, group differences on self-reported frequencies of negative and positive life events (i.e. LES) were assessed in a series of one-way between subjects ANOVAs. Since the assumption of homogeneity of variance was violated when assessing for group differences in total frequency of positive and negative life events in the previous year, two Kruskal-Wallis one-way between-subjects analyses were completed. There was not a significant effect for frequency of positive life events [$X^2(3, N=39) = 2.34, ns$] or frequency of negative life events [$X^2(3, N=39) = 4.68, ns$]. Please refer to Table 3.18 for group averages.

Table 3.18. Frequency of negative and positive life events in past year

	PTSD & Pain	PTSD Only	Pain Only	Control
LES: Freq of Negative Life Events	9.14_{a} (6.59)	6.33_{a} (3.94)	4.44_{a} (2.70)	4.36_{a} (5.05)
LES: Freq of Positive Life Events	3.00_{a} (2.58)	2.33_{a} (1.58)	2.33_{a} (1.32)	4.43_{a} (3.63)

Note. Standard deviations are in parentheses. Means with different subscripts differ significantly at $p < .05$.

Hypothesis Seven

As mentioned earlier, participants in the three clinical groups were predicted to report greater use of general negative coping strategies (i.e., denial, behavioral disengagement, self-blame), while participants in the control group were predicted to report greater use of general positive coping strategies (i.e., active coping, positive reframing, acceptance).

Planned comparisons between the clinical group and the control group with regards to general use of coping strategies indicated significant group differences in levels of self-blame [$t = 2.01$, df = 38, $p = .05$]. In particular, the three clinical groups combined reported higher levels of self-blame than the control group. Planned comparisons did not indicate any significant group differences in denial [$t = -.20$, df = 38, *ns*], behavioral disengagement [$t = .15$, df = 38, *ns*], active coping [$t = -.10$, df = 38, *ns*], positive reframing [$t = -.97$, df = 38, *ns*], or acceptance [$t = -.85$, df = 38, *ns*]. Refer to Table 3.19 for planned comparison group averages.

Table 3.19. General Brief COPE

Subscales	Control	Clinical Group
Active Coping	2.75_{a} (.85)	2.73_{a} (.92)
Positive Reframing	2.68_{a} (.97)	2.39_{a} (.86)
Acceptance	2.96_{a} (.89)	2.71_{a} (.89)
Denial	1.21_{a} (.47)	1.18_{a} (.46)
Behavioral Disengagement	1.39_{a} (.71)	1.41_{a} (.51)
Self-Blame	1.50_{ab} (.71)	1.89_{a} (.72)

Note. Standard deviations are in parentheses. Means with different subscripts differ significantly at $p < .05$.

A series of one-way ANOVAs were completed to further assess for group differences. There were significant group differences in self-blame [$F(3,41) = 5.82$, $p<.01$]. Post-hoc analyses revealed that the PTSD only group reported significantly higher levels of self-blame than the pain only and control groups. The PTSD and pain group also reported significantly higher levels of self-blame than the pain only group. No significant group differences were demonstrated for denial [$F(3,41) = 1.19$, *ns*], behavioral disengagement [$F(3,41) = 1.32$, *ns*], active coping [$F(3,41) = .87$, *ns*], positive reframing [$F(3,41) = .74$, *ns*], or acceptance [$F(3,41) = .40$, *ns*]. Individual group averages are presented in Table 3.20.

Table 3.20. General Brief COPE: Individual Groups

Subscales	PTSD & Pain	PTSD Only	Pain Only	Control
Active Coping	2.72_{a} (.67)	2.39_{a} (.99)	3.05_{a} (1.01)	2.75_{a} (.85)
Positive Reframing	2.56_{a} (.85)	2.11_{a} (.93)	2.50_{a} (.85)	2.68_{a} (.97)
Acceptance	2.72_{a} (.83)	2.56_{a} (.95)	2.85_{a} (.97)	2.96_{a} (.89)
Denial	1.39_{a} (.70)	1.17_{a} (.35)	1.0_{a} (.00)	1.21_{a} (.47)
Behavioral Disengagement	1.67_{a} (.50)	1.44_{a} (.58)	1.15_{a} (.34)	1.39_{a} (.71)
Self-Blame	2.17_{bc} (.66)	2.28_{c} (.67)	1.30_{a} (.42)	1.50_{ab} (.71)

Note. Standard deviations are in parentheses. Means with different subscripts differ significantly at $p < .05$.

Hypothesis Eight

It was predicted that only participants in the PTSD group and PTSD and pain group would report evidence of a PTSD specific learned alarm. Similarly, it was predicted that only participants in the pain group and PTSD and pain group would report evidence of a pain specific learned alarm. All three clinical groups were predicted to report higher levels of anxiety sensitivity than the control group. Self-report measures (i.e., IES-Revised, PASS-20, ASI) associated with evidence of a learned alarm were assessed in a series of one-way between subjects ANOVAs.

Pain Anxiety Symptom Scale. No significant group differences between the two chronic pain groups and the PTSD only and control groups were demonstrated with planned comparisons for pain related avoidance [$t = 1.44$, df = 37, *ns*], cognitions [$t = 1.23$, df = 37, *ns*], physiological symptoms [$t = 1.08$, df = 37, *ns*], or total PASS scores [*t*

= 1.59, df = 37, *ns*]; however, a marginal significant difference was found for pain related fear [*t* = 1.90, df = 37, *p* = .06]. Refer to Table 3.21 for planned comparison group averages.

Table 3.21. Pain Related Anxiety

	Control & PTSD Only	PTSD & Pain & Pain Only
PASS: Total	31.59_a (21.55)	41.63_a (17.73)
PASS: Fear	5.91_a (4.33)	8.89_a (5.50)
PASS: Avoidance	9.05_a (5.67)	11.47_a (4.67)
PASS: Cognitive	10.73_a (6.95)	13.53_a (5.36)
PASS: Physiological	5.91_a (4.51)	7.74_a (5.26)

Note. Standard deviations are in parentheses. Means with different subscripts differ significantly at $p < .05$.

A one-way ANOVA did not indicate any significant group differences in the total score for pain related anxiety [$F(3,40) = 1.40$, *ns*]. Similarly, no significant group differences were found on the avoidance [$F(3,40) = .85$, *ns*], cognitive [$F(3,40) = 1.07$, *ns*], or physiological [$F(3,40) = 1.90$, *ns*] subscales of the PASS; however, a marginal significant group difference was found for pain related fear [$F(3,40) = 2.36$, $p = .09$]. In particular, the PTSD and pain group reported the highest levels of pain related fear, followed by the pain only group, PTSD only group, and control groups respectively. Please refer to Table 3.24 for individual group averages.

Impact of Event Scale – Revised. Planned contrasts between the two PTSD groups and the pain only and control groups indicated significant group differences for total PTSD related symptoms on the IES-R [$t = 2.14$, df = 39, $p < .05$] and symptoms of hyperarousal [$t = 3.21$, df = 39, $p < .01$]. Inspection of the data suggested that participants with PTSD reported significantly higher levels of total PTSD symptoms and symptoms of hyperarousal than those without PTSD. Planned contrasts did not indicate significant group differences for levels of avoidance [$t = 1.70$, df = 39, *ns*] or intrusion [$t = .94$, df = 39, *ns*]. Refer to Table 3.22 for planned comparison group averages.

Table 3.22. PTSD Related Anxiety

	Control & Pain Only	PTSD & Pain & PTSD Only
IES: Avoidance	1.03_a (.84)	1.45_a (.80)
IES: Intrusion	1.25_a (.80)	1.51_a (.95)
IES: Hyperarousal	$.87_a$ (.73)	1.69_b (.86)
IES: Total	23.40_a (14.28)	33.89_b (16.67)

Note. Standard deviations are in parentheses. Means with different subscripts differ significantly at $p < .05$.

A series of one-way between subjects ANOVAs indicated significant group differences for symptoms of hyperarousal [$F(3,42) = 2.59$, $p = .01$]. Post-hoc analyses revealed that the PTSD and pain group reported significantly higher levels of hyperarousal than the control group. No significant group differences were found for total scores on the IES-R [$F(3,42) = 1.69$, *ns*] or for the avoidance [$F(3,42) = 1.50$, *ns*]

and intrusion subscales [$F(3,42) = .46$, *ns*]. Please refer to Table 3.23 for individual group averages.

Anxiety Sensitivity Index. Planned contrasts between the three clinical groups and the control group did not reveal significant group differences [$t = 1.11$, df = 38, *ns*]. Refer to Table 3.21 for planned comparison group averages. However, results of a one-way between subjects ANOVA indicated significant group differences in levels of anxiety sensitivity on the ASI, [$F(3,41) = 3.62$, $p < .02$]. Post-hoc analyses revealed that the PTSD and pain group reported significantly higher levels of anxiety sensitivity than the pain only group. Please refer to Table 3.24 for individual group averages.

Table 3.23. Anxiety Sensitivity

	Control	Combined Clinical Groups
ASI	17.64_a (11.24)	21.82_a (14.59)

Note. Standard deviations are in parentheses. Means with different subscripts differ significantly at $p < .05$.

Table 3.24. Measures of a Learned Alarm: Individual Groups

	PTSD & Pain	PTSD Only	Pain Only	Control
ASI	30.22_{a} (14.66)	23.67_{ab} (15.22)	12.60_{b} (8.55)	17.64_{ab} (11.24)
PASS: Total	45.78_{a} (21.30)	35.00_{a} (14.80)	37.90_{a} (13.88)	29.64_{a} (21.55)
PASS: Fear	10.89_{a} (5.90)	6.62_{a} (3.02)	7.10_{a} (4.70)	5.50_{a} (4.99)
PASS: Avoidance	10.67_{a} (5.31)	8.75_{a} (5.85)	12.20_{a} (4.16)	9.21_{a} (5.78)
PASS: Cognitive	14.44_{a} (5.29)	12.37_{a} (6.02)	12.70_{a} (5.56)	9.78_{a} (7.48)
PASS: Physiological	9.78_{a} (6.65)	7.25_{a} (3.49)	5.90_{a} (2.85)	5.14_{a} (4.96)
IES: Avoidance	1.21_{a} (.86)	1.69_{a} (.69)	$.94_{a}$ (.81)	1.09_{a} (.88)
IES: Intrusion	1.37_{a} (.73)	1.65_{a} (1.16)	1.27_{a} (.98)	1.23_{a} (.68)
IES: Hyperarousal	1.83_{a} (.87)	1.55_{ab} (.88)	1.01_{ab} (.98)	$.78_{b}$ (.53)
IES: Total	31.67_{a} (16.60)	36.11_{a} (17.44)	23.60_{a} (18.66)	23.27_{a} (11.22)

Note. Standard deviations are in parentheses. Means with different subscripts differ significantly at $p < .05$.

Chapter 4

DISCUSSION

The purpose of the current study was to test aspects of the triple vulnerability model (Barlow, 2000; Barlow, 2002; Otis et al., 2003) in PTSD and chronic musculoskeletal pain populations. According to this model, individuals have a generalized biological vulnerability (e.g., inherited personality traits that lead individuals to react to environmental stressors in a defensive manner), generalized psychological vulnerability (e.g., sense of uncontrollability and perceived inability to cope), and a specific psychological vulnerability (e.g., development of learned alarms) that lead individuals to develop both PTSD and chronic pain (Barlow, 2000; Barlow, 2002; Keane & Barlow, 2002; Otis et al., 2003). Furthermore, PTSD and chronic pain may serve as a reminder of each other, and may therefore maintain and worsen symptomatology of each condition (Otis et al., 2003). Results of the current study are assessed within the context of the vulnerability (i.e., generalized biological, generalized psychological, & specific psychological) that they were hypothesized to represent.

