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PTSD and the Experience of Pain: Research and Clinical Implications of Shared Vulnerability and Mutual Maintenance Models

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It is common for individuals with symptoms of posttraumatic stress disorder (PTSD) to present with co-occurring pain problems, and vice versa. However, the relation between these conditions often goes unrecognized in clinical settings. In this paper, we describe potential relations between PTSD and chronic pain and their implications for assessment and treatment. To accomplish this, we discuss phenomenological similarities of these conditions, the prevalence of chronic pain in patients with PTSD, and the prevalence of PTSD in patients with chronic pain. We also present several possible explanations for the co-occurrence of these disorders, based primarily on the notions of shared vulnerability and mutual maintenance. The paper concludes with an overview of future research directions, as well as practical recommendations for assessing and treating patients who present with co-occurring PTSD or chronic pain symptoms.

Clinical Implications

Clinicians treating patients with posttraumatic stress disorder (PTSD) need to pay careful attention to co-occurring symptoms of pain.

Likewise, those treating patients with chronic pain need to be aware of the potential influence of PTSD symptoms on clinical presentation.

Use of propranolol may be effective in simultaneous relief of co-occurring PTSD and pain symptoms.

Limitation

The positions suggested in this review warrant careful empirical scrutiny.

Key Words: *posttraumatic stress disorder, pain, chronic pain, shared vulnerability, mutual maintenance, assessment, treatment*

It is not atypical for patients with posttraumatic stress disorder (PTSD) to present with several concomitant physical and mental health problems. These most often include increased reporting of physical symptoms and physical health problems, increased alcohol consumption, and depressed mood (1,2). Recent evidence suggests that pain is one of the most commonly reported symptoms of patients with PTSD, regardless of the nature of their traumatic experience (for example, military combat, motor vehicle accident, or sexual assault). Similarly, patients who have persistent, chronic pain associated with musculoskeletal injury, serious burn injuries, and other pathologies (such as fibromyalgia, cancer, or AIDS) frequently present with symptoms of PTSD. In the past decade, investigation into the relation(s) between PTSD and the experience of pain has flourished. To a lesser degree, researchers have developed models that attempt to explain how the conditions may be linked (3).

This paper has several purposes. First, we provide summary definitions of the conditions under discussion. Second, we highlight symptoms from each condition that have similar characteristics. Third, we summarize the literature on prevalence rates of pain experiences in PTSD populations, and vice versa. Fourth, we articulate potential explanations for the observed association between pain and PTSD. Finally, we discuss future directions for empirical investigation and clinical practice that stem from this line of inquiry.

Definitions

PTSD typically develops following exposure to a situation or event that is, or is perceived to be, threatening to the well-being of oneself or another person. As presented in the DSM-IV-TR (4), symptoms are grouped into 3 clusters: reexperiencing of the event (for example, intrusive thoughts and nightmares), avoidance and emotional numbing (for example, restricted affect), and hyperarousal (for example, sleep difficulties and hypervigilance). To satisfy criteria for a diagnosis of PTSD, a person must be exposed to a traumatic event with actual or perceived threat. In addition, the individual must experience intense fear or helplessness; must have at least 1 re-experiencing symptom, at least 3 avoidance and numbing symptoms, and at least 2 hyperarousal symptoms; must be bothered by these symptoms for more than 1 month; and must be significantly distressed or impaired in social, occupational, or other functioning. Recent findings indicate that prevalence rates for PTSD in the general population range between 7% and 12% (5); in certain at-risk groups (for example, survivors of sexual assault, motor vehicle accidents, or military combat), the rates can be substantially higher (4).

Accumulating evidence from factor analytic investigations (6–9) suggests that the PTSD symptom clusters outlined in the DSM-IV-TR diagnostic criteria may not best conceptualize PTSD symptom profiles. While the optimal symptom cluster arrangement differs slightly among each of these investigations, a consistent finding is that the 2 elements of the current avoidance and numbing cluster should be considered independently. In short, the empirical evidence suggests that PTSD comprises 4 distinct symptom clusters: reexperiencing, avoidance, numbing, and hyperarousal. Recent treatment studies show that it is important to distinguish among the 4 clusters because important effects of intervention—particularly in the avoidance and numbing domains—are lost if the 4 clusters are not examined separately (10,11). This has not become clinical convention, but as will become evident below, it is an increasingly common consideration in empirical work.

