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A Different Kind of Comorbidity: Understanding Posttraumatic Stress Disorder and Chronic Pain

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Many traumatic events leave lingering physical injuries and chronic pain in their wake, in addition to trauma-related psychopathology. In this review, we provide an overview of developments in the recent literature on comorbid posttraumatic stress disorder (PTSD) and chronic pain. Starting with the conceptual models presented by Sharp and Harvey (2001) and Asmundson, Coons, Taylor, and Klatz (2002), this review summarizes newer studies that examine prevalence of these comorbid conditions. Additionally, we present an updated synthesis of research on factors that may maintain both chronic physical pain and PTSD in trauma survivors. Consideration of the impact of this comorbidity on psychosocial assessment and treatment also is discussed, with particular attention to issues that warrant additional research.

Keywords: PTSD, chronic pain, comorbidity, treatment, assessment

It is well recognized that posttraumatic stress disorder (PTSD) often is complicated by additional pathologies (Kessler, Chiu, Demler, Merikangas, & Walters, 2005). Whereas a substantial literature has examined the implications of psychiatric comorbidity on the assessment and treatment of PTSD, research that explores the impact of physical health comorbidity, specifically chronic pain, is limited. Because many traumatic events involve physical injuries that leave chronic pain in their wake, the interplay of PTSD and physical pain becomes salient for many trauma survivors. Two significant reviews (e.g., Asmundson, Coons, Taylor, & Klatz, 2002; Sharp & Harvey, 2001) first focused attention on this co-occurrence and comorbidity. These reviews have guided research by highlighting factors that may contribute to the development, mutual maintenance, or both, of PTSD and physical pain. This review aims to synthesize developments in the empirical literature, following these earlier publications. Specifically we review current prevalence estimates of comorbid PTSD–pain as well as developments in the empirical evidence that supports factors identified in these two reviews. Research that examines the impact of PTSD–pain comorbidity on assessment and treatment also is reviewed with attention to areas where further research is needed.

Definition of Terms

PTSD is an anxiety disorder characterized by a collection of specific symptoms, following exposure to a traumatic event (American Psychiatric Association, 2000). Within the current diagnostic framework, traumatic events include those that involve experiencing or witnessing actual or threatened death, serious injury, or physical threat and which elicit a subjective sense of fear, helplessness, and horror. Symptoms of PTSD include reexperiencing (e.g., intrusive thoughts of the event), avoidance (e.g., not wanting to think or talk about the event), emotional numbing (e.g., diminished affect), and physiological hyperarousal (e.g., excessive startle). Symptoms must persist for at least one month following the trauma and result in significant distress or functional impairment. A recent meta-analysis suggests that PTSD exerts a large negative impact on physical health, social functioning, and relationships with close others relative to other anxiety disorders (Olatunji, Cisler, & Tolin, 2007). Once established, PTSD symptoms tend to become chronic if not treated (Riggs, Rothbaum, & Foa, 1995).

In contrast, chronic physical pain is not recognized as a condition within formal psychiatric nosology (Sharp & Harvey, 2001). Chronic pain is defined as an unpleasant sensory–emotional experience associated with actual or perceived tissue damage (Otis, Pincus, & Keane, 2006). It is important to note that there are no specific symptoms for chronic physical pain as this condition can involve a variety of pain syndromes (e.g., headache, back pain) and can originate from a range of physical conditions (Smith, Elliott, & Hannaford, 2004). Despite this heterogeneity, chronic pain is considered a unitary syndrome, owing to similarities in response to treatment (Addison, 1984). Pain is considered chronic after persisting for a period of 3 months or more (International Association for the Study of Pain, 1986) and is associated with functional impairment. As a consequence, chronic pain demonstrates a robust relationship with ongoing health care utilization and is one of the more expensive benign conditions that affects working-age adults (Smith, Macfarlane, & Torrance, 2007). Current models recognize multiple sources of influence to pain beyond tissue or structural damage including emotional, environmental, social, and financial factors (e.g., Fordyce, 1995). In considering

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this definition of chronic pain, the areas of overlap with PTSD are notable, including ongoing disability, the contribution of emotional and social factors, and the persistent course of both conditions.

The Starting Point: Summary of Sharp and Harvey (2001) and Asmundson et al. (2002)

As noted by Asmundson et al. (2002) and Sharp and Harvey (2001), comorbidity between PTSD and chronic pain has only been noted recently within the empirical literature. Because these two previous reviews form the backdrop for this article, a brief summary of these papers is the starting point for this review.

