Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript

HHS Public Access

Author manuscript

Prog Neuropsychopharmacol Biol Psychiatry. Author manuscript; available in PMC 2018 December 20.

Published in final edited form as:

Prog Neuropsychopharmacol Biol Psychiatry. 2018 December 20; 87(Pt B): 168–182. doi:10.1016/j.pnpbp.2018.01.017.

Evaluating Psychosocial Contributions to Chronic Pain Outcomes

S.M. Meints, Ph.D.[1] and **R.R. Edwards, Ph.D.**[1]

[1]Department of Anesthesiology Pain Management Center Brigham and Women's Hospital Harvard Medical School Chestnut Hill, MA, USA

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

Corresponding Author: Samantha M. Meints, 850 Boylston St., Ste. 209, Chestnut Hill, MA 02467, Phone: 857-307-5405, smeints@bwh.harvard.edu.

Author Contributions:
All authors have made substantial contributions to the conception and design of this manuscript, have assisted in drafting and revising the manuscript, and have read and approved the final manuscript.

Declarations of interest: None.
Funding: None.

Publisher's Disclaimer: This is a PDF file of an unedited manuscript that has been accepted for publication. As a service to our customers we are providing this early version of the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting proof before it is published in its final form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

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., distress[20–22], 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

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

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 affect [33–35]. 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 anxiety [21]. 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 disability [47], healthcare costs [48, 49], mortality [50–53], 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

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

for degenerative spondylolisthesis, patients were followed post-surgically for 2 years to determine the predictors of functional outcomes [56]. 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 ⅓ less likely to return to work and, among those who did return, took nearly twice as long post-operatively to begin working again [56].

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 catastrophizing [57]. 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 movement[62]. 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 affect [58].

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 ones[63]. There is preliminary evidence suggesting that PAIs can be effective in the treatment of chronic pain. In one study by Hausmann and colleagues [64], 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 condition [65].

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 pain[23]. 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 adulthood [23]. Furthermore, individuals with childhood reports of distressing events such as

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

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 status [67]

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 children [67]. 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 outcomes [77]. 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 function [78], affects approximately 1.4 million people in the US each year [79]. According to a metaanalysis of 23 studies, more than 50% of people diagnosed with TBI report chronic pain complaints [80], likely due to the associated physical injuries at the time of the TBI[81]. 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 support [86]. 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

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

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 amputation [85]. 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 leukemia [92], 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-analysis [97]. 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 colleagues[101] 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 surgery [115], and to the child's disability [116]. 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 period [123]. 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 health [91]. Patients with partners showing avoidant and anxious attachment styles are more likely to

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

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 health [128], for reduced engagement in physical activity [129], and for less treatment- related improvement in affective outcomes among patients participating in a multidisciplinary treatment program [130].

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 later [133]. 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 colleagues [134] 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 disability [135]. 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 intervention [139].

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 outcomes [140]. 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,

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

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 regimens [147].

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, Unruh [155] 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 men [161]. 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 reflexes [165] and show increased pupil dilation [166] as well as amplified cerebral activation in response to pain [167].

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 faciliatory 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

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

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 pain [192] 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 life [193]. 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 responses [199]. 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 pain [200].

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 sensitivity [208]. 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

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

disparities in pain, Anderson and colleagues[215] 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 colleagues [228] 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 inhibition [229]. 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 perception [230].

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 sensitivity [236]. 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

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

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 colleagues [244] 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 Americans [247]. 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 stimuli [251]. 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 distress [255]. 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 strategies [256]. 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

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

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 complains, 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 outcomes [302]. Indeed, when controlling for depression and anxiety, catastrophizing remains significantly correlated with return to work [303–305], pain related disability [306], pain intensity [307], and pain tolerance [308]. 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 pain [13].

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 analgesics [313], oral analgesics [309], and psychosocial treatments[314, 315]. In a study aimed at identifying predictors of treatment responders for persistent orofacial pain, researchers found that

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

baseline levels of catastrophizing were more than one standard deviation higher in the non-responder group than for responders [316]. 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 treatment [317]. Recent reviews have identified catastrophizing as a key phenotypic factor to assess in trials of analgesic treatments in patients with chronic pain [11].

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 decades [322] and its effects can last for months or years after treatment [323]. 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 disability [328]. 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 disability [330]. 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 catastrophizing [329]. 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

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

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 support [103], and perceived critical responses to pain [106] have been observed. Further, both solicitous and punishing responses to pain are associated with higher pain intensity [108] 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 dyspareunia [344]. 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 factorfrom 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

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

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 factorfor children, adolescents, and adults with chronic pain [270], 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 pain [372]. Similarly, self-efficacy mediates the relationship between pain and performance of valued activities in patients with rheumatoid arthritis [373]. 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 disability [374]

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 disability [315]. 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-efficacy [376], 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 pain [381–383]. 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

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

enhanced microglial activation relative to pain-free controls [388]. 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 patients [396], and pain-free adults [397]. 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 catastrophizing [385]. 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 regions [395]. 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 come [403].

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.

References

1. Engel GL. The need for a new medical model: a challenge for biomedicine. Science 1977;196:129–136. [PubMed: 847460]
2. Fordyce WE. Behavioral methods in chronic pain and illness. St. Louis: Mosby, 1976.

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

3. Fordyce W Behavioral concepts in chronic pain and illness In: Davidson P, ed. The Behavioral Management of Anxiety, Depression, and Pain. New York: Bruner/Mazel, 1979: 147–188.
4. Loeser JD, Black RG. A taxonomy of pain. Pain 1975;1:81–84. [PubMed: 1235978]
5. Loeser JD. The history of pain project. Pain 1997;73:1–2. [PubMed: 9414050]
6. Loeser JD. Pain and suffering. Clin J Pain 2000;16:S2–S6. [PubMed: 10870733]
7. Gatchel RJ, McGeary DD, McGeary CA, Lippe B. Interdisciplinary chronic pain management: past, present, and future. Am Psychol 2014;69:119–130. [PubMed: 24547798]
8. Jensen MP. Psychosocial approaches to pain management: an organizational framework. Pain 2011;152:717–725. [PubMed: 21168972]
9. Jimenez N, Galinkin JL. Personalizing pediatric pain medicine: using population-specific pharmacogenetics, genomics, and other -omics approaches to predict response. Anesth Analg 2015;121:183–187. [PubMed: 26086515]
10. Bruehl S Personalized pain medicine: pipe dream or reality? Anesthesiology 2015;122:967–968. [PubMed: 25751235]
11. Edwards RR, Dworkin RH, Turk DC, et al. Patient phenotyping in clinical trials of chronic pain treatments: IMMPACT recommendations. Pain 2016;157:1851–1871. [PubMed: 27152687]
12. Jensen MP, Turk DC. Contributions of psychology to the understanding and treatment of people with chronic pain: why it matters to ALL psychologists. Am Psychol 2014;69:105–118. [PubMed: 24547797]
13. Edwards RR, Cahalan C, Mensing G, Smith M, Haythornthwaite JA. Pain, catastrophizing, and depression in the rheumatic diseases. Nat Rev Rheumatol 2011;7:216–224. [PubMed: 21283147]
14. Linton SJ, Nicholas MK, Macdonald S, et al. The role of depression and catastrophizing in musculoskeletal pain. Eur J Pain 2010.
15. Nicholas MK, Linton SJ, Watson PJ, Main CJ. Early identification and management of psychological risk factors ("yellow flags") in patients with low back pain: a reappraisal. Phys Ther 2011;91:737–753. [PubMed: 21451099]
16. Bair MJ, Robinson RL, Katon W, Kroenke K. Depression and pain comorbidity: a literature review. Arch Intern Med 2003;163:2433–2445. [PubMed: 14609780]
17. Asmundson GJ, Katz J. Understanding the co-occurrence of anxiety disorders and chronic pain: state-of-the-art. Depress Anxiety 2009;26:888–901. [PubMed: 19691031]
18. Williams DA. The importance of psychological assessment in chronic pain. Curr Opin Urol 2013;23:554–559. [PubMed: 24080806]
19. Wright MOD, Masten AS, Narayan AJ. Resilience Processes in Development: Four Waves of Research on Positive Adaptation in the Context of Adversity In: Goldstein S, Brooks RB, eds. Handbook of Resilience in Children. Boston, MA: Springer US, 2013: 15–37.
20. Jackson T, Tian P, Wang Y, Iezzi T, Xie W. Toward Identifying Moderators of Associations Between Presurgery Emotional Distress and Postoperative Pain Outcomes: A Meta-Analysis of Longitudinal Studies. J Pain 2016;17:874–888. [PubMed: 27163836]
21. Lerman SF, Rudich Z, Brill S, Shalev H, Shahar G. Longitudinal associations between depression, anxiety, pain, and pain-related disability in chronic pain patients. Psychosom Med 2015;77:333–341. [PubMed: 25849129]
22. Noyman-Veksler G, Lerman SF, Joiner TE, et al. Role of Pain-Based Catastrophizing in Pain, Disability, Distress, and Suicidal Ideation. Psychiatry 2017;80:155–170. [PubMed: 28767335]
23. Afari N, Ahumada SM, Wright LJ, et al. Psychological trauma and functional somatic syndromes: a systematic review and meta-analysis. Psychosom Med 2014;76:2–11. [PubMed: 24336429]
24. Brennstuhl MJ, Tarquinio C, Montel S. Chronic Pain and PTSD: Evolving Views on Their Comorbidity. Perspect Psychiatr Care 2014.
25. Crombez G, Eccleston C, Van Damme S, Vlaeyen JW, Karoly P. Fear-avoidance model of chronic pain: the next generation. Clin J Pain 2012;28:475–483. [PubMed: 22673479]
26. Somers TJ, Wren AA, Shelby RA. The context of pain in arthritis: self-efficacy for managing pain and other symptoms. Curr Pain Headache Rep 2012;16:502–508. [PubMed: 23054977]

