

Chronic Pain, Psychopathology, and DSM-5 Somatic Symptom Disorder

Joel Katz, PhD¹; Brittany N Rosenbloom, MSc²; Samantha Fashler, MA²

¹Professor, Department of Psychology, York University, Toronto, Ontario.

Correspondence: Department of Psychology, BSB 232, York University, 4700 Keele Street, Toronto, ON, M3J 1P3; jkatz@yorku.ca.

²Graduate Student, Department of Psychology, York University, Toronto, Ontario.

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Unlike acute pain that warns us of injury or disease, chronic or persistent pain serves no adaptive purpose. Though there is no agreed on definition of chronic pain, it is commonly referred to as pain that is without biological value, lasting longer than the typical healing time, not responsive to treatments based on specific remedies, and of a duration greater than 6 months. Chronic pain that is severe and intractable has detrimental consequences, including psychological distress, job loss, social isolation, and, not surprisingly, it is highly comorbid with depression and anxiety. Historically, pain without an apparent anatomical or neurophysiological origin was labelled as psychopathological. This approach is damaging to the patient and provider alike. It pollutes the therapeutic relationship by introducing an element of mutual distrust as well as implicit, if not explicit, blame. It is demoralizing to the patient who feels at fault, disbelieved, and alone. Moreover, many medically unexplained pains are now understood to involve an interplay between peripheral and central neurophysiological mechanisms that have gone awry. The new Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, somatic symptom disorder overpsychologizes people with chronic pain; it has low sensitivity and specificity, and it contributes to misdiagnosis, as well as unnecessary stigma. Adjustment disorder remains the most appropriate, accurate, and acceptable diagnosis for people who are overly concerned about their pain.

Pain has survival value. It serves as a warning that all is not well, frequently signalling injury or disease. It encourages us to seek medical help, contributes to the healing process by promoting rest and recovery, and lets us know, by its absence, when to resume activities. Pain reminds us of past harmful events and situations, it teaches us what to avoid in the future, and motivates us to act to terminate it. People born without the capacity to feel pain often do not live beyond childhood because they fail to appreciate the implications of injury and disease.¹ These protective functions concern acute pain, a relatively short, time-limited experience that abates when the injury heals or the disease is cured.

However, there is another kind of pain—chronic pain or persistent pain—that serves no adaptive purpose. When chronic pain is severe and intractable, it lodges itself in the core of the person and causes distress and suffering. Chronic pain ruins marriages and families. It leads to job loss and other financial problems, social isolation, worry, anxiety, depression, and, at times, suicide. The IASP defines pain as “an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage.”^{2, p 210} In contrast, we do not have a generally agreed on definition of chronic pain. Chronic pain was traditionally defined by the length of time that pain persists,³ but a time-based approach ignores many other important features. Recent conceptualizations have introduced a more nuanced approach.² The IASP currently defines chronic pain variously as “pain without apparent biological value,” pain “that has persisted beyond the normal tissue healing time . . . as determined by common medical experience,” and (or) as “a persistent pain that is not amenable, as a rule, to treatments based upon specific remedies.”^{2, p xii} But even these refinements do not capture all the varieties of persistent pain, and an acceptable definition continues to elude us. For example, some chronic pain conditions, such as rheumatoid arthritis, will likely never heal, and others, such as migraine headaches, remit (that is, heal) and then recur.² Notwithstanding the challenges associated with defining chronic pain and the problems with a solely time-based definition, for research purposes, chronic nonmalignant pain is typically defined as pain that persists for longer than 6 months.⁴⁻⁶

From a neurobiological perspective, pain involves nociceptive, inflammatory, and (or) neuropathic components.⁷ Pain arising from nociceptive afferent input is

Highlights

- About 1 in 5 Canadian adults suffers with chronic pain of at least moderate intensity.
- Many strange and unusual pain symptoms once thought to be caused by psychopathology can now be explained by peripheral and central neurophysiological mechanisms that have gone awry.
- DSM-5 SSD with predominant pain replaces DSM-IV-TR pain disorder, but, because the diagnostic criteria for SSD are overly inclusive, many medical patients with significant symptoms of emotional distress will receive an inappropriate psychiatric diagnosis.

the first stage of an “early-warning physiological protective system”^{7, p 3742} designed to minimize contact with tissue-damaging and other noxious stimuli. It involves activation of high-threshold receptors (nociceptors) on sensory neurons located in skin, muscle, and viscera. Pain due to inflammatory inputs is usually caused by tissue-damaging stimuli, such as injury, trauma, and surgery. Tissue damage activates a cascade of neuromolecular immune responses that results in swelling and tenderness, which as noted above, encourages rest and promotes recovery. Nociceptive and inflammatory pains are adaptive and protective; they typically comprise what is referred to as acute pain in that they are time-limited and resolve when the noxious stimulus has been removed or healing occurs.