Generalized Biological Vulnerability

According to Barlow (2000, 2002), a generalized biological vulnerability is thought to be characterized by a genetic tendency to demonstrate emotional instability and reactivity in response to changes and stressors in one's environment. Inherited personality traits that are indicative of this vulnerability may consist of being high-strung, nervousness, emotionality, negative affectivity, or neuroticism (Barlow, 2002; Clark et al., 1994). Furthermore, neuroticism is characterized as experiencing emotions associated with the fight or flight response (e.g., fear, anxiety, distress), which may lead

to increased experience of negative moods (Eysenck, 1967, 1981). In assessing for a generalized biological vulnerability for PTSD and chronic pain, three different hypotheses were assessed.

Salivary Cortisol. If a biological vulnerability to demonstrate stress reactivity to stressful events is similar for PTSD and chronic pain, then baseline levels of physiological hyperarousal and physiological reactivity to environmental stressors were predicted to be similar in the three clinical groups as opposed to the control group. In particular, participants in the three clinical groups were hypothesized to demonstrate lower baseline levels of cortisol prior to the experimental stressor and blunted cortisol levels following the experimental stressor. Contrary to this hypothesis, results of the current study did not demonstrate significant group differences in baseline levels of cortisol between the three clinical groups and the control group. This finding is similar to previous research comparing baseline levels of cortisol among fibromyalgia and control group samples (Wingenfeld et al., 2008). Previous research studies assessing for basal cortisol levels among participants with PTSD have also generally demonstrated conflicting results (Meewisse, Reitsma, De Vries, Gersons, & Olff, 2007).

Visual inspection of the data suggested that the PTSD only group reported the greatest increase in cortisol following the TSST; however, no significant group differences were demonstrated between groups across all time points. Limited research has assessed for the role of cortisol reactivity in response to the TSST among individuals with PTSD or chronic pain, although McRae and colleagues (2006) reported that participants with PTSD demonstrated higher cortisol reactivity in response to the TSST than to a cold pressor task. The PTSD and pain and pain only groups appeared to

demonstrate a blunted cortisol response following the TSST; however, this trend was also unexpectedly demonstrated by the control group. Previous research has demonstrated similar blunted cortisol reactivity in response to the TSST among participants with fibromyalgia (Wingenfeld et al., 2008), although no previous studies have been found that demonstrated a blunted response by a control group. It is possible that this may be a result of publication biases in which researchers are less likely to publish non-significant or atypical results (Meewisse et al., 2007). Levels of cortisol declined in all groups from 20 minutes to 40 minutes following the experimental stressor, although they began to increase again in the control group from 40 to 60 minutes as participants were listening to the relaxation soundtrack. It is possible that participants in this group began to feel restless toward the end of their participation in the study.

As expected on the visual analog stress scale, all groups reported that the TSST was perceived as a moderately stressful experience. Taking this into consideration, it appears as though the TSST was successful in eliciting the level of stress reactivity that was intended. Although several variables were controlled for in the current study (e.g., food intake, exercise, smoking, menstrual cycle), it is possible that cortisol levels may have been influenced by the difficulty of controlling for other external variables (e.g., comorbid diagnoses, type of trauma). In a meta-analysis that assessed for basal cortisol levels in participants with PTSD and control groups across 37 studies, results demonstrated that basal levels of cortisol tend to be highly variable across studies (Meewisse et al., 2007). Results also indicated that PTSD tends to be associated with lower basal cortisol levels when participants experienced physical or sexual abuse; however, no significant group differences were demonstrated between participants in the

PTSD groups and control groups when other forms of trauma were assessed (Meewisse et al., 2007). Controlling for type of trauma was beyond the scope of the current study, although it is possible that type of trauma may have influenced study results.

Anxiety. It was hypothesized that self-reports of anxious mood during the lab visit would exist on a continuum. Participants in the PTSD and pain group were predicted to report the highest levels of anxiety across each occasion, followed by the PTSD only and pain only groups, and then by the control group. This hypothesis was partially supported. The PTSD and pain group reported significantly higher levels of anxiety across most time points; however, no significant group differences were demonstrated between the PTSD only, pain only, and control groups. No significant group differences were demonstrated at 10 minutes following the TSST, although, trends indicate that the PTSD and pain and PTSD only groups reported the highest levels of anxiety. The pain only and control groups reported similar increases in anxiety at 10 minutes following the TSST. Although not significantly different, the PTSD only group reported the second highest levels of anxiety at 10 minutes following the TSST, while the pain only and control groups continued to report similar levels of anxiety. After listening to the relaxation track in the final 20 minutes of follow-up, the PTSD only, pain only, and control groups reported minimal levels of anxiety, while the PTSD and pain group continued to report significantly higher levels of anxiety. Given that the TSST is supposed to induce moderate physiological and psychological stress (e.g., Dorn et al., 2003; Jones, Rollman, & Brooke, 1997; McRae et al., 2006; Simeon et al., 2007; Wingenfeld et al., 2008), results of the current study are in line with previous research demonstrating that individuals with PTSD tend to report high levels of anxiety in response to everyday

stressful life events than those without PTSD (Koopman, Gore-Felton, Classen, Kim, Spiegel, 2001).

Overall, these findings support the hypothesis that comorbid diagnoses of PTSD and pain are associated with increased anxiety both prior to and following the experimental stressor; however, this trend was not the same for the PTSD only and pain only groups. Although not significantly different, the PTSD only group reported higher levels of anxiety than the pain only and control groups immediately following the experimental stressor, although this trend was not demonstrated at other time points. Finally, the pain only and control groups reported similar levels of anxiety at all time points. As a result, it appears as though the combined influence of PTSD and pain may lead to increased self-reports of anxious mood for individuals with these diagnoses. Although it does not appear that the PTSD only and pain only groups are linked to a similar vulnerability to respond to stressful situations with similar increases in self-reported anxious mood.

Depression. It was predicted that self-reported symptoms of depression would also exist on a continuum. Participants in the PTSD and pain group were predicted to report the highest levels of depression during the previous two weeks, followed by the PTSD only and pain only groups. Participants in the control group were predicted to report the lowest symptoms of depression. Contrary to this hypothesis, the PTSD and pain and PTSD only groups reported similar and significantly higher levels of depression than the pain only and control groups. These findings are similar to that of previous research that has assessed for the high comorbidity rates between PTSD and depression (Taft, Resick, Watkins, & Panuzio, 2009); however it is surprising that participants in the

pain only group did not report higher levels of depression because high comorbidities between these diagnoses have been reported in previous research (e.g., Gormsen, Rosenberg, Bach, & Jensen, 2010). Findings in the current study suggest that symptoms of depression may be associated with having a diagnosis of PTSD and not necessarily chronic pain. When considering depression as a component of negative affectivity and negative mood (Watson, Clark, & Carey, 1988), it does not appear as though PTSD and chronic pain have the same vulnerability for these personality traits.

Family History of PTSD and Chronic Pain. Finally, self-report data indicated that participants in the PTSD and pain and PTSD only groups were significantly more likely to report a family history of PTSD than the pain only and control groups. This finding is similar to that of previous research reporting that individuals with PTSD are more likely to report a family history of PTSD than those without PTSD (Davidson et al., 1985; McFarlane, 1988). Similarly, participants in the PTSD and pain and pain only groups were significantly more likely to report a family history of chronic pain than participants in the PTSD only and control groups. This finding also supports previous research assessing for a family history of chronic pain among individuals with generalized chronic widespread pain or a specific diagnosis of fibromyalgia (Bergman, 2005). Overall, these findings support the notion that there may be a genetic vulnerability toward developing PTSD and/or chronic pain due to the high family history rates of each diagnosis. Given that participants in the chronic pain only group did not report significant frequencies of a family history of PTSD, and participants in the PTSD only group did not report significant frequencies of a family history of chronic pain,

results suggest that genetic vulnerabilities toward developing each diagnosis may be independent of one another.

In summary, results of the current study do not provide support for the hypothesis that PTSD and chronic pain have a similar biological vulnerability to respond to stressful situations with an alarm reaction. Results do provide initial support for the potential role of a biological vulnerability for individuals with PTSD to respond to stressful situations with an alarm reaction. This was evident with regards to anxiety reactivity, heightened symptoms of depression, and self-reported family histories of PTSD; however, it is unclear as to whether several of these factors represent an actual vulnerability or a reaction to having the diagnosis. Participants with chronic pain also reported significantly higher family history rates of chronic pain, although participants in the pain only group did not report heightened levels of anxiety in response to the experimental stressor or general levels of depression. As such, evidence for this vulnerability was not readily evident among participants with chronic pain without PTSD.

Given that no significant results were demonstrated with cortisol analyses, additional research will be needed to determine the role of cortisol reactivity in both populations. As hypothesized, participants with comorbid PTSD and chronic pain reported the highest levels of anxiety reactivity. This group also reported similarly high levels of depression to that of the PTSD only group. As a result, it may be possible that several of these factors served as an initial vulnerability for PTSD, although it is also possible that having both conditions may serve to worsen symptoms experienced by this population. This hypothesis would be in line with Otis and colleagues proposal that

PTSD and chronic pain may work together to maintain and worsen symptomatology of each condition (Otis et al., 2003).

Generalized Psychological Vulnerability

A generalized psychological vulnerability is thought to refer to a general sense of uncontrollability and perceived inability to cope with unpredictable negative life events (Barlow, 2000, 2002). Barlow (2002) also highlighted the importance of inducing a stress response to measure changes in coping strategies. Otis and colleagues (2003, 2006) notion that a generalized psychological vulnerability may be applied to both PTSD and chronic pain was assessed within three hypotheses.

Learned Helplessness. According to the fourth hypothesis, all three clinical groups were predicted to report higher levels of learned helplessness than the control group. Results of the current study indicate that there were no significant group differences for learned helplessness factors of internality, stability, or globality. Consequently, results suggest that the construct of learned helplessness may not serve as a psychological vulnerability for PTSD or chronic pain. This lack of significant group differences conflicts with limited research that has found PTSD to be associated with a helpless attributional style (Casella & Motta, 1990; Gibb, 2002), although results are similar to those previously found within the chronic pain literature that did not demonstrate an increase in learned helplessness among participants with chronic pain (Ingram et al., 1990). As the Attributional Style Questionnaire (ASQ; Peterson & Villanova, 1988) does not specifically target symptoms associated with PTSD and chronic pain populations, it is possible that responses to the ASQ scenarios do not generalize to real life experiences.

Life experiences. If individuals with PTSD and chronic pain experience a general sense of uncontrollability and perceived inability to cope with negative life events, then it was hypothesized that the three clinical groups would report greater levels of negative life events, perception of stress over the past month, and intolerance of uncertainty. Results of the current study support the hypothesis that having a diagnosis of PTSD and/or chronic pain is associated with greater impact of negative life events than the control group. When considering all four groups independently, results revealed that the PTSD and pain and PTSD only groups, in particular, reported significantly more negative impact ratings of stressful events than the control group. Of particular interest, is that the control group was the only group that rated the average impact of their life events as being in the positive range.