Sharp and Harvey (2001) were the first to formally draw attention to the overlap between PTSD and chronic pain. At the time, Sharp and Harvey observed that research on this form of comorbidity was lacking. These authors noted that the early literature on the prevalence of comorbid PTSD and chronic physical pain was derived from specialized settings, in particular individuals who were seeking professional help for one condition or the other. In these studies, the prevalence of chronic pain within *trauma samples* ranged from 20%–80%. Alternatively, the prevalence of PTSD within *chronic pain samples* ranged from 10%–50%. These data provide preliminary evidence for a substantial comorbidity between PTSD and pain among help-seeking samples.

Additionally, Sharp and Harvey reviewed the extant literature that concerns factors that have been shown to maintain PTSD and chronic pain individually and proposed a series of seven processes that may explain the mutual maintenance of these two conditions. The first factor involved *attentional biases* toward both trauma-related and pain-related cues. Sharp and Harvey speculated that pain sensations may serve as trauma reminders and generate further attentional biases in a recursive fashion. Second, *anxiety sensitivity* (AS) is postulated as a potential maintaining factor of comorbid PTSD and chronic pain. AS, the fear of arousal-related sensations based on beliefs that these sensations are harmful (Reiss & McNally, 1985), is postulated to fuel anxious interpretation of bodily sensations (including pain) in comorbid patients. Third, *persistent reminders of the trauma* are highlighted as a possible maintaining factor. Although situational reminders can trigger reexperiencing symptoms (a PTSD symptom), Sharp and Harvey postulated that pain sensations likewise can be interpreted as a trauma reminder, particularly given the link between the traumatic event and pain-producing physical injuries.

Avoidant coping style is postulated as the fourth maintaining factor for comorbid pain and PTSD. Although avoidance of specific physical activities may be necessary for individuals with injuries, prolonged avoidance of pain cues can lead to physical deconditioning and distress while avoidance of trauma cues can prevent fear extinction and relearning safety to these cues. Fifth, *depression and reduced levels of behavioral activity* are believed to serve as mutual maintenance factors by contributing to disability in pain patients while also preventing exposure to trauma cues. Sixth, *pain perception,* heightened by anxiety, is proposed to operate in a feed-forward fashion, increasing levels of perceived pain, emotional distress, and disability in comorbid patients. Last, Sharp and Harvey recognized that both PTSD and chronic pain demand *cognitive resources* and suggest that these demands limit one's capacity to engage in more adaptive strategies for managing distress and disability.

Following Sharp and Harvey's (2001) review, Asmundson et al. (2002) proposed a shared vulnerability model, emphasizing the role of trait AS in understanding comorbidity between PTSD and pain. In this model, AS is believed to predispose an individual to both chronic pain and PTSD by enhancing the perceived sense of alarm during a traumatic event, which increases awareness of psychological threat and physical injury. This increased sensitivity is postulated to exacerbate overall emotionality and increase the risk for development of PTSD. Asmundson et al.'s model differs from Sharp and Harvey's (2001) in that they emphasized the role that AS plays in heightening risk for developing comorbid PTSD and pain following a trauma, delineating its role as a preexisting vulnerability factor. Although the distinction between etiological–vulnerability factors and maintaining factors may seem semantic, these two types of processes can exert different influences. Unfortunately, research has not advanced to the point to allow determination of which of these processes serve as risk versus maintaining factors for PTSD–chronic pain comorbidity.

In many respects, these two reviews focused awareness of the comorbidity of PTSD and chronic pain and provided the context for a number of subsequent research studies. In the next section, we first review recent studies that examined the prevalence and course of comorbid PTSD–chronic pain. Then, we review the available literature on factors that might explain this comorbidity, using the seven processes proposed by Sharp and Harvey (2001). In organizing this review, only studies that focused on samples with both PTSD and chronic pain conditions are included, given current conceptual models.

