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Behavioral, Psychological, Neurophysiological, and Neuroanatomic Determinants of Pain

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Pain has historically been conceptualized as a linear relationship between an underlying injury or pathology and observed symptomatology. In other words, pain is expected to be proportional to the extent of tissue damage. However, much study has indicated extensive modulation of the pain signal, which may vary between individuals and across contexts. The emerging biopsychosocial model of pain[1,2] recognizes the important contributions of biological, psychological, and social modulators of pain. Indeed, there is an increasing emphasis on identifying the interrelationships between behavioral, psychological, neurophysiological, and neuroanatomic modulators, as well as their influence on clinical pain severity and impact, to optimize treatment outcomes for individual patients.

As one example of a common musculoskeletal pain condition, knee osteoarthritis is one of the leading causes of pain and disability in the United States and globally, accounting for millions of years lived with disability, substantially reduced quality of life, and hundreds of billions of dollars in direct and indirect costs every year in the United States alone[3–7]. Prominent symptoms of knee osteoarthritis include a decreased range of motion, stiffness, limitations in physical mobility, and function-limiting pain. As with many chronic pain conditions, the biological mechanisms underlying pain in knee osteoarthritis are complex and incompletely understood, and numerous studies have documented that objective indices of pathology such as radiographic markers of disease severity are relatively weak correlates of actual pain severity and disability[8–12]. For example, surveys of patients with knee osteoarthritis awaiting joint arthroplasty have revealed no significant association of radiographic scores with pain or function[13], suggesting that other patient-related factors, including a wide array of biopsychosocial mechanisms, may contribute to individual variability in the experience of joint pain in patients with osteoarthritis[14–18].

Biopsychosocial influences predominate in the realm of surgical pain as well. For example, total joint replacement is, for many patients in the later stages of osteoarthritis, an effective intervention to reduce pain and improve physical functioning[19–24]. However, recent decades have brought a growing recognition of the tremendous inter-patient variability in pain-

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related outcomes after surgical procedures[25–29]. Following nearly any operative procedure, including seemingly minor operations[30], a substantial percentage of patients have reported persistent postoperative pain[27,31,32]. Collectively, around one-third of patients experience moderate to severe acute pain following a surgical procedure[33], leading to increased exposure to opioids in the months after the surgical procedure[34]. Total knee arthroplasty outcomes show great variability, with some patients reporting full resolution of knee pain and large increases in physical capacity, and others reporting continuing, or even worsening, pain. Recent reviews have suggested that approximately 15% to 30% of patients undergoing total knee arthroplasty do not achieve satisfactory outcomes following the surgical procedure, prompting interest in identifying factors that predict positive or negative outcomes[4,35–39].

In this review, we will provide a non-exhaustive discussion of important behavioral, psychological, neurophysiological, and neuroanatomic factors that have been studied as contributors to bone and joint pain. The influence of these variables appears to generalize across conditions, having been studied across both operative and nonoperative populations.

Negative Affect

Common psychological variables including depression, anxiety, and general distress remain some of the most robust predictors of the development of chronic pain, especially musculoskeletal pain[15,40,41]. Indeed, high levels of general negative affect and pain-specific distress are both associated with poorer outcomes for treatments targeting pain relief[42,43]. Assessing psychosocial functioning preoperatively is also important among patients undergoing joint replacement surgery. Findings have indicated that patients with greater preoperative anxiety and depression report less benefit, more complications, and poorer function following total knee or total hip replacement[44–46]. Anxiety and catastrophizing are prominent risk factors[47,48], and recent systematic reviews have supported the importance of negative affective and cognitive processes[35,49,50]. For example, Sorel et al. reported poorer outcomes in patients who preoperatively had elevated scores on measures of pain catastrophizing, more symptoms of anxiety and depression, and somatization[49].

Catastrophizing

In addition to measures of general negative affect (i.e., depression and anxiety), there are pain-specific psychosocial processes that shape pain outcomes and treatment responses. Catastrophizing involves helplessness, rumination, and magnification of pain. Although catastrophizing is associated with general negative affect, it also uniquely and specifically influences pain-related outcomes. Indeed, pretreatment catastrophizing is the most important risk factor associated with poor treatment outcomes for pain-relieving interventions[51,52]. Preoperatively, catastrophizing is associated with amplified acute pain severity, with an elevated risk of persistent pain after a surgical procedure and with a higher likelihood of prolonged and high-dose opioid use after a surgical procedure[35,49,53–57]. Our own work has also suggested that catastrophizing promotes enhanced pain sensitivity and interferes with pain modulation in patients with a variety of pain conditions, including low back pain, osteoarthritis, fibromyalgia, and others[58–62].

