

## An examination of the relationship between chronic pain and post-traumatic stress disorder

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**Abstract**—Chronic pain and post-traumatic stress disorder (PTSD) are frequently observed within the Department of Veterans Affairs healthcare system and are often associated with a significant level of affective distress and physical disability. Clinical practice and research suggest that these two conditions co-occur at a high rate and may interact in such a way as to negatively impact the course of either disorder; however, relatively little research has been conducted in this area. This review summarizes the current literature pertaining to the prevalence and development of chronic pain and PTSD. Research describing the comorbidity of both conditions is reviewed, and several theoretical models are presented to explain the mechanisms by which these two disorders may be maintained. Future directions for research and clinical implications are discussed.

**Key words:** anxiety, avoidance, chronic pain, post-traumatic stress disorder, vulnerability.

### INTRODUCTION

Over the past 25 years, the development of a greater understanding of the functional, psychological, and social problems commonly associated with chronic pain has gradually evolved. As a result, substantial literature currently exists documenting the relationship between chronic pain and disorders such as substance abuse [1], depression [2], and anxiety disorders [3,4], and researchers and clinicians acknowledge that these conditions, as well as others, can play a role in the experience of chronic pain. Importantly, interest in the relationship between

chronic pain and its comorbid conditions has had a significant impact on the field of pain research and has contributed to improvements in the delivery of treatment.

While some chronic pain conditions may have an organic etiology and develop gradually over time, other conditions may develop because of an injury sustained in a traumatic event such as a motor vehicle accident (MVA), work-related injury, or participation in military combat. Most recently, there has been burgeoning interest in the relationship between pain and post-traumatic stress disorder (PTSD). Clinical practice and research indicate that the two disorders frequently co-occur and may interact in such a way as to negatively impact the course and outcome of treatment of either disorder. Despite this recent interest, a review of the relevant literature indicates that neither empirical studies investigating theoretical models to explain the comorbidity of the two disorders nor well-controlled studies investigating the efficacy of tailoring treatments for individuals for which

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**Abbreviations:** ASI = anxiety sensitivity index, MVA = motor vehicle accident, PTSD = post-traumatic stress disorder.

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pain and PTSD co-occur have been done. The lack of controlled research in this area is unfortunate because such studies could significantly advance theory development and improve treatment efficacy.

This paper primarily provides a critical review and synthesis of the existing literature investigating the relationship between chronic pain and PTSD. The paper will begin with a presentation of the diagnostic criteria, prevalence, and theoretical models of chronic pain and PTSD. Research will then be presented that describes the co-occurrence of the two disorders, and several models will be highlighted that may explain the similar mechanisms by which these two disorders may be maintained. Finally, the paper will close with a call for continued research and refinements of the proposed models.

## DISCUSSION

### Chronic Pain

Pain is defined as an unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage [5]. Although pain is typically a transient experience, for some people, pain persists past the point where it is considered adaptive and contributes to affective distress, to impaired occupational and social functioning, and to increased use of healthcare system resources [6–8]. Pain that persists for 6 months or longer is typically referred to as “chronic” pain [5]. Chronic pain is the most common complaint made by patients to their primary care providers and accounts for an estimated \$75 to \$100 billion a year in the United States in lost productivity and healthcare costs [9].

Vlaeyen and Linton propose a cognitive-behavioral fear-avoidance model of chronic pain to explain the role of fear and avoidance behaviors in the development and maintenance of chronic pain and related functional limitations [10]. According to this model, the experience of pain may be interpreted by some patients as overly threatening, a process called “catastrophizing.” Research consistently indicates that catastrophizing is a significant predictor of negative outcomes for patients with pain [11]. Vlaeyen and Linton propose that catastrophizing contributes to a fear of pain and may lead to a variety of events [10], including avoidance of activities that may elicit pain, guarding behaviors, and hypervigilance to bodily sensations. Such avoidance may also contribute to disabili-

ty and depressive symptoms. As an individual becomes more depressed and inactive, the cycle of pain is fueled even further, and fear and avoidance are further increased. However, in the absence of serious somatic pathology, individuals who confront the pain rather than catastrophize are more likely to have quicker recoveries because of their greater participation in daily activities [12].

### Post-Traumatic Stress Disorder

A person typically develops PTSD following an exposure to a situation or event that is, or is perceived to be, threatening to his or her well being or to that of another person. High levels of anxiety, depression, panic, and substance abuse are frequently observed in individuals with PTSD [13]. Its distinctive profile of symptoms includes (1) reexperiencing the event in the form of intrusive thoughts, nightmares, dissociative flashbacks to elements of the original traumatic event, and psychophysiological reactivity to cues of the traumatic event and preoccupation with that event; (2) avoidance of thoughts, people, and places that resemble the traumatic event, emotional numbing, an absence of emotional attachments, and an inability to feel the range of positive emotions; and (3) symptoms of hyperarousal, including heightened startle sensitivity, sleep problems, attentional difficulties, hypervigilance, and the presence of irritability, anger, or rage [14].

