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Evaluating Psychosocial Contributions to Chronic Pain Outcomes

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Abstract

The biopsychosocial model of pain dominates the scientific community's understanding of chronic pain. Indeed, the biopsychosocial approach describes pain and disability as a multidimensional, dynamic integration among physiological, psychological, and social factors that reciprocally influence one another. In this article, we review two categories of studies that evaluate the contributions of psychosocial factors to the experience of chronic pain. First, we consider general psychosocial variables including distress, trauma, and interpersonal factors. Additionally, we discuss pain-specific psychosocial variables including catastrophizing, expectations, and pain-related coping. Together, we present a diverse array of psychological, social, and contextual factors and highlight the need to consider their roles in the development, maintenance, and treatment of chronic pain conditions.

Keywords

chronic pain; biopsychosocial; mechanisms

Introduction

Historically, the concept of pain, even chronic pain, depended on a linear relationship between identifiable organic pathology and patient-reported symptoms. Thus, the amount of pain was expected to be perfectly proportional to the amount of tissue damage “causing” the pain. Psychological factors were presumed to be primary mechanistic contributors to pain only in those cases where no identifiable pathology was present and the pain could be labeled as “psychogenic”. Over the past 4–5 decades, however, a biopsychosocial

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understanding has come to dominate the scientific community's characterization of chronic pain. One of the biopsychosocial model's strengths is its flexibility, and the term has undergone substantial reconceptualizations over the past half-century. In a 1977 article in *Science*, psychiatrist George Engel called for a new "medical model" that would supplant the old biomedical model of disease and would incorporate social, psychological, and behavioral dimensions of illness ¹. Similarly, Fordyce's seminal work on the contribution of behavioral conditioning and contextual factors ^{2,3}, and Loeser's synthesis of biopsychosocial principles ⁴⁻⁶, along with the patient benefits of multidisciplinary pain care, helped to establish a growing acceptance of the biopsychosocial model of pain in principle if not practice ^{7,8}. Finally, the dawning era of personalized pain medicine emphasizes the importance of characterizing the inter-relationships between psychological states, social/contextual forces, and neurobiological processes for the individual patient, with the goal of optimizing treatment outcomes ⁹⁻¹¹.

Collectively, the biopsychosocial approach describes pain and disability as a multidimensional, dynamic interaction among physiological, psychological, and social factors that reciprocally influence one another, resulting in chronic and complex pain syndromes^{7, 12}. A good deal of empirical evidence supports the biopsychosocial model, though in practice, psychosocial factors are often assigned secondary status and viewed largely as reactions to pain. As we will describe below, longitudinal, observational research supports a strong bidirectional link between mood disorders and persistent pain; the development of an enduring pain condition confers a substantially increased vulnerability for the subsequent diagnosis of an affective disorder, while psychosocial variables such as depression, anxiety, and distress are among the most potent and robust predictors of the transition from acute to chronic pain ¹³⁻¹⁸. This should perhaps not be surprising, as the report of pain is always subjective, influenced by a broad range of cognitive, behavioral, and affective constructs and processes. The biopsychosocial model shifts the emphasis from exclusive reliance on the pathophysiology involved in the initiation of nociception to involvement of the patient's cognitive and emotional state, or psychological vulnerability (i.e., susceptibility to undesirable outcomes ¹⁹), and conditioned responses that influence his or her pain experiences and subsequent behavior. From this perspective, assessment, diagnosis, prognosis, and consequently treatment of the patient with persistent pain requires a broad strategy that examines and incorporates a wide range of psychosocial and behavioral factors in addition to, but not to the exclusion of, biomedical ones.

In this review we discuss 2 broad categories of "mechanistic" studies that evaluate the contributions of psychosocial factors to the experience of chronic pain. In the first category, psychosocial processes either exist within an individual as pre-existing "vulnerability" factor [e.g., distress²⁰⁻²², trauma^{23, 24}] or emerge for the first time in response to the experience of ongoing pain [e.g., fear-avoidance behavior ²⁵, self-efficacy^{26, 27}]. These psychosocial factors then influence individual variability in pain-related outcomes. For example, specific psychosocial characteristics place individuals at elevated risk for the transition from acute to persistent pain, or for the development of pain-related disability in the context of a persisting pain condition ^{15, 22, 28, 29}. The second category of mechanistic study involves delivery of some type of pain-related intervention that is hypothesized to directly affect a psychological factor, presuming that changing that psychosocial factor (e.g., reducing distress, improving

pain-coping, etc) will be associated with a subsequent change in one or more pain-related outcomes. That is, the psychological factor acts as a mediator (though not necessarily a causal mechanism) through which a treatment confers its benefits^{30, 31}. This type of study parallels, in its structure, many biological and pharmacological studies in which a medication acts on a specific receptor, a TENS unit reduces the transmission of pain-related information in the distribution of a nerve, or a physical therapy regimen strengthens a particular group of muscles, which then impacts the recipient's reported experience of pain.

In the following sections, we discuss a number of psychosocial factors (see Table 1) that have been studied as contributors to the development and long-term outcomes of persistent pain as well as factors influencing treatment-related outcomes. These variables are not condition-specific; indeed most have been studied across diagnostic categories with similar results. We have organized these constructs as "general" psychosocial factors present in the general population and "pain-specific" factors unique to individuals experiencing pain. While this review is not exhaustive, we have highlighted some of the most influential and commonly studied psychosocial factors that influence the pain experience. While many of the constructs discussed below overlap at least to a moderate degree, we discuss their unique contributions to the experience of pain.