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

27. Wright LJ, Zautra AJ, Going S. Adaptation to early knee osteoarthritis: the role of risk, resilience, and disease severity on pain and physical functioning. Ann Behav Med 2008;36:70–80. [PubMed: 18716855]
28. Bergbom S, Boersma K, Linton SJ. Both early and late changes in psychological variables relate to treatment outcome for musculoskeletal pain patients at risk for disability. Behav Res Ther 2012;50:726–734. [PubMed: 23000845]
29. Pincus T, Santos R, Breen A, Burton AK, Underwood M. A review and proposal for a core set of factors for prospective cohorts in low back pain: a consensus statement. Arthritis Rheum 2008;59:14–24. [PubMed: 18163411]
30. Burns JW, Day MA, Thorn BE. Is reduction in pain catastrophizing a therapeutic mechanism specific to cognitive-behavioral therapy for chronic pain? Transl Behav Med 2012;2:22–29. [PubMed: 24073095]
31. Thorn BE, Burns JW. Common and specific treatment mechanisms in psychosocial pain interventions: the need for a new research agenda. Pain 2011;152:705–706. [PubMed: 21227586]
32. Edwards RR, Dworkin RH, Sullivan MD, Turk DC, Wasan AD. The Role of Psychosocial Processes in the Development and Maintenance of Chronic Pain. J Pain 2016;17:T70–92. [PubMed: 27586832]
33. Turk DC, Fillingim RB, Ohrbach R, Patel KV. Assessment of Psychosocial and Functional Impact of Chronic Pain. J Pain 2016;17:T21–49. [PubMed: 27586830]
34. Burke AL, Mathias JL, Denson LA. Psychological functioning of people living with chronic pain: A meta-analytic review. Br J Clin Psychol 2015.
35. Howe CQ, Robinson JP, Sullivan MD. Psychiatric and Psychological Perspectives on Chronic Pain. Phys Med Rehabil Clin N Am 2015;26:283–300. [PubMed: 25952065]
36. Fillingim RB, Ohrbach R, Greenspan JD, et al. Psychological factors associated with development of TMD: the OPPERA prospective cohort study. J Pain 2013;14:T75–T90. [PubMed: 24275225]
37. Diatchenko L, Fillingim RB, Smith SB, Maixner W. The phenotypic and genetic signatures of common musculoskeletal pain conditions. Nat Rev Rheumatol 2013;9:340–350. [PubMed: 23545734]
38. Huijnen IP, Rusu AC, Scholich S, Meloto CB, Diatchenko L. Subgrouping of low back pain patients for targeting treatments: evidence from genetic, psychological, and activity-related behavioral approaches. Clin J Pain 2015;31:123–132. [PubMed: 24681821]
39. Clauw DJ. Fibromyalgia and Related Conditions. Mayo Clin Proc 2015;90:680–692. [PubMed: 25939940]
40. Ip HY, Abrishami A, Peng PW, Wong J, Chung F. Predictors of postoperative pain and analgesic consumption: a qualitative systematic review. Anesthesiology 2009;111:657–677. [PubMed: 19672167]
41. Pincus T, Burton AK, Vogel S, Field AP. A systematic review of psychological factors as predictors of chronicity/disability in prospective cohorts of low back pain. Spine 2002;27:E109–E120. [PubMed: 11880847]
42. Shipton EA. The transition from acute to chronic post surgical pain. Anaesth Intensive Care 2011;39:824–836. [PubMed: 21970126]
43. Hall AM, Kamper SJ, Maher CG, Latimer J, Ferreira ML, Nicholas MK. Symptoms of depression and stress mediate the effect of pain on disability. Pain 2011;152:1044–1051. [PubMed: 21306826]
44. Lee H, Hubscher M, Moseley GL, et al. How does pain lead to disability? A systematic review and meta-analysis of mediation studies in people with back and neck pain. Pain 2015;156:988–997. [PubMed: 25760473]
45. Ross C, Juraskova I, Lee H, et al. Psychological distress mediates the relationship between pain and disability in hand or wrist fractures. J Pain 2015.
46. Edwards RR, Klick B, Buenaver L, et al. Symptoms of distress as prospective predictors of pain-related sciatica treatment outcomes. Pain 2007;130:47–55. [PubMed: 17156925]
47. Kenardy J, Heron-Delaney M, Warren J, Brown EA. Effect of mental health on long-term disability after a road traffic crash: results from the UQ SuPPORT study. Arch Phys Med Rehabil 2015;96:410–417. [PubMed: 25450121]

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

48. Baumeister H, Knecht A, Hutter N. Direct and indirect costs in persons with chronic back pain and comorbid mental disorders-a systematic review. J Psychosom Res 2012;73:79–85. [PubMed: 22789408]
49. Parsons B, Schaefer C, Mann R, et al. Economic and humanistic burden of post-trauma and post-surgical neuropathic pain among adults in the United States. J Pain Res2013;6:459–469.
50. Kadam UT, Thomas E, Croft PR. Is chronic widespread pain a predictor of all-cause morbidity? A 3 year prospective population based study in family practice. J Rheumatol 2005;32:1341–1348. [PubMed: 15996075]
51. Macfarlane GJ, McBeth J, Silman AJ. Widespread body pain and mortality: prospective population based study. BMJ 2001;323:662–665. [PubMed: 11566829]
52. McBeth J, Silman AJ, Macfarlane GJ. Association of widespread body pain with an increased risk of cancer and reduced cancer survival: a prospective, population-based study. Arthritis Rheum 2003;48:1686–1692. [PubMed: 12794837]
53. Smith D, Wilkie R, Uthman O, Jordan JL, McBeth J. Chronic pain and mortality: a systematic review. PLoS One 2014;9:e99048. [PubMed: 24901358]
54. Ilgen MA, Kleinberg F, Ignacio RV, et al. Noncancer pain conditions and risk of suicide. JAMA Psychiatry 2013;70:692–697. [PubMed: 23699975]
55. Hassett AL, Aquino JK, llgen MA. The risk of suicide mortality in chronic pain patients. Curr Pain Headache Rep 2014;18:436. [PubMed: 24952608]
56. Parker SL, Godil SS, Zuckerman SL, Mendenhall SK, Devin CJ, McGirt MJ. Extent of preoperative depression is associated with return to work after lumbar fusion for spondylolisthesis. World Neurosurg 2015;83:608–613. [PubMed: 25527877]
57. Hanssen MM, Peters ML, Vlaeyen JW, Meevissen YM, Vancleef LM. Optimism lowers pain: evidence of the causal status and underlying mechanisms. PAIN® 2013;154:53–58. [PubMed: 23084002]
58. Hassett AL, Finan PH. The role of resilience in the clinical management of chronic pain. Current pain and headache reports 2016;20:39. [PubMed: 27115770]
59. Finan PH, Quartana PJ, Smith MT. Positive and negative affect dimensions in chronic knee osteoarthritis: effects on clinical and laboratory pain. Psychosomatic Medicine 2013;75:463–470. [PubMed: 23697467]
60. Strand EB, Kerns RD, Christie A, Haavik-Nilsen K, Klokkerud M, Finset A. Higher levels of pain readiness to change and more positive affect reduce pain reports-a weekly assessment study on arthritis patients. PAIN 2007;127:204–213. [PubMed: 16997472]
61. Litt MD, Shafer D, Napolitano C. Momentary mood and coping processes in TMD pain. Health Psychology 2004;23:354. [PubMed: 15264971]
62. Meulders A, Meulders M, Vlaeyen JW. Positive affect protects against deficient safety learning during extinction of fear of movement-related pain in healthy individuals scoring relatively high on trait anxiety. The Journal of Pain 2014;15:632–644. [PubMed: 24650796]
63. Sin NL, Lyubomirsky S. Enhancing well-being and alleviating depressive symptoms with positive psychology interventions: A practice-friendly meta-analysis. Journal of Clinical Psychology 2009;65:467–487. [PubMed: 19301241]
64. Hausmann LR, Parks A, Youk AO, Kwoh CK. Reduction of bodily pain in response to an online positive activities intervention. The Journal of Pain 2014;15:560–567. [PubMed: 24568751]
65. Müller R, Gertz KJ, Molton IR, et al. Effects of a tailored positive psychology intervention on well-being and pain in individuals with chronic pain and a physical disability: a feasibility trial. The Clinical Journal of Pain 2016;32:32–44. [PubMed: 25724020]
66. Burke NN, Finn DP, McGuire BE, Roche M. Psychological stress in early life as a predisposing factor for the development of chronic pain: Clinical and preclinical evidence and neurobiological mechanisms. Journal of neuroscience research 2017;95:1257–1270. [PubMed: 27402412]
67. Jones GT, Power C, Macfarlane GJ. Adverse events in childhood and chronic widespread pain in adult life: Results from the 1958 British Birth Cohort Study. Pain 2009;143:92–96. [PubMed: 19304391]

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

68. McBeth J, Morris S, Benjamin S, Silman AJ, Macfarlane GJ. Associations between adverse events in childhood and chronic widespread pain in adulthood: are they explained by differential recall? J Rheumatol 2001;28:2305–2309. [PubMed: 11669174]
69. Romans S, Belaise C, Martin J, Morris E, Raffi A. Childhood Abuse and Later Medical Disorders in Women. an epidemiological study. Psychother Psychosom 2002;71:141–150. [PubMed: 12021556]
70. Moeller-Bertram T, Keltner J, Strigo IA. Pain and post traumatic stress disorder - review of clinical and experimental evidence. Neuropharmacology 2012;62:586–597. [PubMed: 21586297]
71. Jenewein J, Wittmann L, Moergeli H, Creutzig J, Schnyder U. Mutual influence of posttraumatic stress disorder symptoms and chronic pain among injured accident survivors: a longitudinal study. J Trauma Stress 2009;22:540–548. [PubMed: 19924822]
72. Jenewein J, Moergeli H, Wittmann L, Buchi S, Kraemer B, Schnyder U. Development of chronic pain following severe accidental injury. Results of a 3-year follow-up study. J Psychosom Res 2009;66:119–126. [PubMed: 19154854]
73. Wuest J, Ford-Gilboe M, Merritt-Gray M, et al. Abuse-related injury and symptoms of posttraumatic stress disorder as mechanisms of chronic pain in survivors of intimate partner violence. Pain Med 2009;10:739–747. [PubMed: 19453953]
74. Wuest J, Ford-Gilboe M, Merritt-Gray M, et al. Pathways of chronic pain in survivors of intimate partner violence. J Womens Health (Larchmt) 2010;19:1665–1674. [PubMed: 20718626]
75. Lang AJ, Laffaye C, Satz LE, et al. Relationships among childhood maltreatment, PTSD, and health in female veterans in primary care. Child Abuse Negl 2006;30:1281–1292. [PubMed: 17116330]
76. Powers A, Fani N, Pallos A, Stevens J, Ressler KJ, Bradley B. Childhood abuse and the experience of pain in adulthood: the mediating effects of PTSD and emotion dysregulation on pain levels and pain-related functional impairment. Psychosomatics 2014;55:491–499. [PubMed: 24360527]
77. Raphael KG, Widom CS. Post-traumatic stress disorder moderates the relation between documented childhood victimization and pain 30 years later. Pain 2011;152:163–169. [PubMed: 21050659]
78. Menon DK, Schwab K, Wright DW, Maas AI. Position statement: definition of traumatic brain injury. Archives of Physical Medicine and Rehabilitation 2010;91:1637–1640. [PubMed: 21044706]
79. Langlois J, Rutland-Brown W, Thomas K. Traumatic Brain Injury in the United States: Emergency Department Visits, Hospitalizations, and Deaths. Atlanta: Division of Injury and Disability Outcomes and Programs. National Center for Injury Prevention and Control CDC 2004.
80. Nampiaparampil DE. Prevalence of chronic pain after traumatic brain injury: a systematic review. JAMA 2008;300:711–719. [PubMed: 18698069]
81. Lahz S, Bryant RA. Incidence of chronic pain following traumatic brain injury. Archives of Physical Medicine and Rehabilitation 1996;77:889–891. [PubMed: 8822679]
82. Khoury S, Benavides R. Pain with traumatic brain injury and psychological disorders. Progress in Neuro-Psychopharmacology and Biological Psychiatry 2017.
83. Stein MB, McAllister TW. Exploring the convergence of posttraumatic stress disorder and mild traumatic brain injury. American Journal of Psychiatry 2009;166:768–776. [PubMed: 19448186]
84. George E, Elman I, Becerra L, Berg S, Borsook D. Pain in an era of armed conflicts: Prevention and treatment for warfighters and civilian casualties. Prog Neurobiol 2016;141:25–44. [PubMed: 27084355]
85. Jensen MP, Moore MR, Bockow TB, Ehde DM, Engel JM. Psychosocial factors and adjustment to chronic pain in persons with physical disabilities: a systematic review. Arch Phys Med Rehabil 2011;92:146–160. [PubMed: 21187217]
86. Cohen S, Wills TA. Stress, social support, and the buffering hypothesis. Psychologicl Bulletin 1985;98:310–357.
87. Leonard MT, Cano A, Johansen AB. Chronic pain in a couples context: a review and integration of theoretical models and empirical evidence. J Pain 2006;7:377–390. [PubMed: 16750794]

