

Pain Catastrophizing: Mechanisms and Clinical Implications

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Objective: This study aims to summarize current perspectives on the underlying mechanisms, conceptual frameworks, and potential clinical implications of pain catastrophizing (PC).

Methods: A narrative review approach was employed.

Results: Existing studies suggest that PC may be associated with functional activity in several brain regions, including the anterior cingulate cortex, amygdala, and prefrontal cortex, which are involved in pain perception and emotional regulation. From a psychosocial perspective, catastrophizing has been linked to psychological distress, social support, and behavioral responses to pain. Various interventions, including psychological therapies, pain neuroscience education, exercise-based approaches, and complementary therapies have been explored as potential strategies for addressing PC. However, findings across studies remain heterogeneous.

Discussion: This study integrates current evidence on neurobiological mechanisms and psychosocial models related to PC and discusses their potential clinical implications. By summarizing existing findings and highlighting areas of uncertainty, this work aims to provide a more comprehensive understanding of the multidimensional nature of PC and may inform future research and clinical approaches in chronic pain management.

Keywords: pain, pain catastrophizing, neurobiological mechanisms, psychosocial factors, chronic pain

Introduction

Psychosocial factors have increasingly been recognized as significant moderators and essential elements of chronic pain.¹ Among various psychological factors, catastrophism has received considerable empirical attention.^{1,2} Catastrophizing is generally regarded as a maladaptive cognitive pattern in response to stress, both within the chronic pain literature and in the broader psychological context, and is characterized by diminished confidence and a reduced sense of control.³⁻⁵ It is commonly observed among individuals with anxiety,^{6,7} depressive disorders,⁸ and chronic pain conditions.⁹

Pain catastrophizing (PC) is conceptualized as a maladaptive cognitive-emotional response characterized by an exaggerated negative orientation toward actual or anticipated pain experiences, encompassing three core dimensions: rumination, magnification, and perceived helplessness.¹⁰ Available evidence suggests that PC may account for a substantial proportion of the variance in the transition from acute to chronic pain, indicating its potential relevance in pain chronification.¹¹ PC has been associated with higher levels of perceived pain, longer hospital stays, and an increased risk of opioid misuse in patients undergoing total joint arthroplasty.^{12,13}

Previous studies have also reported associations between PC and a range of adverse outcomes, including functional disability, mood disorders, and substance use in patients with chronic musculoskeletal conditions.¹⁴⁻¹⁹ In addition, PC has been linked to poorer treatment adherence and unfavorable clinical outcomes.²⁰⁻²² Together, these findings suggest that PC may contribute to substantial individual and societal burden of chronic pain, potentially increasing the demand for analgesics and antidepressants and contributing to rising healthcare costs.^{23,24}



Current evidence suggests that PC may involve amplification of distress and pain-facilitating neural processes.²⁵ Individuals with higher levels of catastrophizing tend to show greater attention to pain signals and heightened emotional responses.²⁶ While increased pain awareness may facilitate early recognition of potential tissue damage in some contexts, excessive attention to pain-related cues may also contribute to maladaptive pain experiences.²⁷ Additionally, PC has been associated with alterations in neural mechanisms related to pain processing, attention, emotion, physical activity, and reduced top-down pain inhibition.^{28,29} However, the available evidence remains heterogeneous, and the underlying pathways are not yet fully understood.

Nevertheless, relatively few studies have systematically summarized PC from underlying mechanisms to clinical management. Therefore, integrating findings from functional neuroimaging with anatomical, physiological, and psychosocial investigations may help clarify the mechanisms of PC and its potential clinical implications. In this review, we proposed a framework for interpreting the influence of PC on clinical outcomes (Figure 1). Specifically, this review examines the mechanistic underpinnings, conceptual and psychosocial frameworks, and therapeutic approaches related to PC, with the aim of providing a clearer overview of current evidence and informing future research and clinical practice.

Methods

A narrative review was conducted to identify studies relevant to pain catastrophizing (PC), its underlying mechanisms, and clinical implications. Electronic databases, including PubMed, Web of Science, and Scopus, were searched for articles published up to March 2025. The search strategy combined terms related to PC and its associated mechanisms, including “pain catastrophizing”, “catastrophizing”, “pain perception”, “neurobiological mechanisms”, “cognitive mechanisms”, “behavioral mechanisms”, and “clinical management”. In addition, the reference lists of relevant articles were manually screened to identify further studies.

Studies were considered eligible if they examined PC or related cognitive processes and reported findings on neurobiological, psychological, behavioral, or clinical aspects relevant to the scope of this review. Only articles published in English in peer-reviewed journals were included. Conference abstracts, editorials, commentaries, and studies not directly relevant to the topic were excluded.