The traditional medical model conceptualizes pain as a pure sensory experience arising from noxious stimulation (for example, organic pathology and physical injury). Contemporary models now recognize that biological, psychological (for example, cognitive, affective, and behavioural), and social factors are involved in the experience (12,13). These contemporary models conceptualize pain as a complex, subjective, perceptual phenomenon involving numerous dimensions (for example, intensity, quality, time course, and personal meaning). Pain is typically acute and occurrs in response to actual or potential tissue damage to motivate adaptive processes that facilitate escape and promote recuperative behaviour (14). However, for about 30% of adults in developed countries, the experience of pain persists over long periods of time, whether it is related to injury or to some organic pathology (15); it often leads to distress, suffering, and functional disability (16,17); and it is associated with inappropriate use of medical services, costly insurance claims, and work absenteeism (18,19). When pain persists for at least 3 months, it is considered chronic (20). Many, but not all, patients with chronic pain exhibit considerable anxiety, avoidance behaviour, and general emotional lability, as well as attentional biases and hypervigilance for pain cues (21). In Canada, chronic musculoskeletal pain is the second most common chronic health condition, costing the health care system approximately $18 billion annually.

Symptom Overlap

A close look at the definitions provided above indicates that there is some degree of symptom overlap between PTSD and chronic pain. In particular, anxiety and hyperarousal, avoidance behaviour, emotional lability, and elevated somatic focus are frequently observed in both conditions. Both PTSD and chronic pain are characterized by hypervigilance (21,22) and attentional bias (23,24) for stimuli that are specific to each condition. There are also data suggesting that startle responses are intensified during states of negative affect (25), to which both conditions can be a major contributor (26). Further, preliminary data suggest that stress responses and pain modulation are dysregulated in both conditions (27,28). Collectively, these findings indicate that PTSD and chronic pain share similar response patterns in the cognitive, behavioural, and physiological domains; in the opinion of some (3,29), the data suggest an intricate connection between the 2 conditions.

Prevalence Estimates

One method for gaining preliminary insight into the relation(s) between PTSD and pain—albeit a limited one in that it does not allow for conclusions regarding the nature of the association (see below)—is to assess the degree to which they co-occur. As might be anticipated, based on the preceding discussion, the literature indicates a high degree of co-occurrence, regardless of whether pain is being assessed in patients with PTSD, or vice versa.

Pain in Patients With PTSD

PTSD and PTSD symptoms are associated with greater reporting of physical health problems and symptoms (30–34). They are also strongly associated with current pain, overall pain ratings, and pain-related disability (29,35); with functional impairment (31,32); and with increased health care utilization (36). For military veterans, these findings appear to hold regardless of the theatre (for example, Vietnam or the Persian Gulf) or the nature of the trauma experienced. At the acute level, then, there is evidence suggesting that PTSD symptoms and pain—as well as functional impairments that might be attributed to either or both—frequently co-occur.

Does this association hold in cases wherein pain persists beyond the acute phase?

In one of the first studies to assess co-occurrence of PTSD and chronic pain, White and others reported that approximately 1 in 5 military veterans with PTSD developed chronic pain (37). More striking, Beckham and others observed that the symptoms reported by 80% of consecutive outpatient military combat veterans sampled (103/129) satisfied the criteria for current chronic pain in 1 or more sites, including the back in 77% of cases (29). McFarlane and others found that almost one-half of a sample of volunteer firefighters with PTSD reported significant musculoskeletal pain, primarily in the back, compared with 21% of those without PTSD (38). Others (39,40) have noted that between 20% and 30% of individuals from community and mental health outpatient samples with current PTSD report persistent pain symptoms. In a recent large-scale study of community outpatients, PTSD was associated with an increasing number of pain reports over time and an increased risk of somatic symptoms, even after the researchers controlled for comorbid disorders (41).

PTSD in Patients With Chronic Pain

A growing number of studies have shown that PTSD symptoms tend to be elevated in, and to impact on, patients with chronic pain and fibromyalgia (33,39,42–46). Indeed, it appears that between 10% and 50% of patients receiving tertiary care treatment for chronic pain and related conditions have symptoms that satisfy diagnostic criteria for PTSD, compared with approximately 8% of the general population. Benedikt and Kolb, for example, have reported that 10% of a sample of military veterans being treated for chronic pain also satisfied diagnostic criteria for PTSD (47). In a study of patients with chronic musculoskeletal pain associated with work-related injury, we found that 34.7% had symptoms consistent with a diagnosis of PTSD (42). We also noted that a significant number of patients (18.2%) who did not present with full PTSD had symptoms sufficient for clinical attention; that is, they met the criteria for 2 of the 3 PTSD symptom clusters. Even more striking are the various reports from the motor-vehicle accident literature: these indicate that upward of 50% of patients receiving treatment for chronic pain present with co-occurring PTSD (48,49). All these findings are supported by recent data from the National Comorbidity Study indicating that patients with musculoskeletal pain are 4 times more likely to develop PTSD than are those without musculoskeletal pain (50).