In contrast, pain of neuropathic origin, which arises from direct lesion or damage to somatosensory system, is considered pathological.⁸ Neuropathic pain involves profound alterations in the normal peripheral and central neural processing of afferent input. As a consequence of injury or disease, nociceptive neurons change their response properties; they may display spontaneous activity, an increase in responsiveness, and a reduction in activation threshold to normal and subthreshold inputs (Table 1).^{9,10} Pain of neuropathic origin is often described as burning, aching, and electric shock-like in quality. It is typically more severe and less responsive to conventional treatments than are nociceptive and inflammatory pain. Pathological pain with similar features also occurs in people who have not sustained an injury or who have no discernable disease, such as in fibromyalgia, irritable bowel syndrome, and tension headaches.^{8,10} Regardless of the presence or absence of an identifiable etiological trigger, when neuroplasticity goes awry in certain at-risk people, the pain becomes the disease.^{7,11,12}

Recent epidemiologic studies reveal surprisingly high prevalence estimates for chronic pain. Using the World Mental Health version of the World Health Organization CIDI, the 12-month prevalence of chronic pain was found to be about 37% in developed countries and 41% in developing countries.¹³ When only moderate-to-severe pain is considered, lifetime prevalence rates drop to about 25% of the general population.¹⁴ These data are consistent with 3 Canadian surveys showing a prevalence rate for

Abbreviations

CIDI	Composite International Diagnostic Interview
CNS	central nervous system
DSM	Diagnostic and Statistical Manual of Mental Disorders
IASP	International Association for the Study of Pain
OR	odds ratio
PTSD	posttraumatic stress disorder
SSD	somatic symptom disorder

chronic noncancer pain between 19% and 29%, with most respondents reporting pain of moderate to severe intensity.^{5,15,16} Chronic pain prevalence increases with age, is greater among females than males, and among people with lower, compared with higher, socioeconomic status.^{5,13,17,18} Common causes of chronic noncancer pain include traumatic injury, surgery, and arthritis.^{5,17,19} The most frequent body locations for chronic pain include the low back, knee joints, head, and neck.^{5,6}

Comorbidity Between Chronic Pain and the Mood and Anxiety Disorders

Chronic pain is highly comorbid with anxiety and depression. Space limitations preclude a thorough review. Two recent, large-scale, population-based surveys examined the comorbidity between chronic neck and (or) back pain and the DSM-IV mood and anxiety disorders.^{20,21} Both surveys used the third version of the CIDI to assess mood and anxiety disorders as well as interviews to determine the 12-month prevalence of comorbid neck and (or) back pain. Von Korff et al²¹ surveyed 5692 community-dwelling people in the United States. Demyttenaere et al²⁰ conducted a cross-national survey of 85 088 community-dwelling people in 18 countries around the world. The ORs (adjusted for age, sex, race and [or] ethnicity, and education) associated with having comorbid neck and (or) back pain and any anxiety disorder or major depression relative to neck and (or) back pain alone (that is, the added risk of having a mental disorder in people with neck and [or] back pain) for the 2 studies were remarkably similar.

Depression

In the US-based study,²¹ the 12-month prevalence of comorbid chronic pain and any mood disorder was 17.5% (SE [standard error] 1.1), with major depression comprising the most frequent comorbid mood disorder (12.6%, SE 0.07). ORs for any mood disorder ranged from 2.5 to 3.2, with an OR of 2.5 for major depression. In the cross-national study,²⁰ the 12-month prevalence of comorbid chronic pain and major depression ranged from 2.5% to 15.7%, with ORs greater than zero in 16 out of 18 of the countries and a pooled OR of 2.3 (95% CI 2.1 to 2.5). The STOP-PAIN Project evaluated the burden of chronic pain in 728 people waiting for treatment at 1 of 8 university-based multidisciplinary pain treatment facilities in Canada.²² Eighty-two per cent endorsed symptoms of depression (as measured by the Beck Depression Inventory), with about 56% reporting moderate to extremely severe levels and 34.6% reporting suicidal ideation.

Anxiety

ORs ranged from 1.5 (95% CI 0.9 to 2.4) for agoraphobia to 2.6 (95% CI 2.1 to 3.3) for PTSD, with a pooled OR of 2.3 (95% CI 1.9 to 2.7) for any anxiety disorder in the US-based study²¹ and pooled across countries from 1.9 (95% CI 1.7 to 2.2) for social phobia to 2.7 (95% CI 2.4 to 3.1) for generalized anxiety disorder with a pooled OR

of 2.2 (95% CI 2.1 to 2.4) for any anxiety disorder for the cross-national study.²⁰ It is notable that, with the exception of agoraphobia without panic disorder,²¹ all of the anxiety disorders in both surveys were significantly more likely to occur in people with neck and (or) back pain than in people without. Moreover, the highest ORs were found for generalized anxiety disorder (OR 2.6; 95% CI 2.0 to 3.5²¹ and OR 2.7; 95% CI 2.4 to 3.1²⁰) and PTSD (OR 2.614; 95% CI 2.2 to 3.0²⁰ and OR 2.6; 95% CI 2.1 to 3.3²¹). The substantial overlap in comorbidity and symptomatology between the anxiety disorders and chronic pain has been documented both when examining the presence of anxiety disorders in people with chronic pain and the presence of chronic pain in people with anxiety (mainly PTSD).^{23–25} Interested readers are referred to recent reviews describing the theoretical models and mechanisms proposed to underlie the high comorbidity between chronic pain and the anxiety disorders.^{23–25}