General Psychosocial Factors

AFFECTIVE FACTORS

Depression, anxiety, and general indices of emotional distress along with a cluster of negative emotions, thoughts, and behaviors termed, "negative affect," are probably the most commonly-assessed psychological factors in patients with persistent pain^{11, 32, 33}. Recent systematic reviews indicate that chronic pain patients, relative to pain-free controls, show elevations in all of these indices of self-reported negative affect³³⁻³⁵. Although psychological symptomatology is often interpreted as a consequence of chronic pain, prospective studies suggest that pre-morbid psychological dysfunction represents a risk factor for the future development of numerous chronic pain conditions including musculoskeletal pain and functional pain disorders^{14, 36-39}. Furthermore, results of a 20-month longitudinal study indicate that depression and anxiety longitudinally predict pain and pain-related disability, however neither pain nor pain-related disability predicted depression and anxiety²¹. Emotional distress also predicts pain and function. In recent meta-analyses and reviews, researchers found that higher levels of pre-surgical emotional distress were associated with more postsurgical pain and impairment^{20, 40}. Moreover, emotional distress and psychosocial stress have been shown to increase the likelihood of transitioning from acute to chronic musculoskeletal pain^{18, 36, 37, 41, 42}.

Overall, there is a wealth of evidence that symptoms of depression, anxiety, and emotional distress contribute strongly (more strongly than pain intensity, in many studies) to key long-term outcomes of persistent pain such as physical disability^{35, 43-46}, work disability⁴⁷, healthcare costs^{48, 49}, mortality⁵⁰⁻⁵³, and suicide^{22, 54, 55}. In general, these studies establish the association of pain with the deleterious outcomes of interest, and then show that some or all of that association can be statistically accounted for by indices of depression, anxiety, or emotional distress. For example, in a recent study of lumbar fusion

for degenerative spondylolisthesis, patients were followed post-surgically for 2 years to determine the predictors of functional outcomes⁵⁶. In multivariate analyses, high pre-operative symptoms of depression remained the only significant predictor of failure to return to work after surgery, even in multivariate models with pre- and post-operative pain intensity included. In this study, depression fully mediated the prospective association between pain intensity and occupational disability. Furthermore, patients in the upper half of the distribution of pre-operative depression scores were approximately $\frac{1}{3}$ less likely to return to work and, among those who did return, took nearly twice as long post-operatively to begin working again⁵⁶.

Just as negative affect is associated with increased pain and disability, positive affect and optimism are associated with less pain and dysfunction^{57, 58}. Dispositional optimism has not only been linked to lower pain sensitivity, but a recent study by Hanssen and colleagues identified that optimism improves pain intensity through its effects on reducing situational pain catastrophizing⁵⁷. Likewise, state-based positive affect has been identified as a mediator of resilience in the wake of chronic pain^{59–61}. There is also evidence suggesting that trait-based positive affect may buffer maladaptive pain-related behaviors such as fear of movement⁶². Further, in patients with low back pain, positive affect has been shown to buffer the effects of negative affect such that patients with high levels of both positive and negative affect reporting less pain and depression and better functioning than those with high levels of negative affect and low levels of positive affect⁵⁸.

Because positive affect is highly malleable, it has been identified as a target for resilience-oriented interventions such as Positive Activity Interventions (PAIs). PAIs are aimed at raising positive feelings, cognitions, and behaviors rather than reducing negative ones⁶³. There is preliminary evidence suggesting that PAIs can be effective in the treatment of chronic pain. In one study by Hausmann and colleagues⁶⁴, participants who were asked to complete positive activities (e.g., listing three good things about their day) reported improvements in pain scores from baseline to 6 months. In another study of patients with chronic pain, individuals assigned to tailored positive activities reported greater improvements in pain intensity and pain control compared to those in an active control condition⁶⁵.

TRAUMA

Increasing evidence suggests an association between both psychological and physical trauma and chronic pain. There are strong prospective links between early-life traumatic experiences and the subsequent development of chronic pain^{23, 24, 66–69}. In a recent metaanalysis, the authors reported that the presence of past traumas is associated with a 2- to 3-fold increase in the development of chronic widespread pain²³. Early-life trauma can take many forms, and childhood physical, sexual, and psychological abuse have all been demonstrated as risk factors for the development of chronic pain conditions in adulthood including fibromyalgia, irritable bowel syndrome, chronic pelvic pain, and temporomandibular joint disorders^{23, 69, 70}. In fact, abuse in childhood conferred a 97% increase in risk for having a painful somatic syndrome such as fibromyalgia in adulthood²³. Furthermore, individuals with childhood reports of distressing events such as

hospitalizations or loss of a family member showed double the risk for the development of adult chronic pain even after adjusting for other important (and potentially confounding) predictors such as psychological distress and socioeconomic status⁶⁷

It remains unclear whether the association between trauma and later chronic pain is the result of the trauma or is driven by affective, cognitive, and behavioral responses to the traumatic event (e.g. fear and avoidance). However, there is evidence to suggest some categories of traumatic stress are stronger risk factors for pain than others. For example, some childhood stressful events such as prolonged hospitalization are associated with chronic pain in adulthood while childhood surgery conferred no additional risk, despite the likelihood that surgery is a painful and distressing experience for children⁶⁷. Furthermore, in adult veterans, combat exposure and post-traumatic stress disorder (PTSD) have been shown to have the strongest statistical association with chronic pain^{23, 24}. PTSD, a psychiatric condition resulting from exposure to a traumatic event and characterized by hyperarousal, avoidance, and reexperiencing, is not only a risk factor for chronic pain^{71, 72}, but is associated with increased risk for the transition from acute to chronic pain. It is also associated with elevated pain severity and disability among abuse victims^{73, 74}. In addition, researchers have demonstrated the role of PTSD as a mediator between trauma and pain outcomes. Indeed, PTSD symptoms have been shown to mediate the relationship between childhood abuse and maltreatment and pain and pain-related limitations later in life^{75, 76}. Moreover, results of a 3-decade prospective study suggest the presence of PTSD symptoms amplify the predictive effect of child abuse on later-life pain outcomes⁷⁷. Demonstration of such effects over a 30-year time frame are a reminder of just how entrenched and enduring the deleterious effects of early-life abuse can be.