It is, however, important in this context to consider the possibility that the observed rates of PTSD may depend partly on the heterogeneous ways in which patients with chronic pain respond to their symptoms and general situation. This heterogeneity can be operationalized using the Multidimensional Pain Inventory (MPI) (51) and an associated empirical clustering procedure called the Multiaxial Assessment of Pain (MAP) (52,53). These tools identify 3 primary subgroups of patients with chronic pain, including 1) those who are adaptive copers (displaying lower pain severity, pain interference, and affective distress), 2) those who are interpersonally distressed (displaying lower perceived social support), and 3) those who are considered dysfunctional (displaying higher pain severity, pain interference, elevated affective distress and fear, and lower activity). As anticipated, we found evidence to support the hypothesis that PTSD is most prevalent in patients with chronic musculoskeletal pain who are classified as dysfunctional, compared with interpersonally distressed or adaptive copers (54). Indeed, we observed that approximately 70% of dysfunctional copers satisfy diagnostic criteria for PTSD, compared with 21% of interpersonally distressed and 35% of adaptive copers. Others have recently reported similar findings (unpublished data).

Potential Mechanisms

The foregoing review indicates that pain symptoms and chronic pain are prevalent in patients with PTSD and that PTSD symptoms are common in patients with chronic pain, particularly in those with higher pain severity, more interference in daily living, and higher negative affect. However, establishing co-occurrence provides neither an understanding of the nature of the relation(s) between the conditions nor an understanding of the mechanisms by which they are linked. Indeed, the data reviewed thus far indicate several possible relation scenarios. For any 2 variables (or conditions), possible relations are as follows: 1) they co-occur but are unrelated, 2) one causes the other (that is, PTSD causes pain, or vice versa), 3) each influences the other in some way (for example, chronic pain exacerbates symptoms of PTSD, or vice versa), or 4) some third factor (for example, a genetic predisposition) causes both. It should be noted that the third and fourth possibilities are not mutually exclusive. In most cases, the mounting evidence reviewed below does not strongly support the first 2 possibilities. It does, however, suggest several mechanisms through which PTSD and chronic pain are closely linked and influence each other. These mechanisms include shared vulnerability and mutual maintenance.

Shared Vulnerability

Since PTSD and chronic pain frequently co-occur, it seems plausible that there may be individual difference factors predisposing people to develop one or both of these conditions. Consequently, clinical investigators have been working to identify potential predisposing factors. While several constructs hold promise, including the constructs of trait negative affectivity and harm avoidance, it is anxiety sensitivity that has been proving most fruitful as a predisposing factor. Anxiety sensitivity denotes a dispositional tendency to become fearful and, more specifically, refers to the fear of anxiety symptoms based on the belief that they may have harmful consequences (55,56). For example, a person who has high levels of anxiety sensitivity is likely to become fearful in response to chest tightness and shortness of breath, thinking that these symptoms may signal an impending heart attack. Conversely, a person with low levels of anxiety sensitivity would likely find these symptoms to be nothing more than unpleasant. Research suggests that there are 3 basic dimensions of anxiety

sensitivity: fear of publicly observable anxiety reactions, fear of cognitive dyscontrol, and fear of somatic sensations (56).

Anxiety sensitivity has been shown to be elevated in patients with PTSD (10,57,58) and in some (59), but not all (60,61), samples of patients with chronic pain. Patients with persistent headache pain and those with musculoskeletal pain who are classified as dysfunctional tend to have elevated anxiety sensitivity relative to other patients with pain (for a review, see 54). These are the same patients with pain who are more likely to have PTSD (26,54). It has also been shown that the severity of anxiety sensitivity is positively correlated with severity of PTSD symptoms (62). Based on these data, it seems plausible that the anxiety sensitivity construct represents the bridge, or shared vulnerability, between PTSD and chronic pain.