Previous research has demonstrated that PTSD or chronic pain populations are more likely to experience negative life events than individuals without these diagnoses (e.g., Naidoo & Pillay, 1994; Solomon, Zur-Noah, Horesh, Zerach, & Keinan, 2008). When assessing for the frequency of negative life events reported by participants in the current study, no significant group differences were demonstrated; however, participants in the PTSD and pain group did report the highest frequencies of negative life events, followed by the PTSD only, pain only, and control groups respectively. These findings also provide preliminary support for the hypothesis that individuals with PTSD and chronic pain tend to report life events more negatively, which may suggest that they may experience greater difficulty in coping with these events for them to rate the impact of these events in this way.

Stress. As mentioned above, it was hypothesized that participants in the three clinical groups would report greater perceptions of stress over the past month than the control group. Planned comparisons indicated that the three clinical groups combined reported significantly greater perceptions of stress than the control group. When considering the four groups independently, results revealed that the PTSD and pain group reported significantly higher perceptions of stress than the pain only and control groups. Although not significantly different from the pain only and control groups, the PTSD only group also reportedly similar levels of stress as the PTSD and pain group. The high rates of general stress reported by participants with PTSD provides further support for previous research that has also indicated that individuals with PTSD tend to report high levels of stress (Fincham, Altes, Stein, & Seedat, 2009). As a result, it appears as though having a co-morbid diagnosis of PTSD and pain is associated with the highest perceptions of stress over the past month, followed by the PTSD only, control, and pain only groups respectively.

Intolerance of Uncertainty. It was also predicted that the three clinical groups would report higher levels of intolerance of uncertainty than the control group. Planned comparisons between the three clinical groups and the control group did not indicate significant group differences in levels of this construct. Similarly, analysis of the four groups independently did not demonstrate any significant group differences for intolerance of uncertainty. As a result, it does not appear that intolerance of uncertainty plays a major role as a psychological vulnerability for either PTSD or chronic pain. These findings conflict with previous research that has demonstrated a relationship between intolerance of uncertainty and symptoms of anxiety (Kirby & Yardly, 2009).

Previous research has also demonstrated that uncertainty regarding illness within a fibromyalgia population was associated with increased difficulty in coping with symptoms of fibromyalgia (Johnson, Zautra, & Davis, 2006). No known research studies have been found that directly assessed for the relationship between intolerance of uncertainty within PTSD or chronic pain populations.

Coping Strategies. Finally, it was predicted that the clinical groups would report greater use of negative coping strategies than the control group. Conversely, it was predicted that the control group would report greater use of positive coping strategies than the three clinical groups. Coping strategies were assessed as both general use of coping strategies and coping strategies utilized in response to the experimental stressor.

Planned comparisons of general coping strategies indicated that the three clinical groups combined reported significantly higher levels of self-blame than the control group. However, planned comparisons did not indicate significant group differences in levels of denial, behavioral disengagement, active coping, positive reframing, or acceptance. When each group was considered independently, results further indicated that the PTSD only group reported using significantly higher levels of self-blame coping than the pain only and control groups. Although not significantly different from the control and pain only groups, the PTSD and pain group reported similar levels of self-blame to that of the PTSD only group. The pain only group actually reported the lowest levels of self-blame. These results are similar to that of previous research that has not found an increase in self-blame within a chronic pain population that reported comorbid symptoms of depression (Lopez-Lopez, Montorio, Izal, & Velasco, 2008). With regards to symptoms of PTSD, the current results support previous research that has found

symptoms of PTSD to be positively correlated with self-blame (e.g., Najdowski, & Ullman, 2009).

When considering coping strategies in response to the experimental stressor, planned comparisons did not suggest any significant group differences in self-blame, behavioral disengagement, denial, acceptance, positive reframing, or active coping. Similar to that of general coping strategies that were reported, analysis of individual group differences demonstrated that participants in the PTSD only group reported higher levels of self-blame coping in response to the TSST than the pain only group, although this was only a marginal result. The pain only group continued to report the lowest levels of self-blame when compared to the other three groups. No other significant group differences in coping strategies were demonstrated in response to the experimental stressor.

Overall, these findings suggest that individuals with PTSD are significantly more likely to utilize negative coping strategies such as self-blame than individuals who have chronic pain without PTSD. No significant group differences were reported with regards to the use of positive coping strategies. Furthermore, this trend held true when assessing for both general use of coping strategies and coping strategies utilized in response to the experimental stressor. Thus, the current results support the potential role of negative coping strategies among individuals with chronic pain, although this trend was not supported for individuals with chronic pain without PTSD. It is possible that analyses of group coping strategies were limited by the small sample size.

In summary, results of the current study suggest that PTSD may be associated with a generalized psychological vulnerability. This was evident by greater perceptions

of stress over the past month, negative impact ratings of significant life events over the past year, and greater report of negative coping strategies (i.e., self-blame) than the pain only group. The only indicator of a psychological vulnerability that was supported for the clinical group was the negative mean impact ratings of life events. However, having a comorbid diagnosis of both PTSD and chronic pain appears to be associated with the greatest levels of stress and negative impact ratings of life events. Similar to findings focusing on a generalized biological vulnerability, results of the current study suggest that having both diagnoses may maintain and even worsen symptoms experienced by these individuals. The findings among the PTSD and pain group are not surprising as previous research has also demonstrated that comorbid diagnoses of PTSD and pain are associated with greater psychological distress and disability (Sherman et al., 2000).

Specific Psychological Vulnerability

According to Barlow (2002), a specific psychological vulnerability refers to the development of a learned alarm, which may occur after the experience of a traumatic event. With regards to chronic pain, activities and sensations that are associated with the experience of pain may also serve as learned alarms (Asmundson et al., 1999; McCracken et al., 1993; Otis et al., 2003). To assess for this vulnerability, the current study predicted that PTSD and chronic pain would be associated with learned alarms that are specific to the particular diagnosis. It was hypothesized that participants in the PTSD and pain and PTSD only groups would report specific learned alarms associated with PTSD, while participants with chronic pain would report evidence of a learned alarm associated with chronic pain. All three clinical groups were predicted to report higher levels of anxiety sensitivity than the control group.

PTSD Related Anxiety. As expected, results of the current study suggested evidence of a related learned alarm among participants with PTSD. This was indicated by higher levels of total PTSD related symptoms and symptoms of hyperarousal among participants with PTSD. No significant group differences were reported for the intrusion and avoidance subscales of the IES-R. All participants in the current study met DSM-IV diagnostic criteria for PTSD on a structured clinical interview, so it is likely that results of the current study were limited due to the small number of participants. Despite this limitation, evidence for a PTSD specific learned alarm was still evident among participants with PTSD as opposed to those without PTSD.

Pain Related Anxiety. No significant group differences were demonstrated for total scores for pain-related anxiety. Similarly, no significant group differences were demonstrated for the avoidance, cognitive, and physiological subscales of the PASS; however, near significant group differences were demonstrated for pain- related fear. In particular, the PTSD and pain group reported the highest levels of pain- related fear, followed by the pain only group, PTSD only group, and control groups respectively. This suggests that significant group differences in pain- related fear would likely be evident with a larger sample size. Given that participants in the PTSD and pain group reported the highest levels of pain- related fear, results of the current study provide preliminary support for the role of a learned alarm among individuals with chronic pain. Furthermore, it appears as though having a comorbid diagnosis of PTSD and chronic pain contributes to increased pain-related fear among this population. It is possible that pain related fear is elevated within this group because symptoms of chronic pain may serve as a reminder of an individual's traumatic event (Straub & Straub, 2009).

Anxiety Sensitivity. With regards to anxiety sensitivity, planned comparisons between the three clinical groups and the control group did not demonstrate any significant group differences. Analyses of individual group differences demonstrated that the PTSD and pain group reported significantly higher levels of anxiety sensitivity than the pain only group. Although not significantly different, the PTSD only group reported the second highest levels of anxiety sensitivity, followed by the control and pain only groups respectively. These results are in line with results of the current study given that participants with PTSD and chronic pain reported the highest levels of pain related fear. Previous research has also demonstrated that pain anxiety may be accounted for by anxiety sensitivity (Greenburg & Burns, 2003). The relationship between anxiety sensitivity and PTSD has also been well established within the extant literature (Kilic et al., 2008; Taylor, 2004). Overall, results of the current study support the potential role of anxiety sensitivity as a vulnerability for PTSD; however, results do not support the hypothesis that anxiety sensitivity serves as a vulnerability for chronic pain without PTSD. Having comorbid diagnoses of PTSD and chronic pain appears to lead to greater self-reports of anxiety sensitivity for individuals with these diagnoses.

In summary, preliminary evidence for a specific learned alarm associated with PTSD was demonstrated. This was evidenced by total PTSD related symptoms and symptoms of hyperarousal. Similarly, preliminary evidence for a specific learned alarm among participants with chronic pain was also demonstrated. Although there was only a marginal significant difference, the PTSD and pain group reported the highest levels of pain related fear, followed by the pain only, PTSD only, and control groups respectively. Finally, results did not support the role of anxiety sensitivity in all three clinical groups;

however, the PTSD and pain group reported significantly higher levels of anxiety sensitivity than the pain only group.

Summary of Findings

The purpose of the current study was to test aspects of the triple vulnerability model (Barlow, 2000; Barlow, 2002; Otis et al., 2003) in PTSD and chronic musculoskeletal pain populations. This model proposed that individuals have a generalized biological vulnerability (e.g., inherited personality traits that lead individuals to react to environmental stressors in a defensive manner), generalized psychological vulnerability (e.g., sense of uncontrollability and perceived inability to cope), and a specific psychological vulnerability (e.g., development of learned alarms) to develop both PTSD and chronic pain (Barlow, 2000; Barlow, 2002; Keane & Barlow, 2002; Otis et al., 2003). PTSD and chronic pain may also serve as a reminder of each other, which may maintain and worsen symptoms of each condition (Otis et al., 2003).

Results of the current study do not provide support for the hypothesis that PTSD and chronic pain have a similar biological vulnerability to respond to stressful situations with an alarm reaction; however, initial support for the role of a biological vulnerability for individuals with PTSD to respond to stressful situations with an alarm reaction was demonstrated. This was evident by participants' anxiety reactivity, heightened symptoms of depression, and self-reported family histories of PTSD. Participants with chronic pain also reported significantly higher family history rates of chronic pain, although measures of anxiety and depression were not heightened in the pain only group. No significant results were demonstrated with cortisol analyses, so additional research is needed to assess for cortisol reactivity in both populations. As hypothesized, participants with

comorbid PTSD and chronic pain reported the highest levels of anxiety in response to the experimental stressor. This group also reported high levels of depression. As a result, it may be possible certain vulnerabilities may exist for PTSD to initially develop, and that having both diagnoses may serve to worsen and maintain symptoms experienced by this population. This result also supports, Otis and colleagues' contention that PTSD and chronic pain may maintain and worsen symptoms of each condition (Otis et al., 2003).

Preliminary support for a generalized psychological vulnerability among participants with PTSD was demonstrated. This was evident by greater perceptions of stress over the past month, negative impact ratings of significant life events over the past year, and greater report of negative coping strategies (i.e., self-blame) than the pain only group. Having a diagnosis of chronic pain was only associated with negative mean impact ratings of life events when combined with the other clinical groups. Having both PTSD and chronic pain is associated with the greatest levels of stress and negative impact ratings of life events. As such, results of the current study further suggest that having both conditions may maintain and potentially worsen psychological symptoms experienced by these individuals.