Prevalence of Comorbid PTSD and Chronic Pain

Whereas earlier reviews by Sharp and Harvey (2001) and Asmundson et al. (2002) reported on comorbidity of PTSD and pain in specific patient groups, recent epidemiologic studies extend this literature by providing prevalence estimates of comorbidity among the general population. Within the National Comorbidity Replication Survey, 7.3% of individuals with chronic back pain met criteria for PTSD (Von Korff et al., 2005). Similarly, data from the Canadian Community Health Survey indicated a greater occurrence of chronic pain conditions among community members with PTSD with differences ranging from 7.7% among individuals with fibromyalgia to 46.0% among those with back pain (Sareen et al., 2007). Research by Buchwald and colleagues (2005) provided the only study to examine the prevalence of comorbid PTSD and pain within a minority (American Indian) sample. This report suggested a prevalence of lifetime PTSD of 16% among Southwestern tribe members and 14% among Northern Plains tribe members who experience chronic pain. Given increased interest in cross-cultural issues in trauma response, more work in this area is needed.

Thus, ongoing research supports the relationship between PTSD and pain observed in early reviews. Although the prevalence of comorbid PTSD and chronic pain is considerably higher within treatment settings, the association between these conditions remains robust in the general community and suggests a modal prevalence rate around 7%–8%. Rates among minorities may be higher. To date, most studies have not specified whether the source of pain was a function of the same event used to diagnose PTSD.

Failure to differentiate between events that contribute to either PTSD or chronic pain creates ambiguity in the available data; future research would be strengthened by a focus on differentiating events when applicable. However, the available data strongly suggest that these two conditions are likely to co-occur, supporting earlier research.

Change Over Time in PTSD and Pain Following Trauma That Produces Physical Injury

Although the comorbidity of PTSD and chronic pain is well recognized, only recently has research explored the course of these two conditions over time. One of the earliest efforts followed patients who sought care at a hospital emergency service following a motor vehicle accident (MVA; Mayou & Bryant, 2001). Individuals were assessed initially and again at 3 and 12 months afterward. At 3 and 12 months, 7% of the individuals reported difficulty in physical recovery. At the 3-month assessment, 23% reported PTSD at diagnostic severity on the PTSD Symptom Scale–Self-Report (PSS-SR; Foa, Riggs, Dancu, & Rothbaum, 1993), which reduced to 17% at one-year post-MVA. Self-report of physical recovery at one year was significantly predicted by PTSD at the 3-month assessment. Considering psychological recovery at one year, the severity of physical injury and ongoing health problems emerged as significant predictors of PTSD. A similar report sampled more severely injured hospital inpatients (Zatzick et al., 2007). Approximately 23% of survivors had a probable diagnosis of PTSD 12 months after their hospitalization based on structured interviews. A number of factors were associated with PTSD, including greater posttrauma levels of physical pain as well as greater physical pain at 3-months posttrauma. It is important to note that the trajectory of physical pain complaints appeared to parallel posttrauma symptomatology.

Sterling and colleagues examined the trajectory of PTSD and pain in greater detail through a series of reports that followed MVA survivors with whiplash injuries (Sterling, Jull, Vicenzino, Kenardy, & Darnell, 2005; Sterling & Kenardy, 2006; Sterling, Kenardy, Jull, & Vicenzino, 2003). Participants initially were assessed within one month of the MVA and again at 2-, 3-, and 6-month intervals. The initial report by Sterling et al. (2003) examined trajectories of emotional distress, global functioning, and posttraumatic symptomology across three patient subsamples derived from responses on the Neck Disability Index (Vernon & Mior, 1991): (a) recovered, (b) mild pain and disability, and (c) moderate–severe pain and disability. Initial psychological distress was evident, with some reduction over time. Whereas the moderate-to-severe group's distress levels remained elevated at 6-months postaccident, the recovered and mild groups' scores were in the normative range by 2–3 months postaccident. Although slightly diminished by 3-months postaccident, PTSD symptoms reported in the moderate-to-severe group were higher at the initial assessment and remained high over time. Overall, decreases in psychological distress were paralleled by decreases in self-reported physical pain and disability, suggesting that recovery in these domains is closely linked. Subsequent analyses of these data observed that initial trauma symptomology was a significant predictor of disability, along with a collection of demographic and pain-related variables (Sterling et al., 2005).

Sterling and Kenardy (2006) used a similar group-based approach to examine factors related to the trajectory of posttrauma symptoms. Using initial and 6-month Impact of Event Scale (IES; Horowitz, Wilner, & Alvarez, 1979) scores, researchers classified individuals into three groups: (a) those with low levels of PTSD throughout the study ($n = 52$), (b) those with high initial levels of PTSD that fell below diagnostic threshold at 6 months ($n = 14$), and (c) those whose scores remained elevated throughout the study ($n = 14$). Mediation analysis suggested that the association between initial pain sensitivity and symptoms of PTSD at 6-months was fully mediated by the severity of initial pain and disability. These data suggest that pain severity and interference play a notable role in posttrauma psychological symptoms.