Furthermore, functional neuroimaging studies suggest that catastrophizing is associated with altered brain responses to painful stimuli, as well as alterations in brain structure[61,63–76]. According to functional magnetic resonance imaging, greater catastrophizing is related to enhanced activation of the anterior insular cortex among both patients with and without chronic pain[77,78]. The results of other imaging studies have also acknowledged increased activity or connectivity in affectively oriented brain circuits and areas such as the amygdala[79,80]. We showed that patients with fibromyalgia demonstrated enhanced pain facilitation in the form of elevated painful after-sensations that were linked with reduced post-stimulus deactivation of the amygdala and with high levels of pain-related catastrophizing[70]. Patients with chronic back pain show exaggerated connectivity between the amygdala and central executive network structures, a finding that is strongest among patients who exhibit greater catastrophizing[81]. A recent neuroimaging review highlighted the maladaptive influence of catastrophizing on default mode network, salience-related, and pain-inhibitory neurocircuitry[72].

Expectations

Expectations, vital to placebo effects, also influence outcomes from active treatments including opioid analgesics[82], surgical procedures[83,84], and complementary and alternative medicine approaches such as acupuncture[85]. Indeed, both patient and provider expectations are potent predictors of treatment response for acupuncture[85,86]. Likewise, preoperative expectations for pain relief are associated with greater pain relief following lumbar spine surgery[87], and patient preoperative expectations are good predictors of both pain and function at 1 year following both total knee arthroplasty and total hip arthroplasty[88]. It is also important to note that expectations are modifiable and, thus, may be a good non-pharmacological therapeutic target to help to improve patient outcomes[89].

Sleep

Experimental, clinical, and epidemiologic studies have suggested that sleep disruption or deprivation has a variety of negative effects, both within the general population and also specifically in patients with pain, including enhancing pain sensitivity, reducing pain inhibition, exacerbating chronic pain severity and disability, and increasing the frequency and impact of daily musculoskeletal pain[90–92]. In longitudinal studies, individuals with a sleep disturbance are at an elevated long-term risk for developing clinically relevant pain, especially persistent musculoskeletal pain. Most researchers in the field have concluded that pain and sleep disruption exhibit reciprocal, bidirectional influences. A number of studies have indicated that greater preoperative sleep disturbance is associated with elevated indices of postoperative pain, including more severe acute postoperative pain following a surgical procedure, greater and more prolonged postoperative opioid consumption, and increased risk of persistent postoperative pain and disability, following a variety of operative procedures[31,93–95].

Somatosensory Function

The center of pain processing is the spinal cord, which involves a complex internal pharmacology to receive, process, and transmit input from afferent nociceptors to the brain. However, descending input from the brain also feeds into this processing, with 2 main mechanistic modulatory families: descending, anti-nociceptive (inhibitory) input[96], and descending faciliatory pathways[96–98]. Depending on the relative degree and balance of these opposing inputs, an augmentation or dampening of nociceptive transmission may occur. Quantitative sensory testing refers to a set of psychophysical methods used to quantify different aspects of the somatosensory system. It utilizes a standardized measurement of responses to calibrated, graded, innocuous, or noxious stimuli (generally mechanical or thermal) to diagnose and monitor sensory neuropathies and pain disorders, to investigate pain mechanisms, and to characterize individual differences in somatosensory functioning in various pain disorders[99–105]. As with psychosocial phenotyping, the application of quantitative sensory testing to measure and quantify sensitization-related processes has become increasingly common[10,106–113]. As reviews have noted, a tip in the balance toward enhanced facilitation and decreased inhibition has become evident in this testing, with findings including widespread hyperalgesia, elevated temporal summation of pain, and deficits in endogenous pain inhibition present in a substantial proportion of patients with knee osteoarthritis[106,108,110]. Variation in quantitative sensory testing measurements may have the potential to predict long-term, pain-related outcomes, including outcomes after joint replacement surgery[42,108,114]. Several studies have shown that indices of sensitization (e.g., high levels of temporal summation of pain, widespread evidence of low pain thresholds), assessed preoperatively, are associated with worse short-term and long-term outcomes following total knee arthroplasty, including elevated pain severity, greater opioid use, lower patient satisfaction, and reduced physical function[48,115–122]. Similarly, elevated temporal summation has been associated with pain after hip arthroplasty[118], after caesarean section[123], after thoracotomy[124], and after lumpectomy and mastectomy[60,125].