In the United States, the prevalence of PTSD is estimated to be 6 percent in males and 12 percent in females [15]. However, exposure to traumatic events has been estimated to be as high as 70 percent of the adult population [16,17]. These numbers suggest that trauma and PTSD are among the most frequent psychological disorders, ranking behind substance abuse and depression. In the most comprehensive study of the effects of war on its combatants, the National Vietnam Veterans Readjustment Study found a lifetime rate of PTSD of 30 percent and a current rate of 15 percent [18]. An international study of women estimated a lifetime rate of PTSD of 12 percent and a current rate of 5 percent [17]. While there are no reliable estimates of PTSD in developing countries, several authorities suggest that the rates may be higher because of the frequency of traumatic events and the absence of resources to buffer their effects [19].

Not everyone who is exposed to a potentially traumatic event develops PTSD. The literature suggests that although the experience of a traumatic event certainly contributes to the development of PTSD, personal vulnerabilities also play an important role [20]. Other important

factors may include prior exposure to traumatic events, age at the time of combat exposure, premilitary family instability, and postmilitary factors, including social support, additional life stressors, and personal hardiness [21,22]. Thus, psychosocial factors and personal characteristics apparently may significantly influence the impact a traumatic event has on a person's life.

The current behavioral conceptualizations of PTSD are based on the two-factor learning theory developed by Mowrer [23]. The theory proposes that fear is learned via classical conditioning as the first stage of a process that can sustain emotional learning despite the influence of naturally occurring processes that would reduce it. The second stage is marked by avoidance behavior that minimizes the contact time with the conditioned cues, thus impairing extinction of the learned fear. Keane, Zimmering, and Caddell extended this model to humans and proposed that an extreme stressor, such as a traumatic event, acts as an unconditioned stimulus (UCS) that can create learned associations with internal and external cues (e.g., sights, sounds, or smells) that are present during the stressor [24]. After these conditioned associations are established, previously neutral cues can then elicit strong autonomic and physiological responses that are similar to those experienced at the time of the stressor. These responses may be so aversive that the individual begins to avoid the triggering cues as a way to decrease their own fearful reactions [25].

Cognitive and information-processing models of PTSD have been developed from Lang's bio-informational theory of emotion [26]. According to Lang [26], "fear networks" store memory representation of anxiety-provoking events and contain information about a feared stimulus or situation; the person's cognitive, psychophysiological, and behavioral response to the stimuli or situation; and information about the meaning of the feared stimuli. Anxiety disorders develop when the fear network contains faulty connections and information that do not truly represent the state of the world. Foa and Kozak have proposed that when compared to other anxiety disorders, the size of the fear network in PTSD is larger, the networks are more easily activated, and the affective and physiological response elements of the network are more intense [27].

### **Comorbid Pain and PTSD**

A number of studies have been conducted to assess the co-occurrence of PTSD and chronic pain symptoms.

For example, Benedikt and Kolb reported that 10 percent of a sample of 225 patients referred to a Veterans Administration pain clinic met criteria for PTSD [28]. Muse reported that 9.5 percent of a sample of patients attending a multidisciplinary chronic pain center met criteria for "post-traumatic pain syndrome" [29]. When patients are referred for the assessment of a chronic pain problem resulting from a traumatic event, the prevalence of PTSD increases. Asmundson, Norton, Allerdings, Norton, and Larson performed a study to assess the extent to which work-related injuries were associated with PTSD [30]. Assessments were conducted on 139 injured workers with chronic pain who were referred to a rehabilitation program. The results indicated that 34.7 percent of the sample reported symptoms consistent with PTSD. High rates of PTSD have been reported by Hickling and colleagues for patients referred for psychological treatment following an MVA [31,32]. Research indicates that rates of PTSD in patients for which pain is secondary to an MVA range from 30 to 50 percent [31,33,34]. Studies suggest that from 24 to 47 percent of fibromyalgia patients attribute the onset of their symptoms to a physical injury associated with an MVA [35–37]. Hospitalized burn patients have been found to have high rates of PTSD (45%) at 12 months postinjury [38]. Geisser, Roth, Bachman, and Eckert examined the self-report of pain, affective distress, and disability in pain patients with and without PTSD symptoms [39]. Their results indicated that patients with accident-related pain and high PTSD symptoms reported higher levels of pain and affective distress relative to patients with accident-related pain and without PTSD, or nonaccident-related pain.