Chronic Pain and Psychopathology

There are many pains whose cause is not known. If a diligent search has been made in the periphery and no cause is found, we have seen that clinicians act as though there was only one alternative. They blame faulty thinking, which for many classically-thinking doctors is the same thing as saying that there is no cause and even no disease. They ignore a century's work on disorders of the spinal cord and brainstem and target the mind. . . . These are the doctors who repeat again and again to a Second World War amputee in pain that there is nothing wrong with him and that it is all in his head.^{26, p 107}

Pains that do not conform to present-day anatomical and neurophysiological knowledge are often attributed to psychopathology. This view of medically unexplained pain persists, notwithstanding evidence to the contrary. Psychological dysfunction has been proposed to cause phantom limb pain,²⁷ dyspareunia,²⁸ orofacial pain,^{29,30} fibromyalgia,³¹ pelvic pain,³² and a host of others, including abdominal pain, chest pain, and headache.³³ However, the complexity of the pain transmission circuitry means that many pains that are currently poorly understood will ultimately be explained without resorting to a psychopathological etiology.¹⁰ Pain that is nonanatomical in distribution, spread of pain to noninjured territory, pain that is said to be out of proportion to the degree of injury, and pain in the absence of injury have all, at one time or another, been used as evidence to support the idea that psychological disturbance underlies the pain. Notably, each of these features once thought to be caused by psychopathology can now be explained by peripheral and central neurophysiological mechanisms that have gone awry (Table 1).^{29,34}

Spontaneous pain, pain from a stimulus, such as gentle touch, that does not normally provoke pain (allodynia), and increased pain from a stimulus that normally provokes pain (hyperalgesia) are perceptual correlates of well-documented mechanisms of central sensitization that

Table 1 Mechanisms of dorsal horn neuronal central sensitization and other physiological findings associated with pathological pain states showing possible associated experiences and (or) symptoms that in the past were medically unexplained and contributed to the misdiagnosis of chronic pain patients as having mental illness (for example, hysteria, hypochondriasis, malingering, pain-prone, psychogenic pain disorder, somatization)

Physiological mechanism	Experience (symptom)
Development of and (or) increase in spontaneous neural activity ⁹	Stimulus-independent pain (spontaneous pain)
Reduction in neural threshold for activation ⁹	Pain in response to a stimulus that does not normally produce pain (allodynia)
Increased neural response to repeated, fixed-intensity, C-fibre strength stimulus ⁷²	Increased pain from a stimulus that normally provokes pain (hyperalgesia)
Enlargement of neuronal receptive fields ⁹	Spread of pain to adjacent, undamaged tissue (secondary hyperalgesia)
Injury-induced unmasking of previously ineffective synaptic connections ³⁷	Spread of pain to distant body regions (remote hyperalgesia)
Cross-system, viscerovisceral interactions within the central nervous system ⁴⁰	Spread of pain to distant body regions (remote hyperalgesia)
Ipsilateral injury-induced, contralateral peripheral neurite loss ⁴⁹	Mirror-image pain (allochiria)

develop after injury to the peripheral or CNS.^{9,10,35} There is also a less-well-known body of literature on the expression of novel receptive fields^{36,37} and the expansion of existing receptive fields^{38,39} in response to alterations in the flow of afferent input. Further, previously undocumented cross-system, viscerovisceral interactions within the CNS have recently been described among women with chronic pelvic pain.⁴⁰ This literature provides a neurophysiological basis for clinical observations in humans that pain may occur in the absence a known trigger, spread to distant, previously nonpainful, and otherwise healthy body regions,^{9,40} as well as a rationale for why a local intervention applied to one body region may relieve pain at another.^{40,41}

Recent data linking the immune system and the CNS have provided an explanation for another medically unexplained pain problem. Mirror-image pain or allochiria has puzzled clinicians and basic scientists ever since it was first documented in the late-1800s.⁴² Injury to one side of the body is experienced as pain at the site of injury as well as at the contralateral, mirror-image point.^{43,44} Recent animal studies show induction of a sciatic inflammatory neuritis by peri-sciatic microinjection of immune system activators results in both an ipsilateral hyperalgesia and hyperalgesia at the mirror-image point on the opposite side in the territory of the contralateral healthy sciatic nerve.⁴⁵ Moreover, both the ipsilateral and contralateral hyperalgesia are prevented or reversed by intrathecal injection of various proinflammatory cytokine antagonists.⁴⁶