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

88. Gatchel RJ, Peng YB, Peters ML, Fuchs PN, Turk DC. The biopsychosocial approach to chronic pain: scientific advances and future directions. Psychol Bull 2007;133:581–624. [PubMed: 17592957]

89. Helmhout PH, Staal JB, Heymans MW, Harts CC, Hendriks EJ, de Bie RA. Prognostic factors for perceived recovery or functional improvement in non-specific low back pain: secondary analyses of three randomized clinical trials. Eur Spine J 2010;19:650–659. [PubMed: 20035358]

90. Schreiber KL, Martel MO, Shnol H, et al. Persistent pain in postmastectomy patients: comparison of psychophysical, medical, surgical, and psychosocial characteristics between patients with and without pain. Pain 2013;154:660–668. [PubMed: 23290256]

91. Novy DM, Aigner Cj. The biopsychosocial model in cancer pain. Curr Opin Support Palliat Care 2014;8:117–123. [PubMed: 24690764]

92. Morrison EJ, Flynn JM, Jones J, Byrd JC, Andersen BL. Individual differences in physical symptom burden and psychological responses in individuals with chronic lymphocytic leukemia. Ann Hematol 2016;95:1989–1997. [PubMed: 27539615]

93. Badr H, Milbury K. Associations between depression, pain behaviors, and partner responses to pain in metastatic breast cancer. Pain 2011;152:2596–2604. [PubMed: 21889263]

94. Badr H, Shen MJ. Pain catastrophizing, pain intensity, and dyadic adjustment influence patient and partner depression in metastatic breast cancer. Clin J Pain 2014;30:923–933. [PubMed: 24402001]

95. Leach CR, Troeschel AN, Wiatrek D, et al. Preparedness and Cancer-Related Symptom Management among Cancer Survivors in the First Year Post-Treatment. Ann Behav Med 2017;51:587–598. [PubMed: 28224276]

96. Canoui-Poitrine F, Reinald N, Laurent M, et al. Geriatric assessment findings independently associated with clinical depression in 1092 older patients with cancer: the ELCAPA Cohort Study. Psychooncology 2016;25:104–111. [PubMed: 26123351]

97. Johannsen M, Frederiksen Y, Jensen AB, Zachariae R. Psychosocial predictors of posttreatment pain after nonmetastatic breast cancer treatment: a systematic review and metaanalysis of prospective studies. J Pain Res 2018;11:23–36. [PubMed: 29317846]

98. Roberts MH, Klatzkin RR, Mechlin B. Social Support Attenuates Physiological Stress Responses and Experimental Pain Sensitivity to Cold Pressor Pain. Ann Behav Med 2015;49:557–569. [PubMed: 25623896]

99. Eisenberger NI, Master SL, Inagaki TK, et al. Attachment figures activate a safety signal- related neural region and reduce pain experience. Proc Natl Acad Sci U S A 2011;108:11721–11726. [PubMed: 21709271]

100. Inagaki TK, Eisenberger NI. Neural correlates of giving support to a loved one. Psychosom Med 2012;74:3–7. [PubMed: 22071630]

101. Guillory J, Chang P, Henderson CR, Jr, et al. Piloting a text message-based social support intervention for patients with chronic pain: establishing feasibility and preliminary efficacy. The Clinical Journal of Pain 2015;31:548. [PubMed: 25565587]

102. Martire LM, Schulz R, Helgeson VS, Small BJ, Saghafi EM. Review and meta-analysis of couple-oriented interventions for chronic illness. Ann Behav Med 2010;40:325–342. [PubMed: 20697859]

103. Keefe FJ, Lipkus I, Lefebvre JC, et al. The social context of gastrointestinal cancer pain: a preliminary study examining the relation of patient pain catastrophizing to patient perceptions of social support and caregiver stress and negative responses. Pain 2003;103:151–156. [PubMed: 12749969]

104. Keefe FJ, Ahles TA, Sutton L, et al. Partner-guided cancer pain management at the end of life: a preliminary study. J Pain Symptom Manage 2005;29:263–272. [PubMed: 15781177]

105. Fordyce WE, Fowler RS, Lehmann JF, DeLateur B, Sand PL, Trieschmann RB. Operant conditioning in the treatment of chronic pain. Arch Phys Med Rehabil 1973;54:399–408. [PubMed: 4729785]

106. Boothby JL, Thorn BE, Overduin LY, Ward LC. Catastrophizing and perceived partner responses to pain. Pain 2004;109:500–506. [PubMed: 15157712]

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

107. Jensen MP, Ehde DM, Hoffman AJ, Patterson DR, Czerniecki JM, Robinson LR. Cognitions, coping and social environment predict adjustment to phantom limb pain. Pain 2002;95:133–142. [PubMed: 11790476]
108. Giardino ND, Jensen MP, Turner JA, Ehde DM, Cardenas DD. Social environment moderates the association between catastrophizing and pain among persons with a spinal cord injury. Pain 2003;106:19–25. [PubMed: 14581106]
109. Hanley MA, Jensen MP, Ehde DM, Hoffman AJ, Patterson DR, Robinson LR. Psychosocial predictors of long-term adjustment to lower-limb amputation and phantom limb pain. Disabil Rehabil 2004;26:882–893. [PubMed: 15497917]
110. Palermo TM, Holley AL. The importance of the family environment in pediatric chronic pain. JAMA Pediatr 2013;167:93–94. [PubMed: 23403868]
111. Palermo TM, Valrie CR, Karlson CW. Family and parent influences on pediatric chronic pain: a developmental perspective. Am Psychol 2014;69:142–152. [PubMed: 24547800]
112. Rabbitts JA, Groenewald CB, Tai GG, Palermo TM. Presurgical psychosocial predictors of acute postsurgical pain and quality of life in children undergoing major surgery. J Pain 2015;16:226–234. [PubMed: 25540939]
113. Rabbitts JA, Fisher E, Rosenbloom BN, Palermo TM. Prevalence and Predictors of Chronic Postsurgical Pain in Children: A Systematic Review and Meta-Analysis. J Pain 2017;18:605–614. [PubMed: 28363861]
114. Rhee H Physical symptoms in children and adolescents. Annu Rev Nurs Res 2003;21:95–121. [PubMed: 12858694]
115. Noel M, Rabbitts JA, Tai GG, Palermo TM. Remembering pain after surgery: a longitudinal examination of the role of pain catastrophizing in children's and parents' recall. Pain 2015;156:800–808. [PubMed: 25630028]
116. Hechler T, Vervoort T, Hamann M, et al. Parental catastrophizing about their child's chronic pain: are mothers and fathers different? Eur J Pain 2011;15:515–519. [PubMed: 20971665]
117. Claar RL, Simons LE, Logan DE. Parental response to children's pain: the moderating impact of children's emotional distress on symptoms and disability. Pain 2008;138:172–179. [PubMed: 18221837]
118. Simons LE, Sieberg CB, Claar RL. Anxiety and impairment in a large sample of children and adolescents with chronic pain. Pain Res Manag 2012;17:93–97. [PubMed: 22518371]
119. Page MG, Campbell F, Isaac L, Stinson J, Katz J. Parental risk factors for the development of pediatric acute and chronic postsurgical pain: a longitudinal study. J Pain Res 2013;6:727–741. [PubMed: 24109194]
120. Burns JW, Peterson KM, Smith DA, et al. Temporal associations between spouse criticism/hostility and pain among patients with chronic pain: a within-couple daily diary study. Pain 2013;154:2715–2721. [PubMed: 23932908]
121. Geisser ME, Cano A, Leonard MT. Factors associated with marital satisfaction and mood among spouses of persons with chronic back pain. J Pain 2005;6:518–525. [PubMed: 16084466]
122. Pence L, Cano A, Thorn B, Ward LC. Perceived spouse responses to pain: the level of agreement in couple dyads and the role of catastrophizing, marital satisfaction, and depression. J Behav Med 2006;29:511–522. [PubMed: 16960758]
123. Lam M, Lehman AJ, Puterman E, DeLongis A. Spouse depression and disease course among persons with rheumatoid arthritis. Arthritis Rheum 2009;61:1011–1017. [PubMed: 19644902]
124. Gauthier LR, Rodin G, Zimmermann C, et al. The communal coping model and cancer pain: the roles of catastrophizing and attachment style. J Pain 2012;13:1258–1268. [PubMed: 23182231]
125. Thorn BE, Keefe FJ, Anderson T. The communal coping model and interpersonal context: problems or process? Pain 2004;110:505–507. [PubMed: 15288389]
126. Porter LS, Davis D, Keefe FJ. Attachment and pain: recent findings and future directions. Pain 2007;128:195–198. [PubMed: 17321050]
127. Porter LS, Keefe FJ, Davis D, Rumble M, Scipio C, Garst J. Attachment styles in patients with lung cancer and their spouses: associations with patient and spouse adjustment. Support Care Cancer 2012;20:2459–2466. [PubMed: 22246596]

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

128. Laird KT, Preacher KJ, Walker LS. Attachment and adjustment in adolescents and young adults with a history of pediatric functional abdominal pain. Clin J Pain 2015;31:152–158. [PubMed: 24751542]
129. Andrews NE, Meredith PJ, Strong J, Donohue GF. Adult attachment and approaches to activity engagement in chronic pain. Pain Res Manag 2014;19:317–327. [PubMed: 25337857]
130. Kowal J, McWilliams LA, Peloquin K, Wilson KG, Henderson PR, Fergusson DA. Attachment insecurity predicts responses to an interdisciplinary chronic pain rehabilitation program. J Behav Med 2015;38:518–526. [PubMed: 25716120]
131. Melloh M, Elfering A, Stanton TR, et al. Who is likely to develop persistent low back pain? A longitudinal analysis of prognostic occupational factors. Work 2013;46:297–311. [PubMed: 24004738]
132. Melloh M, Elfering A, Chapple CM, et al. Prognostic occupational factors for persistent low back pain in primary care. Int Arch Occup Environ Health 2013;86:261–269. [PubMed: 22434236]
133. Li X, Gignac MA, Anis AH. Workplace, psychosocial factors, and depressive symptoms among working people with arthritis: a longitudinal study. J Rheumatol 2006;33:1849–1855. [PubMed: 16881094]
134. Anema JR, Schellart AJ, Cassidy JD, Loisel P, Veerman TJ, van der Beek AJ. Can cross country differences in return-to-work after chronic occupational back pain be explained? An exploratory analysis on disability policies in a six country cohort study. J Occup Rehabil 2009;19:419–426. [PubMed: 19760488]
135. McWilliams LA, Kowal J, Verrier MJ, Dick BD. Do Pain-Related Support Preferences Moderate Relationships Between Chronic Pain Patients' Reports of Support Received and Psychosocial Functioning? Pain Med 2017.
136. Keefe FJ, Caldwell DS, Baucom D, et al. Spouse-assisted coping skills training in the management of osteoarthritic knee pain. Arthritis Care Res 1996;9:279–291. [PubMed: 8997917]
137. Keefe FJ, Caldwell DS, Baucom D, et al. Spouse-assisted coping skills training in the management of knee pain in osteoarthritis: long-term followup results. Arthritis Care Res 1999;12:101–111. [PubMed: 10513498]
138. Keefe FJ, Blumenthal J, Baucom D, et al. Effects of spouse-assisted coping skills training and exercise training in patients with osteoarthritic knee pain: a randomized controlled study. Pain 2004;110:539–549. [PubMed: 15288394]
139. Abbasi M, Dehghani M, Keefe FJ, Jafari H, Behtash H, Shams J. Spouse-assisted training in pain coping skills and the outcome of multidisciplinary pain management for chronic low back pain treatment: a 1-year randomized controlled trial. Eur J Pain 2012;16:1033–1043. [PubMed: 22337646]
140. Martin DJ, Garske JP, Davis MK. Relation of the therapeutic alliance with outcome and other variables: a meta-analytic review. J Consult Clin Psychol 2000;68:438–450. [PubMed: 10883561]
141. Burns JW, Higdon LJ, Mullen JT, Lansky D, Wei JM. Relationships among patient hostility, anger expression, depression, and the working alliance in a work hardening program. Ann Behav Med 1999;21:77–82. [PubMed: 18425658]
142. Hall AM, Ferreira PH, Maher CG, Latimer J, Ferreira ML. The influence of the therapist- patient relationship on treatment outcome in physical rehabilitation: a systematic review. Phys Ther 2010;90:1099–1110. [PubMed: 20576715]
143. Hall AM, Ferreira ML, Clemson L, Ferreira P, Latimer J, Maher CG. Assessment of the therapeutic alliance in physical rehabilitation: a RASCH analysis. Disabil Rehabil 2012;34:257–266. [PubMed: 21999716]
144. Burns JW, Nielson WR, Jensen MP, Heapy A, Czlapinski R, Kerns RD. Does Change Occur for the Reasons We Think It Does? A Test of Specific Therapeutic Operations During Cognitive-Behavioral Treatment of Chronic Pain. Clin J Pain 2015;31:603–611. [PubMed: 25119513]
145. Burns JW, Nielson WR, Jensen MP, Heapy A, Czlapinski R, Kerns RD. Specific and general therapeutic mechanisms in cognitive behavioral treatment of chronic pain. J Consult Clin Psychol 2015;83:1–11. [PubMed: 24979313]