Studies examining the prevalence of chronic pain in individuals with a primary diagnosis of PTSD have reported even higher copevalence rates. McFarlane, Atchison, Rafalowicz, and Papay reported that in a sample of PTSD patients reporting physical symptoms [40], pain was the most common physical complaint (45% back pain and 34% headaches). Beckham et al. performed a study to investigate chronic pain patterns in Vietnam veterans with PTSD [41]. A sample of 129 combat veterans with PTSD completed self-report questionnaires assessing PTSD symptoms and current pain status. The results indicated that 80 percent reported the presence of a chronic pain condition. In addition, increased levels of PTSD reexperiencing symptoms were associated with increased pain level and pain-related disability. White and Faustman performed a review of discharge

summaries of 543 veterans treated for PTSD to assess the frequency and nature of medical problems [42]. Their results indicated that 60 percent had an identified medical problem and that 1 in 4 showed some type of musculoskeletal or pain problem.

The co-occurrence of chronic pain and PTSD may have implications in terms of an individual's experience of both conditions. Research indicates that patients with chronic pain related to trauma or PTSD experience more intense pain and affective distress [39,43], higher levels of life interference [44], and greater disability than pain patients without trauma or PTSD [37]. For example, Chibnall and Duckro examined a sample of chronic post-traumatic headache patients and found that PTSD pain patients had higher levels of depression and suppressed anger than non-PTSD pain patients [33]. Similarly, Tushima and Stoddard found that patients with post-traumatic headache reported more frequent pain and had a poorer prognosis than did nontraumatic headache patients [45]. Thus, these studies suggest that the presence of both PTSD and chronic pain can increase the symptom severity of either condition.

### **Chronic Pain and PTSD: Examination of Theoretical Models**

The high rate of comorbidity and symptom overlap between chronic pain and PTSD suggests that the two disorders may be related in some way. Clearly, this review of studies establishing the co-occurrence between pain and PTSD does not explain the mechanisms by which they are linked. Although theoretical models have been proposed to account for the co-occurrence of pain and PTSD, these theoretical models have yet to be tested. However, these models present numerous factors that may be implicated in the etiology and maintenance of both conditions. For clarity of presentation, each model will be presented in turn.

#### *Mutual Maintenance Model*

According to Sharp and Harvey's mutual maintenance model [46], there are seven specific factors by which mutual maintenance of chronic pain and PTSD may occur:

1. Attentional biases may be present in chronic pain and PTSD patients such that they attend to threatening or painful stimuli.
2. Anxiety sensitivity may contribute toward a vulnerability to catastrophize.

3. Pain may be a reminder of the traumatic event, triggering an arousal response, avoidance of the cause of pain, and any memories of the trauma.
4. In both disorders, avoidance may be adopted as a means to minimize pain and disturbing thoughts.
5. Fatigue and lethargy associated with depression may contribute to both disorders.
6. General anxiety may contribute to both disorders.
7. Cognitive demands from symptoms of pain and PTSD limit the use of adaptive coping strategies.

Although many of the factors described in this model have not been empirically investigated, they are useful in that they may stimulate further, more critical examinations of the comorbidity of chronic pain and PTSD and may provide several possible directions for future research. In a critique of Sharp and Harvey's mutual maintenance model [46], Asmundson, Coons, Taylor, and Katz suggest that given the multidimensional nature of both pain and PTSD [47], specific dimensions of the factors described likely may be involved while others may not. They also suggest that a distinction should be made between models of mutual maintenance and those that describe a shared vulnerability.

#### *Shared Vulnerability Model*

Asmundson et al. have proposed a shared vulnerability model of chronic pain and PTSD in which anxiety sensitivity is a predisposing factor contributing to the development of both conditions [47]. According to this model, a person with high levels of anxiety sensitivity is likely to become fearful in response to physical sensations such as heart pounding and breathlessness, thinking that these symptoms may signal impending doom. When people with high-anxiety sensitivity encounter either a traumatic stressor or pain (or both), they are believed to respond with more fear than those with low-anxiety sensitivity. Thus, the tendency to respond with fear to physical symptoms of anxiety is seen as a shared vulnerability contributing to the development of either disorder. In the case of PTSD, the degree of alarm caused by the stressor is combined with the alarm of physiological sensations to further exacerbate the emotional reaction, thereby increasing the risk of developing PTSD. In the case of chronic pain, anxiety sensitivity heightens fear and avoidance of activities that may induce pain, which further increase the chances that pain will be maintained over time.