Preliminary evidence for a specific learned alarm associated with PTSD was demonstrated. Participants with PTSD reported higher total PTSD related symptoms and symptoms of hyperarousal. Preliminary evidence for a specific learned alarm among participants with chronic pain was also demonstrated. Although there was only a marginal significant difference, the PTSD and pain group reported the highest levels of pain related fear, followed by the pain only, PTSD only, and control groups respectively.

Finally, the PTSD and pain group reported significantly higher levels of anxiety sensitivity than the pain only group.

Overall, results of the current study support the role of a generalized biological, generalized psychological, and specific psychological vulnerabilities toward developing PTSD. Limited findings have supported the potential role of these vulnerabilities toward developing chronic pain; however, results of these measures were not similar to that of PTSD (e.g., family history of chronic pain). As such, it is not thought that PTSD and chronic pain are associated with the same vulnerabilities. Across many measures, having PTSD and chronic pain was associated with an increase in symptoms. At this point in time, it is thought that having both diagnoses may serve to maintain and possibly worsen symptoms experienced by this population.

Study Limitations

There are several limitations associated with the current study. Small sample sizes may have limited the ability to find significant group differences. Due to difficulties with recruiting participants, women were recruited from both community and student populations. As a result, participants in the PTSD only and control groups were significantly younger than those in the pain only group. Conflicting results have been found regarding whether or not there are differences in cortisol reactivity across the lifespan (Gotthardt et al., 1995; Kudielka et al., 1999; Kudielka et al., 2000; Kudielka et al., 2004). This may have increased variability in both cortisol levels and self-report measures, although no significant group differences in cortisol were demonstrated when age was utilized as a covariate. It is also possible that undergraduate students may respond differently to stressful situations than the general population.

Due to financial constraints, salivary cortisol was the only indicator of stress reactivity in the HPA axis that was measured. The current results may have been limited by not assessing for other changes in the HPA axis (e.g., CRH, ACTH, DHEA). Variability in stress reactivity may also have been increased by including participants who were both premenopausal and postmenopausal. In an attempt to control for this, participants who were premenopausal were scheduled during the premenstrual phase of their menstrual cycle; however, this may not have eliminated all variability (Kudielka et al., 2004). Similarly, an inability to control for use of oral contraceptives may have influenced cortisol analyses (Kirschbaum et al., 1999).

Significant group differences were also demonstrated with regards to body mass index. In particular, the pain only group had significantly higher BMI ratings than the PTSD only and control groups. It is possible that BMI may have influenced cortisol reactivity, although this is unlikely given that previous research has not demonstrated significant group differences between obese and non-obese women with regards to cortisol reactivity in response to the TSST (Therrien et al., 2010).

Participants in the current study were primary Caucasian females. This may limit generalizability of study results to other racial and ethnic groups. Ability to generalize results to male populations may be limited as well.

The use of the TSST as a stress inducing event may not have been stressful enough to induce levels of cortisol across all populations. Although all groups reportedly perceived the TSST as being a moderately stressful event on the VASS, it is concerning that the control group did not demonstrate an increase in cortisol levels following the TSST as has been reported in previous research (e.g., Kirschbaum et al., 1993). It is also

possible that levels of cortisol in the control group were influenced by other axis I diagnoses that were not ruled out (Young, Abelson, & Cameron, 2004). Individual differences in raters for the TSST may have also influenced cortisol levels for the control group. Male and female raters were used but approximately 10 different sets of raters were used in the current study. In addition, college students may be less reactive to a stress task given IRB requirements regarding harm and previous experience with psychological experiments.

Due to the constraints of the current study, no formal medical chart review was conducted to confirm chronic pain diagnoses. Although the current study attempted to limit variability within the chronic pain groups by only including participants with chronic musculoskeletal pain, it is possible that including participants with different types of musculoskeletal pain could have influenced cortisol reactivity. Similarly, the current study was unable to account for type of traumatic events among participants with PTSD. It is possible that this may have also influenced cortisol reactivity among these participants (Meewisse et al., 2007). Participants with differential conditions of PTSD and or chronic pain may also have varying sleep-wake schedules. Although time of day was controlled for in the current study, it is possible that variations in sleep-wake cycles may have influenced cortisol reactivity (e.g., Neylan, Otte, Yehuda, Marmar, 2006).

Finally, participants in the current study appeared to be relatively high functioning when demographic information was examined (e.g., education, family income). Individuals who experience more severe levels of PTSD and chronic pain may have been less likely to respond to recruitment efforts. Approximately 50% - 60% of participants who completed the initial phone screening did not show up for their laboratory visit.

Several efforts were made to reschedule these participants, however many of them never participated. It is unknown if severity in symptoms of PTSD, chronic pain, or disability may have played a role in these individuals not showing up for their research appointment.

Future Directions

Additional research will be needed to further assess for the role of biological and psychological factors that may influence the high comorbidity rates of PTSD and chronic pain. It is likely that study analyses were limited due to the small number of participants and high variability among several of the factors. As such, a larger sample size would likely be beneficial. Furthermore, future research will be needed to generalize study findings to other populations (e.g., male, racial/ethnic minorities).

The current study was only able to assess for salivary cortisol reactivity due to financial constraints. It will be helpful for future research to assess for the role of other hormones in the HPA axis in attempt to obtain a complete picture of HPA axis functioning. Although it would be extremely difficult, longitudinal data is needed to determine the temporal course of cortisol reactivity, in addition to other biological and psychological markers, before and after experiencing a traumatic event. Changes in pain sensitivity before and after experiencing a stressful event would also provide interesting information regarding the effects of daily stressors on chronic pain.

Finally, the current study provides preliminary support for several indicators of biological and psychological vulnerabilities toward developing PTSD and/or chronic pain. Results also indicate that having comorbid diagnoses of PTSD and chronic pain is associated with increased symptoms on many of the measures utilized in this study. As

such, it will be imperative for future research to consider treatment implications for individuals with both diagnoses. It would be interesting to investigate if symptoms of chronic pain lessen when symptoms of PTSD are treated. Furthermore, preventative efforts should be developed that address the potential vulnerabilities demonstrated in the current study.

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APPENDICES
Appendix A
CONSENT FORMS

Traumatic Events and Chronic Pain
Informed Consent Form – Community Participants

Because you are a female 18 years of age or older, and you responded to recruitment efforts, you are invited to participate in a research project investigating the relationship between posttraumatic stress disorder (PTSD) and chronic musculoskeletal pain. This study is being conducted by Anna Cassel, a graduate student in the Psychology department at the University of Maine, and is being supervised by Dr. Sandy Sigmon, a professor in the Psychology department at the University of Maine.

How do I qualify for this study?

• You have already responded to some brief questions over the phone.
• Next, you will be asked to come into the lab to answer some questions from a diagnostic interview (e.g., In the past six months, have you felt particularly nervous or anxious?).
• You will also be asked to draw the location of your pain on a human figure and answer a few brief questions regarding your pain condition.
• There are four ways that you may qualify for this study:
• Meet diagnostic criteria for PTSD without a chronic pain condition (PTSD group)
• Meet diagnostic criteria for PTSD and chronic musculoskeletal pain (PTSD and pain group)
• Meet diagnostic criteria for chronic musculoskeletal pain without PTSD (pain group)
• Never had a diagnosis of either PTSD or chronic pain

What does this study involve?

• If you qualify based on information that you have given regarding the initial phone screening criteria, you will be scheduled to come into the lab at the University of Maine. During this visit, you will be asked to answer further questions (discussed above) to confirm your eligibility for this study. It is estimated that it will take approximately 60 minutes for this portion of the study. Next, you will complete two mentally challenging tasks. Following the challenging tasks, you will be asked to stay for an additional hour. During the last 20 minutes of this hour, you will listen to some relaxing music. Saliva samples and pain sensitivity will be assessed at various points throughout a 90 minute period. Overall, it is estimated that the laboratory visit will take approximately 2 to 2.5 hours.

    • Levels of pain sensitivity will be measured using a device that will apply a small amount of pressure on to your thumb. You will be asked to report when you first perceive symptoms of pain.

- Salivary cortisol samples will be collected using a plastic tube. You will be asked to drool into the plastic tube.

• Following the completion of your laboratory visit, you will be asked to complete a packet of questionnaires. Questionnaires may be completed on surveymonkey.com, or you may be given a packet of questionnaires with a return envelope. It is estimated that it will take approximately 45 minutes to complete the packet of questionnaires. You will be asked questions about:

- Your reactions to various situations and life events (e.g., "You confront a serious conflict in your values")
- Life experiences (e.g., "Serious injury or Illness")
- Coping strategies (e.g., "I've been expressing my negative feelings")
- Symptoms associated with a stressful experience ("Pictures about it popped into my mind")
- Symptoms associated with the experience of pain (e.g., "I think that if my pain gets too severe, it will never decrease")
- Symptoms of anxiety (e.g., "It is important to me not to appear nervous")
- Feelings of uncertainty (e.g., "I can't stand being taken by surprise")
- Feelings of stress (e.g., "In the past month, how often have you felt that you were unable to control the important things in your life?")
- Symptoms of depression (e.g., "I am so sad or unhappy that I can't stand it.")
- Basic demographic questions (e.g., "Current household income")

Voluntary

Your participation in this research project is voluntary. You have the right to withdraw from this study at any point. You may also choose to not answer any question that you do not feel comfortable answering. If you do withdraw from the study, then you will be compensated for the parts of the study that you participated in.

Are there any potential risks?

In previous research, any risk associated with participating in the mentally challenging tasks has not been any greater than what you would normally encounter in your daily life. While measuring pain sensitivity, you may temporarily experience a minimal level of pain. This method of measuring pain sensitivity has been used in previous research studies with no other adverse side effects being reported. There are no known risks associated with collecting and analyzing the saliva samples. No identifying information is sent with the samples when they are analyzed. In addition, there have been no long lasting effects of completing questionnaires included in this study. All questionnaires and tasks utilized in this study have been used in previous research with no reported negative effects. You may, however, experience some discomfort when answering questions that involve stressful events or situations. Anna Cassel (the principal investigator) and Dr. Sandy Sigmon (her supervisor) will be available to talk with you if you would like to discuss this discomfort. You may also request referral information for psychological services at any time.

Potential risks associated with completing online questionnaires on SurveyMonkey.com are thought to be no greater than those encountered during routine access of the internet. SurveyMonkey.com has taken precautions to secure the website and protect its users from fraud or compromised confidentiality. In addition, they will not utilize customers' data for their own purposes

What are the benefits of participating in this study?

Your responses may help us to gain a better understanding of the relationship between chronic pain and PTSD. You will receive $20 for completing participation in this study. If you do not qualify for participation, then you will receive $5 for the diagnostic interview. You may also be eligible to participate in a free treatment study. This treatment study will involve treatment for individuals who are still bothered by traumatic events that have happened to them. You will have the option of participating or not in this treatment study. The treatment consists of learning new skills in how to deal with recurring thoughts and images that you may be bothered with.

Will my answers be confidential?

Your participation and all of your answers will be confidential. All study materials will be stored in Dr. Sigmon's locked laboratory. You will be assigned a participant number for the study that will be written on all information that you give us. A list that contains your identifying information will be stored in a separate location. This list will be destroyed after the study is completed. Your answers will only be used for research purposes. If any data becomes published or presented at a conference, then all of your answers will remain anonymous and be compiled in a group format. The data will be kept in Dr. Sigmon's lab for an indefinite period of time.