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

146. Barton JL, Imboden J, Graf J, Glidden D, Yelin EH, Schillinger D. Patient-physician discordance in assessments of global disease severity in rheumatoid arthritis. Arthritis Care Res (Hoboken) 2010;62:857–864. [PubMed: 20535797]

147. Haugli L, Strand E, Finset A. How do patients with rheumatic disease experience their relationship with their doctors? A qualitative study of experiences of stress and support in the doctor-patient relationship. Patient Educ Couns 2004;52:169–174. [PubMed: 15132522]

148. Fillingim RB. Sex, gender, and pain: women and men really are different. Curr Rev Pain 2000;4:24–30. [PubMed: 10998712]

149. Greenspan JD, Craft RM, LeResche L, et al. Studying sex and gender differences in pain and analgesia: a consensus report. Pain 2007;132 Suppl 1:S26–S45.

150. Bartley EJ, Fillingim RB. Sex differences in pain: a brief review of clinical and experimental findings. Br J Anaesth 2013;111:52–58. [PubMed: 23794645]

151. Berkley KJ, Holdcroft A. Sex and gender differences in pain In: Wall PD, Melzack R, eds. Textbook of Pain, 4 ed. Edinburgh: Churchill-Livingstone, 1999: 951–965.

152. Bingefors K, Isacson D. Epidemiology, co-morbidity, and impact on health-related quality of life of self-reported headache and musculoskeletal pain--a gender perspective. Eur J Pain 2004;8:435–450. [PubMed: 15324775]

153. Racine M, Tousignant-Laflamme Y, Kloda LA, Dion D, Dupuis G, Choiniere M. A systematic literature review of 10years of research on sex/gender and pain perception - Part 2: Do biopsychosocial factors alter pain sensitivity differently in women and men? Pain 2012;153:619–635. [PubMed: 22236999]

154. Hampton SB, Cavalier J, Langford R. The Influence of Race and Gender on Pain Management: A Systematic Literature Review. Pain Manag Nurs 2015;16:968–977. [PubMed: 26697821]

155. Unruh AM. Gender variations in clinical pain experience. Pain 1996;65:123–167. [PubMed: 8826503]

156. Cairns BE, Gazerani P. Sex-related differences in pain. Maturitas 2009;63:292–296. [PubMed: 19595525]

157. Wijnhoven HA, de Vet HC, Picavet HS. Prevalence of musculoskeletal disorders is systematically higher in women than in men. Clin J Pain 2006;22:717–724. [PubMed: 16988568]

158. Wijnhoven HA, de Vet HC, Picavet HS. Explaining sex differences in chronic musculoskeletal pain in a general population. Pain 2006;124:158–166. [PubMed: 16716517]

159. Shinal RM, Fillingim RB. Overview of orofacial pain: epidemiology and gender differences in orofacial pain. Dent Clin North Am 2007;51:1–18, v. [PubMed: 17185057]

160. Theis KA, Helmick CG, Hootman JM. Arthritis burden and impact are greater among U.S. women than men: intervention opportunities. J Womens Health (Larchmt) 2007;16:441–453. [PubMed: 17521246]

161. Riley JL, Robinson ME, Wise EA, Myers CD, Fillingim RB. Sex differences in the perception of noxious experimental stimuli: a meta-analysis. Pain 1998;74:181–187. [PubMed: 9520232]

162. Meints SM, Stout M, Abplanalp S, Hirsh AT. Pain-Related Rumination, But Not Magnification or Helplessness, Mediates Race and Sex Differences in Experimental Pain. J Pain 2017;18:332–339. [PubMed: 27908838]

163. Fillingim RB, Ness TJ. Sex-related hormonal influences on pain and analgesic responses. Neuroscience and Biobehavioral Reviews 2000;24:485–501. [PubMed: 10817845]

164. Fillingim RB. Sex, Gender, and Pain. Seattle: IASP Press, 2000.

165. Serrao M, Rossi P, Sandrini G, et al. Effects of diffuse noxious inhibitory controls on temporal summation of the RIII reflex in humans. Pain 2004;112:353–360. [PubMed: 15561391]

166. Ellermeier W, Westphal W. Gender differences in pain ratings and pupil reactions to painful pressure stimuli. Pain 1995;61:435–439. [PubMed: 7478686]

167. Paulson PE, Minoshima S, Morrow TJ, Casey KL. Gender differences in pain perception and patterns of cerebral activation during noxious heat stimulation in humans. Pain 1998;76:223–229. [PubMed: 9696477]

168. Woolf CJ. Central sensitization: implications for the diagnosis and treatment of pain. Pain 2011;152:S2–15. [PubMed: 20961685]

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

169. Staud R Abnormal endogenous pain modulation is a shared characteristic of many chronic pain conditions. Expert Rev Neurother 2012;12:577–585. [PubMed: 22550986]

170. Arendt-Nielsen L Central sensitization in humans: assessment and pharmacology. Handb Exp Pharmacol 2015;227:79–102. [PubMed: 25846615]

171. Fillingim RB, Maixner W, Kincaid S, Silva S. Sex differences in temporal summation but not sensory-discriminative processing of thermal pain. Pain 1998;75:121–127. [PubMed: 9539681]

172. Fillingim R, Maixner W, Kincaid S, Silva S. Sex differences in temporal summation but not sensory-discriminative processing of thermal pain. Pain 1998;75:121–127. [PubMed: 9539681]

173. Sarlani E, Greenspan JD. Gender differences in temporal summation of mechanically evoked pain. Pain 2002;97:163–169. [PubMed: 12031789]

174. Sarlani E, Grace EG, Reynolds MA, Greenspan JD. Sex differences in temporal summation of pain and aftersensations following repetitive noxious mechanical stimulation. Pain 2004;109:115–123. [PubMed: 15082133]

175. Yarnitsky D Conditioned pain modulation (the diffuse noxious inhibitory control-like effect): its relevance for acute and chronic pain states. Curr Opin Anaesthesiol 2010;23:611–615. [PubMed: 20543676]

176. van Wijk G, Veldhuijzen DS. Perspective on diffuse noxious inhibitory controls as a model of endogenous pain modulation in clinical pain syndromes. J Pain 2010;11:408–419. [PubMed: 20075013]

177. Ge HY, Madeleine P, Arendt-Nielsen L. Sex differences in temporal characteristics of descending inhibitory control: an evaluation using repeated bilateral experimental induction of muscle pain. Pain 2004;110:72–78. [PubMed: 15275754]

178. Granot M, Weissman-Fogel I, Crispel Y, et al. Determinants of endogenous analgesia magnitude in a diffuse noxious inhibitory control (DNIC) paradigm: Do conditioning stimulus painfulness, gender and personality variables matter? Pain 2007.

179. Edwards RR, Ness TJ, Weigent DA, Fillingim RB. Individual differences in diffuse noxious inhibitory controls (DNIC): association with clinical variables. Pain 2003;106:427–437. [PubMed: 14659526]

180. France CR, Suchowiecki S. A comparison of diffuse noxious inhibitory controls in men and women. Pain 1999;81:77–84. [PubMed: 10353495]

181. Tousignant-Laflamme Y, Page S, Goffaux P, Marchand S. An experimental model to measure excitatory and inhibitory pain mechanisms in humans. Brain Res 2008;1230:73–79. [PubMed: 18652808]

182. Lipton RB, Scher AI, Kolodner K, Liberman J, Steiner TJ, Stewart WF. Migraine in the United States: epidemiology and patterns of health care use. Neurology 2002;58:885–894. [PubMed: 11914403]

183. LeResche L, Saunders K, Von Korff MR, Barlow W, Dworkin SF. Use of exogenous hormones and risk of temporomandibular disorder pain. Pain 1997;69:153–160. [PubMed: 9060026]

184. LeResche L Gender considerations in the epidemiology of chronic pain In: Crombie IK, ed. Epidemiology of Pain. Seattle: IASP Press, 1999: 43–52.

185. Heitkemper MM, Jarrett M. Pattern of gastrointestinal and somatic symptoms across the menstrual cycle. Gastroenterology 1992;102:505–513. [PubMed: 1732122]

186. Heitkemper MM, Jarrett M. Gender differences and hormonal modulation in visceral pain. Curr Pain Headache Rep 2001;5:35–43. [PubMed: 11252136]

187. Heitkemper MM, Cain KC, Jarrett ME, Burr RL, Hertig V, Bond EF. Symptoms across the menstrual cycle in women with irritable bowel syndrome. Am J Gastroenterol 2003;98:420–430. [PubMed: 12591063]

188. Heitkemper MM, Cain KC, Jarrett ME, Burr RL, Crowell MD, Woods NF. Relationship of bloating to other GI and menstrual symptoms in women with irritable bowel syndrome. Dig Dis Sci 2004;49:88–95. [PubMed: 14992441]

189. LeResche L, Mancl L, Sherman JJ, Gandara B, Dworkin SF. Changes in temporomandibular pain and other symptoms across the menstrual cycle. Pain 2003;106:253–261. [PubMed: 14659508]