Clinical pain research supports a relationship between anxiety sensitivity and pain. For example, Asmundson and Norton found that patients with higher anxiety sensitivity were more likely to experience greater anxiety and fear of pain, more negative affect, and greater avoidance of activities [48]. Asmundson and Taylor found that anxiety sensitivity directly increased fear of pain [49]; however, anxiety sensitivity indirectly influenced avoidance and escape behaviors through fear of pain. More recently, Zvolensky, Eifert, Lejuez, Hopko, and Forsyth evaluated anxiety sensitivity, depression, and pain severity as potential predictors of pain-related fear in a heterogeneous chronic pain population [50]. Their findings indicated that anxiety sensitivity, as measured by the Anxiety Sensitivity Index (ASI), was a better predictor of fear and anxiety about pain than other relevant variables [51]. Additionally, in a study of the relationship between anxiety sensitivity and fear of pain in healthy adolescents, anxiety sensitivity was found to account for a unique proportion of the variance in pain anxiety symptoms, even after controlling for other potential predictors of fear of pain [52]. Finally, Greenberg and Burns examined pain-related anxiety in a group of chronic musculoskeletal pain patients who underwent an experimentally induced pain induction procedure (i.e., cold pressor) and had to complete mental arithmetic tasks [53]. Results indicated that almost all effects of pain anxiety on task responses were accounted for by anxiety sensitivity. Taken together, results of all of these studies support the hypothesis that anxiety sensitivity may represent a vulnerability factor in the development and maintenance of pain-related anxiety and avoidance behaviors.

While several studies indicate that anxiety sensitivity may play a role in the development of anxiety disorders, such as panic, few studies support the model of anxiety sensitivity as a vulnerability factor in PTSD [54]. In a study that examined the presence of anxiety sensitivity in 313 individuals with anxiety disorders, Taylor, Koch, and McNally found that patients with PTSD were the second highest on the ASI measure, with patients diagnosed with panic being the highest [55]. Lang, Kennedy, and Stein found that anxiety sensitivity was a significant predictor of PTSD symptoms in women [56]. In another study, anxiety sensitivity was related to severity of PTSD symptoms, and a reduction in anxiety sensitivity after cognitive behavioral therapy (CBT) was related to a reduction in PTSD symptoms [57]. Thus, while preliminary research is supportive, additional research on the interac-

tion of anxiety sensitivity and PTSD would help clarify the role of anxiety sensitivity as a potential vulnerability factor contributing to the development of PTSD.

#### *Fear-Avoidance Model*

Norton and Asmundson recently proposed an amended fear-avoidance model of chronic pain that largely emphasizes the contributions of physiological symptoms and arousal [58,10]. According to the model, physiological symptoms (e.g., increased blood flow, heart rate, or muscle tension) may directly increase pain sensations and reinforce fears and negative beliefs that activities will be painful. When fears and negative beliefs are confirmed, avoidance behavior increases. In addition, physiological arousal may produce bodily sensations such as muscle tension, which could be misinterpreted as being pain-related. The misinterpretations (e.g., catastrophizing) are thought to be influenced by an individual's tendency to respond with fear to sensations that are anxiety-provoking (e.g., anxiety sensitivity, negative affect). While insufficient empirical evidence exists to support that physiological arousal can cause pain [59], Norton and Asmundson suggest that physiological reactivity may have an impact on anxiety, fear, catastrophizing, and performance [58].

Several similar features can be found between the fear-avoidance model of chronic pain and the models of PTSD previously presented [24,25]. First, models of chronic pain and PTSD suggest that a personal vulnerability may contribute to the development and maintenance of each disorder. As previously discussed, anxiety sensitivity may represent one such vulnerability because it has been found to be elevated in some chronic pain and panic patients; however, further research is needed to support a proposed relationship between anxiety sensitivity and PTSD.

Avoidance is also significant in models of chronic pain and PTSD. In chronic pain, fear and avoidance generally refer to the avoidance of movements or activities, such as exercise or work, for fear of causing increased pain or injury. Research indicates that avoidance can contribute to decreased feelings of self-efficacy related to pain, negative expectations and beliefs about an individual's ability to cope with pain, and increased disability [60]. Since avoidance occurs prior to pain rather than in response to pain, there are fewer opportunities for false pain-related beliefs and expectancies to be challenged. The avoidance of reinforcing activities and social situations can contribute to

affective distress, which can further exacerbate the experience of pain [61]. Similarly, for a person with PTSD, fear of reexperiencing disturbing thoughts of events and avoidance of reminders associated with the trauma are core components of this disorder. This fear and avoidance can help prevent effective processing of the event and may lead to the maintenance of intrusive symptoms and arousal [25].

#### *Triple Vulnerability Model*

Keane and Barlow propose a model of the development of PTSD that may also be applied to the development of chronic pain [20]. According to the triple vulnerability model of anxiety and PTSD [62,63], an integrated set of triple vulnerabilities needs to be present for developing an anxiety disorder: a generalized biological vulnerability, a generalized psychological vulnerability based on early experiences of control over salient events, and a more specific psychological vulnerability in which one learns to focus anxiety on specific situations. While the triple vulnerability model applies to the development of anxiety in general, Keane and Barlow propose a model of the development of PTSD specifically [20]. According to their model, a true or false alarm develops during exposure to situations that symbolize or resemble an aspect of a traumatic event. However, the experience of alarm or other intense emotions alone is not sufficient for PTSD development. To develop PTSD, one must develop anxiety or the sense that these events, including one's own emotional reactions to them, are preceding in an unpredictable and uncontrollable manner. Thus, when negative affect and a sense of uncontrollability develop, PTSD may emerge. While this model implies that a psychological and biological vulnerability to develop the disorder exists, anxiety has been found to be always moderated to some extent by variables, such as the presence of adequate coping skills and social support [20].