Who do I contact if I have any questions?

In the event that you have any questions regarding this study, you may contact Anna Cassel (581-2030, 301 Little Hall, Orono, ME, 04469 or by email: anna.cassel@umit.maine.edu) or her advisor, Dr. Sandy Sigmon (581-2049, 376 Little Hall, Orono, ME, 04469 or by email: sandra.Sigmon@umit.maine.edu). In the event that you have questions regarding your right as a research participant, you may contact Gayle Anderson who is the Assistant to the Protection of Human Subjects Review Board (581-1498, 114 Alumni Hall, Orono, ME, 04469, or by email: gayle.anderson@umit.maine.edu).

If you would like a copy of the results of this study when it is completed, please indicate below. Also, please indicate if you would like to be contacted regarding the free treatment study.

You will receive a copy of this consent form.

Participant Signature: _____Date: _____

Print Name Here: _____ Phone: _____

Please indicate if you would like a summary of study results: _____ yes _____ no

Would you like to be contacted regarding the free treatment study? _____ yes _____ no

If you would like the results of this study, please write down your permanent address:

_____

_____

Traumatic Events and Chronic Pain
Informed Consent Form – Student Version

Because you are a female 18 years of age or older, and you responded to recruitment efforts, you are invited to participate in a research project investigating the relationship between posttraumatic stress disorder (PTSD) and chronic musculoskeletal pain. This study is being conducted by Anna Cassel, a graduate student in the Psychology department at the University of Maine, and is being supervised by Dr. Sandy Sigmon, a professor in the Psychology department at the University of Maine.

How do I qualify for this study?

- You have already responded to some brief questions over the phone.
- Next, you will be asked to come into the lab to answer some questions from a diagnostic interview (e.g., In the past six months, have you felt particularly nervous or anxious?).
- You will also be asked to draw the location of your pain on a human figure and answer a few brief questions regarding your pain condition.
- There are four ways that you may qualify for this study:
    - Meet diagnostic criteria for PTSD without a chronic pain condition (PTSD group)
    - Meet diagnostic criteria for PTSD and chronic musculoskeletal pain (PTSD and pain group)
    - Meet diagnostic criteria for chronic musculoskeletal pain without PTSD (pain group)
    - Never had a diagnosis of either PTSD or chronic pain

What does this study involve?

- If you qualify based on information that you have given regarding the initial phone screening criteria, you will be scheduled to come into the lab at the University of Maine. During this visit, you will be asked to answer further questions (discussed above) to confirm your eligibility for this study. It is estimated that it will take approximately 60 minutes for this portion of the study. Next, you will complete two mentally challenging tasks. Following the challenging tasks, you will be asked to stay for an additional hour. During the last 20 minutes of this hour, you will listen to some relaxing music. Saliva samples and pain sensitivity will be assessed at various points throughout a 90 minute period. Overall, it is estimated that the laboratory visit will take approximately 2 to 2.5 hours.
    - Levels of pain sensitivity will be measured using a device that will apply a small amount of pressure on to your thumb. You will be asked to report when you first perceive symptoms of pain.
    - Salivary cortisol samples will be collected using a plastic tube. You will be asked to drool into the plastic tube.

• Following the completion of your laboratory visit, you will be asked to complete a packet of questionnaires. Questionnaires may be completed on surveymonkey.com, or you may be given a packet of questionnaires with a return envelope. It is estimated that it will take approximately 45 minutes to complete the packet of questionnaires. You will be asked questions about:

- Your reactions to various situations and life events (e.g., "You confront a serious conflict in your values")
- Life experiences (e.g., "Serious injury or Illness")
- Coping strategies (e.g., "I've been expressing my negative feelings")
- Symptoms associated with a stressful experience ("Pictures about it popped into my mind")
- Symptoms associated with the experience of pain (e.g., "I think that if my pain gets too severe, it will never decrease")
- Symptoms of anxiety (e.g., "It is important to me not to appear nervous")
- Feelings of uncertainty (e.g., "I can't stand being taken by surprise")
- Feelings of stress (e.g., "In the past month, how often have you felt that you were unable to control the important things in your life?")
- Symptoms of depression (e.g., "I am so sad or unhappy that I can't stand it.")
- Basic demographic questions (e.g., "Current household income")

Voluntary

Participation in this research project is voluntary. You have the right to withdraw from this study at any point. You may also choose to not answer any question that you do not feel comfortable answering. If you do withdraw from the study, then you will be compensated for the parts of the study that you participated in. You will receive 1 credit for completing the initial interview, 1 credit for participation in the mentally challenging tasks, 1 credit for remaining in the lab for an additional hour while saliva samples are collected, and then 1 credit for completing the questionnaires.

Are there any potential risks?

In previous research, any risk associated with participating in the mentally challenging tasks has not been any greater than what you would normally encounter in your daily life. While measuring pain sensitivity, you may temporarily experience a minimal level of pain. This method of measuring pain sensitivity has been used in previous research studies with no other adverse side effects being reported. There are no known risks associated with collecting and analyzing the saliva samples. No identifying information is sent with the samples when they are analyzed. In addition, there have been no long lasting effects of completing questionnaires included in this study. All questionnaires and tasks utilized in this study have been used in previous research with no reported negative effects. You may, however, experience some discomfort when answering questions that involve stressful events or situations. Anna Cassel (the principal investigator) and Dr. Sandy Sigmon (her supervisor) will be available to talk with you if you would like to discuss this discomfort. You may also request referral information for psychological services at any time.

Potential risks associated with completing online questionnaires on SurveyMonkey.com are thought to be no greater than those encountered during routine access of the internet. SurveyMonkey.com has taken precautions to secure the website and protect its users from fraud or compromised confidentiality. In addition, they will not utilize customers' data for their own purpose.

What are the benefits of participating in this study?

Your responses may help us to gain a better understanding of the relationship between chronic pain and PTSD. You will receive 5 credits for completing participation in this study. If you do not qualify for participation, then you will receive 1 credit for the diagnostic interview. If you do qualify for the study, you will receive 1 credit for completing the diagnostic interview, 1 credit for participation in the mentally challenging tasks, 1 credit for remaining in the lab for an additional hour while saliva samples are collected, and then 1 credit for completing the questionnaires. You may also be eligible to participate in a free treatment study. This treatment study will involve treatment for individuals who are still bothered by traumatic events that have happened to them. You will have the option of participating or not in this treatment study. The treatment consists of learning new skills in how to deal with recurring thoughts and images that you may be bothered with.

Will my answers be confidential?

Your participation and all of your answers will be confidential. All study materials will be stored in Dr. Sigmon's locked laboratory. You will be assigned a participant number for the study that will be written on all information that you give us. A list that contains your identifying information will be stored in a separate location. This list will be destroyed after the study is completed. Your answers will only be used for research purposes. If any data becomes published or presented at a conference, then all of your answers will remain anonymous and be compiled in a group format. The data will be kept in Dr. Sigmon's lab for an indefinite period of time.

Who do I contact if I have any questions?

In the event that you have any questions regarding this study, you may contact Anna Cassel (581-2030, 301 Little Hall, Orono, ME, 04469 or by email: anna.cassel@umit.maine.edu) or her advisor, Dr. Sandy Sigmon (581-2049, 376 Little Hall, Orono, ME, 04469 or by email: sandra.Sigmon@umit.maine.edu). In the event that you have questions regarding your right as a research participant, you may contact Gayle Anderson who is the Assistant to the Protection of Human Subjects Review Board (581-1498, 114 Alumni Hall, Orono, ME, 04469, or by email: gayle.anderson@umit.maine.edu).

If you would like a copy of the results of this study when it is completed, please indicate below. Also, please indicate if you would like to be contacted regarding the free treatment study.

You will receive a copy of this consent form.

Participant Signature: _____Date: _____

Print Name Here: _____Phone: _____

Please indicate if you would like a summary of study results: _____ yes _____ no

Would you like to be contacted regarding the free treatment study? _____ yes _____ no

If you would like the results of this study, please write down your permanent address:

_____

_____

Appendix B

SHORT SCREENING SCALE FOR DSM-IV PTSD

1. Did you avoid being reminded of this experience by staying away from certain places, people, or activities? (REMIND RESPONDENT OF LIFE EVENT IF NECESSARY.)

1. YES
2. NO

2. Did you lose interest in activities that were once important or enjoyable? (REMIND RESPONDENT OF LIFE EVENT IF NECESSARY.)

1. YES
2. NO

3. Did you begin to feel more isolated or distant from other people? (REMIND RESPONDENT OF LIFE EVENT IF NECESSARY.)

1. YES
2. NO

4. Did you find it hard to have love or affection for other people? (REMIND RESPONDENT OF LIFE EVENT IF NECESSARY.)

1. YES
2. NO

5. Did you begin to feel that there was no point in planning for the future? (REMIND RESPONDENT OF LIFE EVENT IF NECESSARY.)

1. YES
2. NO

6. After this experience, were you having more trouble than usual falling asleep or staying asleep? (REMIND RESPONDENT OF LIFE EVENT IF NECESSARY.)

1. YES
2. NO

7. Did you become jumpy or get easily startled by ordinary noises or movements? (REMIND RESPONDENT OF LIFE EVENT IF NECESSARY.)

1. YES
2. NO

Appendix C

MPQ

Appendix D

EXPANDED ASQ

Interpretations of Events

Please try to imagine yourself in the situations that follow. If such a situation happened to you, what would you feel would have caused it? While events may have many causes, we want you to pick only one—The MAJOR CAUSE IF THIS EVENT HAPPENED TO YOU.

Please write the cause in the blank provided after each event. Next we want you to answer three questions about the cause you provided. First, is the cause of this event something about you or something about other people or circumstances? Second, is the cause of this event something that will persist across time or something that will never again be present? Third, is the cause of this event something that affects all situations in your life or something that just affects this type of event?