Mirror-image pain is likely not a unitary phenomenon and other nonimmune mechanisms may also be involved.⁴⁷ For example, recent human^{48,49} and animal evidence⁵⁰ point to a potential combination of central and peripheral contributions to mirror-image pain as nerve injury to 1 side of the body has been shown to result in a 50% reduction in the innervation of the territory of the same nerve on the opposite side of the body in uninjured skin.⁵⁰ Interestingly, while documented contralateral neurite loss can occur

in the absence of contralateral pain or hyperalgesia, pain intensity at the site of the injury correlates significantly with the extent of contralateral neurite loss.⁴⁹ This raises the intriguing possibility that the intensity of pain at the site of an injury may be facilitated by contralateral neurite loss induced by the ipsilateral injury⁵⁰—a situation that most clinicians would never have imagined possible. More recently, tactile allochiria (for example, touch applied to a finger on the right hand was experienced only as pain in the corresponding left finger) occurred in patients with complex regional pain syndrome—type I who also showed evidence of possible parietal lobe dysfunction.⁴⁸

Taken together, these novel mechanisms explain some of the most puzzling pain symptoms. But there remain many other pains that defy present-day understanding. This should keep us mindful that emotional distress and psychological disturbance in our patients are not the cause of the pain. Attributing pain to a psychological disturbance is damaging to the patient and provider alike. It pollutes the therapeutic relationship by introducing an element of mutual distrust as well as implicit, if not explicit, blame. It is demoralizing to the patient who feels at fault, disbelieved, and alone.

Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, and Chronic Pain

The golden rule: an underlying medical illness or medication side effect has to be ruled out before ever deciding that someone's symptoms are caused by mental disorder. . . . There are serious risks attached to over-psychologizing somatic symptoms and mislabeling the normal reactions to being sick – especially when the judgments are based on vague wording that can't possibly lead to reliable diagnosis. DSM-5 as it now stands will add to the suffering of those already burdened with all the cares of having a medical illness.^{51, p 484}

Table 2 DSM-5 diagnostic criteria for somatic symptom disorder

A.	One or more somatic symptoms that are distressing or result in significant disruption of daily life.
B.	Excessive thoughts, feelings, or behavior[s] related to the somatic symptoms or associated health concerns as manifested by at least one of the following: <ol style="list-style-type: none"> 1. Disproportionate and persistent thoughts about the seriousness of one's symptoms. 2. Persistently high level of anxiety about health or symptoms. 3. Excessive time and energy devoted to these symptoms or health concerns.
C.	Although any one somatic symptom may not be continuously present, the state of being symptomatic is persistent (typically more than 6 months). <i>Specify if:</i> With predominant pain (previously pain disorder): This specifier is for individuals whose somatic symptoms predominantly involve pain. <i>Specify if:</i> Persistent: A persistent course is characterized by severe symptoms, marked by impairment, and long duration (more than 6 months). <i>Specify current severity:</i> Mild: Only one of the symptoms specified in Criterion B is fulfilled. Moderate: Two or more of the symptoms specified in Criterion B are fulfilled. Severe: Two or more of the symptoms specified in Criterion B are fulfilled, plus there are multiple somatic complaints (or one very severe symptom).

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The hard-fought gains to have pain disorder expunged from the DSM-IV⁵² have come at the expense of an overly inclusive and unreliable new DSM-5⁵³ disorder termed SSD. Nevertheless, that DSM-5 does not include a pain-specific disorder (for example, psychogenic pain disorder,⁵⁴ somatoform pain disorder,⁵⁵ and pain disorder⁵²), as in past editions of the DSM, is a welcomed advance (see Merskey^{56,57} for reviews on the history of DSM pain and somatoform diagnoses).

SSD is a single diagnostic entity that replaces 3 of the DSM-IV⁵² somatoform disorders (somatization disorder, pain disorder, and undifferentiated somatoform disorder, and in some cases hypochondriasis). Table 2 lists the diagnostic criteria for SSD, which include 1 or more physical symptoms lasting 6 months or longer that are associated with excessive thoughts, feelings, or behaviours. Three specifiers describe the nature (with predominant pain), duration (persistent, if longer than 6 months), and severity (mild, moderate, or severe) of the symptoms. Brief SSD is diagnosed under other specified somatic symptom and related disorder when the duration of symptoms is less than 6 months. The SSD specifier, persistent, indicating a duration greater than 6 months appears to be redundant and unnecessary, as SSD is diagnosed only at 6 months and brief SSD is diagnosed when the symptoms have persisted for less than 6 months.