190. Hapidou EG, Rollman GB. Menstrual cycle modulation of tender points. Pain 1998;77:151–161. [PubMed: 9766833]

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

191. Gur A, Cevik R, Nas K, Colpan L, Sarac S. Cortisol and hypothalamic-pituitary-gonadal axis hormones in follicular-phase women with fibromyalgia and chronic fatigue syndrome and effect of depressive symptoms on these hormones. Arthritis Res Ther 2004;6:R232–R238. [PubMed: 15142269]
192. Zubieta JK, Smith YR, Bueller JA, et al. mu-opioid receptor-mediated antinociceptive responses differ in men and women. J Neurosci 2002;22:5100–5107. [PubMed: 12077205]
193. Myers CD, Riley JL, III, Robinson ME. Psychosocial contributions to sex-correlated differences in pain. Clin J Pain 2003;19:225–232. [PubMed: 12840616]
194. Robinson ME, Riley JL, III, Myers CD, et al. Gender role expectations of pain: relationship to sex differences in pain. J Pain 2001;2:251–257. [PubMed: 14622803]
195. Thorn BE, Clements KL, Ward LC, et al. Personality factors in the explanation of sex differences in pain catastrophizing and response to experimental pain. Clin J Pain 2004;20:275–282. [PubMed: 15322433]
196. Otto M, Dougher M. Sex differences and personality factors in responsivity to pain. Percept Mot Skills 1985;61:383–390. [PubMed: 4069906]
197. Myers CD, Robinson ME, Riley JL, III, Sheffield D. Sex, gender, and blood pressure: contributions to experimental pain report. Psychosom Med 2001;63:545–550. [PubMed: 11485107]
198. Wise EA, Price DD, Myers CD, Heft MW, Robinson ME. Gender role expectations of pain: relationship to experimental pain perception. Pain 2002;96:335–342. [PubMed: 11973007]
199. Feijó LM, Tarman GZ, Fontaine C, Harrison R, Johnstone T, Salomons T. Sex-specific effects of gender identification on pain study recruitment. The Journal of Pain 2017.
200. Robinson ME, Wise EA, Gagnon C, Fillingim RB, Price DD. Influences of gender role and anxiety on sex differences in temporal summation of pain. J Pain 2004;5:77–82. [PubMed: 15042515]
201. Unruh AM, Ritchie J, Merskey H. Does gender affect appraisal of pain and pain coping strategies? Clin J Pain 1999;15:31–40. [PubMed: 10206565]
202. Sullivan MJL, Tripp DA, Santor D. Gender differences in pain and pain behavior: The role of catastrophizing. Cognitive Therapy and Research 2000;24:121–134.
203. Lynch AM, Kashikar-Zuck S, Goldschneider KR, Jones BA. Sex and age differences in coping styles among children with chronic pain. J Pain Symptom Manage 2007;33:208–216. [PubMed: 17280926]
204. El-Shormilisy N, Strong J, Meredith PJ. Associations between gender, coping patterns and functioning for individuals with chronic pain: a systematic review. Pain Res Manag 2015;20:48–55. [PubMed: 24927488]
205. Edwards RR, Haythornthwaite JA, Sullivan MJ, Fillingim RB. Catastrophizing as a mediator of sex differences in pain: differential effects for daily pain versus laboratory-induced pain. Pain 2004;111:335–341. [PubMed: 15363877]
206. Keefe fJ Lefebvre JC, Egert JR, Affleck G, Sullivan MJ, Caldwell DS. The relationship of gender to pain, pain behavior, and disability in osteoarthritis patients: the role of catastrophizing. Pain 2000;87:325–334. [PubMed: 10963912]
207. Rhudy JL, Williams AE, McCabe KM, Nguyen MA, Rambo P. Affective modulation of nociception at spinal and supraspinal levels. Psychophysiology 2005;42:579–587. [PubMed: 16176380]
208. Rollman GB. Gender differences in pain: role of anxiety. Pain Forum 1995;4:231–234.
209. Kroenke K, Spitzer RL. Gender differences in the reporting of physical and somatoform symptoms. Psychosomatic Medicine 1998;60:150–155. [PubMed: 9560862]
210. Barsky AJ, Peekna HM, Borus JF. Somatic symptom reporting in women and men. J Gen Intern Med 2001;16:266–275. [PubMed: 11318929]
211. Edwards R, Augustson EM, Fillingim R. Sex-specific effects of pain-related anxiety on adjustment to chronic pain. Clin J Pain 2000;16:46–53. [PubMed: 10741818]
212. Robinson ME, Dannecker EA, George SZ, Otis J, Atchison JW, Fillingim RB. Sex differences in the associations among psychological factors and pain report: a novel psychophysical study of patients with chronic low back pain. J Pain 2005;6:463–470. [PubMed: 15993825]

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

213. Marcus SM, Young EA, Kerber KB, et al. Gender differences in depression: findings from the STAR*D study. J Affect Disord 2005;87:141–150. [PubMed: 15982748]

214. Marcus SM, Kerber KB, Rush AJ, et al. Sex differences in depression symptoms in treatment-seeking adults: confirmatory analyses from the Sequenced Treatment Alternatives to Relieve Depression study. Compr Psychiatry 2008;49:238–246. [PubMed: 18396182]

215. Anderson KO, Green CR, Payne R. Racial and ethnic disparities in pain: causes and consequences of unequal care. The Journal of Pain 2009;10:1187–1204. [PubMed: 19944378]

216. Breitbart W, McDonald MV, Rosenfeld B, et al. Pain in ambulatory AIDS patients. I: Pain characteristics and medical correlates. Pain 1996;68:315–321. [PubMed: 9121820]

217. Creamer P, Lethbridge-Cejku M, Hochberg MC. Determinants of pain severity in knee osteoarthritis: effect of demographic and psychosocial variables using 3 pain measures. J Rheumatol 1999;26:1785–1792. [PubMed: 10451078]

218. Green CR, Anderson KO, baker TA, et al. The unequal burden of pain: confronting racial and ethnic disparities in pain. Pain Med 2003;4:277–294. [PubMed: 12974827]

219. Shavers VL, Bakos A, Sheppard VB. Race, ethnicity, and pain among the U.S. adult population. J Health Care Poor Underserved 2010;21:177–220. [PubMed: 20173263]

220. Mossey JM. Defining racial and ethnic disparities in pain management. Clin Orthop Relat Res 2011;469:1859–1870. [PubMed: 21249483]

221. Campbell CM, Edwards RR, Fillingim RB. Ethnic differences in responses to multiple experimental pain stimuli. Pain 2005;113:20–26. [PubMed: 15621360]

222. Meints SM, Hirsh AT. In Vivo praying and catastrophizing mediate the race differences in experimental pain sensitivity. J Pain 2015;16:491–497. [PubMed: 25725173]

223. Rahim-Williams FB, Riley JL, III, Herrera D, Campbell CM, Hastie BA, Fillingim RB. Ethnic identity predicts experimental pain sensitivity in African Americans and Hispanics. Pain 2007;129:177–184. [PubMed: 17296267]

224. Rahim-Williams B, Riley JL, III, Williams AK, Fillingim RB. A Quantitative Review of Ethnic Group Differences in Experimental Pain Response: Do Biology, Psychology, and Culture Matter? Pain Med 2012.

225. Ahn H, Weaver M, Lyon DE, et al. Differences in Clinical Pain and Experimental Pain Sensitivity Between Asian Americans and Whites With Knee Osteoarthritis. Clin J Pain 2017;33:174–180. [PubMed: 28060784]

226. Ostrom C, Bair E, Maixner W, et al. Demographic Predictors of Pain Sensitivity: Results From the OPPERA Study. J Pain 2017;18:295–307. [PubMed: 27884689]

227. Palit S, Kerr KL, Kuhn BL, et al. Exploring pain processing differences in Native Americans. Health Psychol 2013;32:1127–1136. [PubMed: 24219417]

228. Campbell CM, France CR, Robinson ME, Logan HL, Geffken GR, Fillingim RB. Ethnic differences in the nociceptive flexion reflex (NFR). Pain 2008;134:91–96. [PubMed: 17482362]

229. Campbell CM, France CR, Robinson ME, Logan HL, Geffken GR, Fillingim RB. Ethnic differences in diffuse noxious inhibitory controls. J Pain 2008;9:759–766. [PubMed: 18482870]

230. McNeilly M, Zeichner A. Neuropeptide and cardiovascular responses to intravenous catheterization in normotensive and hypertensive blacks and whites. Health Psychol 1989;8:487–501. [PubMed: 2534295]

231. Currie SR, Wang J. More data on major depression as an antecedent risk factor for first onset of chronic back pain. Psychol Med 2005;35:1275–1282. [PubMed: 16168150]

232. 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;129:332–342. [PubMed: 17350169]

233. Gureje O, Von Korff M, Kola L, et al. The relation between multiple pains and mental disorders: results from the World Mental Health Surveys. Pain 2008;135:82–91. [PubMed: 17570586]

234. Gadermann AM, Alonso J, Vilagut G, Zaslavsky AM, Kessler RC. Comorbidity and disease burden in the National Comorbidity Survey Replication (NCS-R). Depress Anxiety 2012;29:797–806. [PubMed: 22585578]

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

235. Bruffaerts R, Demyttenaere K, Kessler RC, et al. The associations between preexisting mental disorders and subsequent onset of chronic headaches: a worldwide epidemiologic perspective. J Pain 2015;16:42–52. [PubMed: 25451620]
236. Roy-Byrne PP, Davidson KW, Kessler RC, et al. Anxiety disorders and comorbid medical illness. Gen Hosp Psychiatry 2008;30:208–225. [PubMed: 18433653]
237. Williams DR, Neighbors HW, Jackson JS. Racial/ethnic discrimination and health: findings from community studies. Am J Public Health 2003;93:200–208. [PubMed: 12554570]
238. Williams DR, Gonzalez HM, Neighbors H, et al. Prevalence and distribution of major depressive disorder in African Americans, Caribbean blacks, and non-Hispanic whites: results from the National Survey of American Life. Arch Gen Psychiatry 2007;64:305–315. [PubMed: 17339519]
239. Griffith DM, Johnson JL, Zhang R, Neighbors HW, Jackson JS. Ethnicity, nativity, and the health of American Blacks. J Health Care Poor Underserved 2011;22:142–156. [PubMed: 21317512]
240. Gara MA, Vega WA, Arndt S, et al. Influence of patient race and ethnicity on clinical assessment in patients with affective disorders. Arch Gen Psychiatry 2012;69:593–600. [PubMed: 22309972]
241. Hudson DL, Neighbors HW, Geronimus AT, Jackson JS. Racial Discrimination, John Henryism, and Depression Among African Americans. J Black Psychol 2016;42:221–243. [PubMed: 27529626]
242. Bates MS, Rankin-Hill L. Control, culture and chronic pain. Social Science & Medicine 1994;39:629–645. [PubMed: 7973863]
243. Bates MS. Biocultural dimensions of chronic pain: implications for treatment of multiethnic populations. Albany: State University of New York Press, 1996.
244. Koyama T, McHaffie JG, Laurienti PJ, Coghill RC. The subjective experience of pain: where expectations become reality. Proc Natl Acad Sci U S A 2005;102:12950–12955. [PubMed: 16150703]
245. Wandner LD, Scipio CD, Hirsh AT, Torres CA, Robinson ME. The perception of pain in others: how gender, race, and age influence pain expectations. J Pain 2012;13:220–227. [PubMed: 22225969]
246. Torres CA, Bartley EJ, Wandner LD, Alqudah AF, Hirsh AT, Robinson ME. The influence of sex, race, and age on pain assessment and treatment decisions using virtual human technology: a cross-national comparison. J Pain Res 2013;6:577–588. [PubMed: 23901291]
247. Meints SM, Miller MM, Hirsh AT. Differences in Pain Coping Between Black and White Americans: A Meta-Analysis. J Pain 2016;17:642–653. [PubMed: 26804583]
248. Shavers VL, Shavers BS. Racism and health inequity among Americans. J Natl Med Assoc 2006;98:386–396. [PubMed: 16573303]
249. Banks KH, Kohn-Wood LP, Spencer M. An examination of the African American experience of everyday discrimination and symptoms of psychological distress. Community Ment Health J 2006;42:555–570. [PubMed: 16897412]
250. Mays VM, Cochran SD, Barnes NW. Race, race-based discrimination, and health outcomes among African Americans. Annu Rev Psychol 2007;58:201–225. [PubMed: 16953796]
251. Williams DR. Race, socioeconomic status, and health. The added effects of racism and discrimination. Ann N Y Acad Sci 1999;896:173–188. [PubMed: 10681897]
252. Edwards RR. The association of perceived discrimination with low back pain. J Behav Med 2008;31:379–389. [PubMed: 18581224]
253. Goodin BR, Pham QT, Glover TL, et al. Perceived racial discrimination, but not mistrust of medical researchers, predicts the heat pain tolerance of African Americans with symptomatic knee osteoarthritis. Health Psychol 2013;32:1117–1126. [PubMed: 24219416]
254. Carlisle SK. Perceived discrimination and chronic health in adults from nine ethnic subgroups in the USA. Ethn Health 2015;20:309–326. [PubMed: 24920185]
255. Lazarus RS, Folkman S. Stress,Appraisal,and Coping. New York: Springer Publishing Company, 1984.
256. Rosenstiel AK, Keefe FJ. The use of coping strategies in chronic low back pain patients: Relationship to patient characteristics and current adjustment. Pain 1983;3:1–8.