Further confounding the association between psychological trauma and chronic pain is the role of traumatic brain injury (TBI). TBI, an injury to the brain that results in sudden acceleration and/or deceleration of brain matter inside the skull leading to an alteration in brain function⁷⁸, affects approximately 1.4 million people in the US each year⁷⁹. According to a metaanalysis of 23 studies, more than 50% of people diagnosed with TBI report chronic pain complaints⁸⁰, likely due to the associated physical injuries at the time of the TBI⁸¹. Likewise, there is significant comorbidity as well as symptom convergence between PTSD and TBI^{82–84}. Because traumatic injuries can result in TBI, pain, and PTSD, it remains difficult to identify the degree to which PTSD and TBI can independently predict the development of chronic pain. Thus, further research is necessary to identify causal relationship between TBI and chronic pain development.

SOCIAL/INTERPERSONAL FACTORS

Social forces also impact pain and pain-related outcomes. To date, most studies have focused on either perceived global social support (i.e., not pain-related) or solicitous social responses to pain behaviors (e.g., offering to take over tasks or encouragement to become less active)⁸⁵. According to the stress-buffering hypothesis, negative consequences of stressors on health outcomes can be buffered by social support⁸⁶. Consistent with this notion, social support is found to be associated with improved physical functioning for individuals with pain conditions^{33, 87–89}. In a review of more than a dozen studies, the authors found that

increased perceived social support was associated with better outcomes such as less pain and improved overall functioning among persons with pain and physical disability related to spinal cord injury, multiple sclerosis, and acquired amputation⁸⁵. There is a substantial literature on the important role of social support specifically in pain caused by cancer and cancer-related treatments^{90, 91}. For example, the presence of a supportive partner has been associated with reduced symptom burden (including less pain), improved quality of life, and fewer symptoms of distress in patients with chronic lymphocytic leukemia⁹², metastatic breast cancer^{93, 94}, and other cancer types such as colorectal cancer^{95, 96}. Moreover, better social support was associated with less postoperative pain after surgical management of breast cancer in a recent meta-analysis⁹⁷. This is consistent with experimental pain studies that demonstrate receiving social support during a pain task not only attenuates blood pressure, heart rate, and cortisol reactivity, but is also associated with reduced pain intensity and unpleasantness ratings and increased pain threshold^{98–100}. Further, social support may serve as a treatment target for chronic pain. Guillory and colleagues¹⁰¹ found that a text message-based social support intervention was effective in reducing pain severity and interference and increasing positive affect. In addition, couples-based interventions for patients with painful cancers appear to show greater efficacy than either treatment as usual or exclusively patient-focused interventions^{102–104}.

On the other hand, the operant conditioning model of chronic pain posits that others' responses to pain behavior play a role in chronic pain and disability^{2, 105}. Indeed, there is considerable evidence that a higher degree of solicitousness in response to pain behaviors predicts increased pain-related disability^{85, 106–108}. The social environment and interaction between social support and solicitousness may be particularly important for persons with acquired amputation during the first few months after the amputation. Several studies found that patients reporting positive general social support but lacking solicitous responses to pain were less likely to develop persistent phantom limb pain post-amputation and report lower levels of physical dysfunction^{85, 107, 109}.

The immediate social environment in the form of parents (for children experiencing pain) and spouses (for married adult patients with pain) exerts a powerful influence on pain-related outcomes. In the case of children with persistent pain, parents' reactions to children's pain have also been shown to play an important determining role in children's pain responses^{110–114} in particular, parental pain catastrophizing is strongly related to the development of children's persistent pain following major surgery¹¹⁵, and to the child's disability¹¹⁶. Parental attention to pain and solicitous behaviors that encourage children to avoid regular activities may provide specific pathways by which parental catastrophizing amplifies a child's pain experience and behavior^{116–118, 119}. It is clear that the interactions between patients with chronic pain and their significant others can either facilitate or impair adjustment to chronic pain^{87, 120–122}. Studies across painful conditions illustrate the important role of significant others. For example, among couples, high levels of spousal depressive symptoms predict worsening patient disability and disease activity in patients with rheumatoid arthritis over a 1-year period¹²³. In cancer pain, social support interpersonal interaction preferences (e.g., discomfort with closeness and interdependence in relationships) seem to play an important role in shaping pain reports and general health⁹¹. Patients with partners showing avoidant and anxious attachment styles are more likely to

report increased pain and decreased well-being^{103, 124–127}. Patients' attachment styles are also important predictors of pain-related outcomes among both children and adults. Individuals with anxious or insecure attachment styles are at elevated risk for poorer mental and physical health¹²⁸, for reduced engagement in physical activity¹²⁹, and for less treatment-related improvement in affective outcomes among patients participating in a multidisciplinary treatment program¹³⁰.

It is also important to understand the nature (e.g., supportive, solicitous, adversarial), of other important social interactions, such as relationships at work. Lack of social support at work, dissatisfaction with co-workers, and interactions with the disability compensation system are among the most potent predictors of pain-related work disability^{89, 131, 132}. For example, Li and colleagues studied workplace support among arthritis patients; those who reported low workplace support were much more likely to develop depressive symptoms and work-related disability 18 months later¹³³. Moreover, differing social and occupational structures across countries appear to contribute to cross-national differences in rates of return to work and occupational disability in the context of painful work injury. Anema and colleagues¹³⁴ compared sustainable return-to-work rates between 6 different countries and found that differences in job characteristics and social disability systems were more important than medical interventions, patient, and injury-related factors in explaining the large between-country differences in rates of return to work following painful occupational injuries.