Physical Activity

Although rest and inactivity have historically been recommended for people experiencing acute and chronic pain (based on the hypothesis [now known to be inaccurate] that engaging in activity would potentially risk injury and slow healing time), regular physical activity has garnered recent attention as a beneficial treatment approach for people in pain. Indeed, various types of physical activity (e.g., cardiovascular training, resistance exercise, stretching and strengthening, movement therapies, and lifestyle-based activity interventions) are associated with lower pain severity ratings and improvements in physical function, psychological function, and quality of life for people with various chronic pain symptoms[126]. There is evidence for the benefit of postoperative physical therapy for recovery[127]. Similar benefits have been touted for the use of physical activity prior to the surgical procedure. For example, a recent meta-analysis found that participation in preoperative physical activity was associated with decreased pain following total knee arthroplasty and total hip arthroplasty[128]. Given the association between physical activity and improved pain outcomes, it is not surprising that there has been a movement toward prehabilitation. Prehabilitation involves enhancing functional capacity in preparation for a

surgical procedure[129] as a strategy to offset postoperative deconditioning and to speed recovery. Exercise may also enhance the effectiveness of some surgical interventions for pain, such as lumbar spine surgery, in which 1 study showed that patients participating in preoperative physiotherapy had a decrease in pain as well as improved quality of life and physical activity levels. However, only the improvements in physical activity were maintained following the surgical procedure[130]. There is more consistent evidence for the benefits of prehabilitation prior to total hip arthroplasty, total knee arthroplasty, and anterior cruciate ligament reconstruction, effectively increasing physical function, reducing pain, and improving strength, range of motion, and functional task performance prior to the surgical procedure, all of which are associated with a shorter hospital stay and faster recovery[131–138]. Patients who underwent prehabilitation were also more likely to meet their outcome expectations following total knee arthroplasty[139] and were less likely to be discharged to a long-term rehabilitation facility following total hip and total knee arthroplasty[134].

Coping

Individual differences in coping or the use of behavioral, emotional, and cognitive strategies used to manage stress may influence pain-related outcomes in important ways[140]. Indeed, pain coping is associated with pain outcomes including severity, adjustment, and psychological and physical functioning[141–144]. Active coping (i.e., techniques used to control pain or continue functioning despite pain) is associated with positive outcomes including positive affect, better psychological adjustment, and decreased depression[145–149]. However, passive coping strategies (i.e., surrendering control of one's pain) are linked to poor outcomes, including increased pain and depression[145,146,150], among patients with chronic pain. Among patients undergoing total knee arthroplasty, greater preoperative passive coping and less problem-focused coping are associated with more pain and worse functioning at 6 to 12 months following the surgical procedure[151,152]. In addition to these broad categorizations of coping, specific strategies are associated with pain-related outcomes. Catastrophizing and praying are associated with greater pain intensity, pain interference, and disability and worse physical function among patients with chronic pain conditions as well as healthy individuals without a history of chronic pain[15,62,153–157]. Preoperative coping may also impact postoperative pain and functioning among patients undergoing total knee arthroplasty. Guarding (a behavior that can involve tensing musculature, limping, or minimizing use to protect an area from additional pain), resting, and seeking social support are associated with greater pain and worse functioning prior to total knee arthroplasty[158].

Conclusions

Consistent with the biopsychosocial model, a broad range of factors contribute to and substantially modulate the experience of pain. These factors increase the likelihood of developing a persistent painful condition, but also contribute to the intensity of acute and chronic pain, as well as the related sequelae such as disability, physical functioning, and quality of life. As described in this review, it is vital to recognize that psychosocial functioning is not solely a secondary reaction to pain, but includes integral, interactive processes that shape the pain experience. Across disparate diagnoses and patient

populations, including those with musculoskeletal pain or undergoing a related surgical procedure, these psychosocial factors should be considered as potential risk factors, protective factors, and treatment targets that might be modulated to minimize the burden of pain.

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- Unlike the biomedical model, the biopsychosocial model recognizes the important contributions of biological, psychological, and social modulators of pain.
- This review provides a discussion of important behavioral, psychological, neurophysiological, and neuroanatomic factors that have been studied as contributors to bone and joint pain.
- We highlight the importance of negative affect, catastrophizing, expectations, sleep, somatosensory function, physical activity, and coping on pain and related outcomes.
- These psychosocial factors should be considered as possible risk and protective factors for the development of pain, as well as treatment targets that might be modulated to minimize the burden of pain.