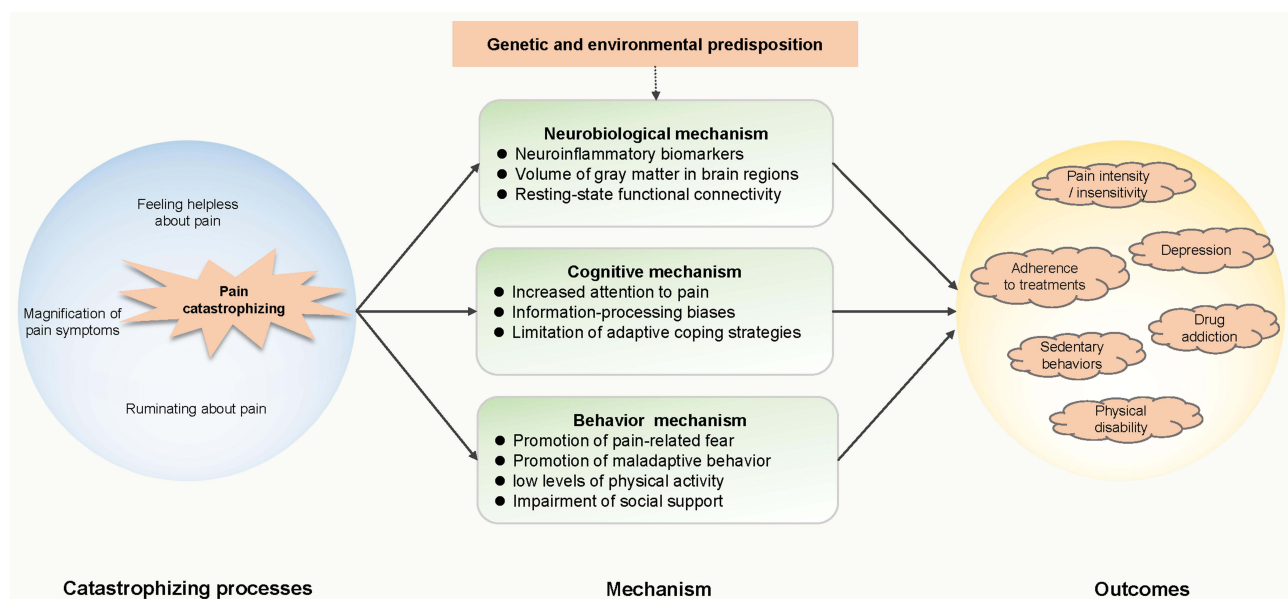


Figure 1 Conceptual framework of pain catastrophizing and its impact on clinical outcomes. The schematic illustrates the self-perpetuating cycle of pain catastrophizing (magnification, rumination, and helplessness) and its influence on adverse clinical prognosis. Underpinned by genetic and environmental predispositions, this cognitive-emotional process may affect clinical endpoints—including pain intensity, depression, and physical disability—through three primary mediating pathways: neurobiological alterations, cognitive biases, and maladaptive behavioral patterns.

Titles and abstracts were screened first, followed by full-text review of potentially relevant articles. Study selection was guided by relevance to the objectives of this review, with emphasis on three main domains: underlying mechanisms, conceptual and psychosocial frameworks, and clinical management strategies related to PC. Given the narrative nature of this review, studies were selected to provide a representative overview of key themes rather than an exhaustive systematic synthesis. Owing to the heterogeneity of study designs, populations, and outcome measures, findings were synthesized narratively.

Neurobiological and Psychosocial Mechanisms of Pain Catastrophizing

Neurobiological Mechanisms of Pain Catastrophizing

Molecular Biomarkers Associated with Pain Catastrophizing

Neurobiological mechanisms may contribute to PC (Figure 2). Both genetic and environmental factors have been suggested to influence the neurobiology of pain, potentially contributing to individual differences in pain perception, emotional regulation, and coping strategies. Pain experience is shaped by prior exposures and learning processes that affect cognitive, emotional, and pain modulatory systems.

Emerging evidence suggests that neuroinflammatory processes may be involved in the biological pathways associated with PC.³⁰ For example, in veterans with chronic residual limb pain, higher levels of PC were associated with increased levels of IL-8, IL-12, TNF- β , PIGF, and ICAM-1, as well as lower levels of IL-13.³⁰ In patients with bladder pain syndrome, higher catastrophizing scores have been associated with lower urinary levels of BDNF and VEGF.³¹ In addition, reduced expression of IL-1 α and Granzyme-B has been reported in arthritis patients with elevated PC, suggesting possible involvement in inflammatory pathways related to pain and disease progression.³² Experimental studies have also suggested that GM-CSF may participate in pain-related processes in animal models.³³

However, biomarker findings related to PC remain heterogeneous across pain conditions and study populations. These differences may reflect variation in study design, sample source, and underlying pain mechanisms. Although several candidate markers have been proposed, current evidence remains preliminary, and their specific roles in PC require further validation.