Although designed to describe the development of PTSD, this model may also relate to the development of chronic pain. It is possible that for some people to develop a chronic pain condition, they must also develop a belief that the pain is preceding in an unpredictable and uncontrollable manner. Numerous studies indicate that many chronic pain sufferers do, in fact, typically perceive a lack of personal control over their pain [64]. The relationship between perceived controllability and pain has been demonstrated in a variety of chronic pain syndromes, including migraine headache patients [65], low

back pain patients, and rheumatoid arthritis patients [66], to name a few. When persons perceive their pain to be uncontrollable, feelings of low self-efficacy may develop, along with negative affect. Thus, a fear may develop of entering situations in which pain may occur, leading to avoidance of situations in daily life. This avoidance will further fuel negative affect and feelings of uncontrollability and low self-efficacy. Similar to the PTSD literature, pain has been found to be always moderated to some extent by variables such as the presence of adequate coping skills and social support [67,68]. Thus, whether the "alarm" is a trauma reminder or pain reminder, the development of a sense of uncontrollability may precede the development of both disorders.

#### **Implications for Assessment and Treatment**

Given the high rates of comorbidity of chronic pain and PTSD, clinicians conducting diagnostic assessments of patients with either condition should assess for both disorders. If they have reason to believe that a history of trauma or pain exists, they can use several well-validated self-report questionnaires to help determine a diagnosis and the severity of symptoms. Self-report measures of pain include the 0 to 10 numerical pain rating scale, the McGill Pain Questionnaire [69], or for a more comprehensive assessment, the West Haven-Yale Multidimensional Pain Inventory [70]. Measures of PTSD include the Posttraumatic Stress Disorder Checklist [71], or the Clinician Administered PTSD Scale Revised (for a diagnosis of PTSD) [72]. Measures of anxiety sensitivity, cognitive and behavioral avoidance, coping style, beliefs and expectations, and self-efficacy could also be included in the assessment to gain a comprehensive understanding of the factors contributing to and maintaining these conditions.

Only a few studies have reported the results of treatments designed to address co-occurring chronic pain and PTSD. Preliminary research suggests that the use of propranolol for pain may also have the secondary benefit of preventing PTSD; however, further research is needed in this area [73]. Muse described a series of cases of co-occurring chronic pain and PTSD in which patients were treated for pain with the use of a multidisciplinary treatment approach [29]. Their results indicated that pain treatment had a minimal effect on symptoms of PTSD and that patients were subsequently successfully treated with systematic desensitization for the PTSD. Hickling et al. described a series of cases in which 20 patients with post-traumatic headache received CBT and exposure

therapy [31]. It was noted that the PTSD group required significantly longer treatment and failed to show positive results with the headache until the PTSD symptoms were addressed. Because few studies have been published currently about treatment of comorbid pain and PTSD, further investigation of effective treatments could help elucidate ways to better help this population, as well as ways to prevent the chronicity of these disorders.

The theoretical models presented on the relationship between chronic pain and PTSD have yet to be fully tested, and further research is needed before clinical treatment protocols are modified to integrate treatments for both disorders. However, data from existing research studies and clinical case studies point toward several techniques that might be useful to incorporate when a person with comorbid pain and PTSD is being treated. The strategies would likely include standard CBT techniques such as cognitive restructuring, coping skills training, and relaxation, all of which are commonly used when pain or PTSD is treated. Programs designed to treat both disorders could include education about the function of cognitive and behavioral avoidance, and patients could be instructed in ways to conduct both situational exposure exercises (e.g., doing activities that were previously avoided) as well as interoceptive exposure exercises (e.g., spinning in chair, running in place, etc.), which are designed to help patients face and cope with uncomfortable physiological sensations. Patients could also be trained in ways to reinforce positive self-efficacy beliefs, correct attentional biases, and help reduce catastrophizing. Overall, it would be important to help patients with both pain and PTSD to understand the ways that these two disorders may maintain each other and to help patients use strategies to minimize both the cognitive and behavioral avoidance that is observed so frequently in both conditions. As patients begin to decrease avoidance and increase their participation in appropriate activities, their high levels of distress and disability may possibly decrease and they will be able to obtain a more positive quality of life.

## CONCLUSIONS

The research summarized in this review suggests that chronic pain and PTSD frequently co-occur and that similar mechanisms, such as fear and avoidance, anxiety sensitivity, and catastrophizing, may exist for maintain-

ing both conditions. Although several models have been proposed to explain the relationship between chronic pain and PTSD, continued research in this area is needed so that these models can be fully developed and empirically tested. Given the high prevalence of chronic pain and PTSD among U.S. veterans, it is imperative that researchers continue to refine the models presented and develop more in-depth assessment and treatment techniques that can best fit the needs of this population. As treatment studies are launched and strategies are used and tested with patients with comorbid pain and PTSD, we may then begin to modify our existing treatment protocols. This can only be done through systematic and well-controlled research.