Of course, the social environment can also be harnessed for adaptive purposes. In a study of patients with chronic pain, social support in the form of encouragement to persist in a task was negatively associated with disability¹³⁵. Keefe and colleagues have added spouse-assisted coping skills training to standard CBT and multidisciplinary pain management programs, hypothesizing that the supportive and reinforcing effects of a spouse will facilitate improved pain-related coping and enhance self-efficacy for managing pain-related symptoms^{136–139}. These interventions generally involve dyadic sessions that teach couples communication skills and use mutual goal setting to assist chronic pain patients in acquiring, maintaining, and effectively employing pain-coping skills. In a recent randomized controlled trial examining patients with LBP and their spouses, the spouse-assisted intervention produced larger decreases in fear of pain and catastrophizing than the standard multidisciplinary intervention¹³⁹.

Other individual social and interpersonal relationships are also important influences on pain-related outcomes. During psychotherapeutic treatment, for example, establishing a sound therapeutic relationship between patient and therapist is crucial for producing good outcomes¹⁴⁰. A handful of studies suggest that an index of the therapeutic relationship (i.e., the working alliance) statistically mediates the positive effects of rehabilitative treatments among people with musculoskeletal pain^{141–145}. Indeed, creating and sustaining an effective therapeutic alliance appears to be a necessary and sufficient condition for promoting the pain-improving effects of diverse interventions^{144–145}. These interpersonal processes can also interact with intrapersonal factors such as distress. For example, a recent study revealed that depression was associated with patient-physician discordance in estimates of disease severity (i.e., depressed patients estimated their disease severity as much worse, on average,

than their physicians did)¹⁴⁶. Such discordance is likely to be common, especially in light of the “invisible” nature of pain, and it can have deleterious effects on patient satisfaction and adherence to treatment regimens¹⁴⁷.

SEX AS A CONTRIBUTING PSYCHOBIOLOGICAL FACTOR

Sex and gender are important factors in the perception and experience of pain^{148–151}. There is considerable evidence suggesting greater pain prevalence in women when compared to men^{150, 152–154}. In a review, Unruh¹⁵⁵ found that, compared to men, women are more likely to experience recurrent pain, more severe pain, more frequent pain, and longer-lasting pain. Furthermore, musculoskeletal pain, rheumatoid arthritis, gastrointestinal, neuropathic, facial pain and headache are all more prevalent among females compared to males^{156–160}.

In addition to clinical pain conditions, women demonstrate a greater sensitivity to multiple modalities of experimental pain when compared with men¹⁶¹. Indeed, compared to males, females demonstrate a decreased threshold and tolerance for pain and increased pain intensity and unpleasantness for heat, cold, ischemic, and pressure pain^{150, 161–164}. Sex differences in physiological responses to pain have also been observed. Compared with males, females demonstrate increased sensitivity to pain-induced muscle reflexes¹⁶⁵ and show increased pupil dilation¹⁶⁶ as well as amplified cerebral activation in response to pain¹⁶⁷.

Sex differences have also been observed in more dynamic models of experimental pain such as temporal summation and diffuse noxious inhibitory controls (DNIC). Temporal summation of pain is used to evaluate differences in the central facilitatory processing of nociceptive stimuli. The temporal response to repetitive nociceptive stimuli is characterized by a gradual increase in subjective pain ratings^{168–171}. Compared to males, females exhibit a more pronounced temporal summation response^{172–174} suggesting differences in the central processing of nociceptive stimuli. DNIC is a form of endogenous pain modulation wherein perception of one painful stimulus is attenuated by a pain stimulus applied at a remote site^{175, 176}. Several studies indicate the DNIC response is more pronounced in males than females, suggesting better functioning of the endogenous pain inhibitory system among males^{176–178}. However, a second subset of studies have found no sex differences in DNIC response^{179–181}. These inconsistencies maybe a consequence of methodological differences or sex-specific mediators, however further investigation is necessary to better understand the relationship between sex and endogenous pain inhibition.

The sex differences observed in the pain experience may be caused by a number of biological, psychological, and sociocultural factors. Regarding biological contributions to sex differences in the pain experience, there is consistent evidence to suggest hormones influence sex differences in clinical pain conditions. This is supported first by studies demonstrating similar prevalence across prepubescent males and females for conditions such as migraine and temporomandibular disorders with differences in prevalence emerging after puberty^{149, 182–184}. Lending further support for the role of hormones in the sex differences in pain, the severity of symptoms for several pain conditions including irritable bowel syndrome, temporomandibular disorders, headache, and fibromyalgia varies across the menstrual cycle^{185–191}. Sex differences in the endogenous opioid system may also account

for sex differences in the pain experience. Indeed, women show a higher μ opioid receptor binding at rest while men exhibit greater μ -opioid receptor binding in response to experimentally induced pain¹⁹² which may contribute to sex differences in pain perception.

Psychosocial mechanisms also contribute to the sex differences in the pain experience. Gender roles, for example, are associated with pain responses. Males often adopt the masculine gender norm of increased pain tolerance while females adopt a norm accepting pain as a normal part of life¹⁹³. A number of studies have demonstrated an association between endorsing masculinity and a higher pain threshold and tolerance and lower pain ratings^{194–198}. Moreover, a recent study demonstrated that males endorsing greater masculine gender traits are more likely to participate pain studies, offering a potential explanation for sex differences in pain responses¹⁹⁹. Researchers have also examined the role of pain-specific gender role expectations in the sex differences observed in experimental pain. Using the Gender Role Expectation of Pain (GREP) questionnaire, Robinson and colleagues found that women were considered more sensitive to pain, less enduring of pain, and more willing to report pain compared to men^{194, 198, 200}. Further, sex differences in pain threshold were no longer significant after controlling for willingness to report pain and sex differences in temporal summation of heat pain was partially mediated by willingness to report pain²⁰⁰.