To summarize, we want you to:

1. Read each situation and vividly imagine it happening to you.

2. Decide what you feel would be the one major cause of the situation if it happened to you.

3. Write the cause in the blank provided.

4. Answer three questions about the cause.

1. You have been looking for a job unsuccessfully for some time.

A. Write down one major cause:

B. Is the cause of this due to something about you or something about other people or circumstances? (circle one number)

totally due to others	1	2	3	4	5	6	7	totally due to me

C. In the future, will this cause be present again? (circle one number)

never present	1	2	3	4	5	6	7	always present

D. Is this cause something that affects just this type of situation, or does it also influence other areas of your life? (circle one number)

just this situation	1	2	3	4	5	6	7	all situations

---

2. A friend comes to you with a problem, and you don't try to help.

A. Write down one major cause:

B. Is the cause of this due to something about you or something about other people or circumstances? (circle one number)

totally due to others	1	2	3	4	5	6	7	totally due to me

C. In the future, will this cause be present again? (circle one number)

never present	1	2	3	4	5	6	7	always present

D. Is this cause something that affects just this type of situation, or does it also influence other areas of your life? (circle one number)

just this situation	1	2	3	4	5	6	7	all situations

3. You give an important talk in front of a group, and the audience reacts negatively.

A. Write down one major cause:

B. Is the cause of this due to something about you or something about other people or circumstances? (circle one number)

totally due to others	1	2	3	4	5	6	7	totally due to me

C. In the future, will this cause be present again? (circle one number)

never present	1	2	3	4	5	6	7	always present

D. Is this cause something that affects just this type of situation, or does it also influence other areas of your life? (circle one number)

just this situation	1	2	3	4	5	6	7	all situations

---

4. You meet a friend who acts hostilely to you.

A. Write down one major cause:

B. Is the cause of this due to something about you or something about other people or circumstances? (circle one number)

totally due to others	1	2	3	4	5	6	7	totally due to me

C. In the future, will this cause be present again? (circle one number)

never present	1	2	3	4	5	6	7	always present

D. Is this cause something that affects just this type of situation, or does it also influence other areas of your life? (circle one number)

just this situation	1	2	3	4	5	6	7	all situations

5. You can't get all the work done that others expect of you.

A. Write down one major cause:

B. Is the cause of this due to something about you or something about other people or circumstances? (circle one number)

totally due to others	1	2	3	4	5	6	7	totally due to me

C. In the future, will this cause be present again? (circle one number)

never present	1	2	3	4	5	6	7	always present

D. Is this cause something that affects just this type of situation, or does it also influence other areas of your life? (circle one number)

just this situation	1	2	3	4	5	6	7	all situations

---

6. You go out on a date, and it goes badly.

A. Write down one major cause:

B. Is the cause of this due to something about you or something about other people or circumstances? (circle one number)

totally due to others	1	2	3	4	5	6	7	totally due to me

C. In the future, will this cause be present again? (circle one number)

never present	1	2	3	4	5	6	7	always present

D. Is this cause something that affects just this type of situation, or does it also influence other areas of your life? (circle one number)

just this situation	1	2	3	4	5	6	7	all situations

7. Your stead romantic relationship ends.

A. Write down one major cause:

B. Is the cause of this due to something about you or something about other people or circumstances? (circle one number)

totally due to others	1	2	3	4	5	6	7	totally due to me

C. In the future, will this cause be present again? (circle one number)

never present	1	2	3	4	5	6	7	always present

D. Is this cause something that affects just this type of situation, or does it also influence other areas of your life? (circle one number)

just this situation	1	2	3	4	5	6	7	all situations

---

8. You experience a major personal injury.

A. Write down one major cause:

B. Is the cause of this due to something about you or something about other people or circumstances? (circle one number)

totally due to others	1	2	3	4	5	6	7	totally due to me

C. In the future, will this cause be present again? (circle one number)

never present	1	2	3	4	5	6	7	always present

D. Is this cause something that affects just this type of situation, or does it also influence other areas of your life? (circle one number)

just this situation	1	2	3	4	5	6	7	all situations

9. You are found guilty of a minor violation of the law.

A. Write down one major cause:

B. Is the cause of this due to something about you or something about other people or circumstances? (circle one number)

totally due to others	1	2	3	4	5	6	7	totally due to me

C. In the future, will this cause be present again? (circle one number)

never present	1	2	3	4	5	6	7	always present

D. Is this cause something that affects just this type of situation, or does it also influence other areas of your life? (circle one number)

just this situation	1	2	3	4	5	6	7	all situations

---

10. You and your family have a serious argument.

A. Write down one major cause:

B. Is the cause of this due to something about you or something about other people or circumstances? (circle one number)

totally due to others	1	2	3	4	5	6	7	totally due to me

C. In the future, will this cause be present again? (circle one number)

never present	1	2	3	4	5	6	7	always present

D. Is this cause something that affects just this type of situation, or does it also influence other areas of your life? (circle one number)

just this situation	1	2	3	4	5	6	7	all situations

11. You are fired from your job.

A. Write down one major cause:

B. Is the cause of this due to something about you or something about other people or circumstances? (circle one number)

totally due to others	1	2	3	4	5	6	7	totally due to me

C. In the future, will this cause be present again? (circle one number)

never present	1	2	3	4	5	6	7	always present

D. Is this cause something that affects just this type of situation, or does it also influence other areas of your life? (circle one number)

just this situation	1	2	3	4	5	6	7	all situations

---

12. After your first term at school, you are on academic probation.

A. Write down one major cause:

B. Is the cause of this due to something about you or something about other people or circumstances? (circle one number)

totally due to others	1	2	3	4	5	6	7	totally due to me

C. In the future, will this cause be present again? (circle one number)

never present	1	2	3	4	5	6	7	always present

D. Is this cause something that affects just this type of situation, or does it also influence other areas of your life? (circle one number)

just this situation	1	2	3	4	5	6	7	all situations

13. Your best friend tells you that you are not to be trusted.

A. Write down one major cause:

B. Is the cause of this due to something about you or something about other people or circumstances? (circle one number)

totally due to others	1	2	3	4	5	6	7	totally due to me

C. In the future, will this cause be present again? (circle one number)

never present	1	2	3	4	5	6	7	always present

D. Is this cause something that affects just this type of situation, or does it also influence other areas of your life? (circle one number)

just this situation	1	2	3	4	5	6	7	all situations

---

14. You have a lot of trouble understanding what your new employer requires of you.

A. Write down one major cause:

B. Is the cause of this due to something about you or something about other people or circumstances? (circle one number)

totally due to others	1	2	3	4	5	6	7	totally due to me

C. In the future, will this cause be present again? (circle one number)

never present	1	2	3	4	5	6	7	always present

D. Is this cause something that affects just this type of situation, or does it also influence other areas of your life? (circle one number)

just this situation	1	2	3	4	5	6	7	all situations

15. You cannot sleep soundly.

A. Write down one major cause:

B. Is the cause of this due to something about you or something about other people or circumstances? (circle one number)

totally due to others	1	2	3	4	5	6	7	totally due to me

C. In the future, will this cause be present again? (circle one number)

never present	1	2	3	4	5	6	7	always present

D. Is this cause something that affects just this type of situation, or does it also influence other areas of your life? (circle one number)

just this situation	1	2	3	4	5	6	7	all situations

---

16. You experience sexual difficulties.

A. Write down one major cause:

B. Is the cause of this due to something about you or something about other people or circumstances? (circle one number)

totally due to others	1	2	3	4	5	6	7	totally due to me

C. In the future, will this cause be present again? (circle one number)

never present	1	2	3	4	5	6	7	always present

D. Is this cause something that affects just this type of situation, or does it also influence other areas of your life? (circle one number)

just this situation	1	2	3	4	5	6	7	all situations

17. You confront a serious conflict in your values.

A. Write down one major cause:

B. Is the cause of this due to something about you or something about other people or circumstances? (circle one number)

totally due to others	1	2	3	4	5	6	7	totally due to me

C. In the future, will this cause be present again? (circle one number)

never present	1	2	3	4	5	6	7	always present

D. Is this cause something that affects just this type of situation, or does it also influence other areas of your life? (circle one number)

just this situation	1	2	3	4	5	6	7	all situations

---

18. Your roommate tells you he/she is switching to a room down the hall.

A. Write down one major cause:

B. Is the cause of this due to something about you or something about other people or circumstances? (circle one number)

totally due to others	1	2	3	4	5	6	7	totally due to me

C. In the future, will this cause be present again? (circle one number)

never present	1	2	3	4	5	6	7	always present

D. Is this cause something that affects just this type of situation, or does it also influence other areas of your life? (circle one number)

just this situation	1	2	3	4	5	6	7	all situations

19. There are few recreational activities in which you are interested.

A. Write down one major cause:

B. Is the cause of this due to something about you or something about other people or circumstances? (circle one number)

totally due to others	1	2	3	4	5	6	7	totally due to me

C. In the future, will this cause be present again? (circle one number)

never present	1	2	3	4	5	6	7	always present

D. Is this cause something that affects just this type of situation, or does it also influence other areas of your life? (circle one number)

just this situation	1	2	3	4	5	6	7	all situations

---

20. Your holiday vacation plans are cancelled.

A. Write down one major cause:

B. Is the cause of this due to something about you or something about other people or circumstances? (circle one number)

totally due to others	1	2	3	4	5	6	7	totally due to me

C. In the future, will this cause be present again? (circle one number)

never present	1	2	3	4	5	6	7	always present

D. Is this cause something that affects just this type of situation, or does it also influence other areas of your life? (circle one number)

just this situation	1	2	3	4	5	6	7	all situations

21. You have trouble with one of your instructors.

A. Write down one major cause:

B. Is the cause of this due to something about you or something about other people or circumstances? (circle one number)

totally due to others	1	2	3	4	5	6	7	totally due to me

C. In the future, will this cause be present again? (circle one number)

never present	1	2	3	4	5	6	7	always present

D. Is this cause something that affects just this type of situation, or does it also influence other areas of your life? (circle one number)

just this situation	1	2	3	4	5	6	7	all situations

---

22. You experience financial difficulties.

A. Write down one major cause:

B. Is the cause of this due to something about you or something about other people or circumstances? (circle one number)

totally due to others	1	2	3	4	5	6	7	totally due to me

C. In the future, will this cause be present again? (circle one number)

never present	1	2	3	4	5	6	7	always present

D. Is this cause something that affects just this type of situation, or does it also influence other areas of your life? (circle one number)

just this situation	1	2	3	4	5	6	7	all situations

23. Your attempt to capture the interest of a specific person of the opposite sex is a failure.

A. Write down one major cause:

B. Is the cause of this due to something about you or something about other people or circumstances? (circle one number)

totally due to others	1	2	3	4	5	6	7	totally due to me

C. In the future, will this cause be present again? (circle one number)

never present	1	2	3	4	5	6	7	always present

D. Is this cause something that affects just this type of situation, or does it also influence other areas of your life? (circle one number)

just this situation	1	2	3	4	5	6	7	all situations

---

24. You feel sick and tired all of the time.

A. Write down one major cause:

B. Is the cause of this due to something about you or something about other people or circumstances? (circle one number)

totally due to others	1	2	3	4	5	6	7	totally due to me

C. In the future, will this cause be present again? (circle one number)

never present	1	2	3	4	5	6	7	always present

D. Is this cause something that affects just this type of situation, or does it also influence other areas of your life? (circle one number)

just this situation	1	2	3	4	5	6	7	all situations

Appendix E

LIFE EXPERIENCES SURVEY

Listed below are a number of events which sometimes bring about change in the lives of those who experience them and which necessitate social readjustment. For **events that have happened to you in the past year**, please indicate the extent to which you viewed the event as having either a positive or negative impact on your life (by **putting a number from -3 to +3 in the blank space**). That is, indicate the type and extent of impact that the event had. A rating of -3 would indicate an extremely negative impact. A rating of 0 suggests no impact either positive or negative. A rating of +3 would indicate an extremely positive impact. Then put a checkmark (√) to indicate when in your lifetime that the event occurred (either in the past year, or in your lifetime)

-3	-2	-1	0	+1	+2	+3
Extremely negative	Moderately negative	Somewhat negative	No impact	Slightly positive	Moderately positive	Extremely positive

Impact Event	**Past year**	**Lifetime**
1. Marriage	____	____
2. Detention in jail or Comparable institution	____	____
3. Death of Spouse	____	____
4. Major change in sleeping habits (much more or much less sleep)	____	____
5. Death of close family	____	____
a. mother	____	____
b. father	____	____
c. brother	____	____
d. sister	____	____
e. grandmother	____	____
f. grandfather	____	____
g. other (specify)	____	____
6. Major change in eating Habits (much more or less food intake)	____	____
7. Foreclosure on Mortgage or loan	____	____
8. Death of close friend	____	____
9. Outstanding personal achievement	____	____
10. Minor law violations (traffic tickets, disturbing the peace, etc.)	____	____
11. Male: Wife/girlfriend's pregnancy	____	____
12. Female: Pregnancy	____	____