DSM-IV pain disorder was rightly criticized on 2 counts: The questionable importance of medically unexplained pain^{58,59} in pain disorder associated with psychological factors; and, the lack of a definition of psychological factors or a description of when they are of sufficient importance or magnitude to play a role in the pain experience in the

presence of a general medical condition made it a diagnosis of exclusion.^{59,60} The DSM-5 SSD represents an advance over DSM-IV pain disorder with the removal of the requirement that the symptoms be medically unexplained and the addition of subjective distress as diagnostic of SSD, but it has ignored the sage advice that this is a diagnosis that should rarely be made.⁵⁶

Notwithstanding these improvements, 2 major criticisms have been levelled against the DSM-5: diagnostic inflation and inadequate field testing.⁶¹ Both appear to apply to the new diagnostic category of SSD. The main criticism of SSD is the high probability of misdiagnosing a medical illness, including chronic pain conditions, as a mental illness.^{61–63} Further, the conditions that qualify for a diagnosis of SSD are extremely variable. They include a highly mixed group of patients with medically unexplained symptoms, medical patients with significant symptoms of emotional distress, patients with typical chronic pain conditions (for example, low-back pain, fibromyalgia, rheumatoid arthritis) and patients with health-related anxiety.⁶⁴ According to Frances,⁶² 15% of cancer patients, 15% of patients with heart disease, and 25% of patients with irritable bowel syndrome and chronic widespread pain would qualify for a diagnosis of SSD. Moreover, the false-positive rate in the general population would approach 7%.

DSM-5 field testing in the United States and Canada showed a test–retest kappa coefficient of 0.61 (95% CI 0.40 to 0.77) for complex SSD (revised), as well as a prevalence for complex SSD (revised) of 0.08 (95% CI 0.06 to 0.11) and a corresponding DSM-IV prevalence of 0.1 for somatoform disorder (excluding conversion and body dysmorphic disorders).⁶⁵ However, note that in the absence of more data

Table 3 Recommended⁷¹ revised DSM-5 diagnostic criteria for somatic symptom disorder

Criteria A, B, C, D, E, F, and G must all be fulfilled to make the diagnosis:

- D. One or more prominent physical symptoms.
- E. Excessive and maladaptive thoughts, feelings, and behavio[u]rs related to the physical symptoms. All three [3] of the following must be present:
 1. clearly disproportionate and intrusive worries about the seriousness of the symptoms,
 2. extreme anxiety about the symptoms, and
 3. excessive time and energy devoted to the symptoms or health concerns.
- F. The concerns have persisted at a clearly problematic and maladaptive level for at least 6 months.
- G. The excessive concerns about physical symptoms are pervasive and cause significant disruption and impairment in daily life.
- H. If a diagnosed medical condition is present, the thoughts, feelings, and behavio[u]rs are grossly in excess of what would be expected, given the nature of the medical condition.
- I. If no medical diagnosis has been made, a thorough medical workup has been performed to rule out possible causes and is repeated at suitable intervals to uncover medical conditions that may declare themselves with the passage of time.
- J. The physical symptom or concern it is not better accounted for by another mental disorder (e.g. [for example], anxiety, depressive, or psychotic disorder).

Reproduced, with permission, from Frances.⁷¹

on the validity of SSD, even a moderately high reliability coefficient is not very meaningful. It is also not clear how the diagnostic criteria for complex SSD (revised), as described in the field testing,^{65,66} map onto those that now appear in the DSM-5 under SSD. Moreover, there is no mention of whether any of the patients in the field trial sample actually had chronic pain conditions (compared with other body system symptoms). Further, although a stated aim of SSD was to simplify and clarify the diagnostic procedure for use in primary care, field testing did not include primary care settings or physicians.⁶⁵ Finally, sample size was inadequate. Considering that field testing usually produces higher reliability estimates than exist in clinical practice,⁶¹ the data on reliability seem unrealistically high for what is an extremely broad range of patients who qualify for a diagnosis of SSD. A subsequent field trial in routine mental health clinical practice settings reported the DSM-5 approach to diagnosis was feasible, useful, and acceptable to clinicians and patients but the sample size diagnosed with SSD was extremely small.⁶⁷

Frances and Chapman⁵¹ list 10 negative consequences of the new DSM-5 SSD diagnostic category, including the following that relate specifically to chronic pain conditions: stigma; overlooked diagnoses owing to a failure to investigate new or worsening somatic symptoms; increased risk of a parent, with a child who has a chronic illness, being diagnosed with SSD, leading to inappropriate claims of parental overinvolvement or of encouraging the child's sick role behaviour; increased risk of receiving inappropriate psychotropic medications; and falling prey to the gender trap⁶⁸ by further marginalizing women in the health care system as they present with physical symptoms such as persistent pain more frequently than do men. This is supported by a comparison of DSM-5 SSD and DSM-IV somatoform disorders assessed in the same group of patients showing that a significantly greater