Sex differences in pain perception can also be accounted for by a number of psychological factors including coping, catastrophizing, and affective distress. Several studies have demonstrated sex differences in pain coping. Indeed, compared to men, women have endorsed more frequent use of catastrophizing, behavioral activities, positive self-statements, social and emotional support, and problem-focused coping^{148, 201–204}. Furthermore, catastrophizing has been shown to mediate the sex differences in pain-related outcomes, clinical pain, and experimental pain^{162, 205, 206}.

Anxiety and depression are components of affective distress that may account for sex differences in pain. Anxiety, which is associated with increased clinical pain and experimental pain sensitivity^{161, 207} has been suggested as a potential mediator of sex differences in pain sensitivity²⁰⁸. Although anxiety is more prevalent among females^{209, 210}, several studies have shown that anxiety is positively correlated with pain sensitivity among males, but not females^{211, 212}. Depression, which is highly comorbid with pain, is also more prevalent in women than men; among individuals with depression, women are more likely to report pain complaints than men^{213, 214} and women with chronic pain are more likely to experience depression. However, it remains unclear whether depression mediates the sex differences in pain perception.

RACE AS A CONTRIBUTING PSYCHOBIOLOGICAL FACTOR

Race, which plays an important role in the development and experience of pain, should always be considered when assessing psychosocial factors related to pain. Although there is considerable racial diversity throughout the United States, the current pain literature has primarily examined race differences between Black and White individuals. Further, racial minority groups are frequently grouped together (e.g. non-White, Asian/other) in the literature, limiting any conclusions that otherwise could be drawn. Indeed, in a review of

disparities in pain, Anderson and colleagues²¹⁵ called for future research focused on Asian Americans, Arab Americans, Native Hawaiians, Pacific Islanders, Native American, and Alaskan Natives. Thus, while we ultimately hope to better understand the broader effect of race on pain, this review will focus only on Black-White differences.

It is well-documented that, compared to White individuals, Black individuals demonstrate increased pain sensitivity. Black individuals have reported higher levels of pain for clinical conditions such as AIDS, glaucoma, arthritis, post-operative pain, post-spinal fusion pain, and low back pain^{216–220}. In addition to being more sensitive to clinical pain, Black individuals demonstrate a lower tolerance and report higher pain intensity and unpleasantness ratings than non-Hispanic Whites during experimental pain tasks^{162, 221–224}. Similar findings have been noted for Asian Americans, who report a greater intensity of daily joint pain than non-Hispanic whites, and who demonstrate enhanced sensitivity to thermal and mechanical pain stimuli applied in a laboratory setting^{225, 226}. Intriguingly, though there is comparatively little research in this area, other ethnic/racial groups may show different patterns of pain responses; for example, Native American participants in a laboratory pain study were actually less pain-sensitive than non-Hispanic Whites on measures of acute pain sensitivity, suggesting that more work with larger samples (and a greater distribution of race/ethnic categories) will be important in this field.²²⁷

Racial differences in clinical and experimental pain sensitivity may be due to a variety of reasons including biological factors. Campbell and colleagues²²⁸ found that compared to Caucasians, African Americans have a lower threshold for the nociceptive flexion reflex, a measurement of pain induced spinal reflexes. Differences in central pain-inhibitory mechanisms may also underlie racial differences in pain sensitivity. In fact, Black individuals experience reduced diffuse noxious inhibitory control (DNIC) of pain compared to non-Hispanic Whites, indicating ethnic differences in endogenous pain inhibition²²⁹. Additionally, Black individuals are more prone than non-Hispanic Whites to experience hypertension and show cardiovascular reactivity to pain, which may, in turn, influence their pain perception²³⁰.

In addition to biological factors, psychosocial factors may account for race differences in pain sensitivity. An abundance of evidence links depression and anxiety to chronic pain and increased pain sensitivity^{11, 13, 32, 41}. Approximately 20% of individuals with chronic pain also have depression, while less than 7% of the general population experiences this disorder, and these findings hold not just for the U.S. but around the world^{231–235}. Likewise, higher levels of anxiety are associated with increased pain sensitivity²³⁶. Although Blacks are less likely than non-Hispanic Whites to report experiencing depression and anxiety, the chronicity of Major Depressive Disorder is higher among Black individuals than Caucasians, and Black patients are less likely to receive effective treatment and more likely than Whites to rate their depression as severe and disabling^{237–241}. The differential experience of psychological distress may well contribute to race differences in pain sensitivity.

Beyond psychological distress, pain-specific factors such as appraisals, expectations, and coping are also related to pain sensitivity and may influence the race differences observed in the pain experience. The meaning of pain (i.e. how individuals appraise pain) can be

influenced by sociocultural factors and ethnic background^{242, 243}. Such pain appraisals can, in turn, impact pain related responses. Expectations regarding pain also impact pain perception. Koyama and colleagues²⁴⁴ found that expectations of decreased pain reduced the subjective experience of pain. Furthermore, these expectations decreased the activation of pain-related brain regions, offering an explanation of how expectations influence pain sensations. Preliminary evidence suggests that expectations for pain sensitivity and willingness to report pain differ based on race and ethnicity, which may help explain race difference in pain sensitivity^{245, 246}. There are also notable race differences in pain coping. In a recent meta-analysis, researchers found that Black Americans more frequently endorse the use of catastrophizing, hoping and praying, and passive coping compared to White Americans²⁴⁷. Furthermore, there is evidence to suggest that both catastrophizing as well as praying mediate race differences in experimental pain^{162, 222}. That is, compared to White individuals, Black individuals more frequently endorse the use of catastrophizing and prayer and these differences in coping partially account for their decreased cold pain tolerance.