These longitudinal studies extend the previous literature by documenting that each of these conditions influences the other, following a traumatic physical injury. Early pain emerges as a robust predictor of chronic PTSD whereas early symptoms of PTSD confer risk for the development of chronic pain. These parallel trajectories suggest that recovery in these domains is closely linked, supporting suppositions offered by Sharp and Harvey (2001) and Asmundson et al. (2002), concerning common processes. The next section will review the current state of research on the seven processes that have been proposed as relevant in understanding PTSD–chronic pain comorbidity.

Current State of Research on Factors That Contribute to PTSD–Chronic Pain Comorbidity

Attention and reasoning biases. Attentional bias is conceptualized as a delayed or a facilitated response to a defined set of stimuli (Williams, Mathews, & MacLeod, 1996). Attentional biases within psychopathology research often are assessed by using a modified Stroop task wherein participants are asked to name the color of words that are presented in a range of colors. Greater delays in naming the color of words that are salient to specific pathology (e.g., "helpless" for a patient with PTSD) relative to neutral words (e.g., "chair") are interpreted as evidence of attentional bias. Sharp and Harvey (2001) reviewed a number of studies that have observed delays in color naming to trauma words in individuals with PTSD. A smaller handful of studies have noted color-naming delays in response to pain-relevant words in chronic pain samples (Pincus & Morley, 2001). To date, only one study has examined attentional biases among individuals with both PTSD and chronic pain. Beck, Freeman, Shipherd, Hamblen, and Lackner (2001) examined three groups of MVA survivors: (a) individuals with comorbid PTSD and chronic pain, (b) individuals with only chronic pain, and (c) control participants without pain or any psychiatric condition. Participants were presented with words from four categories: trauma words, pain words, positive words, and neutral words. Attentional bias showed some disorder-related specificity; the comorbid group evidenced response delays to both trauma and pain-related words, whereas the pain-only group showed delays specific to pain cues. Evidence of generalized responding also was noted; the comorbid group showed slower responding to all word categories, relative to the other two groups. In turn, the pain-only group showed overall slower responding relative to control participants. These effects were independent of depression, age, anxiety, or elapsed time since the accident, and

they provide preliminary support for a synergistic interplay between PTSD and chronic pain in attentional processing.

Reasoning biases (e.g., overestimation of the probability of experiencing pain, trauma, or both) also are presumed to interact in ways that maintain distress and dysfunction. Reasoning biases have been examined both in chronic pain samples (e.g., Sullivan, Stanish, Waite, Sullivan, & Tripp, 1998) and in PTSD samples (e.g., Nortje, Roberts, & Möller, 2004), as they predict distress and disability, but no existing research has examined biases in comorbid patients. This could prove to be a fruitful avenue of research, particularly as these biases interact with other postulated maintaining factors such as avoidance, depression, and heightened anxiety.

Anxiety sensitivity. AS plays a fundamental role both in Sharp and Harvey's (2001) mutual maintenance model and Asmundson et al.'s (2002) shared vulnerability model. Sharp and Harvey's review examined the role of AS among pain and PTSD samples individually, whereas Asmundson et al. included research examining the interrelationships of PTSD, pain, and AS among those with comorbid conditions. Since these initial reviews, one additional study has been published in this domain. Jakupcak et al. (2006) noted that AS serves as a proxy risk factor in the association between PTSD symptoms and somatic complaints among male veterans. A proxy risk factor is similar to a mediator without the requirement of temporal ordering (Kraemer, Stice, Kazdin, Offord, & Kupfer, 2001). This report is notable, owing to use of an alternate measure of anxiety sensitivity (the Body Sensations Questionnaire; Chambless, Caputo, Bright, & Gallagher, 1984). This study highlights the role that anxiety sensitivity plays in maintaining comorbid chronic pain and PTSD symptoms. Future longitudinal research that explores this comorbidity is encouraged to include measures of AS, particularly given its relevance in both Asmundson et al. and Sharp and Harvey's models.