proportion of women than men received the former but not the latter diagnosis.⁶⁹

Several authors have proposed modified diagnostic criteria to reduce the likelihood of diagnostic inflation and the misdiagnosis of medical illness as mental illness.^{62,64,70} Table 3 contains a set of revised diagnostic criteria recommended by Frances.⁷¹ These criteria were developed to establish an appropriate balance between sensitivity and specificity, and to reduce the likelihood of false-positive diagnoses. However, given the problems with current diagnostic criteria for SSD, noted above, alternative diagnoses have been suggested as appropriate for people with chronic pain; including, psychological factors affecting other medical conditions⁷⁰ and adjustment disorder.^{70,71} The distinction between SSD and psychological factors affecting other medical conditions is not clear-cut, especially for people with chronic pain conditions, as both involve a diagnosable medical condition. For SSD, the emphasis is said to be on "maladaptive thoughts, feelings and behavior," whereas for psychological factors affecting other medical conditions the emphasis is on "the exacerbation of the medical condition."^{53, p 324} This distinction seems arbitrary for people with chronic pain, as pain is known to produce worry and worry is known to exacerbate pain. Until the criteria for SSD are appropriately revised, adjustment disorder is a "safer and more accurate" diagnosis when one is needed for someone who is "medically ill or otherwise troubled by puzzling symptoms"^{71, p 531} Chronic pain is highly comorbid with anxiety and depression

Summary and Conclusions

Acute pain is protective in the short term when it signals tissue damage or disease via nociceptive and inflammatory inputs, but chronic pathological pain has no adaptive value. Chronic pain is highly prevalent worldwide and highly comorbid with anxiety and depression. It is associated with substantial

financial, occupational, psychological, and social burden. Pain that is not readily explained medically has frequently been attributed to psychopathology, largely because of puzzling and bizarre symptoms, but recent basic science findings have provided a neurophysiological basis for many of these symptoms. These advances in our understanding of pain mechanisms have had parallel effects in psychiatry where, for the first time in more than 30 years, the most recent edition of the DSM no longer includes a pain-specific mental disorder. DSM-5 SSD with predominant pain replaces DSM-IV pain disorder but it is not recommended for people with chronic pain, because it lacks validity, and is overly inclusive and stigmatizing. Instead, adjustment disorder remains the most appropriate, accurate, and acceptable diagnosis for people who are overly concerned about their pain.