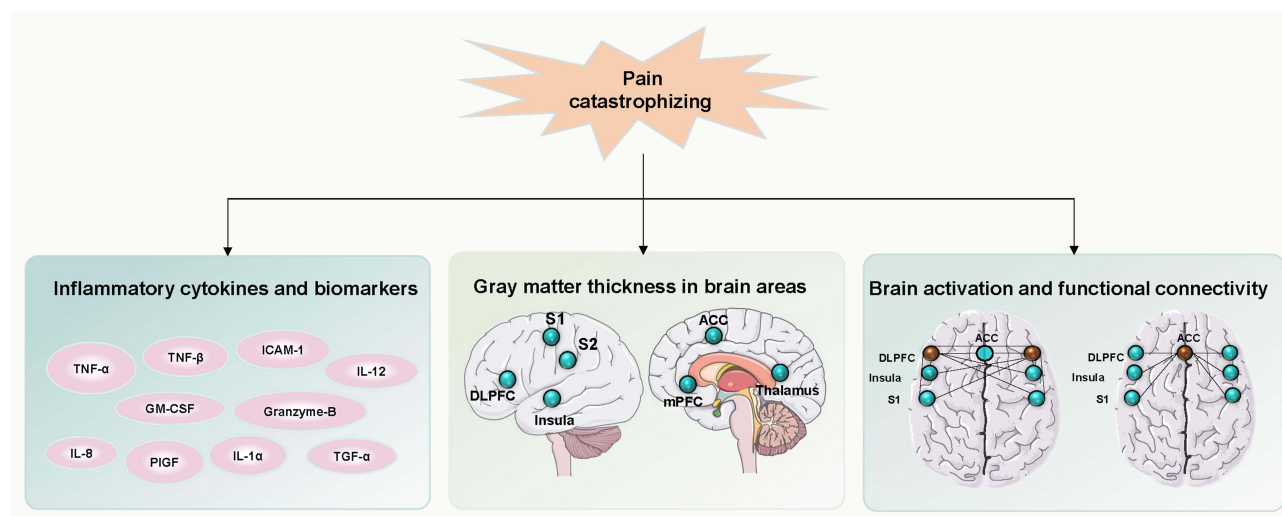


Figure 2 Proposed neurobiological mechanisms associated with pain catastrophizing. The schematic illustrates the multi-dimensional neurobiological framework of pain catastrophizing: Systemic inflammation, characterized by pro-inflammatory cytokines and biomarkers; Structural alterations, indicated by variations in cortical thickness across key pain-processing regions (e.g., S1, S2, ACC, DLPFC); and Functional dysconnectivity, reflecting disrupted neural network communication within the pain matrix. These components may contribute to the transition from acute pain to chronic pain states.

Abbreviations: S1, primary somatosensory cortex; S2, secondary somatosensory area; DLPFC, dorsolateral prefrontal cortex; mPFC, medial prefrontal cortex; ACC, anterior cingulate cortex; ICAM-1, intercellular adhesion molecule 1.

Brain Regions and Functional Connectivity Associated with Pain Catastrophizing

PC has been associated with structural and functional alterations in several brain regions involved in pain processing and emotional regulation. Previous neuroimaging studies have reported that PC is correlated with changes in gray matter morphology and density in multiple brain areas.³⁴ For example, a negative association between PC and cortical thickness in the dorsolateral prefrontal cortex (DLPFC) has been observed in patients with irritable bowel syndrome.³⁵ Similar relationships have been reported in the nucleus accumbens and the left anterior insula among individuals with fibromyalgia.³⁶ Comparable structural associations have also been identified in other regions, including the primary somatosensory cortex (S1), anterior midcingulate cortex (ACC), and prefrontal cortex (PFC) in patients with migraine.³⁷ One study also suggested that reductions in PC following CBT were associated with increased gray matter in regions involved in pain perception (insula, ACC, S1, and PFC) and pain modulation (DLPFC).³⁸

PC has also been associated with alterations in resting-state functional connectivity (rsFC),³⁹ particularly in brain regions related to pain perception, pain cognition, and the default neural network (DMN).⁴⁰ A previous study⁴¹ reported a positive association between pain rumination and activity in regions involved in pain perception, including S1, the anterior insula, and the thalamus, in patients with temporomandibular disorders. Altered functional connectivity between the medial prefrontal cortex (mPFC) and other regions—including the posterior cingulate cortex (PCC), precuneus, thalamus, retrosplenial cortex, and periaqueductal gray (PAG) has also been reported in relation to PC.^{36,37,42}

Catastrophizing-related cognition has also been linked to altered DMN activity,⁴³ and some studies have suggested that individuals with elevated PC show stronger connectivity between the DMN and motor-related networks.^{44,45} Conversely, reductions in pain catastrophizing following treatment have been associated with decreased DMN connectivity.⁴⁶ The DMN is thought to interact with circuits of the medial system, which are involved in affective and evaluative aspects of pain processing. Self-referential tasks have been shown to activate the DMN, particularly in individuals with depressive symptoms.⁴⁷ The insula, a brain region associated with interoception and the subjective awareness of bodily states, has also been reported to show heightened activation in individuals with elevated levels of PC. Additionally, rsFC between the posterior cingulate cortex (PCC) and the DLPFC was positively associated with PC in individuals with migraine, whereas a negative correlation was observed in control subjects.³⁷

Task-based neuroimaging studies have also explored the neural correlates of PC using experimental pain paradigms. Despite differences in experimental stimuli (such as pressure pain and saline infusion), several brain regions have been consistently implicated, including the DLPFC,^{48,49} insula,^{48,50,51} ACC,^{23,42,52} PCC,^{51,53} and portions of the supplementary motor area.⁵⁰ Furthermore, anticipatory brain activity in the anterior and ventrolateral prefrontal cortex and posterior parietal cortex has been negatively associated with PC.^{54,55} In addition, a working memory study reported a positive association between PC and medial prefrontal cortex responses to increasing task load.⁵⁶

However, findings across studies remain heterogeneous, and the direction and magnitude of these associations may vary across pain conditions and experimental paradigms. Overall, although neuroimaging findings suggest that PC is associated with alterations in brain structure and connectivity, the underlying mechanisms remain incompletely understood.