Evidence suggests that anxiety sensitivity amplifies the intensity of emotional reaction, particularly of fear and anxiety, and that it is a predisposing factor in panic attacks (56,63). It has been suggested that elevated anxiety sensitivity may be a predisposing factor both for PTSD (58) and for much of the suffering and disability associated with chronic musculoskeletal pain (26,64). That is, the tendency to respond with fear to symptoms of anxiety is thought to predate the development of PTSD and chronic pain. When people with high anxiety sensitivity levels encounter a traumatic stressor, painful physical injury, or both, they are believed to respond with a more intense emotional reaction than do those with lower levels. In the case of PTSD, the degree of alarm caused by the stressor itself combined with alarm related to the anxiety sensations arising from the stressor amplifies the emotional reaction and thereby increases the risk of developing PTSD (58). In the case of chronic pain, it also appears that anxiety sensitivity amplifies fear, anxiety, and associated avoidance responses when pain-related experiences occur, thereby increasing the likelihood that pain will be maintained over time (21,64–66). When the traumatic stressor and pain-precipitating event are the same or occur in close temporal proximity, anxiety sensitivity may amplify the collective response and may increase vulnerability for development of both conditions.

Growing evidence suggests that there may be a genetic basis for this shared vulnerability. Indeed, data indicate that genetic factors play a role in anxiety sensitivity (67,68), PTSD (69), and pain (70,71). It may be that there are genetic factors common to all 3 conditions. Genetically based dysregulations in serotinergic or GABA-ergic systems may be involved. Consistent with this, dysregulations in both systems have been implicated in pain and in anxiety disorders (71–73).

Mutual Maintenance

Sharp and Harvey have recently proposed a mutual maintenance model to explain the association between PTSD and chronic pain (3). In essence, they posit that certain components of chronic pain (cognitive, affective, and behavioural) maintain or exacerbate symptoms associated with PTSD and, likewise, that components of PTSD (physiological, affective, and behavioural) maintain or exacerbate symptoms associated with chronic pain. To illustrate: the model holds that chronic pain serves as a persistent reminder of the trauma and, conversely, that arousal triggered by the reminder promotes avoidance of pain-related situations. Distress and disability appear to have a unidirectional influence on both PTSD and chronic pain and themselves appear to be influenced directly by the following 7 proposed mechanisms through which mutual maintenance occurs: 1) attentional and reasoning biases, 2) anxiety sensitivity, 3) reminders of the trauma, 4) avoidance, 5) depression and reduced activity levels, 6) anxiety and pain perception, and 7) cognitive demand from symptoms that limits the use of adaptive strategies.

The model and its derivative postulates provide several useful directions for empirical inquiry, some of which we discuss in greater detail below. As well, there are several useful clinical implications. Sharp and Harvey suggest that "reduction in cognitive and behavioural avoidance (via in vivo and imaginal exposure) and aiming to increase activity levels (acting as an exposure intervention) are crucial" (3, p 872) and that there should be an emphasis on "helping the patient to see links between their chronic pain problem and PTSD" (3, p 872). (We also discuss general practice guidelines in greater detail below.) There are, however, some noteworthy conceptual limitations to the proposed model. First, and perhaps foremost, Sharp and Harvey seem to confuse the issues of shared vulnerability and mutual maintenance. It is plausible that anxiety sensitivity may become elevated through associative learning (58,74), and thereby serve as a maintaining factor, as opposed to a preexisting vulnerability factor. However, we feel that progress in the field will be best facilitated by distinguishing between the shared vulnerability and mutual maintenance constructs. Further, Sharp and Harvey provide a somewhat simplified view of the potentially complex relation (3). Indeed, given that both PTSD and pain are multidimensional constructs (for example, both comprise multiple symptom clusters involving cognition, behaviour, and physiology), it is plausible that specific sets of symptoms are causally associated, whereas others are not. Evidence from the clinical setting suggests that subsyndromal presentations of PTSD and their association with pain may also warrant careful consideration in this regard (75). Finally, while the model recognizes the importance of feedback loops, there are occasions in which this feedback is more likely bidirectional than unidirectional. Again, progress in the field will be best facilitated by a fully articulated model.