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References

- Nagasako EM, Oaklander AL, Dworkin RH. Congenital insensitivity to pain: an update. *Pain*. 2003;101(3):213–219.
- Merskey H, Bogduk N. Classification of chronic pain: descriptions of chronic pain syndromes and definitions of pain terms. 2nd ed. Seattle (WA): International Association for the Study of Pain Press; 1994.
- International Association for the Study of Pain. Classification of chronic pain. *Pain*. 1986;Suppl 3:S1–S225.
- Landmark T, Romundstad P, Dale O, et al. Estimating the prevalence of chronic pain: validation of recall against longitudinal reporting (the HUNT pain study). *Pain*. 2012;153(7):1368–1373.
- Schopflocher D, Taenzer P, Jovey R. The prevalence of chronic pain in Canada. *Pain Res Manag*. 2011;16(6):445–450.
- Johannes CB, Le TK, Zhou X, et al. The prevalence of chronic pain in United States adults: results of an Internet-based survey. *J Pain*. 2010;11(11):1230–1239.
- Woolf CJ. What is this thing called pain? *J Clin Invest*. 2010;120(11):3742–3744.
- von Hehn CA, Baron R, Woolf CJ. Deconstructing the neuropathic pain phenotype to reveal neural mechanisms. *Neuron*. 2012;73(4):638–652.
- Latremoliere A, Woolf CJ. Central sensitization: a generator of pain hypersensitivity by central neural plasticity. *J Pain*. 2009;10(9):895–926.
- Woolf CJ. Central sensitization: implications for the diagnosis and treatment of pain. *Pain*. 2011;152(3 Suppl):S2–S15.
- Niv D, Devor M. Chronic pain as a disease in its own right. *Pain Pract*. 2004;4(3):179–181.
- Siddall PJ, Cousins MJ. Persistent pain as a disease entity: implications for clinical management. *Anesth Analg*. 2004;99(2):510–520.
- Tsang A, Von Korff M, Lee S, et al. Common chronic pain conditions in developed and developing countries: gender and age differences and comorbidity with depression-anxiety disorders. *J Pain*. 2008;9(10):883–891.
- Elliott AM, Smith BH, Penny KI, et al. The epidemiology of chronic pain in the community. *Lancet*. 1999;354(9186):1248–1252.
- Boulanger A, Clark AJ, Squire P, et al. Chronic pain in Canada: have we improved our management of chronic noncancer pain? *Pain Res Manag*. 2007;12(1):39–47.
- Moulin DE, Clark AJ, Speechley M, et al. Chronic pain in Canada—prevalence, treatment, impact and the role of opioid analgesia. *Pain Res Manag*. 2002;7(4):179–184.
- Pergolizzi J, Ahlbeck K, Aldington D, et al. The development of chronic pain: physiological CHANGE necessitates a multidisciplinary approach to treatment. *Curr Med Res Opin*. 2013;29(9):1127–1135.
- van Hecke O, Torrance N, Smith BH. Chronic pain epidemiology and its clinical relevance. *Br J Anaesth*. 2013;111(1):13–18.
- Johansen A, Romundstad L, Nielsen CS, et al. Persistent postsurgical pain in a general population: prevalence and predictors in the Tromsø study. *Pain*. 2012;153(7):1390–1396.
- Demyttenaere K, Bruffaerts R, Lee S, et al. Mental disorders among persons with chronic back or neck pain: results from the World Mental Health Surveys. *Pain*. 2007;129(3):332–342.
- Von Korff M, Crane P, Lane M, et al. Chronic spinal pain and physical-mental comorbidity in the United States: results from the National Comorbidity Survey Replication. *Pain*. 2005;113(3):331–339.
- Choiniere M, Dion D, Peng P, et al. The Canadian STOP-PAIN Project—part 1: who are the patients on the waitlists of multidisciplinary pain treatment facilities? *Can J Anaesth*. 2010;57(6):539–548.
- Asmundson GJ, Coons MJ, Taylor S, et al. PTSD and the experience of pain: research and clinical implications of shared vulnerability and mutual maintenance models. *Can J Psychiatry*. 2002;47(10):930–937.
- Asmundson GJ, Katz J. Understanding the co-occurrence of anxiety disorders and chronic pain: state-of-the-art. *Depress Anxiety*. 2009;26(10):888–901.
- Katz J, Pagé MG, Fashler S, et al. Chronic pain and the anxiety disorders: epidemiology, mechanisms and models of comorbidity, and treatment. In: Marchand S, Saravane D, Gaumond I, editors. *Mental health and pain: somatic and psychiatric components of pain in mental health*. Paris (FR): Springer-Verlag; 2014. p 119–155.
- Wall PD. *Pain: the science of suffering*. London (GB): Weidenfeld & Nicolson; 1999.
- Katz J. Individual differences in the consciousness of phantom limbs. In: Kunzendorf RG, Wallace B, editors. *Individual differences in conscious experience: first-person constraints on theories of consciousness, self-consciousness, and subconsciousness*. Amsterdam (NL): John Benjamins Publishing Co; 2000. p 45–97.
- Meana M, Binik YM. Painful coitus: a review of female dyspareunia. *J Nerv Ment Dis*. 1994;182(5):264–272.
- Gagliese L, Katz J. Medically unexplained pain is not caused by psychopathology. *Pain Res Manag*. 2000;5:251–257.
- Graff-Radford SB. Facial pain. *Curr Opin Neurol*. 2000;13(3):291–296.
- Nielson WR, Merskey H. Psychosocial aspects of fibromyalgia. *Curr Pain Headache Rep*. 2001;5(4):330–337.
- Hoffman D. Understanding multisymptom presentations in chronic pelvic pain: the inter-relationships between the viscera and myofascial pelvic floor dysfunction. *Curr Pain Headache Rep*. 2011;15(5):343–346.
- Stoudemire A, Sandhu J. Psychogenic/idiopathic pain syndromes. *Gen Hosp Psychiatry*. 1987;9(2):79–86.
- Mannion RJ, Woolf CJ. Pain mechanisms and management: a central perspective. *Clin J Pain*. 2000;16(3 Suppl):S144–S156.
- Coderre TJ, Katz J, Vaccarino AL, et al. Contribution of central neuroplasticity to pathological pain: review of clinical and experimental evidence. *Pain*. 1993;52(3):259–285.
- Dostrovsky JO, Millar J, Wall PD. The immediate shift of afferent drive to dorsal column nucleus cells following deafferentation: a comparison of acute and chronic deafferentation in gracile nucleus and spinal cord. *Exp Neurol*. 1976;52(3):480–495.
- Dostrovsky JO. Immediate and long-term plasticity in human somatosensory thalamus and its involvement in phantom limbs. *Pain*. 1999;Suppl 6:S37–S43.
- Devor M, Wall PD. Plasticity in the spinal cord sensory map following peripheral nerve injury in rats. *J Neurosci*. 1981;1:679–684.
- Woolf CJ. Evidence for a central component of post-injury pain hypersensitivity. *Nature*. 1983;306(5944):686–688.
- Berkley KJ. A life of pelvic pain. *Physiol Behav*. 2005;86(3):272–280.
- Gracely RH, Lynch SA, Bennett GJ. Painful neuropathy: altered central processing maintained dynamically by peripheral input. *Pain*. 1992;51(2):175–194.