Psychosocial Theories and Models of Pain Catastrophizing

Several theoretical frameworks have been proposed to explain the role of PC in pain perception and related outcomes.²⁶ These frameworks include appraisal theory, the communal coping model, the fear-avoidance model, the bio-psychomotor model, and the behavioral inhibition/activation system (BIS-BAS) model (Figure 3).

Appraisal Theory

PC has been conceptualized as part of the cognitive appraisal process and may influence the coping strategies that individuals subsequently adopt.⁹ Previous studies have suggested that individuals with elevated PC may exhibit a negative cognitive bias.⁵⁷ Such biases may involve selective attention to negative information and reduced attention to positive or neutral stimuli, thereby amplifying the perceived severity of pain and related emotional distress. In addition, individuals with high levels of PC may show cognitive distortions, including overgeneralization and exaggerated expectations regarding negative outcomes.

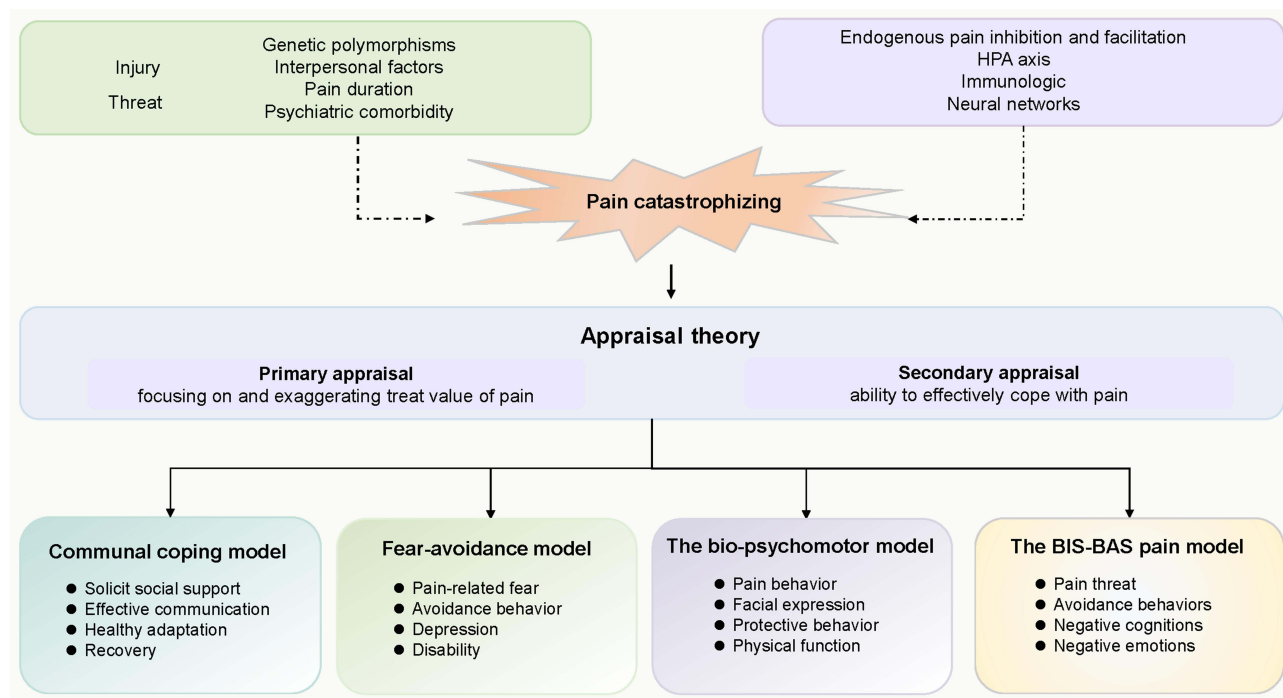


Figure 3 Psychosocial framework and theoretical models of pain catastrophizing. The schematic depicts the theoretical underpinnings of pain catastrophizing within the context of appraisal theory. (Top) Pre-existing genetic, environmental, and neuro-endocrine factors are potential contributors to pain catastrophizing. (Middle) According to the appraisal model, pain catastrophizing involves primary appraisal (overestimation of pain threat) and secondary appraisal (underestimation of coping resources). (Bottom) These cognitive processes manifest through four validated psychosocial models: Communal Coping, Fear-Avoidance, Bio-psychomotor, and BIS-BAS models, which may help explain behavioral and recovery outcomes.

Abbreviations: HPA, hypothalamic-pituitary-adrenal axis; BIS, behavioral inhibition system; BAS, behavioral activation system.