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

257. Obrist PA, Light KC. Active-passive coping and cardiovascular reactivity: Interaction with individual differences and types of baseline In: Gordon WA, Herd JA, Baum A, eds. Perspectives on behavioral medicine, 3 ed. San Diego: Academic Press, Inc., 1988: 109–126.

258. Snow-Turek AL, Norris MP, Tan G. Active and passive coping strategies in chronic pain patients. Pain 1996;64:455–462. [PubMed: 8783309]

259. Kraaimaat FW, Evers AW. Pain-coping strategies in chronic pain patients: psychometric characteristics of the pain-coping inventory (PCI). Int J Behav Med 2003;10:343–363. [PubMed: 14734263]

260. Folkman S, Lazarus RS. Coping as a mediator of emotion. J Personal Soc Psychol 1988;54:3:466–475.

261. Carver CS, Scheier MF, Weintraub JK. Assessing coping strategies: a theoretically based approach. J Pers Soc Psychol 1989;56:267–283. [PubMed: 2926629]

262. Dysvik E, Natvig GK, Eikeland OJ, Lindstrom TC. Coping with chronic pain. Int J Nurs Stud 2005;42:297–305. [PubMed: 15708016]

263. Jensen MP, Turner JA, Romano JM, Karoly P. Coping with chronic pain: A critical review of the literature. Pain 1991;47:249–283. [PubMed: 1784498]

264. Jensen MP, Karoly P. Control beliefs, coping efforts, and adjustment to chronic pain. J Consult Clin Psychol 1991;59:431–438. [PubMed: 2071728]

265. Jensen MP, Keefe FJ, Lefebvre JC, Romano JM, Turner JA. One- and two-item measures of pain beliefs and coping strategies. Pain 2003;104:453–469. [PubMed: 12927618]

266. Tan G, Nguyen Q, Anderson KO, Jensen M, Thornby J. Further validation of the chronic pain coping inventory. J Pain 2005;6:29–40. [PubMed: 15629416]

267. Brown G, Nicassio P, Wallston K. Pain coping strategies and depression in rheumatoid arthritis. J Consult Clin Psychol 1989;57:652–657. [PubMed: 2794186]

268. Holmes JA, Stevenson CA. Differential effects of avoidant and attentional coping strategies on adaptation to chronic and recent-onset pain. Health Psychol 1990;9:577–584. [PubMed: 2226386]

269. Covic T, Adamson B, Hough M. The impact of passive coping on rheumatoid arthritis pain. Rheumatology (Oxford) 2000;39:1027–1030. [PubMed: 10986310]

270. Stewart DE, Yuen T. A systematic review of resilience in the physically ill. Psychosomatics 2011;52:199–209. [PubMed: 21565591]

271. Keefe FJ, Kashikar-Zuck S, Opiteck J, Hage E, Dalrymple L, Blumenthal JA. Pain in arthritis and musculoskeletal disorders: the role of coping skills training and exercise interventions. [Review] [43 refs]. Journal of Orthopaedic & Sports Physical Therapy 1996;24:279–290. [PubMed: 8892142]

272. Keefe FJ, Affleck G, Lefebvre J, et al. Living with rheumatoid arthritis: the role of daily spirituality and daily religious and spiritual coping. J Pain 2001;2:101–110. [PubMed: 14622831]

273. Keefe FJ, Somers TJ. Psychological approaches to understanding and treating arthritis pain. Nat Rev Rheumatol 2010;6:210–216. [PubMed: 20357790]

274. Nicassio PM, Radojevic V, Weisman MH, Culbertson AL, Lewis C, Clemmey P. The role of helplessness in the response to disease modifying drugs in rheumatoid arthritis. J Rheumatol 1993;20:1114–1120. [PubMed: 8103802]

275. Gil KM, Williams DA, Thompson RJ, Jr, Kinney TR. Sickle cell disease in children and adolescents: the relation of child and parent pain coping strategies to adjustment. J Pediatr Psychol 1991;16:643–663. [PubMed: 1744811]

276. Thompson RJ, Jr, Gil KM, Abrams MR, Phillips G. Stress, coping, and psychological adjustment of adults with sickle cell disease. J Consult Clin Psychol 1992;60:433–440. [PubMed: 1619097]

277. Evers AW, Kraaimaat FW, Geenen R, Bijlsma JW. Psychosocial predictors of functional change in recently diagnosed rheumatoid arthritis patients. Behav Res Ther 1998;36:179–193. [PubMed: 9613024]

278. Quartana PJ, Campbell CM, Edwards RR. Pain catastrophizing: a critical review. Expert Rev Neurother 2009;9:745–758. [PubMed: 19402782]

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

279. Wertli MM, Eugster R, Held U, Steurer J, Kofmehl R, Weiser S. Catastrophizing-a prognostic factor for outcome in patients with low back pain: a systematic review. Spine J 2014;14:2639–2657. [PubMed: 24607845]

280. Geisser ME, Robinson ME, Henson CD. The Coping Strategies Questionnaire and chronic pain adjustment: a conceptual and empirical reanalysis. Clin J Pain 1994;10:98–106. [PubMed: 8075472]

281. Robinson ME, Riley JL, III, Myers CD, et al. The Coping Strategies Questionnaire: a large sample, item level factor analysis. Clin J Pain 1997;13:43–49. [PubMed: 9084951]

282. Novy DM, Nelson DV, Hetzel RD, Squitieri P, Kennington M. Coping with chronic pain: sources of intrinsic and contextual variability. Journal of Behavioral Medicine 1998;21:19–34. [PubMed: 9547420]

283. Edwards RR, Moric M, Husfeldt B, Buvanendran A, Ivankovich O. Ethnic similarities and differences in the chronic pain experience: a comparison of african american, Hispanic, and white patients. Pain Med 2005;6:88–98. [PubMed: 15669954]

284. Buckelew SP, Conway RC, Shutty MS, et al. Spontaneous coping strategies to manage acute pain and anxiety during electrodiagnostic studies. Arch Phys Med Rehabil 1992;73:594–598. [PubMed: 1622311]

285. Fauerbach JA, Lawrence JW, Haythornthwaite JA, Richter L. Coping with the stress of a painful medical procedure. Behav Res Ther 2002;40:1003–1015. [PubMed: 12296486]

286. Lefebvre JC, Lester N, Keefe FJ. Pain in young adults. II: The use and perceived effectiveness of pain-coping strategies. Clin J Pain 1995;11:36–44. [PubMed: 7787337]

287. Lester N, Lefebvre JC, Keefe FJ. Pain in young adults--III: Relationships of three pain-coping measures to pain and activity interference. Clin J Pain 1996;12:291–300. [PubMed: 8969874]

288. Turner JA, Clancy S. Strategies for coping with chronic low back pain: relationship to pain and disability. Pain 1986;24:355–364. [PubMed: 2938059]

289. Jordan MS, Lumley MA, Leisen JC. The relationships of cognitive coping and pain control beliefs to pain and adjustment among African-American and Caucasian women with rheumatoid arthritis. Arthritis Care & Research 1998;11:80–88.

290. Haythornthwaite JA, Clark MR, Pappagallo M, Raja SN. Pain coping strategies play a role in the persistence of pain in post-herpetic neuralgia. Pain 2003;106:453–460. [PubMed: 14659529]

291. Dozois DJA, Dobson KS, Wong M, Hughes D, Long A. Predictive utility of the CSQ in low back pain: individual vs. composite measures. Pain 1996;66:171–180. [PubMed: 8880838]

292. Geisser ME, Robinson ME, Riley JL, III. Pain beliefs, coping, and adjustment to chronic pain: let's focus more on the negative. Pain Forum 1999;8:161–168.

293. Sullivan MJ, Bishop SR, Pivik J. The Pain Catastrophizing Scale: Development and Validation. Psychological Assessment 1995;7:524–532.

294. Sullivan MJ, Thorn B, Haythornthwaite JA, et al. Theoretical perspectives on the relation between catastrophizing and pain. Clin J Pain 2001;17:52–64. [PubMed: 11289089]

295. Severeijns R, Vlaeyen JW, Van den Hout MA, Weber WE. Pain catastrophizing predicts pain intensity, disability, and psychological distress independent of the level of physical impairment. Clin J Pain 2001;17:165–172. [PubMed: 11444718]

296. Severeijns R, Van den Hout MA, Vlaeyen JW, Picavet HS. Pain catastrophizing and general health status in a large Dutch community sample. Pain 2002;99:367–376. [PubMed: 12237216]

297. Edwards RR, Calahan C, Mensing G, Smith M, Haythornthwaite JA. Pain, catastrophizing, and depression in the rheumatic diseases. Nat Rev Rheumatol 2011.