## REFERENCES

1. Brown RL, Patterson JJ, Rounds LA, Papanouliotis O. Substance use among patients with chronic back pain. *J Fam Pract* 1996;43:152–60.
2. Banks SM, Kerns RD. Explaining the high rates of depression in chronic pain: A stress diathesis framework. *Psychol Bull* 1996;119:95–110.
3. Asmundson GJG, Jacobson SJ, Allardings M, Norton GR. Social phobia in disabled workers with chronic musculoskeletal pain. *Behav Res Ther* 1996;34:939–43.
4. Burton K, Polatin PB, Gatchel R. Psychosocial factors and the rehabilitation of patients with chronic work-related upper extremity disorders. *J Occup Rehabil* 1997;7:139–53.
5. Merskey H, Bogduk N, editors. Classification of chronic pain. IASP task force on taxonomy. Seattle (WA): IASP Press; 1994. p. 209–14.
6. Gatchel RJ. Psychological disorders and chronic pain: Cause and effect relationships. In: Gatchel RJ, Turk D, editors. *Psychological approaches to pain management: A practitioner's handbook*. New York: Guilford; 1996. p. 33–52.
7. Benedetto MC, Kerns RD, Rosenberg R. Health risk behaviors and healthcare utilization among veterans receiving primary medical care. *J Clin Psychol Med Settings* 1998;5:441–47.
8. Feuerstein M, Huang GD, Pransky G. Work style and work-related upper extremity disorders. In: Gatchel RJ, Turk DC, editors. *Psychosocial factors in pain*. New York: Guilford; 1999. p. 175–92.
9. Weisberg JN, Vaillancourt PD. Personality factors and disorders in chronic pain. *Semin Clin Neuropsychiatry* 1999; 4:155–66.
10. Vlaeyen JWS, Linton SJ. Fear-avoidance and its consequences in musculoskeletal pain: A state of the art. *Pain* 2000;85:317–32.