For example, children who catastrophize in response to pain may also display similar cognitive-emotional patterns when responding to non-pain-related stressors. This generalized tendency has been suggested as one possible explanation for the observed associations between PC and psychological conditions such as anxiety and depression. This interpretation is also supported by findings from a previous meta-analysis.⁵⁸ However, it should be noted that the relationship between PC and negative mood states may, in part, reflect conceptual and measurement overlap between these constructs. Therefore, although associations between PC, anxiety, and depression have been consistently reported, the directionality and underlying mechanisms of these relationships remain unclear.

Communal Coping Model

Sullivan et al⁵⁹ proposed PC as a key component in the communal coping model, which has been suggested as a framework for understanding adjustment to chronic illness.⁶⁰ Within this framework, PC is conceptualized as a coping strategy through which individuals communicate their pain experience in order to elicit support, empathy, and assistance from others.⁶¹ When individuals experience pain-related distress, increased attention to pain and its consequences may contribute to a greater focus on pain interference.⁵⁹

Individuals with higher levels of PC may therefore be more likely to engage in communal coping behaviors, such as expressing distress through verbal or non-verbal cues (eg, grimacing or vocalization), particularly in the presence of others. Such behaviors may function to attract attention and solicit emotional or instrumental support from the social environment. In this context, the expression of pain may influence others' perceptions and responses, potentially enhancing perceived empathy and social connectedness.⁶²⁻⁶⁴ This pattern may be adaptive in certain contexts, particularly when adequate social support is available and responsive.

Nevertheless, individuals who have lower levels of catastrophizing may adopt solitary coping strategies and downplay their discomfort in order to avoid drawing social attention. However, current evidence remains limited, and it is difficult to distinguish adaptive support-seeking from maladaptive reinforcement of pain behaviors.

Fear-Avoidance Model

The fear-avoidance model (FAM) provides a widely used framework for understanding the relationship between catastrophizing and physical function.^{65–68} According to this model, individuals who interpret pain as threatening may engage in PC and threat-related appraisals. Such interpretations may increase fear of future pain, which in turn may lead to avoidance of activities and ultimately contribute to disuse and disability.⁶⁸

Within this framework, PC has been proposed as an important cognitive factor associated with chronic pain and psychological distress, including depressive symptoms.^{69,70} The model suggests that individuals with elevated levels of PC may be more vulnerable to persistent pain-related problems following injury, thereby contributing to a self-perpetuating cycle. Individuals with higher levels of catastrophizing may show heightened responses to actual or perceived threats, resulting in greater attention to pain sensations and prior negative experiences.⁶⁹

Moreover, this model suggests that PC may interfere with engagement in health-related exercise and rehabilitation, which are often important components of chronic pain management.^{24,38,71} This may reduce the likelihood that individuals will use exercise as an effective self-management strategy. As a result, PC may be associated with poorer functional outcomes, although the strength and direction of this relationship may vary across studies. Overall, the role of PC within this model may differ across individuals and clinical conditions.

The Bio-Psychomotor Model

Although catastrophizing has been associated with heightened pain-related communicative behaviors, particularly facial expressions of pain,^{72–74} the bio-psychomotor model provides a useful framework for understanding its relationship with physical function. Within this model, pain behaviors are conceptualized as integral components of the pain system that serve communicative, protective, and social-relational functions.⁷⁵ From this perspective, individuals who interpret pain catastrophically may be more likely to adopt protective behaviors (eg, guarding, bracing), which may contribute to reduced physical function over time.⁷⁶ However, the specific contribution of catastrophizing within this model remains difficult to disentangle from other interacting psychological and motor factors.

BIS-BAS Model

PC has been linked to the Behavioral Inhibition System (BIS) component of the BIS-BAS pain model.⁷⁷ When pain is perceived as a threat, activation of the BIS may be associated with avoidance behaviors (eg, reduced movement), negative cognitions (eg, catastrophic thinking), and anxiety, which may in turn contribute to heightened pain perception. PC has been proposed as an important cognitive factor related to BIS activity and has been associated with greater pain intensity and distress. However, the underlying neurobiological pathways remain to be fully elucidated.

Clinical Management Strategies

Various strategies have been implemented in clinical settings to address PC, and this review focuses on three main therapeutic approaches (Figure 4).

Pharmacotherapeutics

Non-steroidal anti-inflammatory drugs (NSAIDs) are commonly used as first-line agents for pain management because of their analgesic and anti-inflammatory effects.⁷⁸ Opioids are typically reserved for moderate to severe pain requiring central analgesia.⁷⁹ However, their use is limited by potential adverse effects, including gastrointestinal complications, renal impairment, and platelet dysfunction, as well as the risk of dependence.