Impact Event	**Past year**	**Lifetime**
13. Changed work situation (different work responsibility, major change in working conditions, working hours, etc.)	____	____
14. New Job	____	____
15. Serious illness or injury of close family member:	____	____
a. father	____	____
b. mother	____	____
c. sister	____	____
d. brother	____	____
e. grandfather	____	____
f. grandmother	____	____
g. spouse	____	____
h. other (specify)	____	____
16. Sexual difficulties	____	____
17. Trouble with employer (in danger of losing job, being suspended, demoted, etc.)	____	____
18. Trouble with in-laws	____	____
19. Major change in financial status (a lot better or a lot worse off)	____	____
20. Major change in closeness of family members (increased or decreased)	____	____
21. Gaining a new family member (through birth, adoption, family member moving in, etc.)	____	____
22. Change in residence	____	____

-3 Extremely negative	-2 Moderately negative	-1 Somewhat negative	0 No impact	+1 Slightly positive	+2 Moderately positive	+3 Extremely positive

Impact Event	Past year	Lifetime	Impact Event	Past year	Lifetime
23. Marital separation mate (due from to conflict)	____	____	38. Divorce	____	____
24. Major change in church activities (increased or decreased attendance)	____	____	39. Serious injury or illness (you or to a close friend)	____	____
25. Martial reconciliation with mate	____	____	40. Retirement from work	____	____
26. Major change in number of arguments with spouse (a lot more or a lot less arguments)	____	____	41. Son or daughter leaving home	____	____
27. Married male: Change in wife's work outside the home (beginning work, ceasing work, change to a new job, etc.)	____	____	42. Ending of formal schooling	____	____
28. Married female: Change in husband's work, (loss of job, beginning a new job, retirement, etc.)	____	____	43. Separation from spouse (due to work, travel, etc.)	____	____
29. Major change in usual type and/or amount of recreation	____	____	44. Engagement	____	____
30. Borrowing more than $10,000 (buying car, TV, getting school loan, etc.)	____	____	45. Breaking up with boyfriend or girlfriend	____	____
31. Borrowing less than $10,000 (buying car, TV, getting school loan, etc.)	____	____	46. Leaving home for the first time	____	____
32. Being fired from a job	____	____	47. Reconciliation with boyfriend/girlfriend	____	____
33. Male: Wife/girlfriend having an abortion	____	____	48. Beginning a new school experience at a higher academic level (college, graduate school, professional school, etc.)	____	____
34. Female: having an abortion	____	____	49. Changing to a new school at same academic level (undergraduate, graduate, etc.)	____	____
35. Major personal illness or injury	____	____	50. Academic probation	____	____
36. Major change in social activities, e.g. parties, movies, visiting (increased or decreased)	____	____	51. Being dismissed from dormitory or other residence	____	____
37. Major change in living conditions of family (building new home, remodeling, deterioration of home, neighborhood, etc.)	____	_____	52. Failing an important exam	____	____
			53. Changing a major	____	____
			54. Failing a course	____	____
			55. Dropping a course	____	____
			56. Joining a fraternity/ sorority	____	____
			57. Financial problems concerning school (in danger of not having sufficient money to continue)	____	____
			Other recent experiences which have had an impact on your life. List and rate.		
			58. _____	____	____
			59. _____	____	____
			60. _____	____	____

Appendix F

BRIEF COPE

These items deal with ways you've been coping with the stress in your life. There are many ways to try to deal with problems. These items ask what you've been doing to cope with this one. Each item says something about a particular way of coping. I want to know to what extent you've been doing what the item says – How much or how frequently. Don't answer on the basis of whether it seems to be working or not—***just whether or not you're doing it***. Use these response choices. Try to ***rate each item separately*** in your mind from the others. Make your answers as true FOR YOU as you can.

1	**2**	**3**	**4**
I haven't been doing this at all	**I've been doing this a little bit**	**I've been doing this a medium amount**	**I've been doing this a lot**

1. _____ I've been turning to work or other activities to take my mind off things.
2. _____ I've been concentrating my efforts on doing something about the situation I'm in.
3. _____ I've been saying to myself "this isn't real.
4. _____ I've been using alcohol or other drugs to make myself feel better.
5. _____ I've been getting emotional support from others.

6. _____ I've been giving up trying to deal with it.
7. _____ I've been taking action to try to make the situation better.
8. _____ I've been refusing to believe that it has happened.
9. _____ I've been saying things to let my unpleasant feelings escape.
10. _____ I've been getting help and advice from other people.

11. _____ I've been using alcohol or other drugs to help me get through it.
12. _____ I've been trying to see it in a different light, to make it seem more positive.
13. _____ I've been criticizing myself.
14. _____ I've been trying to come up with a strategy about what to do.
15. _____ I've been getting comfort and understanding from someone.

16. _____ I've been giving up the attempt to cope.
17. _____ I've been looking for something good in what is happening.
18. _____ I've been making jokes about it.
19. _____ I've been doing something to think about it less, such as going to movies, watching TV, reading, daydreaming, sleeping, or shopping.

20. _____ I've been accepting the reality of the fact that it has happened.
21. _____ I've been expressing my negative feelings.
22. _____ I've been trying to find comfort in my religion or spiritual beliefs.
23. _____ I've been trying to get advice or help from other people about what to do.
24. _____ I've been learning to live with it.

25. _____ I've been thinking hard about what steps to take.
26. _____ I've been blaming myself for things that happened.
27. _____ I've been praying or meditating.
28. _____ I've been making fun of the situation.

Appendix G

IES – REVISED

Instructions: The following is a list of difficulties people sometimes have after stressful life events. Please read each item, and then indicate how distressing each difficulty has been for you *during the past 7 days* with respect to _____. How much were you distressed or bothered by these difficulties?

	Not at All	A little bit	Moderately	Quite a bit	Extremely
1. Any reminder brought back feelings about it.	0	1	2	3	4
2. I had trouble staying asleep.	0	1	2	3	4
3. Other things kept making me think about it.	0	1	2	3	4
4. I felt irritable and angry.	0	1	2	3	4
5. I avoided letting myself get upset when I thought about it or was reminded of it.	0	1	2	3	4
6. I thought about it when I didn't mean to.	0	1	2	3	4
7. I felt as if it hadn't happened or wasn't real.	0	1	2	3	4
8. I stayed away from reminders about it.	0	1	2	3	4
9. Pictures about it popped into my mind.	0	1	2	3	4
10. I was jumpy and easily startled.	0	1	2	3	4
11. I tried not to think about it.	0	1	2	3	4
12. I was aware that I had a lot of feelings about it, but I didn't deal with them.	0	1	2	3	4
13. My feelings about it were kind of numb.	0	1	2	3	4

	Not at All	A little bit	Moderately	Quite a bit	Extremely
14. I found myself acting or feeling like I was back at that time.	0	1	2	3	4
15. I had trouble falling asleep.	0	1	2	3	4
16. I had waves of strong feelings about it.	0	1	2	3	4
17. I tried to remove it from my memory.	0	1	2	3	4
18. I had trouble concentrating.	0	1	2	3	4
19. Reminders of it caused me to have physical reactions, such as sweating, trouble breathing, nausea, or pounding heart.	0	1	2	3	4
20. I had dreams about it.	0	1	2	3	4
21. I felt watchful and on guard.	0	1	2	3	4
22. I tried not to talk about it.	0	1	2	3	4

Appendix H

PAIN ANXIETY SYMPTOM SCALE

Individuals who experience pain develop different ways to respond to that pain. We would like to know what you do and what you think about when in pain. Please use the rating scale below to indicate how often you engage in each of the following thoughts or activities. Circle any number from 0 (NEVER) to 5 (ALWAYS) for each item.

PASS-20

		NEVER					ALWAYS
1.	I think that if my pain gets too severe, it will never decrease	0	1	2	3	4	5
2.	When I feel pain I am afraid that something terrible will happen	0	1	2	3	4	5
3.	I go immediately to bed when I feel severe pain	0	1	2	3	4	5
4.	I begin trembling when engaged in activity that increases pain	0	1	2	3	4	5
5.	I can't think straight when I am in pain	0	1	2	3	4	5
6.	I will stop any activity as soon as I sense pain coming on	0	1	2	3	4	5
7.	Pain seems to cause my heart to pound or race	0	1	2	3	4	5
8.	As soon as pain comes on I take medication to reduce it	0	1	2	3	4	5
9.	When I feel pain I think that I may be seriously ill	0	1	2	3	4	5

10.	During painful episodes it is difficult for me to think of anything else besides the pain	**0**	**1**	**2**	**3**	**4**	**5**
11.	I avoid important activities when I hurt	**0**	**1**	**2**	**3**	**4**	**5**
12.	When I sense pain I feel dizzy or faint	**0**	**1**	**2**	**3**	**4**	**5**
13.	Pain sensations are terrifying	**0**	**1**	**2**	**3**	**4**	**5**
14.	When I hurt I think about the pain constantly	**0**	**1**	**2**	**3**	**4**	**5**
15.	Pain makes me nauseous (feel sick)	**0**	**1**	**2**	**3**	**4**	**5**
16.	When pain comes on strong I think I might become paralyzed or more disable	**0**	**1**	**2**	**3**	**4**	**5**
17.	I find it hard to concentrate when I hurt	**0**	**1**	**2**	**3**	**4**	**5**
18.	I find it difficult to calm my body down after periods of pain	**0**	**1**	**2**	**3**	**4**	**5**
19.	I worry when I am in pain	**0**	**1**	**2**	**3**	**4**	**5**
20.	I try to avoid activities that cause pain	**0**	**1**	**2**	**3**	**4**	**5**

Appendix I

ASI

Respond to each item by indicating the number of the phrase which best represents the extent to which you agree with the item. If any of the items address something that is not part of your experience (i.e., "it scares me when I feel shaky" for someone who has never trembled or had the "shakes"), answer on the basis of how you think you might feel if you had such an experience. Otherwise answer all items on the basis of your own experience. Be careful to make only one choice for each item and please answer all items.

0	1	2	3	4
Very Little	A Little	Some	Much	Very Much

_____ 1. It is important to me not to appear nervous.

_____ 2. When I cannot keep my mind on a task, I worry that I might be going crazy.

_____ 3. It scares me when I feel "shaky" (trembling).

_____ 4. It scares me when I feel faint.

_____ 5. It is important to me to stay in control of my emotions.

_____ 6. It scares me when my heart beats rapidly.

_____ 7. It embarrasses me when my stomach growls.

_____ 8. It scares me when I am nauseous.

_____ 9. When I notice that my heart is beating rapidly, I worry that I might have a heart attack.

_____ 10. It scares me when I am short of breath.

_____ 11. When my stomach is upset, I worry that I might be seriously ill.

_____ 12. It scares me when I am unable to keep my mind on a task.

_____ 13. Other people notice when I feel shaky.

_____ 14. Unusual body sensations scare me.

_____ 15. When I am nervous, I worry that I might be mentally ill.

_____ 16. It scares me when I am nervous.

Appendix J

IUS-12

Please rate each item based on the following scale.