Reminders of the trauma. Previous reviews of this literature provide a variety of elegant hypotheses, concerning how reminders of the trauma may instigate symptoms of both pain and PTSD (particularly avoidance). In particular, reminders of the trauma can increase individuals' awareness of pain and accompanying disability. Likewise, trauma reminders may be reacted to with avoidance, which is a PTSD symptom. To date, these hypotheses remain unexplored.

Avoidant coping. The role of avoidance has been highlighted separately within the pain (Vlaeyen & Linton, 2000) and the PTSD literatures (Foa, Steketee, & Rothbaum, 1989). Yet, avoidance has not been examined within the context of comorbid pain and PTSD. This oversight is understandable, as it can be difficult to ascertain the function of avoidance in the context of these two disorders. Individuals with comorbid pain and PTSD also may be unable to discern the reason for their avoidance. Although it is possible that such a fine-grained analysis would not be necessary, current theoretical discussions of comorbid pain and PTSD suggest that to understand how avoidance facilitates mutual maintenance of these conditions, understanding the motivation for avoidance is necessary.

Depression and reduced activity levels. Depression is known to be highly comorbid with both chronic pain and PTSD (e.g., Geisser, Roth, Bachman, & Eckert, 1996), and current models suggest that depression can maintain each of these conditions. To date, three studies have examined depression among patients with comorbid PTSD and chronic pain. Jakupcak et al. (2006) observed that depression served as a proxy risk factor in the association between PTSD and somatic complaints in male veterans. A similar report, also drawn from a veteran sample, observed that depression accounted for the association between self-reported PTSD and pain severity (Poundja, Fikretoglu, & Brunet, 2006). More recently, Roth, Geisser, and Bates (2008) examined pathways by which PTSD, depression, and pain interact in patients with accident-related chronic pain. Structural equation modeling suggested that PTSD was directly associated with depression, whereas depression had both a direct influence on pain intensity and an indirect effect by way of its association with disability. These studies each relied on cross-sectional designs. Ideally, depression measures can be included within future longitudinal studies, particularly as this affective state can potentially explain one linkage between PTSD and pain.

Anxiety and pain perception. Sharp and Harvey (2001) proposed that anxiety associated with PTSD may contribute to higher levels of perceived pain among patients with comorbid pain and PTSD, relative to individuals without PTSD. Previous reviews have noted this hypothesized relationship in studies that rely on self-report pain measures (e.g., Geisser et al., 1996). However, when researchers use laboratory-based methods of assessing pain perception, a different pattern of results emerges. In exploring phenomena associated with stress-induced analgesia (Lewis, Cannon, & Liebeskind, 1980), Pitman, van der Kolk, Orr, and Greenberg (1990) examined whether exposure to a trauma-related stimulus would induce analgesia in patients with PTSD. Note that stress-induced analgesia contradicts current models of comorbid PTSD–pain, which predict increased pain sensitivity in comorbid patients. Pitman et al. (1990) examined 8 veterans with PTSD and 8 veterans without any diagnosable disorders. Participants were given either naloxone (an opiate antagonist intended to block stress-induced analgesia) or placebo. Participants then viewed a neutral video followed by a combat video. Consistent with stress-induced analgesia, veterans with PTSD evidenced a decrease in pain sensitivity relative to controls following exposure to the combat video. Administration of naloxone was observed to reverse the analgesic effect. Follow-up research indicated that changes in pain perception between the neutral and combat film were most strongly associated with PTSD (Van der Kolk, Greenberg, Orr, & Pitman, 1989). A similar finding was reported by Geuze et al. (2007), using psychophysical assessment. Participants in this study included 12 combat-exposed veterans with PTSD and 12 control veterans, matched on demographic and service-related parameters. In this report, patients with PTSD rated objectively equal temperatures as subjectively less painful, relative to patients without PTSD. Thus, in both of these studies, PTSD was associated with reduced pain perceptions, contrary to current models.

Aside from methodology, it is important to recognize that anxiety concerning pain may play a salient role in these associations. Those who develop anxiety about their pain may experience higher levels of subjective pain, which may subsequently influence posttrauma symptoms. Van Loey, Maas, Faber, and Taal (2003) explored these associations in a study of 301 burn patients who were evaluated shortly following physical injury and at 8-week intervals for up to one year. Longitudinal analyses indicated that peritraumatic anxiety and dissociation, pain-related anxiety, and gender emerged as significant predictors of PTSD severity at one year. No objective indicator of physical injury severity predicted one-year

trauma symptoms. This report suggests interesting possibilities about the dynamic between PTSD and pain perception. Clearly, this topic merits further exploration, particularly in light of the counterintuitive findings that individuals with PTSD reported attenuated pain perceptions in laboratory paradigms.