Chronic pain frequently coexists with comorbid conditions such as anxiety, depression, and sleep disturbances, which may require adjunctive pharmacotherapy. Nevertheless, there remains a dearth of standardized guidelines regarding optimal dosing and the management of addiction risk in clinical practice. Previous studies have reported associations between PC and substance abuse behaviors, suggesting that interventions targeting PC may help reduce related adverse outcomes.⁸⁰ The mechanism by which PC increases substance use is hypothesized to be driven by the motivation to achieve relief.⁸¹

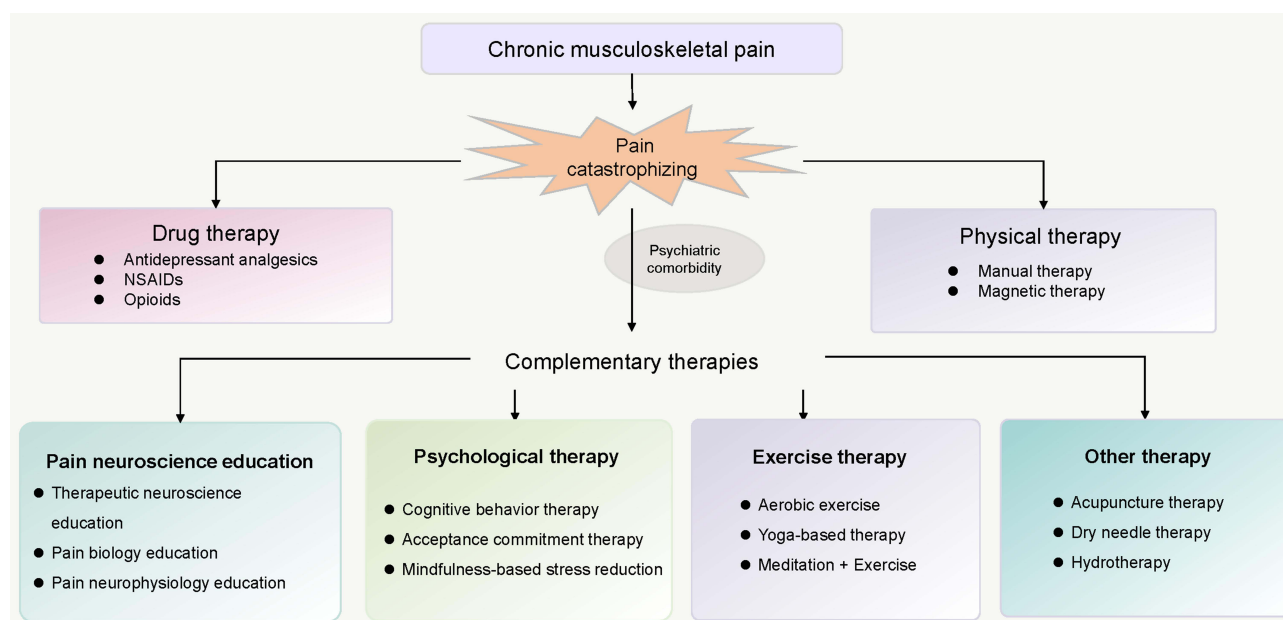


Figure 4 Clinical management strategies for chronic musculoskeletal pain associated with pain catastrophizing. The schematic illustrates the therapeutic pathways targeting chronic musculoskeletal pain through pain catastrophizing (central node). Treatments are structured across conventional (Drug and Physical therapy) and complementary approaches (PNE, psychological, exercise, and other adjunctive therapies). Solid arrows indicate established interventional routes directed at mitigating the reinforcement cycle of catastrophizing.

Participants with high levels of catastrophizing have been reported to have lower awareness of opioid-related risks, including side effects and dependence, which may contribute to increased expectations for opioid prescriptions.^{81,82} However, current evidence does not support a definitive relationship between opioid dependence and the reinforcement of catastrophizing behavior.

Some pharmacological agents have shown potential associations with reductions in both pain severity and catastrophizing. For example, a prospective study suggested that the non-opioid agent (ziconotide) may improve neuropathic pain and related emotional components, including disability and catastrophizing.⁸³ Similarly, limited evidence from specific populations indicates that atypical antipsychotic risperidone for ASD irritability and an ADHD medication, atomoxetine may be associated with improvements in pain, anxiety, depression, and pain-related catastrophic thinking.⁸⁴ However, these findings remain preliminary and may not be generalizable.

Furthermore, the clinical use of glucocorticoids remains variable, with both overuse and underuse reported in practice. Antidepressants are also commonly used in pain management, although their role in targeting PC specifically remains unclear. Overall, pharmacological strategies should be individualized, taking into account patient characteristics, comorbidities, and potential risks.

Manual Therapy

The effects of manual therapy have been attributed to neurophysiological mechanisms as well as placebo-related responses. It may also be associated with improvements in psychological resilience, including potential changes in patients' perceptions and beliefs.^{85,86} Cervical manipulation, a commonly used manual therapy, has been suggested to influence sensory and motor processing and may help modulate communication between the brain and spinal cord.^{87,88} However, previous studies have reported no significant short-term improvement in PC among patients with fibromyalgia, whereas some studies have observed statistically significant changes at longer-term follow-up (eg, one year).⁸⁹

These inconsistent findings may be related to differences in treatment duration, patient characteristics, and study design.^{90,91} Although previous systematic reviews^{86,92,93} have examined the effects of manual therapy on psychological and pain-related outcomes, evidence specifically addressing PC remains limited. Overall, further research is needed to clarify the effectiveness

and consistency of different forms of manual therapy in relation to PC across short-, intermediate-, and long-term follow-up periods.