Not at all characteristic of me				Entirely characteristic of me
1	2	3	4	5

____ 1. Unforeseen events upset me greatly.

____ 2. It frustrates me not having all the information I need.

____ 3. One should always look ahead so as to avoid surprises.

____ 4. A small, unforeseen event can spoil everything, even with the best of planning.

____ 5. I always want to know what the future has in store for me.

____ 6. I can't stand being taken by surprise.

____ 7. I should be able to organize everything in advance.

____ 8. Uncertainty keeps me from living a full life.

____ 9. When it's time to act, uncertainty paralyses me.

____ 10. When I am uncertain I can't function very well.

____ 11. The smallest doubt can stop me from acting.

____ 12. I must get away from all uncertain situations.

Appendix K

POMS SCALE

Directions: Below is a list of words that describe feelings that people have. Please read each one carefully. Then select the number that best describes HOW YOU FEEL RIGHT NOW. Place that number on the small line to the left of each word. Do not skip any items, and print your numbers clearly.

0 = Not at all
1 = A little
2 = Moderately
3 = Quite a bit
4 = Extremely

_____ 1. Tense

_____ 2. Unhappy

_____ 3. Sorry for things done

_____ 4. Shaky

_____ 5. Sad

_____ 6. On edge

_____ 7. Blue

_____ 8. Panicky

_____ 9. Hopeless

_____ 10. Relaxed

_____ 11. Unworthy

_____ 12. Uneasy

_____ 13. Restless

_____ 14. Discouraged

_____ 15. Nervous

_____ 16. Lonely

_____ 17. Miserable

_____ 18. Anxious

_____ 19. Gloomy

_____ 20. Desperate

_____ 21. Helpless

_____ 22. Worthless

_____ 23. Terrified

_____ 24. Guilty

Appendix L

VISUAL ANALOG STRESS SCALE

Please rate the level of stress you felt during the task.

1----------2----------3----------4----------5----------6----------7

Not at all stressed	Moderately stressed	Extremely stressed

Appendix M

PERCEIVED STRESS SCALE

The questions in this scale ask you about your feelings and thoughts during the last month. In each case, you will be asked to indicate *how often* you felt or thought a certain way. Although some of the questions are similar, there are differences between them and you should treat each one as a separate question. The best approach is to answer each question fairly quickly. That is, don't try to count up the number of times you felt a particular way, but rather indicate the alternative that seems like a reasonable estimate. *For each question, choose from the following alternatives:*

0----------1----------2----------3----------4
Never Almost Never Sometimes Fairly Often Very Often

____ 1. In the very last month, how often have you been upset because of something that happened unexpectedly?

____ 2. In the last month, how often have you felt that you were unable to control the important things in your life?

____ 3. In the last month, how often have you felt nervous and "stressed"?

____ 4. In the last month, how often have you dealt successfully with irritating life hassles?

____ 5. In the last month, how often have you felt that you were effectively coping with important changes that were occurring in your life?

____ 6. In the last month, how often have you felt confident about your ability to handle your personal problems?

____ 7. In the last moth, how often have you felt that things were going your way?

____ 8. In the last month, how often have you found that you could not cope with all the things that you had to do?

____ 9. In the last month, how often have you been able to control irritations in your life?

____ 10. In the last month, how often have you felt that you were on top of things?

____ 11. In the last month, how often have you been angered because of things that happened that were outside of your control?

____ 12. In the last month, how often have you found yourself thinking about things that you have to accomplish?

____ 13. In the last month, how often have you been able to control the way you spend your time?

____ 14. In the last month, how often have you felt difficulties were piling up so high that you could not overcome them?

Appendix N

BDI-II

Instructions: This questionnaire consists of 21 groups of statements. Please read each group of statements carefully, and then pick out the **one statement** in each group that best describes the way you have been feeling during the **past two weeks, including today**. Circle the number beside the statement you have picked. If several statements in the group seem to apply equally well, circle the highest number for that group. Be sure that you do not choose more than one statement for any group, including Item 16 (Changes in Sleeping Pattern) or Item 18 (Changes in Appetite).

1. Sadness
   - 0 I do not feel sad.
   - 1 I feel sad much of the time.
   - 2 I am sad all the time.
   - 3 I am so sad or unhappy that I can't stand it.

2. Pessimism
   - 0 I am not discouraged about my future.
   - 1 I feel more discouraged about my future than I used to be.
   - 2 I do not expect things to work out for me.
   - 3 I feel my future is hopeless and will only get worse.

3. Past Failure
   - 0 I do not feel like a failure.
   - 1 I have failed more than I should have.
   - 2 As I look back, I see a lot of failures.
   - 3 I feel I am a total failure as a person.

4. Loss of Pleasure
   - 0 I get as much pleasure as I ever did from the things I enjoy.
   - 1 I don't enjoy things as much as I used to.
   - 2 I get very little pleasure from the things I used to enjoy.
   - 3 I can't get any pleasure from the things I used to enjoy.

5. Guilty Feelings
   - 0 I don't feel particularly guilty.
   - 1 I feel guilty over many things I have done or should have done.

8. Self-Criticalness

0 I don't criticize or blame myself more than usual

1 I am more critical of myself than I used to be.

2 I criticize myself for all of my faults.

3 I blame myself for everything bad that happens.

9. Suicidal Thoughts or Wishes

0 I don't have any thoughts of killing myself.

1 I have thoughts of killing myself, but I would not carry them out.

2 I would like to kill myself.

3 I would kill myself if I had the chance.

10. Crying

0 I don't cry anymore than I used to.

1 I cry more than I used to.

2 I cry over every little thing.

3 I feel like crying, but I can't.

11. Agitation

0 I am no more restless or would up than usual.

1 I feel more restless or wound up than usual.

2 I am so restless or agitated that it's hard to stay still.

3 I am so restless or agitated that I have to keep moving or doing something.

12. Loss of Interest

0 I have not lost interest in other people or activities.

1 I am less interested in other people or things than before.

15. Loss of Energy

0 I have as much energy as ever.

1 I have less energy than I used to have.

2 I don't have enough energy to do very much.

3 I don't have enough energy to do anything.

16. Changes in Sleeping Pattern

0 I have not experienced any change in my sleeping pattern.

1a I sleep somewhat more than usual.

1b I sleep somewhat less than usual.

2a I sleep a lot more than usual.

2b I sleep a lot less than usual

3a I sleep most of the day.

3b I wake up 1-2 hours early and can't get back to sleep.

17. Irritability

0 I am no more irritable than usual.

1 I am more irritable than usual.

2 I am much more irritable than usual.

3 I am irritable all the time.

18. Changes in Appetite

0 I have not experienced any change in my appetite.

1a My appetite is somewhat less than usual.

1b My appetite is somewhat greater than usual.

2a My appetite is much less than before.

2b My appetite is much greater than usual.

3a I have no appetite at all.

3b I crave food all the time.

19. Concentration Difficulty

Appendix O

DEMOGRAPHICS PAGE

Date: _____

Age: _____

Race: _____

Highest Educational Attainment: _____

Current Household Income (circle one):

Under 10,000 10,000-19,999 20,000-29,999

30,000-39,999 40,000-49,999 50,000-59,999

Greater than 60,000

Relationship Status (circle one):

Single Living with partner Married

Divorced Widowed Separated

Current medications: _____

Are you currently taking any form of birth control medications? _____ yes _____no

If so, which one? _____

Weight: _____

Height: _____

Please indicate the actual date of your last menstrual cycle, in other words, what was the date when you began blood flow (e.g., April 15, 2006).

_____

Do you have a family history of posttraumatic stress disorder? _____ yes _____ no

Do you have a family history of chronic pain? _____ yes _____ no

Appendix P

DEBRIEFING STATEMENT

Assessing a Biopsychosocial Model for PTSD and Chronic Pain
Debriefing Statement

The purpose of this study was to look at aspects of a biopsychosocial model that has been proposed to account for the relationship between PTSD and chronic pain. Four groups of women were assessed in this study: women with posttraumatic stress disorder (PTSD) without chronic pain, women with musculoskeletal pain without PTSD, women with both musculoskeletal pain and PTSD, and women who no not have a diagnosis of either PTSD or chronic pain.

During the course of this study, participants were asked to prepare and give a speech about a dream job, and why they would be the perfect applicant for this job. Participants were told that they would be giving the speech in front of two "managers" that are trained to monitor nonverbal behavior. These "managers" were actually research assistants who did not have any training in monitoring nonverbal behavior. In addition, the videotape was not recording during the speech. Participants then began a 5 minute mental arithmetic task, where they were instructed to serially subtract 13 from 1,022 as quickly and accurately as possible for a five minute period. The purpose of these tasks was to induce stress. Participants were also asked to complete questionnaires regarding their thoughts and feelings. In addition, salivary cortisol samples were collected. Cortisol is a hormone in your body that is released in response to stress. These samples were taken to see how participants in each group responded to the stressful event, and to see how long it will take their body to return to their original level of cortisol following the stress event. Below is a list of hypotheses that are being assessed in this study:

Study hypotheses:

1. Individuals who have either PTSD, chronic pain, or both conditions will have lower initial levels of cortisol levels before the experimental stressor, and blunted (i.e. lower) salivary cortisol levels following the experimental stressor.

2. Individuals in all groups will experience symptoms of depression and anxiety on a continuum. Individuals with PTSD and pain group are anticipated to report the highest depressive and anxiety symptomatology (both at baseline and in response to the experimental stressor), followed by individuals with only PTSD or chronic pain. Participants in the control group are anticipated to report to least depressive and anxiety symptomatology.

3. Individuals who have either PTSD, chronic pain, or both conditions will be more likely to make to internal, stabile, and global attributions about events than individuals in the control group.

4. Individuals who have either PTSD, chronic pain, or both conditions will be more likely to report negative life events and general levels of self-perceived stress. Subsequently, these individuals will also be more likely to view ambiguous situations as more threatening than individuals in the control group.

5. Individuals who have either PTSD, chronic pain, or both conditions will be more likely to use negative coping strategies (i.e., denial, behavioral disengagement, self-blame) than individuals in the control group.

6. Individuals who have PTSD or chronic pain and PTSD will be more likely to report symptoms of PTSD. Similarly, individuals with chronic pain or PTSD and chronic pain will be more likely to report anxiety associated with their pain condition.

BIOGRAPHY OF THE AUTHOR

Anna Cassel was born in Brighton, Massachusetts on April 21, 1980. She graduated from Brookline High School in 1998 and the University of Massachusetts in 2003 with a Bachelor's degree in Psychology and Communications and a specialization in Developmental Disabilities. From 2003 until 2005, she worked as a research assistant for the Primary Care Anxiety Project at Brown University. In 2005, Anna entered the Clinical Psychology Doctoral Program at the University of Maine. While at the University of Maine, she was involved in research assessing for aspects of women's health, posttraumatic stress disorder, panic disorder, and binge eating disorder. While working with Dr. Sandra Sigmon, Anna has co-authored 3 publications, 2 symposia presentations, and 17 poster presentations. Anna is a member of the Association for Behavioral and Cognitive Therapies (ABCT), the Women's Issues in Behavior Therapy Special Interest Group, the American Psychological Association (APA), and the American Psychological Association of Graduate Students (APAGS).

Anna completed her predoctoral internship in the Health Psychology track at the VA Maryland Healthcare System/University of Maryland Baltimore Psychology Internship Consortium. She will be completing her postdoctoral residency in the Primary Care Behavioral Health track at the Edith Nourse Rogers Memorial VA Medical Center in Bedford, Massachusetts. Anna is a candidate for the Doctor of Philosophy degree in Psychology from the University of Maine in August, 2010.