Another hypothesis relates to sociocultural factors that cause chronic stress, especially in Black individuals. In addition to the socioeconomic strain often associated with minority status, Black individuals are frequently exposed to racism^{248–250}. Chronic socioeconomic strain and racism lead to high levels of sympathetic nervous system activation and physiological exhaustion making the individual more sensitive to painful stimuli²⁵¹. Multiple published studies have suggested that a greater degree of perceived daily racial discrimination is associated with elevated rates of chronic pain and enhanced sensitivity to pain in the laboratory among samples of African-American participants^{252–254}.

Pain-Specific Psychosocial Constructs

PAIN COPING

Coping is broadly defined as the use of behavioral, emotional, and cognitive techniques to manage symptoms of distress²⁵⁵. Coping strategies are commonly categorized based on process elements. One conceptualization differentiates cognitive from behavioral strategies. This conceptualization served as the basis for the Coping Strategies Questionnaire (CSQ), a widely used measure of 6 cognitive (diverting attention, reinterpreting pain, coping self-statements, ignoring pain, praying/hoping, and catastrophizing) and 2 behavioral (increasing activity and increasing pain behaviors) coping strategies²⁵⁶. Alternatively, some have differentiated active from passive strategies. In broad terms, active coping refers to strategies to control pain or function despite pain while passive coping involves relinquishing control of pain to others^{257–259}. Coping can also be classified into problem-focused versus emotion-focused strategies. Problem-focused strategies involve direct attempts to deal with pain whereas emotion-focused strategies involve managing the emotional reactions to pain^{255, 260–262}.

Whatever classification scheme is used, individual differences in the application of paincoping strategies have an important influence on pain-related outcomes. Differences in pain coping are associated with differences in pain intensity, adjustment to chronic pain, and psychological and physical functioning^{85, 263–266}. Active coping strategies, for example, have been linked to positive affect, better psychological adjustment, and decreased

depression while passive strategies are linked to poorer outcomes such as increased pain and depression^{258, 267–270}. There is also evidence suggesting that, compared to problem-focused coping, emotion-focused coping is associated with worse pain and functioning in individuals with chronic pain^{267, 271–277}. In addition to the relationship between broad categories of coping and pain outcomes, individual strategies have been shown to correlate with pain-related outcomes. For example, catastrophizing is associated with higher pain levels, poorer physical functioning and disability, and increased pain interference^{13, 32, 278, 279}. Likewise, hoping and praying is also correlated with higher levels of disability, more pain interference, and increased pain severity^{280–283}. There is mixed evidence for relationships between other coping strategies and pain-related outcomes. While reinterpreting pain sensations has been shown to be associated with lower pain ratings for patients with headaches as well as those undergoing nerve conduction studies, it shows weak correlations with functional variables such as pain interference and physical disability^{281, 284–287}. There is also mixed evidence for the relationship between ignoring pain and pain-related outcomes. Evidence suggests an association between ignoring pain and both increased and decreased pain interference^{281, 288–290}. On the other hand, the majority of studies indicate that ignoring pain does not predict pain-related outcomes such as pain interference, disability, or pain severity^{291, 292}.

CATASTROPHIZING

Catastrophizing is a cognitive and emotional response to pain consisting of magnification of pain sensations (e.g., “This is the worst pain I’ve ever experienced,”), rumination about pain and pain-related concerns (e.g., “I can’t stop thinking about the pain,”), and helplessness about one’s ability to manage pain (e.g., “There is nothing I can do to make it better”)^{293, 294}. For individuals with chronic low back pain, musculoskeletal pain, and other miscellaneous chronic pain complaints, catastrophizing is associated with increased pain intensity, pain disability, and psychological distress, even when controlling for level of physical impairment^{295, 296}. While catastrophizing positively correlates with general measures of negative affect such as depressive symptoms and anxiety, it also shows a unique and specific influence on pain-related outcomes^{297–299}. Studies in patients with musculoskeletal pain have indicated that catastrophizing is the single most important pre-treatment risk factor that impairs the effectiveness of pain-relieving interventions^{300, 301}, and recent evidence suggests that catastrophizing may be the principal psychosocial driver of persistent pain symptoms, statistically mediating the prospective influence of factors such as anxiety on post-operative pain outcomes³⁰². Indeed, when controlling for depression and anxiety, catastrophizing remains significantly correlated with return to work^{303–305}, pain related disability³⁰⁶, pain intensity³⁰⁷, and pain tolerance³⁰⁸. Further, catastrophizing is a risk factor for the development of chronic pain, pain-related disability, increased healthcare costs, and increased pain sensitivity for low back pain and joint pain¹³.

Catastrophizing also impacts treatment effectiveness for pain-relieving interventions^{300, 301, 309}, including surgery^{310–312}. In randomized controlled trials, individuals with high pretreatment levels of catastrophizing demonstrated less benefit from topical analgesics³¹³, oral analgesics³⁰⁹, and psychosocial treatments^{314, 315}. In a study aimed at identifying predictors of treatment responders for persistent orofacial pain, researchers found that

baseline levels of catastrophizing were more than one standard deviation higher in the non-responder group than for responders³¹⁶. A similar pattern has been found in examining the effectiveness of TENS for post-joint replacement pain. For patients receiving TENS treatment, those with higher baseline levels of catastrophizing experienced less pain reduction and reduced functional outcomes following treatment³¹⁷. Recent reviews have identified catastrophizing as a key phenotypic factor to assess in trials of analgesic treatments in patients with chronic pain¹¹.