42. Basbaum AI. A new way to lose your nerve. *Sci Aging Knowledge Environ*. 2004;2004(15):pe15.
43. Livingston WK. Pain and suffering. Fields HL, editor. Seattle (WA): International Association for the Study of Pain Press; 1998.
44. Maleki J, LeBel AA, Bennett GJ, et al. Patterns of spread in complex regional pain syndrome, type I (reflex sympathetic dystrophy). *Pain*. 2000;88(3):259–266.
45. Chacur M, Milligan ED, Gazda LS, et al. A new model of sciatic inflammatory neuritis (SIN): induction of unilateral and bilateral mechanical allodynia following acute unilateral peri-sciatic immune activation in rats. *Pain*. 2001;94(3):231–244.
46. Milligan ED, Twining C, Chacur M, et al. Spinal glia and proinflammatory cytokines mediate mirror-image neuropathic pain in rats. *J Neurosci*. 2003;23(3):1026–1040.
47. Koltzenburg M, Wall PD, McMahon SB. Does the right side know what the left is doing? *Trends Neurosci*. 1999;22(3):122–127.
48. Cohen H, McCabe C, Harris N, et al. Clinical evidence of parietal cortex dysfunction and correlation with extent of allodynia in CRPS type I. *Eur J Pain*. 2013;17(4):527–538.
49. Oaklander AL, Romans K, Horasek S, et al. Unilateral postherpetic neuralgia is associated with bilateral sensory neuron damage. *Ann Neurol*. 1998;44(5):789–795.
50. Oaklander AL, Brown JM. Unilateral nerve injury produces bilateral loss of distal innervation. *Ann Neurol*. 2004;55(5):639–644.
51. Frances A, Chapman S. DSM-5 somatic symptom disorder mislabels medical illness as mental disorder. *Aust N Z J Psychiatry*. 2013;47(5):483–484.
52. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 4th ed. Text revision. Washington (DC): American Psychiatric Publishing; 2000.
53. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 5th ed. Arlington (VA): American Psychiatric Publishing; 2013.
54. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 3rd ed. Washington (DC): American Psychiatric Publishing; 1980.
55. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 3rd ed. Revised. Washington (DC): American Psychiatric Publishing; 1987.
56. Merskey H. Pain disorder, hysteria or somatization? *Pain Res Manag*. 2004;9(2):67–71.
57. Merskey H. Pain, psychogenesis, and psychiatric diagnosis. *Int Rev Psychiatry*. 2000;12:99–102.
58. Merskey H. Somatization, hysteria, or incompletely explained symptoms? *Can J Psychiatry*. 2004;49(10):649–651.
59. Creed F, Gureje O. Emerging themes in the revision of the classification of somatoform disorders. *Int Rev Psychiatry*. 2012;24(6):556–567.
60. Sullivan MD. DSM-IV pain disorder: a case against the diagnosis. *Int Rev Psychiatry*. 2000;12(2):91–98.
61. Frances AJ, Widiger T. Psychiatric diagnosis: lessons from the DSM-IV past and cautions for the DSM-5 future. *Annu Rev Clin Psychol*. 2012;8:109–130.
62. Frances A. The new somatic symptom disorder in DSM-5 risks mislabeling many people as mentally ill. *BMJ*. 2013;346:f1580.
63. Hauser W, Wolfe F. The somatic symptom disorder in DSM 5 risks mislabelling people with major medical diseases as mentally ill. *J Psychosom Res*. 2013;75(6):586–587.
64. Rief W, Martin A. How to use the new DSM-5 somatic symptom disorder diagnosis in research and practice: a critical evaluation and a proposal for modifications. *Annu Rev Clin Psychol*. 2014;10:339–367.
65. Regier DA, Narrow WE, Clarke DE, et al. DSM-5 field trials in the United States and Canada, part II: test-retest reliability of selected categorical diagnoses. *Am J Psychiatry*. 2013;170(1):59–70.
66. Freedman R, Lewis DA, Michels R, et al. The initial field trials of DSM-5: new blooms and old thorns. *Am J Psychiatry*. 2013;170(1):1–5.
67. Moscicki EK, Clarke DE, Kuramoto SJ, et al. Testing DSM-5 in routine clinical practice settings: feasibility and clinical utility. *Psychiatr Serv*. 2013;64(10):952–960.
68. Malterud K. Symptoms as a source of medical knowledge: understanding medically unexplained disorders in women. *Fam Med*. 2000;32(9):603–611.
69. Voigt K, Wollburg E, Weinmann N, et al. Predictive validity and clinical utility of DSM-5 somatic symptom disorder—comparison with DSM-IV somatoform disorders and additional criteria for consideration. *J Psychosom Res*. 2012;73(5):345–350.
70. Young G. Ill-treatment of pain in the DSM-5. *Psychol Inj Law*. 2013;6(4):307–313.
71. Frances A. DSM-5 somatic symptom disorder. *J Nerv Ment Dis*. 2013;201(6):530–531.
72. Woolf CJ, Thompson SW. The induction and maintenance of central sensitization is dependent on *N*-methyl-D-aspartic acid receptor activation; implications for the treatment of post-injury pain hypersensitivity states. *Pain*. 1991;44(3):293–299.