11. Jensen MP, Romano JM, Turner JA, Good AB, Wald LH. Patient beliefs predict patient functioning: Further support for a cognitive-behavioral model of chronic pain. *Pain* 1999;81:95–104.
12. Vlaeyen JWS, de Jong J, Sieben J, Crombez G. Graded exposure in vivo for pain-related fear. In: Gatchel RJ, Turk DC, editors. *Psychological approaches to pain management*. New York: Guilford Press; 2002. p. 210–33.
13. Keane TM, Wolf J. Comorbidity in post-traumatic stress disorder: An analysis of community and clinical studies. *J Appl Soc Psychol* 1990;20:1776–88.
14. American Psychiatric Association. *Diagnostic and statistical manual of mental disorders*. 4th ed. Washington (DC): American Psychiatric Association; 1994.
15. Kessler RC, Sonnega A, Bromet E, Hughes M, Nelson CB. Posttraumatic stress disorder in the National Comorbidity Survey. *Arch Gen Psychiatry* 1995;52:1048–60.
16. Norris FH. Epidemiology of trauma: Frequency and impact of different potentially traumatic events on different demographic groups. *J Consult Clin Psychol* 1992;60:409–18.
17. Resnick HS, Kilpatrick DG, Dansky BS, Saunders BE, Best CL. Prevalence of civilian trauma and posttraumatic stress disorder in a representative national sample of women. *J Consult Clin Psychol* 1993;61:984–91.
18. Kulka RA, Schlenger WE, Fairbank JA, Hough RL, Jordan KB, Marmar CR, Weiss DS. *Trauma and the Vietnam War generation: Report of findings for the National Vietnam Veterans Readjustment Study*. New York: Brunner/Mazel; 1990.
19. de Girolamo G, McFarlane AC. The epidemiology of PTSD: A comprehensive review of the international literature. In: AJ Marsella, MJ Friedman, ET Gerrity, RM Scurfield, editors. *Ethnocultural aspects of posttraumatic stress disorder: Issues, research, and clinical implications*. Washington (DC): American Psychological Association; 1997. p. 33–86.
20. Keane TM, Barlow DH. Posttraumatic stress disorder. In: Barlow DH, editor. *Anxiety and its disorders*. New York: Guilford; 2002. p. 418–53.
21. King DW, King LA, Foy DW, Gudanowski DM. Prewar factors in combat related posttraumatic stress disorder: Structural equation modeling with a national sample of female and male Vietnam veterans. *J Consult Clin Psychol* 1996;64:520–31.
22. King DW, King LA, Fairbank JA, Keane TM, Adams G. Resilience-recovery factors in posttraumatic stress disorder among female and male Vietnam veterans: Hardiness, post-war social support, and additional stressful life events. *J Pers Soc Psychol* 1998;74:420–34.
23. Mowrer OH. *Learning theory and behavior*. New York: Wiley; 1960.
24. Keane TM, Zimmering RT, Caddell JM. A behavioral formulation of posttraumatic stress disorder in Vietnam veterans. *The Behavior Therapist* 1985; No. 8. p. 9–12.
25. Keane TM, Zimmering RT, Kaloupek DG. Posttraumatic stress disorder. In: Hersen M, Bellack AS, editors. *Psychopathology in adulthood: An advanced text*. Needham Heights (MA): Allyn & Bacon; 2000. p. 208–31.
26. Lang PJ. A bioinformational theory of emotional imagery. *Psychophysiology* 1979;52:1048–60.
27. Foa EB, Kozak MJ. Emotional processing of fear: Exposure to corrective information. *Psychol Bull* 1986;99:20–35.
28. Benedikt RA, Kolb LC. Preliminary findings on chronic pain and posttraumatic stress disorder. *Am J Psychiatry* 1986;143:908–10.
29. Muse M. Stress-related, posttraumatic chronic pain syndrome: Behavioral treatment approach. *Pain* 1986;25:389–94.
30. Asmundson GJG, Norton G, Allardings M, Norton P, Larsen D. Post-traumatic stress disorder and work-related injury. *J Anxiety Disord* 1998;12:57–69.
31. Hickling EJ, Blanchard EB. Post-traumatic stress disorder and motor vehicle accidents. *J Anxiety Disord* 1992;6:285–91.
32. Hickling EJ, Blanchard EB, Silverman DJ, Schwartz SP. Motor vehicle accidents, headaches, and posttraumatic stress disorder: Assessment findings in a consecutive series. *Headache* 1992;32:147–51.
33. Chibnall JT, Duckro PN. Post-traumatic stress disorder and motor vehicle accidents. *Headache* 1994;34:357–61.
34. Taylor S, Koch WJ. Anxiety disorders due to motor vehicle accidents: Nature and treatment. *Clin Psychol Rev* 1995; 15:721–38.
35. Greenfield S, Fitzcharles MA, Esdaile JM. Reactive fibromyalgia syndrome. *Arthritis Rheum* 1992;35:678–81.
36. Turk DC, Okifuji A, Starz TW, Sinclair JD. Effects of type of symptom onset on psychological distress and disability in fibromyalgia syndrome patients. *Pain* 1996;68: 678–81.
37. Sherman JJ, Turk DC, Okifuji A. Prevalence and impact of posttraumatic stress disorder-like symptoms on patients with fibromyalgia syndrome. *Clin J Pain* 2000;16:127–34.
38. Perry S, Cella D, Falkenberg J, Heidrich G, Goodwin C. Pain perception in burn patients with stress disorders. *J Pain Symptom Manage* 1987;2:29–33.
39. Geisser ME, Roth RS, Bachman JE, Eckert TA. The relationship between symptoms of post-traumatic stress disorder and pain, affective disturbance and disability among patients with accident and non-accident related pain. *Pain* 1996;66:207–14.
40. McFarlane AC, Atchison M, Rafalowicz E, Papay P. Physical symptoms in posttraumatic stress disorder. *J Psychosom Res* 1994;42:607–17.
41. Beckham JC, Crawford AL, Feldman ME, Kirby AC, Hertzberg MA, Davidson RJT, Moore S. Chronic post-traumatic stress disorder and chronic pain in Vietnam combat veterans. *J Psychosom Res* 1997;43:379–89.
42. White P, Faustman W. Coexisting physical conditions among inpatients with posttraumatic stress disorder. *Mil Med* 1989;154:66–71.