In addition to being a predictor of treatment effectiveness, many studies have demonstrated that the benefits of analgesic therapies may be explained, in part, by their effects on cognitive emotional processes such as catastrophizing. A number of studies using longitudinal process analyses demonstrate that changes in catastrophizing and negative affect precede changes in clinical pain^{30, 318–321}. There is also evidence suggesting that substantial portions of the variability in end-of-treatment outcomes can be accounted for by early-treatment changes in catastrophizing^{30, 31}. The role of catastrophizing as a mechanism of change has been demonstrated in psychosocial treatments such as Cognitive Behavior Therapy (CBT). CBT, which involves reframing negative automatic cognitions, such as catastrophic cognitions, is effective in reducing catastrophizing in individuals with chronic pain that has persisted for decades³²² and its effects can last for months or years after treatment³²³. Likewise, Acceptance and Commitment Therapy (ACT), which does not directly target catastrophizing, has shown to also be effective in reducing catastrophizing^{324–327}. What is more, catastrophizing has been identified as an indirect mechanism of ACT in reducing pain related disability³²⁸. That is, individuals who participated in an ACT intervention for pain demonstrated a decrease in pain catastrophizing, which was related to later improvement in pain-related disability via its influence on psychological flexibility with pain.

Catastrophizing is also a mechanism of change for non-psychosocial treatments. Indeed studies have shown that multidisciplinary treatment programs that include exercise as well as activity-based physical therapy also improve pain outcomes, in part, through reductions in catastrophizing^{329–332}. For example, for patients with chronic low back pain, interventions aimed at increased engagement in graded physical activity as well as fear-provoking activities have been shown to reduce pain catastrophizing as well as functional disability³³⁰. In a study comparing CBT, physical therapy, and combined CBT and physical therapy, there were no between group differences in the pre- to post-treatment change in catastrophizing³²⁹. However, changes in catastrophizing over the duration of the treatment predicted changes in pain outcomes, even in the group that received only physical therapy, with no specific targeting of catastrophizing-related cognitions.

Although catastrophizing uniquely influences the experience of pain, it also overlaps and interacts with other psychosocial processes including depression, anxiety, distress, fear of pain, self-efficacy, optimism, and other positive factors^{32, 306, 333, 334}. Consistent with the Cognitive Behavioral Model of chronic pain, there is evidence to suggest that pain catastrophizing influences pain-related disability via fear of pain^{25, 335, 336}. Individuals with chronic pain may avoid daily activities that increase pain. However, when this avoidance occurs beyond the expected healing time for an injury, they may experience physical

deconditioning which leads to increased pain and discomfort when performing future activities. The deconditioning and worsened pain then results in increased avoidance. Vlaeyen and colleagues suggest that avoidance behavior persists due to certain beliefs, such as catastrophic cognitions^{25, 337, 338}.

Catastrophizing also overlaps with social processes such as social support^{108, 339, 340}. Within the Communal Coping Model framework, catastrophizing can be conceptualized as a coping strategy that activates the individual's social environment by soliciting support or empathy^{124, 294, 341, 342}. Indeed, associations between catastrophizing and perceived solicitous responses^{339, 340, 343}, perceived instrumental support¹⁰³, and perceived critical responses to pain¹⁰⁶ have been observed. Further, both solicitous and punishing responses to pain are associated with higher pain intensity¹⁰⁸ and increased depressive symptoms^{106, 340}, suggesting these social factors may be a mechanism by which catastrophizing affects pain outcomes.

While most of the literature has examined the relationship between patient catastrophizing and pain outcomes, there is also evidence to suggest that catastrophizing by individuals in supportive roles (e.g. parent, spouse) also influences pain-related outcomes^{112, 115, 344}. In one study partner catastrophizing was associated with increased pain intensity in women with dyspareunia³⁴⁴. A similar pattern has been demonstrated between children and parents. For children undergoing major surgery, parent pain catastrophizing scores predicted the child's post-surgical pain intensity^{116, 119, 345}. Results of these studies highlight the interactive impact of catastrophizing and social support, and more specifically the beliefs of those in supportive roles, on pain outcomes.

EXPECTATIONS

Pain-related expectations also influence the experience of pain as well as treatment outcomes. Expectations are related to overall well-being and the development of chronic pain and are also a critical component of responses to treatment for both placebo and active treatments^{346,347}. Generalized positive outcome expectancy, or dispositional optimism^{348–350}, is related to increased feelings of control, the use of more active coping strategies, and better functional performance^{91, 351–353}. Positive outcome expectancy has been identified as a protective factor from the transition from acute to chronic pain^{354–356}. Pain specific expectations also influence pain intensity ratings in both laboratory and clinical settings^{357–359}.

Pain-specific expectations also play a role in the effectiveness of both active treatments and placebo. Such expectations have been shown to influence treatment outcomes for a number of active interventions including surgery, opioid analgesics, and acupuncture^{360–363}. For example, in a recent analysis of large acupuncture trials revealed that both patient and provider expectations for treatment success were strong predictors of response, with better pretreatment expectations predicting improved post-treatment pain and physical functioning^{362, 364}. Pain-specific expectations have also been identified as one of the core mechanisms underlying placebo analgesia^{365, 366}. Thus, pain expectations are influencing the experience of pain at various stages, from acute pain intensity through the development and treatment of chronic

SELF-EFFICACY

Self-efficacy, or an individual's belief in his ability to perform a behavior or achieve a desired outcome^{26, 273}, determines thoughts, feelings, and behaviors in stressful situations and affects one's ability to cope successfully when confronted with challenges^{367, 368}. Measures of pain-related self-efficacy assess patients' perceived ability to control pain symptoms and function in spite of pain^{33, 369}. Pain-related self-efficacy, a protective factor for patients with persistent pain and a resiliency factor for children, adolescents, and adults with chronic pain²⁷⁰, predicts functional pain outcomes. In samples of adults and children with chronic headaches, pain-related self-efficacy is associated with less disability, better functioning, and fewer depressive symptoms^{370, 371}. Pain-related self-efficacy also mediates the relationship between pain and functioning in women with fibromyalgia pain³⁷². Similarly, self-efficacy mediates the relationship between pain and performance of valued activities in patients with rheumatoid arthritis³⁷³. That is, patients with higher levels of pain have lower self-efficacy, which is associated with increased difficulty performing valued activities. On the other hand, in a longitudinal study of patients with chronic back pain, improvements in self-efficacy partially mediated the relationship between changes in pain and disability over a 12 month period, with improvements in pain leading to increased self-efficacy which in turn resulted in less pain-related disability³⁷⁴.