Outstanding Issues and Future Directions

One line of research that would assist in disentangling the relation between PTSD and chronic pain involves investigating the temporal association between these disorders. To the best of our knowledge, there has been little attention to this issue in the empirical literature. However, since temporal association might hold important implications for the manner in which the disorders are mechanistically linked, progress in the field will likely require careful consideration of this issue. For any given patient, researchers (and clinicians) need to determine whether PTSD or pain came first and the extent to which they are related. In some cases, it may be that the traumatic event

that led to PTSD involved a physically painful injury or experience (for example, regaining consciousness during surgery). In such cases, pain may be a somatic flashback to the original traumatic event, much like flashbacks in other modalities (76). To the extent that this is the case, it may be possible to prevent or minimize the risk of developing pain-related PTSD flashbacks by early treatment with analgesics or other agents. Along these lines, it has been shown that secondary prevention of PTSD may be possible with propranolol (77). What about cases wherein pain predated PTSD? We speculate that PTSD (and, likely, its subsyndromal manifestations) leads to increased reporting of pain and that this is influenced by various cognitive and physiological factors.

It is also important to consider the extent to which specific PTSD symptoms are influenced by pain. We have used the factor analytic approach in preliminary attempts to clarify this issue. This approach is based on the assumption that distinct factors correspond to discrete mechanisms (that is, separate operational processes for each symptom domain [78]). If this is the case, then disentangling the association between symptoms of PTSD and (chronic) pain may help us to better understand the phenomenology of each—particularly when they co-occur. To this end, we have shown that a 4-factor intercorrelated model (that is, a PTSD model comprising 4 interrelated factors of reexperiencing, avoidance, numbing, and hyperarousal) and a hierarchical 2-factor model (that is, a PTSD model comprising 2 lower-order factors of reexperiencing–avoidance and numbing–hyperarousal) provided a good fit to the PTSD-symptom data derived from groups of United Nations peacekeepers with and without chronic pain (75). Of particular importance here, the final models for each group were different in several important ways: specifically, we observed significant differences on 1 reexperiencing item (physical reactions to reminders of trauma), 2 numbing items (emotional numbing and sense of foreshortened future), and 1 hyperarousal item (being hypervigilant).

This suggests that chronic pain may have a cumulative negative impact upon these particular PSTD symptoms. That is, physical reaction to reminders of the trauma, feeling emotionally numb, having a sense of foreshortened future, and hypervigilance may take on particular significance when pain co-occurs with PTSD. This is not surprising when one considers (as noted above) that these symptoms are common to many patients with chronic pain. Along these lines, it has been suggested that PTSD symptoms are aggravated by stimuli and situations that resemble some aspect of the original traumatic event (79). It remains to be determined how specific mechanisms (for example, hypervigilance or attentional bias for threat cues and dysregulation of the endogenous opioid system) operate in individuals with both PTSD and chronic pain, although it seems likely that their operations are not mutually exclusive.

Of particular relevance to future investigation in the area is the notion that co-occurring chronic pain and PTSD are not necessarily distinct disorders but, rather, intimately connected and overlapping (29). This idea, supported by much of the empirical work reviewed above, suggests several testable postulates of the relation between these 2 conditions. General postulates—based on notions of shared vulnerability and mutual maintenance (3)—are as follows:

1. Vulnerability factors, particularly anxiety sensitivity, have been shown to be elevated in patients with PTSD and in chronic pain patients classified as dysfunctional. If elevated anxiety sensitivity is a vulnerability for both PTSD and chronic pain, then those with this vulnerability (prospectively measured) who experience a painful injury should be more likely to develop PTSD. An intriguing question is whether those who are vulnerable but experience a trauma that is not associated with a painful injury (for example, witnessing an accident or death) are equally likely to experience significant and persistent pain.

2. Reminders of trauma typically trigger arousal. If pain is a reminder of trauma, then patients with comorbid PTSD and pain should actively avoid activities and events that trigger acute pain sensations. This avoidance, if persistent, will have a direct relation to functional limitations and disability. It may also prevent activation of the fear network, which in turn will maintain symptoms over time.

3. Attentional biases have been observed in both PTSD and chronic pain (particularly in those who are pain-phobic). If pain does serve as a reminder of trauma, then patients with comorbid PTSD and pain should demonstrate attentional bias toward both trauma-relevant cues and pain cues. There is preliminary evidence from a modified Stroop paradigm suggesting that this is indeed the case (80).

4. Pain threshold and tolerance are affected by elevations in anxiety. Since anxiety is a central feature of PTSD, the experience of PTSD may reduce pain threshold and pain tolerance. In combination with aforementioned factors, these reductions may influence distress and disability levels. In the context of pain threshold and tolerance, it will be interesting to test the notion that comorbid PTSD and chronic pain overtax available coping recourses in patients with both conditions.