43. Toomey TC, Seville JL, Abashian SW, Finkel AG, Mann JD. Circumstances of chronic pain onset: Relationship to pain description, coping and psychological distress [abstract]. Miami Beach (FL): American Pain Society, A-76; 1994.
44. Turk DC, Okifuji A. Perception of traumatic onset, compensation status, and physical findings: Impact on pain severity, emotional distress, and disability in chronic pain patients. *J Behav Med* 1996;19:435–53.
45. Tushima WT, Stoddard VM. Ethnic group similarities in the biofeedback treatment of pain. *Med Psychother* 1990; 3:69–75.
46. Sharp TJ, Harvey AG. Chronic pain and posttraumatic stress disorder: Mutual maintenance? *Clin Psychol Rev* 2001;21:857–77.
47. Asmundson GJG, Coons MJ, Taylor S, Katz J. PTSD and the experience of pain: Research and clinical implications of shared vulnerability and mutual maintenance models. *Can J Psychiatry* 2002;47:930–37.
48. Asmundson GJG, Norton G. Anxiety sensitivity in patients with physically unexplained chronic back pain: A preliminary report. *Behav Res Ther* 1995;33:771–77.
49. Asmundson GJG, Taylor S. Role of anxiety sensitivity in pain-related fear and avoidance. *J Behav Med* 1996;19: 577–86.
50. Zvolensky MJ, Eifert GH, Lejuez CW, Hopko DR, Forsyth JP. Assessing the perceived predictability of anxiety-related events: A report on the perceived predictability index. *J Behav Ther Exp Psychiatry* 2000;31:201–18.
51. Reiss S, Peterson R, Gursky D, McNally R. Anxiety sensitivity, anxiety frequency, and the prediction of fearfulness. *Behav Res Ther* 1986;24:1–8.
52. Muris P, Vlaeyen J, Meesters C. The relationship between anxiety sensitivity and fears of pain in healthy adolescents. *Behav Res Ther* 2001;39:1357–68.
53. Greenberg J, Burns JW. Pain anxiety among chronic pain patients: Specific phobia or manifestation of anxiety sensitivity? *Behav Res Ther* 2003;41:223–40.
54. Taylor S. Understanding and treating panic disorder: Cognitive behavioral approaches. Chichester (NH): Wiley; 2000.
55. Taylor S, Koch WJ, McNally RJ. How does anxiety sensitivity vary across the anxiety disorders? *J Anxiety Disorders* 1992;6:249–59.
56. Lang AJ, Kennedy CM, Stein MB. Anxiety sensitivity and PTSD among female victims of intimate partner violence. *Depress Anxiety* 2002;16:77–83.
57. Fedorff IC, Taylor S, Asmundson GJG, Koch WJ. Cognitive factors in traumatic stress reactions: Predicting PTSD symptoms from anxiety sensitivity and beliefs about harmful events. *Behav Cognitive Psychother* 2000;28:5–15.
58. Norton PJ, Asmundson GJG. Amending the fear-avoidance model of chronic pain: What is the role of physiological arousal? *The Behavior Therapist* 2003; No. 34. p. 17–30.
59. Turk DC. Psychological aspects of chronic pain and disability. *J Musculoskeletal Pain* 1996;4:145–53.
60. Waddell G, Newton M, Henderson I, Somerville D, Main CJ. Fear avoidance beliefs questionnaire (FABQ) and the role of fear-avoidance beliefs in chronic low back pain and disability. *Pain* 1993;52:157–68.
61. Romano JM, Turner JA. Chronic pain and depression: Does the evidence support a relationship? *Psychol Bull* 1985;97:18–34.
62. Barlow DH. Unraveling the mysteries of anxiety and its disorders from the perspective of emotion theory. *Am Psychol* 2000;55:1247–63.
63. Barlow DH. Anxiety and its disorders. 2nd ed. New York: Guilford Press; 2002.
64. Turk DC, Rudy TE. Toward an empirically derived taxonomy of chronic pain patients: Integration of psychological assessment data. *J Consult Clin Psychol* 1988;56:233–38.
65. Mizener D, Thomas M, Billings R. Cognitive changes of migraineurs receiving biofeedback training. *Headache* 1988;28(5):339–43.
66. Flor H, Turk DC. Chronic back pain and rheumatoid arthritis: Predicting pain and disability from cognitive variables. *J Behav Med* 1988;11:251–65.
67. Kerns RD, Rosenberg R, Otis JD. Self-appraised problem-solving competence and pain relevant social support as predictors of the experience of chronic pain. *Ann Behav Med* 2002;24(2):100–5.
68. Kerns RD, Otis JD, Wise E. Treating families of chronic pain patients: Application of a cognitive-behavioral transactional model. In: Gatchel RJ, Turk DC, editors. *Psychological approaches to pain management*. New York: Guilford Press; 2002. p. 256–75.
69. Melzack R. McGill Pain Questionnaire: Major properties and scoring methods. *Pain* 1975;1:277–99.
70. Kerns RD, Turk DC, Rudy TE. West Haven-Yale multidimensional pain inventory (WHYMPI). *Pain* 1985;23:345–56.
71. Weathers FW, Litz BT, Herman DS, Huska JA, Keane TM. The PTSD Checklist (PCL): Reliability, validity, and diagnostic utility. Annual Meeting of the International Society for Traumatic Stress Studies; 1993 Oct; San Antonio, Texas.
72. Blake DD, Weathers FW, Nagy LM, Kaloupek DG, Klauminzer G, Charney DS, Keane TM. A clinician rating scale for assessing current and lifetime PTSD: The CAPS-1. *The Behavior Therapist*; 1990. p. 187–88.
73. Pitman RK, Sanders KM, Zusman RM, Healey AR, Cheema F, Lasko NB. Pilot study of secondary prevention of posttraumatic stress disorder with propranolol. *Biol Psychiatry* 2002;51:189–92.

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