Due to the pervasive and persistent nature of chronic pain, patients must make constant adjustments in order to live with their disease^{26, 369}. Thus, a number of non-pharmacologic treatments for pain target pain-related self-efficacy as a process variable with the aim of improving self-efficacy for making such adjustments and living with pain. Indeed, self-efficacy has been shown to be a key mechanism of change in Cognitive-Behavioral Therapy (CBT) for chronic pain^{145, 315, 375}. In a study of patients with persistent orofacial pain, self-efficacy mediated the CBT-related improvements in pain and disability³¹⁵. Likewise, Mindfulness-based Stress Reduction (MBSR) has similar effects on self-efficacy, with no significant differences between CBT and MBSR in their effect on improving self-efficacy³⁷⁶, highlighting the broad importance of self-efficacy as a process factor in pain-management interventions.

PAIN-RELATED CNS PATHWAYS

Progress in structural and functional neuroimaging has been exponential in recent years producing evidence of alterations in both brain structure and function among patients with chronic pain^{377, 378}. Recent reviews summarize the literature comparing patients with a variety of persistent pain complaints to pain-free controls^{379, 380}. Currently, there is evidence for chronic pain-related alterations a number of cortical regions that are considered to be important for the perception of pain including the primary and secondary somatosensory cortices, insular and anterior cingulate cortices, the prefrontal cortices, and many subcortical areas. There are reductions in gray matter in the dorsolateral prefrontal cortex and insula among patients with persistent pain³⁸¹⁻³⁸³. Further, studies show changes in functional connectivity in chronic pain patients. Indeed, studies of the brain's default mode network and other resting state networks suggest long-lasting functional brain changes related to the presence of chronic pain^{379, 384-387}. Non-neural components of the central nervous system also appear to be affected with chronic low back pain patients showing

enhanced microglial activation relative to pain-free controls³⁸⁸. Collectively, these findings highlight the broad array of structural and functional changes that appear to characterize many chronic pain conditions.

It is important to note that many of the psychosocial factors and, more specifically, pain-specific cognitive and affective processes, have strong associations with functional brain responses to pain. A growing number of neuroimaging studies have suggested that variability in catastrophizing is associated with individual differences in the functional brain response to a variety of stimuli, as well as potential alterations in brain morphology^{359, 384, 387, 389–395}. Higher levels of catastrophizing are related to enhanced fMRI responses to calibrated noxious stimuli in areas such as anterior insular cortex among both fibromyalgia patients³⁹⁶, and pain-free adults³⁹⁷. Other neuroimaging work has highlighted maladaptive activity or connectivity in the amygdala^{396, 398, 399}. A study in patients with chronic low back pain reported that patients had exaggerated and abnormal amygdala connectivity with central executive network structures, and this connectivity was most exaggerated in patients with the greatest pain catastrophizing³⁸⁵. Interestingly, a very recent study in patients with fibromyalgia reported reduced connectivity between periaqueductal gray (a critical hub for pain-inhibitory circuits) and prefrontal regions, and the patients highest in catastrophizing showed the highest degree of anti-correlation between these regions³⁹⁵. To date, the vast majority of mechanism-driven FM research has involved adults. However, negative affective responses to pain, and pain-related catastrophizing are as prominent in adolescent patients^{400–402}, while very few functional neuroimaging studies have examined the neural underpinnings of pediatric chronic pain, this is likely to be a leading area of work in the years to come⁴⁰³.

CONCLUSIONS

The presence of chronic pain has a number of psychosocial and functional consequences in multiple areas of functioning - cognition, emotion, and behavior. Since chronic pain persists overtime, each of these areas will, in turn, affect the experience and reporting of pain and related symptoms. Moreover, the context in which individuals reside will also have an important role. As the tenets of the biopsychosocial model suggest, a broad range of psychosocial variables act as risk or resilience factors, influencing the probability of developing a chronic pain condition, the severity of pain-related consequences such as disability, and the success or failure of various pain treatments. As we describe in this review, it is important to acknowledge that psychological and social factors are not solely secondary reactions to persistent pain; rather, they form an interactive complex of biopsychosocial processes that characterizes chronic pain. Across biological diagnoses, this diverse array of psychological, social and contextual factors need to be considered in their roles as potential risk factors, protective factors, and process variables within the dynamic system of forces that constitutes a chronic pain condition.

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Highlights

- We review psychosocial factors that influence the experience of chronic pain.
- We first review the history of the biopsychosocial model of pain.
- Then we discuss the impact of general psychosocial variables.
- Finally, we identify pain-specific variables including catastrophizing and coping.

Table 1.

Biopsychosocial constructs influencing the pain experience

General psychosocial constructs	
<i>Affect</i>	Anxiety Depression Negative affect Optimism Positive Affect
<i>Trauma</i>	Physical trauma Traumatic brain injury Psychological trauma Post-traumatic stress disorder
<i>Social/interpersonal factors</i>	Social environment Social interactions Social support Therapeutic relationship
<i>Sex-related disparities</i>	Biological mechanisms Endogenous opioid system Hormones Psychosocial mechanisms Affective distress Catastrophizing Coping Gender roles
<i>Race-related disparities</i>	Biological mechanisms Central pain-inhibitory mechanisms Sympathetic nervous system response Psychosocial mechanisms Affective distress Appraisals Catastrophizing Coping Expectations Socioeconomic strain
Pain-specific psychosocial constructs	
	<i>Altered central nervous system pathways</i>
	<i>Catastrophizing</i>
	<i>Coping</i>
	<i>Expectations</i>
	<i>Self efficacy</i>