Cognitive demand. As noted by Sharp and Harvey (2001), both PTSD and chronic pain are associated with cognitive demands in the form of symptoms (e.g., flashbacks) and cognitive methods of management (e.g., distraction), which reduce mental resources for adaptive coping strategies. Although interesting, this hypothesis has not been examined empirically.

Summary of research to date. Research that examines mechanisms that underlie the comorbidity of chronic pain and PTSD following traumatic, injuring events is uneven. Notable attention has been paid to anxiety sensitivity, depression, and pain perception, whereas research on other potential maintaining factors is limited. In reviewing this work, two issues emerge. First, because longitudinal research suggests that pain symptoms and PTSD symptoms run a parallel course following an injuring trauma, prospective studies should include assessments of proposed maintaining factors in an effort to explore these processes over time. Second, issues that have not been addressed thoroughly include the role of attentional and reasoning biases, whether trauma reminders can become learned cues for both pain and PTSD, how avoidance perpetuates both kinds of symptoms, and whether cognitive demand from each condition can maintain this comorbidity. Although the field has progressed to some extent since Sharp and Harvey (2001), there is a need for more systematic work, given the salience of this comorbidity within clinical settings.

As work continues in this area, it is possible that factors other than those identified by Sharp and Harvey (2001) and Asmundson et al. (2002) will emerge as relevant for understanding the unique comorbidity between PTSD and chronic pain. Possible candidates for additional study might include perceptions of life control (e.g., Palyo & Beck, 2005) and dissociation (DePrince & Freyd, 2007), based on preliminary speculation.

Consideration of the clinical implications of pain–PTSD comorbidity is a natural extension for this review and a new development since the publication of Asmundson et al. (2002) and Sharp and Harvey (2001). In the next two sections, we address the implications for assessment and treatment of these patients.

Implications for Clinical Assessment

Unfortunately, few guidelines exist for the clinical assessment of comorbid PTSD and chronic pain. It is important to acknowledge that much of what is known about the assessment of this comorbidity is derived from clinical practice. Clinical experience is immensely valuable, but hypotheses derived from this source, like any other hypotheses, need to be tested. Given that chronic pain and PTSD are both accompanied by depression, general anxiety, and functional impairment, assessment becomes particularly challenging. Moreover, the comorbid patient often is unable to distinguish physical-pain complaints from PTSD symptoms, especially when these conditions stem from the same traumatic event. An important component of clinical assessment involves assisting the patient in understanding which symptoms arise from physical pain, which from PTSD, and which are the likely result of the comorbidity of these conditions.

Within research settings that focus on PTSD, the Clinician-Administered PTSD Scale (CAPS; Weathers, Keane, & Davidson, 2001) often is used. In administering the CAPS to individuals with comorbid pain and PTSD, probe questions may be included to differentiate symptoms that are attributable to pain, so as to exclude these from the PTSD ratings (e.g., Beck et al., 2009). One may question whether patients themselves can make this differentiation when completing a self-report inventory. Although no study has addressed this question directly, several reports have examined the impact of comorbidity on measurement properties of various questionnaires. Coffey, Gudmundsdottir, Beck, Palyo, and Miller (2006) empirically derived cutoff scores for PTSD on the IES and the PSS-SR by using a sample of 229 MVA survivors. Sixty-nine percent of the sample experienced chronic pain, and 43% were diagnosed with PTSD through the CAPS. Results indicated that a cutoff score of 14 or greater on the PSS-SR classified 90 of 99 patients with PTSD. On the IES, a cutoff score of 27 or greater was able to classify 90 of 99 patients with PTSD. Coffey et al. (2006) noted that classification efficiency did not differ for either measure as a function of chronic pain. These data suggest that both the IES and the PSS-SR might be good candidates as screening measures within settings that serve individuals with chronic-pain complaints that stem from traumatic events.