5. There are several other concomitants to both PTSD and chronic pain, depression being the most common. It is possible that depression-related changes, such as fatigue and reduced activity levels, exacerbate and maintain both PTSD and chronic pain symptoms, as well as associated disability. In tests of the shared vulnerability and mutual maintenance model, careful consideration needs to be given to depression and other third-party factors.

General Clinical Implications

The literature reviewed above indicates that PTSD and pain often co-occur in patients presenting for treatment of either condition alone. As well, there is some evidence to support the notion that there may be a shared vulnerability and that, when symptoms do co-occur, they are mutually maintaining. As such, it is imperative that clinicians be aware of the relation between PTSD and pain when assessing and treating patients for these conditions, regardless of whether this occurs in a psychiatric or pain specialty setting. In this section, we present general recommendations to assist in assessing and treating patients who present with co-occurring symptoms of PTSD or pain.

Assessment

We recommend that clinicians who conduct diagnostic assessments of patients presenting with PTSD symptoms also screen for the presence of existing pain conditions (for example, fibromyalgia or chronic musculoskeletal pain). This can be accomplished by including questions about the nature and locations of a patient's pain, about triggers of pain, about the duration of pain, and about pain-related disability or interference with daily activities. Some may prefer using a structured clinical interview format (for example, the Somatoform Disorders Section of the Structured Clinical Interview for DSM-IV [SCID-I/NP] [81]) as a means of collecting this information. However, we recommend self-report measures like the McGill Pain Questionnaire-Short Form (82), the Multidimensional Pain Inventory (83), and where appropriate, the Pain Anxiety Symptoms Scale (84). We also recommend that patients presenting with pain complaints, particularly when these are chronic, be assessed for the presence of PTSD symptoms. This can be achieved by including questions from structured clinical interviews (for example, the PTSD section of the SCID [81] or the Clinician Administered PTSD Scale revised [85]) or self- report measures (like the PTSD Symptom Scale [86] or the Posttraumatic Stress Disorder Checklist [87]). In these cases, full and subsyndromal PTSD may have an impact on pain and pain-related behaviours. To facilitate appropriate treatment, it is important to consider the presence of co-occurring pain symptoms in those seeking treatment for PTSD, and vice versa. However, we also recommend that levels of anxiety sensitivity be assessed and considered in treatment planning (see below). Anxiety sensitivity can be efficiently assessed using the 16-item Anxiety Sensitivity Index (88).

Treatment

When PTSD and pain symptoms co-occur, it is likely that clinicians will be required to modify treatment protocols accordingly. For example, clinicians may have to adapt existing cognitive-behavioural therapy programs to address both PTSD symptoms and pain management strategies. Descriptions of existing treatment protocols for PTSD (11,58,89) and chronic pain (90,91) can be found elsewhere . Within the context of these protocols, it appears that incorporating treatments to reduce anxiety sensitivity may improve the treatment of PTSD (58) and chronic pain (64), or quite likely, both, when they co-occur. Current evidence suggests that the most potent method for reducing anxiety sensitivity is interoceptive exposure therapy, although most studies to date have been conducted in patients with panic disorder (56,63). Similar considerations apply to health care providers prescribing medication regimens, alone or in combination with psychotherapy, for the treatment of PTSD and chronic pain. To the best of our knowledge, there are no systematic empirical data to suggest whether treatment efficacy would be improved by treating one condition or the other first, rather than addressing them simultaneously. As noted above, pilot data from a small randomized trial indicate that propranolol, a beta blocker often used for its analgesic properties, may be effective in the secondary treatment of PTSD (77); thus, it may be effective in simultaneous symptom relief. Regardless of the specific treatment approach, it appears that special attention needs to be paid to the common co-occurrence of these conditions when discussing treatment options with patients.

Conclusions

PTSD and chronic pain are conditions that commonly co-occur. Researchers have found that to some extent these conditions share phenomenological characteristics, including anxiety and hyperarousal, behavioural avoidance, emotional lability, and an attentional bias toward somatic cues. Shared vulnerability and mutual maintenance models hold promise in improving our understanding of the relation. However, further research is required to help delineate the relation so that we can develop a better overall understanding of these 2 disorders, together with more effective assessment and treatment protocols for use when they co-occur. Clinically, a failure to appreciate the intricacies of co-occurring PTSD and chronic pain may result in reduced treatment efficacy and continuing negative outcomes.

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