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Beyond biomechanics: a narrative review on the cognitive, emotional, and metacognitive mechanisms of exercise in chronic pain

Gianluca Regazzo,[1,2] Daniele Coraci,[1,2] Stefano Masiero[1,2]

[1]Section of Rehabilitation, Department of Neuroscience-DNS, University of Padova, Italy; [2]Physical Medicine and Rehabilitation School, Department of Neuroscience, University of Padua, Italy

Abstract

Chronic pain is increasingly recognized as a multidimensional condition sustained by interacting cognitive, behavioral, emotional, attentional, and metacognitive processes. Within this framework, physical exercise has gained relevance as a non-pharmacological intervention that may influence psychological mechanisms involved in pain persistence and disability. This narrative review provides an updated synthesis of recent evidence on the effects and mechanisms of physical exercise on cognitive, behavioral, emotional, and metacognitive dimensions of chronic pain. A structured literature search was conducted across PubMed/MEDLINE, Scopus and Web of Science, focusing on peer-reviewed studies published between January 2016 and December 2025. Evidence was synthesized narratively across mechanistic domains, integrating findings from rehabilitation science and clinical psychology. The literature suggests that exercise may act as an active experiential intervention capable of targeting several psychosocial factors in chronic pain. Across studies, exercise was associated with reductions in catastrophizing and maladaptive pain beliefs, improvements in pain self-efficacy, and attenuation of kinesiophobia. Exercise may also modulate attentional bias and hypervigilance, and psychologically informed exercise approaches appear particularly relevant for metacognitive processes, including reduced rumination, increased decentering, and improved non-reactive awareness of bodily sensations. In parallel, exercise shows beneficial effects on emotional aspects of chronic pain, likely mediated by both neurobiological and psychosocial mechanisms pathways. Overall, physical exercise should be conceptualized as a comprehensive intervention capable of acting on cognitive, behavioral, emotional, and metacognitive processes that sustain chronic pain. Future research should clarify modality-specific mechanisms,

identify moderators of response and incorporate process-based outcomes to support personalized exercise prescriptions.

Key words: chronic pain, physical exercise, catastrophizing, kinesiophobia, self-efficacy.

Chronic pain, defined as pain persisting for more than three months, represents a pervasive global health challenge. Affecting approximately 20% to 40% of the adult population worldwide, this condition extends far beyond physiological nociception to encompass profound alterations in central nervous system function, cognitive processing, and emotional regulation.[1] Historically, the biomedical model viewed pain as a direct symptom of tissue pathology; however, the frequent dissociation between objective tissue damage and the subjective experience of pain has necessitated a fundamental shift toward the biopsychosocial model. Within this contemporary framework, chronic pain is conceptualized not merely as a sensation but as a dynamic, subjective experience constructed by the brain.[2] Specifically, current literature emphasizes the fundamental role of maladaptive cognitive, metacognitive and emotional processes in the chronification and maintenance of pain.[3] For example, neuroimaging studies shows that specific structural and functional alterations in brain regions implicated in pain processing are associated with maladaptive cognitive patterns such as catastrophizing.[4] This bidirectional interplay between psychological states and neuroplastic changes suggests that interventions targeting emotional regulation and cognitive-behavioral processes may help to reduce maladaptive brain reorganization and attenuate pain perception. Furthermore, positive cognitive factors such as self-efficacy play a protective role in chronic pain by modulating brain regions involved in pain processing, particularly the Anterior Cingulate Cortex (ACC) and prefrontal cortex, that exerts top-down control over nociceptive signals.[5] Functional imaging studies show that better executive functions and cognitive control enhance activation in prefrontal areas during tasks that reduce pain perception, supporting the role of these regions in cognitive modulation of pain.[6]

In this biopsychosocial framework, physical exercise is increasingly recognized as a key component in the management of chronic pain, given its potential to influence not only physical function but also the cognitive and affective processes that shape pain perception.[7] Regular physical activity induces significant neurophysiological adaptations that support cognitive and emotional benefits and lead to enhanced top-down inhibition of pain signals, thanks to mechanisms involving the release of growth factors that promote neuroplasticity, neurogenesis, and angiogenesis.[8] For example, emerging evidence suggests that in Complex Regional Pain Syndrome exercise-based interventions may act through mechanisms that extend beyond peripheral tissue conditioning, engaging central neurophysiological and neuroimmune pathways implicated in pain chronification and functional impairment.[9] Overall, these findings support the role of structured physical exercise in addressing both physical function and maladaptive psychological factors that influence chronic pain outcomes; beyond its role as a mechanical stimulus for tissue repair, exercise acts as a neurobehavioral intervention that challenges maladaptive beliefs, regulates affective distress, and induces neuroplastic changes in pain-modulatory brain regions.[10]

Therapeutic value of physical exercise therefore also lies in its capacity to influence how pain is interpreted, anticipated, emotionally processed, and behaviorally managed. Accordingly, exercise may be better understood not simply as a rehabilitative adjunct, but as a central component of chronic pain management strategies aimed at restoring function, reducing disability, and promoting more adaptive engagement in daily life despite the persistence of pain. To date, however, the literature addressing these issues remains relatively scarce, heterogeneous, and not yet fully systematized, underscoring the need for more structured and comprehensive research. Within this context, the present narrative review seeks to provide an updated synthesis of the available literature on the effects of physical exercise on the cognitive, behavioral, emotional, and metacognitive dimensions of chronic pain, while also exploring the mechanisms through which exercise may influence these processes.