In the pain literature, Poundja, Fikretoglu, Guay, and Brunet (2007) examined a similar issue with the Brief Pain Inventory (BPI; Cleeland, 1989) in a sample of 131 veterans with PTSD. The BPI is an 11-item measure with two subscales, pain severity and pain interference. Internal consistency was high in this sample and an exploratory factor analysis upheld the two-factor structure. Significant correlations between the BPI and related measures of pain and quality of life were noted. Social desirability was not correlated with either subscale. The authors conclude that the BPI appears to be a valid pain measure in patients with combat-related PTSD.

To our knowledge, no other studies have examined the measurement properties of published scales in samples with comorbid PTSD and chronic pain. Although both studies suggest that self-report scales can be used to accurately assess PTSD and pain respectively, additional research is needed. In particular, assessment of symptoms common to both conditions (i.e., sleep difficulties, trouble concentrating, and irritability) is complicated in comorbid patients. Comorbid patients may be able to identify and accurately report condition-specific symptoms (e.g., flashbacks, soreness) but may struggle with symptoms that could originate from either PTSD or pain. Thus, the development of self-report devices that separate measurement of both PTSD and chronic pain is important.

Implications for Treatment

Given the overlap of chronic pain and PTSD, one might question whether standard treatments are effective for the comorbid patient. Early on, some writers felt that physical pain may interfere with a patient's ability to respond to PTSD treatment (e.g., Koch & Taylor, 1995). Conversely, the presence of mental health problems was suggested to interfere with effective pain management (Sanders, Harden, Benson, & Vincente, 1999). Fortunately, re-

search provides little evidence in support of either hypothesis. In considering recent advances in the treatment literature, some authors also have examined treatment generalization, which is an important clinical consideration with this population of patients.

Efficacy and generalization of PTSD treatments in comorbid patients. A number of psychological interventions for PTSD have received empirical support for their efficacy (Foa, Keane, & Friedman, 2000), but the literature examining efficacy of PTSD treatment for comorbid patients is limited largely to cognitive behavioral therapies (CBT). These treatments contain exposure exercises, cognitive restructuring, psychoeducation, and general anxiety management. To date, one controlled trial has examined the influence of cognitive behavioral treatment for PTSD on comorbid patients. Beck, Coffey, Foy, Keane, and Blanchard (2009) presented data comparing Group CBT (GCBT) with a minimal contact comparison condition that used 44 individuals with MVA-related PTSD. Eight-five percent of this sample reported chronic pain complaints from injuries sustained during the MVA. Patients receiving GCBT showed a significant reduction in PTSD symptoms relative to controls at postassessment across both clinician interview and self-report measures. It is important to note that 88.3% of the GCBT patients did not satisfy criteria for PTSD following treatment compared with 31.3% of the comparison group. Although the analysis examining pain severity was underpowered, an effect size of $g = 0.57$ was noted at posttreatment, showing significant reduction in pain in the GCBT versus the comparison group. These data suggest that treatments for PTSD can be effective for patients with comorbid PTSD and chronic pain and may generalize to influence pain severity.

A naturalistic report by Shipherd et al. (2007) also suggests that PTSD-focused treatment may show a radiating influence to pain complaints. Participants in this trial included 85 male veterans receiving a 32-session GCBT that focused on PTSD, grief processing, relapse prevention, and wellness skills. Pain severity was assessed by an independent care provider. Decreases in pain ratings were observed across sessions and 4 months after treatment. The uncontrolled nature of this study precludes determination of causality but suggests that PTSD-focused treatment may generalize to lessen severity of perceived pain.

Aside from empirically supported treatments for PTSD, a number of emerging interventions are under study, including expressive writing as a means of addressing posttrauma symptoms. Expressive writing (Pennebaker & Beall, 1986) involves brief, repeated writing sessions focused on personal stressful experiences and is associated with reductions in anxiety, depression, and improvement in physical health. To examine the effects of expressive writing on PTSD and pain, Koopman et al. (2005) randomly assigned 59 women who had experienced intimate partner violence to an expressive writing or a neutral writing condition. The expressive writing group was instructed to write about their most traumatic experiences and encouraged to explore their feelings about these events. The neutral writing group was instructed to write about how they used their time and encouraged to be as objective as possible. Both groups completed four writing sessions and were reevaluated 4 months later. No significant between-condition effects in self-reported PTSD symptoms were observed. With respect to pain, women with higher baseline levels of pain benefited more from the neutral writing condition, relative to expressive writing. On the basis of one report to date, expressive writing does not appear to show efficacy as an intervention for comorbid PTSD and pain.