Electrostimulation

Transcutaneous electrical nerve stimulation (TENS) and transcranial direct current stimulation (tDCS) have been extensively investigated as electrotherapeutic modalities for the management of chronic pain syndrome, primarily due to their potential to modulate nociceptive processing. These interventions are thought to exert their effects by activating descending central inhibitory pathways and attenuating excitatory neurotransmission within the dorsal horn of the spinal cord.⁹⁴ Notably, anodal tDCS applied to the left primary motor cortex has been associated with improvements in pain intensity and sleep quality, possibly through its neuromodulatory effects on central pain processing.⁹⁵

Although the existing evidence remains limited, anodal tDCS applied to the DLPFC has been associated with improvements in both pain perception and cognitive performance in patients with fibromyalgia.^{96,97} However, findings are not consistent. For example, a trial combining multicomponent therapy (including aerobic training, relaxation, education, and swimming) with electrostimulation did not demonstrate additional reductions in PC compared to multicomponent treatment alone.⁹⁸ This suggests that observed improvements in PC may be primarily driven by non-electrostimulation components, although the specific contribution of each component remains unclear.

In addition, higher levels of PC have been associated with lower adherence to treatment, including medication use, clinical follow-up, and engagement in physical therapy.⁹⁹ This may further influence treatment outcomes and complicate the interpretation of intervention effects.

Psychotherapy

Psychological therapies encompass diverse modalities aimed at addressing pain syndromes. As a typical psychotherapy, CBT combines the principles of fundamental cognitive and behavioral techniques to modify maladaptive thoughts and behaviors, thereby supporting adaptive changes in pain-related outcomes.^{100–104} It is targeted at specific problems such as pain interference, psychological distress and sleep disturbances.^{105,106} CBT has been associated with improvements in psychological symptoms in individuals with chronic musculoskeletal conditions.^{107,108} As an extension of CBT, acceptance and commitment therapy (ACT) focuses on enhancing psychological flexibility and promoting acceptance of pain-related experiences.^{109,110} Similarly, mindfulness-based stress reduction (MBSR) is a structured training program that emphasizes psychological flexibility. Specifically, it aims to help patients become more aware of the present experience and contextual changes, as well as to evaluate their physical and psychological circumstances in a more accepted and nonjudgmental way.^{111,112}

Previous reviews have suggested that CBT may reduce PC among individuals with various chronic musculoskeletal disorders.¹¹³ In clinical practice, CBT typically involves active patient participation in structured interventions delivered over 8–12 sessions. One study reported greater improvements in PC scores in the CBT group compared with a health education control at 3-month follow-up, with a mean reduction of 10.94 points (approximately 48.4% from baseline).¹¹⁴

The association between PC and psychological comorbidity suggests that interventions targeting catastrophic thinking may also potentially alleviate anxiety and depressive symptoms. A prior systematic review has also indicated that ACT, administered individually or in groups, may improve function, anxiety, and depression in patients with chronic pain.¹¹⁵

MBSR training has also been suggested to modulate immune-inflammatory pathways in conditions such as fibromyalgia. Some studies have reported a trend toward improvement in the pro/anti-inflammatory (IL-6/IL-10) ratio in fibromyalgia patients who attend six or more sessions of MBSR. However, these findings remain preliminary. Despite the potential benefits of these psychotherapeutic interventions, time, cost, and therapist availability may impede patient access.¹¹⁶ There is a need for more accessible and scalable approaches, particularly for patients with higher levels of PC.

Other psychotherapeutic approaches, such as hypnotherapy¹¹⁷ and guided imagery therapy,¹¹⁸ have also been explored, although they typically require delivery by trained professionals. In clinical practice, interventions should be individualized based on patient characteristics and clinical context.

Pain Neuroscience Education

Education enables individuals to better understand their condition and may facilitate self-management.^{119,120} Pain neuroscience education (PNE), a form of biopsychosocial education, aims to help individuals reconceptualize their pain experience. Multiple terminologies for PNE have been employed in the literature, such as Explain Pain,^{121,122} therapeutic neuroscience education,¹²³ pain biology education,¹²⁴ and pain neurophysiology education.¹²⁵

Previous research has suggested that PNE may modulate maladaptive pain perceptions and reduce PC and fear-avoidance behaviors in patients with chronic low back pain,¹²⁶ osteoarthritis,^{127,128} and fibromyalgia.¹²⁹ A fundamental tenet of PNE posits that pain perception does not always correspond directly to the extent of tissue damage.¹²¹ The traditional format of PNE is educational booklets, videos, or manuals, either individually or in group settings. However, some studies have reported limited long-term effects of PNE on PC reduction in fibromyalgia.¹³⁰ Generally, fibromyalgia symptoms are often considered to be related to altered threat processing within neural networks, which may be influenced by maladaptive beliefs, cultural factors, and prior experiences.¹²⁹ In contrast, structured face-to-face PNE interventions have shown more favorable outcomes in some studies, possibly by facilitating changes in maladaptive cognitions and health perceptions.¹³⁰ PNE has also been proposed to support gradual re-exposure to activities by modifying maladaptive interpretations of pain and symptoms.¹³¹

A previous systematic review has suggested that PNE may be associated with improvements in pain, psychological distress, physical activity, and healthcare utilization.¹³² Given its relatively low cost, PNE may represent a promising complementary approach for reducing reliance on pharmacological therapy.¹³³ However, further research is needed to optimize its delivery and determine its long-term effectiveness.