298. Theunissen M, Peters ML, Bruce J, Gramke HF, Marcus MA. Preoperative anxiety and catastrophizing: a systematic review and meta-analysis of the association with chronic postsurgical pain. Clin J Pain 2012;28:819–841. [PubMed: 22760489]

299. Vissers MM, Bussmann JB, Verhaar JA, Busschbach JJ, Bierma-Zeinstra SM, Reijman M. Psychological factors affecting the outcome of total hip and knee arthroplasty: a systematic review. Semin Arthritis Rheum 2012;41:576–588. [PubMed: 22035624]

300. Hill JC, Lewis M, Sim J, Hay EM, Dziedzic K. Predictors of poor outcome in patients with neck pain treated by physical therapy. Clin J Pain 2007;23:683–690. [PubMed: 17885347]

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

301. Karels CH, Bierma-Zeinstra SM, Burdorf A, Verhagen AP, Nauta AP, Koes BW. Social and psychological factors influenced the course of arm, neck and shoulder complaints. J Clin Epidemiol 2007;60:839–848. [PubMed: 17606181]
302. Pinto PR, McIntyre T, Almeida A, Araujo-Soares V. The mediating role of pain catastrophizing in the relationship between presurgical anxiety and acute postsurgical pain after hysterectomy. Pain 2012;153:218–226. [PubMed: 22115922]
303. Sullivan MJ, Ward LC, Tripp D, French DJ, Adams H, Stanish WD. Secondary prevention of work disability: community-based psychosocial intervention for musculoskeletal disorders. J Occup Rehabil 2005;15:377–392. [PubMed: 16119228]
304. Gauthier N, Sullivan MJ, Adams H, Stanish WD, Thibault P. Investigating risk factors for chronicity: the importance of distinguishing between return-to-work status and self-report measures of disability. J Occup Environ Med 2006;48:312–318. [PubMed: 16531836]
305. Wideman TH, Sullivan MJ. Differential predictors of the long-term levels of pain intensity, work disability, healthcare use, and medication use in a sample of workers' compensation claimants. Pain 2011;152:376–383. [PubMed: 21147513]
306. Hirsh AT, Bockow TB, Jensen MP. Catastrophizing, pain, and pain interference in individuals with disabilities. Am J Phys Med Rehabil 2011;90:713–722. [PubMed: 21814133]
307. Archer KR, Abraham CM, Song Y, Obremskey WT. Cognitive-behavioral determinants of pain and disability two years after traumatic injury: A cross-sectional survey study. J Trauma Acute Care Surg 2012;72:473–479. [PubMed: 21808211]
308. Trost Z, Strachan E, Sullivan M, Vervoort T, Avery AR, Afari N. Heritability of pain catastrophizing and associations with experimental pain outcomes: a twin study. Pain 2015;156:514–520. [PubMed: 25599234]
309. Toth C, Brady S, Hatfield M. The importance of catastrophizing for successful pharmacological treatment of peripheral neuropathic pain. J Pain Res 2014;7:327–338. [PubMed: 25028563]
310. Riddle DL, Wade JB, Jiranek WA, Kong X. Preoperative pain catastrophizing predicts pain outcome after knee arthroplasty. Clin Orthop Relat Res 2010;468:798–806. [PubMed: 19585177]
311. Lewis GN, Rice DA, McNair PJ, Kluger M. Predictors of persistent pain after total knee arthroplasty: a systematic review and meta-analysis. Br J Anaesth 2015;114:551–561. [PubMed: 25542191]
312. Schreiber KL, Kehlet H, Belfer I, Edwards RR. Predicting, preventing and managing persistent pain after breast cancer surgery: the importance of psychosocial factors. Pain Manag 2014;4:445–459. [PubMed: 25494696]
313. Mankovsky T, Lynch M, Clark A, Sawynok J, Sullivan MJ. Pain catastrophizing predicts poor response to topical analgesics in patients with neuropathic pain. Pain Res Manag 2012;17:10–14. [PubMed: 22518362]
314. Desrochers G, Bergeron S, Khalife S, Dupuis MJ, Jodoin M. Provoked vestibulodynia: psychological predictors of topical and cognitive-behavioral treatment outcome. Behav Res Ther 2010;48:106–115. [PubMed: 19879555]
315. Turner JA, Holtzman S, Mancl L. Mediators, moderators, and predictors of therapeutic change in cognitive-behavioral therapy for chronic pain. Pain 2007;127:276–286. [PubMed: 17071000]
316. Litt MD, Porto FB. Determinants of pain treatment response and nonresponse: identification of TMD patient subgroups. J Pain 2013;14:1502–1513. [PubMed: 24094979]
317. Rakel BA, Zimmerman MB, Geasland K, et al. Transcutaneous electrical nerve stimulation for the control of pain during rehabilitation after total knee arthroplasty: A randomized, blinded, placebo-controlled trial. Pain 2014;155:2599–2611. [PubMed: 25270585]
318. Thorn BE, Pence LB, Ward LC, et al. A randomized clinical trial of targeted cognitive behavioral treatment to reduce catastrophizing in chronic headache sufferers. J Pain 2007;8:938–949. [PubMed: 17690017]
319. Thorn BE, Day MA, Burns J, et al. Randomized trial of group cognitive behavioral therapy compared with a pain education control for low-literacy rural people with chronic pain. Pain 2011;152:2710–2720. [PubMed: 21920668]

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

320. Burns JW, Kubilus A, Bruehl S, Harden RN, Lofland K. Do changes in cognitive factors influence outcome following multidisciplinary treatment for chronic pain? A cross-lagged panel analysis. J Consult Clin Psychol 2003;71:81–91. [PubMed: 12602428]
321. Burns JW, Glenn B, Bruehl S, Harden RN, Lofland K. Cognitive factors influence outcome following multidisciplinary chronic pain treatment: a replication and extension of a cross-lagged panel analysis. Behav Res Ther 2003;41:1163–1182. [PubMed: 12971938]
322. Morley S, Williams A, Hussain S. Estimating the clinical effectiveness of cognitive behavioural therapy in the clinic: evaluation of a CBT informed pain management programme. Pain 2008;137:670–680. [PubMed: 18394806]
323. Turner JA, Mancl L, Aaron LA. Short- and long-term efficacy of brief cognitive-behavioral therapy for patients with chronic temporomandibular disorder pain: a randomized, controlled trial. Pain 2006;121:181–194. [PubMed: 16495014]
324. McCracken LM, Carson JW, Eccleston C, Keefe FJ. Acceptance and change in the context of chronic pain. Pain 2004;109:4–7. [PubMed: 15082120]
325. Vowles KE, McCracken LM, Eccleston C. Processes of change in treatment for chronic pain: the contributions of pain, acceptance, and catastrophizing. Eur J Pain 2007;11:779–787. [PubMed: 17303452]
326. McCracken LM, Vowles KE. Acceptance and commitment therapy and mindfulness for chronic pain: model, process, and progress. Am Psychol 2014;69:178–187. [PubMed: 24547803]
327. McCracken LM, Davies M, Scott W, Paroli M, Harris S, Sanderson K. Can a psychologically based treatment help people to live with chronic pain when they are seeking a procedure to reduce it? Pain Med 2015;16:451–459. [PubMed: 25530233]
328. Trompetter HR, Bohlmeijer ET, Fox JP, Schreurs KM. Psychological flexibility and catastrophizing as associated change mechanisms during online Acceptance & Commitment Therapy for chronic pain. Behav Res Ther 2015;74:50–59. [PubMed: 26409158]
329. Smeets RJ, Vlaeyen JW, Kester AD, Knottnerus JA. Reduction of pain catastrophizing mediates the outcome of both physical and cognitive-behavioral treatment in chronic low back pain. J Pain 2006;7:261–271. [PubMed: 16618470]
330. Leeuw M, Goossens ME, Van Breukelen GJ, et al. Exposure in vivo versus operant graded activity in chronic low back pain patients: results of a randomized controlled trial. Pain 2008;138:192–207. [PubMed: 18242858]
331. de Rooij A, de Boer MR, van der LM, Roorda LD, Steultjens MP, Dekker J. Cognitive mechanisms of change in multidisciplinary treatment of patients with chronic widespread pain: a prospective cohort study. J Rehabil Med 2014;46:173–180. [PubMed: 24322580]
332. de Rooij M, van der Leeden M, Heymans MW, et al. Prognosis of Pain and Physical Functioning in Patients With Knee Osteoarthritis: A Systematic Review and Meta-Analysis. Arthritis Care Res (Hoboken) 2016;68:481–492. [PubMed: 26316234]
333. Hirsh AT, Kupper AE, Carter GT, Jensen MP. Psychosocial factors and adjustment to pain in individuals with postpolio syndrome. Am J Phys Med Rehabil 2010;89:213–224. [PubMed: 20068433]
334. Edwards RR, Bingham CO, III, Bathon J, Haythornthwaite JA. Catastrophizing and pain in arthritis, fibromyalgia, and other rheumatic diseases. Arthritis Rheum 2006;55:325–332. [PubMed: 16583384]
335. Vlaeyen JW, Kole-Snijders Am, Boeren RG, van Eek H. Fear of movement/(re)injury in chronic low back pain and its relation to behavioral performance. Pain 1995;62:363–372. [PubMed: 8657437]
336. Wideman TH, Asmundson GG, Smeets RJ, et al. Rethinking the fear avoidance model: toward a multidimensional framework of pain-related disability. Pain 2013;154:2262–2265. [PubMed: 23748115]
337. Vlaeyen JW, Linton SJ. Fear-avoidance model of chronic musculoskeletal pain: 12 years on. Pain 2012;153:1144–1147. [PubMed: 22321917]
338. Volders S, Boddez Y, De Peuter S, Meulders A, Vlaeyen JW. Avoidance behavior in chronic pain research: A cold case revisited. Behav Res Ther 2015;64:31–37. [PubMed: 25506905]

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

339. Buenaver LF, Edwards RR, Haythornthwaite JA. Pain-related catastrophizing and perceived social responses: Inter-relationships in the context of chronic pain. Pain 2007;127:234–242. [PubMed: 17011706]

340. Cano A Pain catastrophizing and social support in married individuals with chronic pain: the moderating role of pain duration. Pain 2004;110:656–664. [PubMed: 15288406]

341. Thorn BE, Ward LC, Sullivan MJ, Boothby JL. Communal coping model of catastrophizing: conceptual model building. Pain 2003;106:1–2. [PubMed: 14581103]

342. Lackner JM, Gurtman MB. Pain catastrophizing and interpersonal problems: a circumplex analysis of the communal coping model. Pain 2004;110:597–604. [PubMed: 15288400]

343. McCracken LM. Social context and acceptance of chronic pain: the role of solicitous and punishing responses. Pain 2005;113:155–159. [PubMed: 15621376]

344. Lemieux AJ, Bergeron S, Steben M, Lambert B. Do romantic partners' responses to entry dyspareunia affect women's experience of pain? The roles of catastrophizing and self-efficacy. J Sex Med 2013;10:2274–2284. [PubMed: 23809759]

345. Pielech M, Ryan M, Logan D, Kaczynski K, White MT, Simons LE. Pain catastrophizing in children with chronic pain and their parents: proposed clinical reference points and reexamination of the Pain Catastrophizing Scale measure. Pain 2014;155:2360–2367. [PubMed: 25180013]

346. Atlas LY, Wielgosz J, Whittington RA, Wager TD. Specifying the non-specific factors underlying opioid analgesia: expectancy, attention, and affect. Psychopharmacology (Berl) 2014;231:813–823. [PubMed: 24096537]

347. Benedetti F, Amanzio M. Mechanisms of the placebo response. Pulm Pharmacol Ther 2013;26:520–523. [PubMed: 23370213]

348. Scheier M, Carver C, Bridges MW. Distiguishing Optimism from Neuroticism (and Trait Anxiety, Self-Mastery, and Self-Esteem): A Reevalution of the Life Orientation Test. The Journal of Personality and Social Psychology 1994;67:1063–1078. [PubMed: 7815302]

349. Scheier MF, Bridges MW. Person variables and health: Personality and predisposition and acute psychological states as shared determinants for disease. Psychosom Med 1995;57:255–268. [PubMed: 7652126]

350. Cruz-Almeida Y, King CD, Goodin BR, et al. Psychological profiles and pain characteristics of older adults with knee osteoarthritis. Arthritis Care Res (Hoboken) 2013;65:1786–1794. [PubMed: 23861288]

351. Affleck G, Tennen H, Zautra A, Urrows S, Abeles M, Karoly P. Women's pursuit of personal goals in daily life with fibromyalgia: a value-expectancy analysis. J Consult Clin Psychol 2001; 69:587–596. [PubMed: 11550725]

352. Brenes GA, Rapp SR, Rejeski WJ, Miller ME. Do optimism and pessimism predict physical functioning? J Behav Med 2002;25:219–231. [PubMed: 12055774]

353. Novy DM, Nelson DV, Hetzel RD, Squitieri P, Kennington M. Coping with chronic pain: Sources of intrinsic and contextual validity. J Beh Med 1998;21:19–34.