Thus, to date, one controlled study supports the use of CBT to treat the patient with comorbid PTSD and chronic pain. Contrary to early speculations, treatment efficacy was demonstrated in the comorbid patients, including a hint that treatment could generalize to reduce pain. One naturalistic study also suggests that CBT might generalize to impact pain severity although more systematic study of this issue is warranted. Expressive writing has not been shown to be effective for patients with comorbid PTSD and chronic pain. Ideally, investigators will begin to recognize chronic pain as an important comorbidity within randomized controlled trials and include measurement of pain as an outcome dimension.

Efficacy and generalization of pain treatments in comorbid patients. Although the PTSD literature has begun to examine whether empirically supported treatments are effective with patients who have comorbid PTSD and pain, few efforts have been made to examine whether traditional pain-management programs are efficacious for comorbid patients. DeCarvalho and Whealin (2006) have noted that a number of effective interventions for PTSD may already be included within traditional pain protocols, such as relaxation training and basic cognitive therapy, which are often a component of pain management. As such, it is important to examine the effects of traditional pain management approaches for comorbid patients, particularly given the prevalence of these individuals in specialized care environments.

Interventions specific to comorbid pain and PTSD. As the PTSD literature has evolved, treatments have developed to address patients with multiple, comorbid disorders. To date, similar efforts have not appeared to address comorbid PTSD and chronic pain. Because it is unclear whether PTSD treatments can reduce pain intensity, the development of integrated interventions that target both chronic pain and PTSD is timely and important. In developing these interventions, consideration should be given to the type of care environment in which they are to be used, given demands that are placed on comorbid patients from associated health care providers. Recent developments within the pain literature have suggested that interventions such as hypnosis might be helpful for reducing both pain and anxiety (e.g., DePascalis, Magurano, Bellusci, & Chen, 2001; Lynn, Shindler, & Meyer, 2003). Clearly, the development of treatments to address this special form of comorbidity has enormous potential, given the prevalence of these patients.

Conclusion

As noted throughout this review, a considerable amount of progress has been made in understanding the comorbidity between PTSD and chronic pain. To begin, it is well-established at this point that chronic pain and PTSD co-occur frequently and this finding is not isolated to patient populations or to specialized care environments. Because of the high prevalence of this unique form of comorbidity, greater efforts should be made to screen individuals who are seeking help in the aftermath of an injuring trauma. For individuals who are struggling with pain complaints, screening for PTSD would seem important and likewise, individuals who are seeking help for PTSD should be assessed for chronic-pain complaints. Because this form of comorbidity bisects mental health and

medical arenas, efforts to identify and assist these patients requires an interdisciplinary effort.

On the basis of longitudinal studies, it appears that pain and posttrauma symptomatology develop in parallel, following an injuring trauma. Unlike psychiatric comorbidity, which can develop in sequence, the simultaneous trajectories of pain and PTSD suggest that inclusion of potential contributory factors in future longitudinal studies of this process would be extremely useful. As noted in our review of current research, considerable progress has been made in understanding the role of factors such as anxiety sensitivity, depression, and pain perception. Other processes, such as attention and reasoning biases, reminders of the trauma, avoidance, and cognitive demand require more study. Identification of processes that contribute to the simultaneous development of PTSD and chronic pain is an essential step in understanding and hopefully preventing this outcome in the wake of an injuring trauma.

As noted in this review, one area that has received very little attention is the clinical assessment of this unique form of comorbidity. Although methodologically challenging, research in this arena is sorely needed. At present, little information exists concerning which assessment devices perform best within specialized care environments. Likewise, little is known about how PTSD–chronic pain comorbidity affects psychometric properties of established measures of PTSD and pain. In light of growing awareness of the prevalence of comorbid pain–PTSD patients, additional research on the clinical assessment of these patients is needed. Lastly, this review highlights the progress that has occurred in understanding the treatment of patients with comorbid PTSD and chronic pain. Contrary to early concerns, it appears that these individuals can be treated effectively with CBT focused on PTSD symptoms. At the present time, nothing is known about how PTSD influences the efficacy of traditional pain-management approaches. Future efforts to develop and test an integrated treatment that targets both chronic pain and PTSD would be highly desirable. In sum, although considerable progress has been made, we have only just begun to understand the comorbidity between chronic pain and PTSD.

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