Exercise Therapy

Among conservative non-pharmacological therapies, exercise therapy, particularly aerobic exercise, has emerged as a widely accepted modality due to its cost-effectiveness and potential benefits in improving sleep quality and mood disorders.^{134–136} Exercise therapy is widely recognized as a cornerstone in the management of chronic musculoskeletal pain and is consistently endorsed in clinical practice guidelines.^{137,138} Regular exercise may influence hypothalamic-pituitary function and neurotransmitter levels, particularly endorphins, which are linked to improved pain sensitivity and psychological symptoms. In addition, physical activity may promote the production of anti-inflammatory cytokines such as IL-10 and reduce TNF- α levels, as reported in healthy populations¹³⁹ and in patients with cardiovascular and autoimmune diseases.^{140–142}

Additionally, increasing acceptance may have substantial effects on the rehabilitation of severe pain.^{61,143} Exercise therapy combined with PNE (eg, twice weekly sessions of 60 minutes over 10 weeks) has been associated with greater improvements in PC and disease impact than exercise therapy alone in the treatment of chronic musculoskeletal pain.^{144–146} It is possible that PNE as an adjunct reduces participants' fear and anxiety regarding movement, thereby enhancing exercise engagement and reducing the affective–emotional burden of pain.¹²¹ This effect, in conjunction with mechanisms of exercise-induced hypoalgesia, may explain why these interventions produce greater benefits when combined.¹⁴⁷ Therefore, future research should consider the impact of exercise dosage and cumulative exposure to PNE on disability outcomes.¹⁴⁸

Moreover, lack of adherence is common among patients with chronic conditions. Kinesiophobia represents a major barrier to exercise and is associated with more sedentary behavior.^{137,149} It remains challenging for patients to maintain consistent exercise routines and incorporate them into daily life. Practice-based interventions in natural settings may help improve adherence to physical activity.^{150–152} In addition, interventions incorporating meditation and mindfulness-based breathing strategies into home-based exercise programs, such as yoga, have been associated with reductions in PC.¹⁵³ These approaches may enhance awareness of maladaptive responses and make physical activity more tolerable, meaningful, and sustainable.¹⁵³

Furthermore, for patients with restricted mobility, such as those with spinal cord injury, hydrotherapy has been reported as a potentially beneficial intervention.¹⁵⁴ This modality may reduce mechanical stress on musculoskeletal structures and improve peripheral circulation and joint mobility. Some studies have suggested that this approach may improve neuropathic pain symptoms and reduce PC.

Regular exercise is generally beneficial for overall health. However, promoting sustained engagement remains a key challenge. The implementation of these interventions should consider practical constraints, economic factors, and resource utilization within healthcare systems.

Other Complementary Therapies

Several complementary therapeutic modalities have been explored in relation to PC. Acupuncture,¹⁵⁵ a systemic intervention derived from traditional Chinese medicine, is thought to exert analgesic effects through multiple mechanisms, including stimulation of specific acupoints that may influence neural transmission and circulation. Specifically, it has been suggested that acupuncture may reduce pain-related cognition and PC, potentially through modulation of neural activity and the release of endogenous opioids such as endorphins. Additionally, dry needling therapy^{156,157} targets myofascial trigger points in muscles and has been associated with improvement in PC. However, these interventions typically require delivery by trained practitioners to ensure safety and appropriate application.

Conclusion

PC has emerged as an important cognitive–emotional construct associated with pain perception, psychological distress, and functional outcomes in individuals with chronic pain. Growing evidence suggests that PC may influence pain experiences through complex interactions involving neurobiological mechanisms, psychosocial processes, and maladaptive behavioral responses. Nevertheless, the strength and consistency of the available evidence vary across studies, and the underlying pathways remain incompletely understood.

Recent research integrating neuroimaging findings, psychological assessments, and inflammatory biomarkers has provided preliminary insights into the potential mechanisms linking catastrophizing to altered pain processing. These findings highlight the multifactorial nature of PC and suggest that cognitive and behavioral factors should be considered when interpreting pain-related outcomes. Interventions such as PNE, CBT-based psychological approaches, and structured physical activity have been associated with reductions in catastrophizing and improvements in some pain-related outcomes in certain populations. Multidisciplinary care models integrating medical and nursing perspectives may further support patient education, self-management, and individualized treatment planning.

Despite these advances, current evidence remains heterogeneous and, in some cases, limited by methodological variability. Future longitudinal and mechanistic studies are needed to clarify causal relationships and to determine how interventions targeting catastrophizing may contribute to improved clinical outcomes across different pain conditions.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors declare no conflicts of interest in this work.

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