354. Bruce J, Thornton AJ, Powell R, et al. Psychological, surgical, and sociodemographic predictors of pain outcomes after breast cancer surgery: a population-based cohort study. Pain 2014;155:232–243. [PubMed: 24099954]

355. Mavros MN, Athanasiou S, Gkegkes ID, Polyzos KA, Peppas G, Falagas ME. Do psychological variables affect early surgical recovery? PLoS One 2011;6:e20306. [PubMed: 21633506]

356. Sobol-Kwapinska M, Babel P, Plotek W, Stelcer B. Psychological correlates of acute postsurgical pain: A systematic review and meta-analysis. Eur J Pain 2016;20:1573–1586. [PubMed: 27136510]

357. Colloca L, Benedetti F. Placebos and painkillers: is mind as real as matter? Nat Rev Neurosci 2005;6:545–552. [PubMed: 15995725]

358. Keltner JR, Furst A, Fan C, Redfern R, Inglis B, Fields HL. Isolating the modulatory effect of expectation on pain transmission: a functional magnetic resonance imaging study. J Neurosci 2006;26:4437–4443. [PubMed: 16624963]

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

359. Brown CA, El Deredy W, Jones AK. When the brain expects pain: common neural responses to pain anticipation are related to clinical pain and distress in fibromyalgia and osteoarthritis. Eur J Neurosci 2013.
360. Bingel U, Wanigasekera V, Wiech K, et al. The effect of treatment expectation on drug efficacy: imaging the analgesic benefit of the opioid remifentanil. Sci Transl Med 2011;3:70ra14.
361. Kalauokalani D, Cherkin DC, Sherman KJ, Koepsell TD, Deyo RA. Lessons from a trial of acupuncture and massage for low back pain: patient expectations and treatment effects. Spine 2001;26:1418–1424. [PubMed: 11458142]
362. Witt CM, Schutzler L, Ludtke R, Wegscheider K, Willich SN. Patient characteristics and variation in treatment outcomes: which patients benefit most from acupuncture for chronic pain? Clin J Pain 2011;27:550–555. [PubMed: 21317771]
363. Zywiel MG, Mahomed A, Gandhi R, Perruccio AV, Mahomed NN. Measuring expectations in orthopaedic surgery: a systematic review. Clin Orthop Relat Res 2013;471:3446–3456. [PubMed: 23633186]
364. Witt CM, Martins F, Willich SN, Schutzler L. Can I help you? Physicians' expectations as predictor for treatment outcome. Eur J Pain 2012;16:1455–1466. [PubMed: 22528659]
365. Benedetti F How the doctor's words affect the patient's brain. Eval Health Prof 2002;25:369–386. [PubMed: 12449081]
366. Benedetti F, Pollo A, Lopiano L, Lanotte M, Vighetti S, Rainero I. Conscious expectation and unconscious conditioning in analgesic, motor, and hormonal placebo/nocebo responses. J Neurosci 2003;23:4315–4323. [PubMed: 12764120]
367. Bandura A Social foundations of thought and action: a social cognitive theory. Englewood Cliffs: Prentice Hall, 1986.
368. Bandura A Human agency in social cognitive theory. American Psychologist 1989;44:1175–1184. [PubMed: 2782727]
369. Somers TJ, Keefe FJ, Godiwala N, Hoyler GH. Psychosocial factors and the pain experience of osteoarthritis patients: new findings and new directions. Curr Opin Rheumatol 2009;21:501–506. [PubMed: 19617836]
370. French DJ, Holroyd KA, Pinell C, Malinoski PT, O'Donnell F, Hill KR. Perceived self-efficacy and headache-related disability. Headache 2000;40:647–656. [PubMed: 10971661]
371. Kalapurakkel S, Carpino EA, Lebel A, Simons LE. "Pain Can't Stop Me": Examining Pain Self-Efficacy and Acceptance as Resilience Processes Among Youth With Chronic Headache. J Pediatr Psychol 2014.
372. Miro E, Martinez MP, Sanchez AI, Prados G, Medina A. When is pain related to emotional distress and daily functioning in fibromyalgia syndrome? The mediating roles of self-efficacy and sleep quality. Br J Health Psychol 2011;16:799–814. [PubMed: 21988065]
373. Ahlstrand I, Vaz S, Falkmer T, Thyberg I, Bjork M. Self-efficacy and pain acceptance as mediators of the relationship between pain and performance of valued life activities in women and men with rheumatoid arthritis. Clin Rehabil 2017;31:824–834. [PubMed: 27146888]
374. Costa LC, Maher CG, McAuley JH, Hancock MJ, Smeets RJ. Self-efficacy is more important than fear of movement in mediating the relationship between pain and disability in chronic low back pain. Eur J Pain 2011;15:213–219. [PubMed: 20655254]
375. Brister H, Turner JA, Aaron LA, Mancl L. Self-efficacy is associated with pain, functioning, and coping in patients with chronic temporomandibular disorder pain. J Orofac Pain 2006;20:115–124. [PubMed: 16708829]
376. Turner JA, Anderson ML, Balderson BH, Cook AJ, Sherman KJ, Cherkin DC. Mindfulness-based stress reduction and cognitive behavioral therapy for chronic low back pain: similar effects on mindfulness, catastrophizing, self-efficacy, and acceptance in a randomized controlled trial. Pain 2016;157:2434–2444. [PubMed: 27257859]
377. Denk F, McMahon SB, Tracey I. Pain vulnerability: a neurobiological perspective. Nat Neurosci 2014;17:192–200. [PubMed: 24473267]
378. Mackey SC. Central neuroimaging of pain. J Pain 2013;14:328–331. [PubMed: 23548485]
379. Baliki MN, Mansour AR, Baria AT, Apkarian AV. Functional reorganization of the default mode network across chronic pain conditions. PLoS One 2014;9:e106133. [PubMed: 25180885]

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

380. Mansour AR, Farmer MA, Baliki MN, Apkarian AV. Chronic pain: the role of learning and brain plasticity. Restor Neurol Neurosci 2014;32:129–139. [PubMed: 23603439]

381. Kregel J, Meeus M, Malfliet A, et al. Structural and functional brain abnormalities in chronic low back pain: A systematic review. Semin Arthritis Rheum 2015;45:229–237. [PubMed: 26092329]

382. Apkarian AV, Sosa Y, Sonty S, et al. Chronic back pain is associated with decreased prefrontal and thalamic gray matter density. J Neurosci 2004;24:10410–10415. [PubMed: 15548656]

383. Farmer MA, Chanda ML, Parks EL, Baliki MN, Apkarian AV, Schaeffer AJ. Brain functional and anatomical changes in chronic prostatitis/chronic pelvic pain syndrome. J Urol 2011;186:117–124. [PubMed: 21571326]

384. Kucyi A, Moayedi M, Weissman-Fogel I, et al. Enhanced medial prefrontal-default mode network functional connectivity in chronic pain and its association with pain rumination. J Neurosci 2014;34:3969–3975. [PubMed: 24623774]

385. Jiang Y, Oathes D, Hush J, et al. Perturbed connectivity of the amygdala and its subregions with the central executive and default mode networks in chronic pain. Pain 2016;157:1970–1978. [PubMed: 27168362]

386. Becerra L, Bishop J, Barmettler G, Kainz V, Burstein R, Borsook D. Brain network alterations in the inflammatory soup animal model of migraine. Brain Res 2017;1660:36–46. [PubMed: 28167076]

387. Schreiber KL, Loggia ML, Kim J, Cahalan CM, Napadow V, Edwards RR. Painful After Sensations in Fibromyalgia are Linked to Catastrophizing and Differences in Brain Response in the Medial Temporal Lobe. J Pain 2017;18:855–867. [PubMed: 28300650]

388. Loggia ML, Chonde DB, Akeju O, et al. Evidence for brain glial activation in chronic pain patients. Brain 2015;138:604–615. [PubMed: 25582579]

389. Vase L, Egsgaard LL, Nikolajsen L, Svensson P, Jensen TS, Arendt-Nielsen L. Pain catastrophizing and cortical responses in amputees with varying levels of phantom limb pain: a high-density EEG brain-mapping study. Exp Brain Res 2012;218:407–417. [PubMed: 22349560]

390. Hubbard CS, Khan SA, Keaser ML, Mathur VA, Goyal M, Seminowicz DA. Altered Brain Structure and Function Correlate with Disease Severity and Pain Catastrophizing in Migraine Patients. eNeuro 2014;1:e20. [PubMed: 25893216]

391. Loggia ML, Berna C, Kim J, et al. The Lateral Prefrontal Cortex Mediates the Hyperalgesic Effects of Negative Cognitions in Chronic Pain Patients. J Pain 2015.

392. Plazier M, Ost J, Stassijns G, De Ridder D, Vanneste S. Pain characteristics in fibromyalgia: understanding the multiple dimensions of pain. Clin Rheumatol 2015;34:775–783. [PubMed: 25048743]

393. Kim J, Loggia ML, Cahalan CM, et al. The somatosensory link in fibromyalgia: functional connectivity of the primary somatosensory cortex is altered by sustained pain and is associated with clinical/autonomic dysfunction. Arthritis Rheumatol 2015;67:1395–1405. [PubMed: 25622796]

394. Lazaridou A, Kim J, Cahalan CM, et al. Effects of Cognitive-Behavioral Therapy (CBT) on Brain Connectivity Supporting Catastrophizing in Fibromyalgia. Clin J Pain 2017;33:215–221. [PubMed: 27518491]

395. Coulombe MA, Lawrence KS, Moulin DE, et al. Lower Functional Connectivity of the Periaqueductal Gray Is Related to Negative Affect and Clinical Manifestations of Fibromyalgia. Front Neuroanat 2017;11:47. [PubMed: 28642688]

396. Gracely RH, Geisser ME, Giesecke T, et al. Pain catastrophizing and neural responses to pain among persons with fibromyalgia. Brain 2004;127:835–843. [PubMed: 14960499]

397. Seminowicz DA, Davis KD. Cortical responses to pain in healthy individuals depends on pain catastrophizing. Pain 2006;120:297–306. [PubMed: 16427738]

398. Elman I, Borsook D. Common Brain Mechanisms of Chronic Pain and Addiction. Neuron 2016;89:11–36. [PubMed: 26748087]

399. Borsook D, Edwards R, Elman I, Becerra L, Levine J. Pain and analgesia: the value of salience circuits. Prog Neurobiol 2013;104:93–105. [PubMed: 23499729]

400. Libby CJ, Glenwick DS. Protective and exacerbating factors in children and adolescents with fibromyalgia. Rehabil Psychol 2010;55:151–158. [PubMed: 20496969]

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

401. Goulart R, Pessoa C, Lombardi IJ. Psychological aspects of juvenile fibromyalgia syndrome: a literature review. Rev Bras Reumatol Engl Ed 2016;56:69–74. [PubMed: 27267336]

402. de Tommaso M, Sciruicchio V, Delussi M, et al. Symptoms of central sensitization and comorbidity for juvenile fibromyalgia in childhood migraine: an observational study in a tertiary headache center. J Headache Pain 2017;18:59. [PubMed: 28560539]

403. Kashikar-Zuck S, King C, Ting TV, Arnold LM. Juvenile Fibromyalgia: Different from the Adult Chronic Pain Syndrome? Curr Rheumatol Rep 2016; 18:19. [PubMed: 26984803]

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

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.

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

Table 1.

Biopsychosocial constructs influencing the pain experience

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