Materials and Methods

This manuscript is a narrative (non-systematic) review aimed at providing a comprehensive overview of the cognitive and emotional mechanisms underlying the therapeutic effects of exercise on chronic pain. The objective was to integrate contemporary evidence from rehabilitation sciences and

psychology within a multidimensional framework of pain modulation. A structured literature search was conducted across major electronic databases, including PubMed/MEDLINE, Scopus, Web of Science, and PsycINFO, to minimize database-specific bias and ensure broad interdisciplinary coverage. The search was restricted to peer-reviewed articles published between January 2016 and December 2025, in order to capture the most recent theoretical and mechanistic advancements in the field, while retaining earlier seminal contributions when conceptually foundational. Search terms were organized into three primary conceptual clusters: i) population (e.g., chronic pain, nociplastic pain, central sensitization); ii) intervention (e.g., physical exercise, resistance training, aerobic exercise, mind-body interventions); and iii) mechanisms (e.g., catastrophizing, self-efficacy, fear-avoidance, kinesiophobia, neuroplasticity, metacognition, emotional regulation). Both Medical Subject Headings (MeSH) and free-text keywords were used and combined with Boolean operators. Inclusion criteria prioritized Randomized Controlled Trials (RCTs), systematic reviews, meta-analyses, and pivotal observational studies examining the interaction between physical activity and cognitive or emotional variables in adult populations with chronic pain. Particular emphasis was placed on cross-disciplinary research bridging physiotherapy, pain neuroscience, and clinical psychology. Reference lists of eligible articles and key reviews were screened to identify additional relevant studies. Studies were excluded if they: focused exclusively on acute pain models; investigated pediatric populations without adult comparators; addressed exercise solely as a fitness outcome without cognitive or emotional measures; or consisted of purely descriptive reports without empirical data.

Results

The main findings are summarized in Table 1.

Exercise as cognitive restructuring: catastrophizing, pain beliefs, and self-efficacy

Modern neurophysiology models explains persistent pain through hierarchical processes where brain regions like the cingulate and insula cortices integrate bottom-up nociceptive signals with top-down expectations, generating internal models to anticipate sensory inputs.[11-13] Under physiological conditions the discrepancy between expected and actual sensory input updates these internal models

to maximize correctness; however, in chronic pain, the brain may disproportionately weight expectations of pain while discounting somatic input, perpetuating a state of persistent nociplasticity.[14-15] Several cognitive mediators have been identified as moderators in pain perception and evaluation, acting on expectancies and other cognitive and perceptual processes.[16] Pain catastrophizing, a maladaptive cognitive style characterized by rumination, magnification of threat, and helplessness, is consistently identified as a predictor of chronicity, pain intensity and related disability.[17,18] Another important cognitive mediator is pain self-efficacy, an individual's belief in their capacity to perform goal-oriented behaviors despite the presence of pain; patients with high self-efficacy report lower disability even when pain intensity remains elevated, suggesting a protective effect.[19-20] Furthermore, specific maladaptive beliefs regarding the nature of pain act as a cognitive maintainers of chronic pain states; among these the pain-as-harm belief (the conviction that pain is an accurate signal of tissue damage) and beliefs concerning the uncontrollability of pain are strongly predictive of poor rehabilitation outcomes.[21,22] Neuroimaging studies show that these maladaptive cognitive factors are associated with altered brain activity in regions involved in pain processing, attention, and emotion regulation, affecting endogenous analgesic systems.[23]

Exercise can significantly influence the cognitive dimensions of pain, as demonstrated by several studies showing altered activity in brain regions involved in central pain processing, including the prefrontal cortex, anterior cingulate cortex, insula, amygdala, and somatosensory cortices.[24] Physical exercise as Tai Chi and resistance training reduce catastrophizing, suggesting a process of embodied cognitive restructuring that modifies maladaptive pain cognitions.[25-27] The mechanism underlying this reduction is likely exposure-based and experiential: as patients engage in motor behavior the magnification of threat decreases, helplessness diminishes through improved physical competence, and functional capacity restoration counters beliefs about the immutability of the condition.[28] In chronic low back pain, exposure treatments effectively reduce harm expectations and increase pain self-efficacy, with effects generalizing to novel activities in different contexts.[29] Overall, exercise appears effective at fostering cognitive restructuring by providing patients with mastery experiences, which counter maladaptive beliefs about pain, reduce pain intensity and foster active self-management.[30-32]

Exercise as a behavioral experiment: disrupting the fear-avoidance cycle

The behavioral component associated with these cognitive dimensions is known as kinesiophobia, defined as an excessive, irrational fear of physical movement arising from a perceived susceptibility to painful injury.[33] Kinesiophobia drives avoidance behaviors, leading to physical inactivity and deconditioning, psychological distress and negative coping strategies. These consequences paradoxically lower the pain threshold and reinforce the original fear, establishing a maladaptive positive feedback loop that perpetuates disability, as described in The Fear-Avoidance Model (FAM).[34] Neurobiologically, this cycle is maintained by the amygdala and hippocampus, which encode fear memories associated with movement, triggering anticipatory anxiety responses and avoidance behaviors.[35] Research shows that kinesiophobia is more influential than pain in limiting physical activity, especially among frail individuals, though intervention studies remain scarce.[36] Early identification and management of kinesiophobia are crucial to improving rehabilitation outcomes and quality of life across affected populations.[37]

While psychological and educational interventions can address the cognitive basis of movement safety, exercise functions as a mechanism of graded exposure in vivo.[38] To unlearn the fear of movement, the patient needs to systematically perform feared movements without the anticipated catastrophic consequences (pain or injury), causing a weakening of the association between movement and threat.[39] By progressively engaging in these feared movements, the patient gathers behavioral evidence that contradicts their negative expectations, a process known as expectancy violation, breaking the fear-avoidance cycle through the principles of Pavlovian inhibitory learning.[40] Graded exposure explicitly targets the fear, asking patients to perform the specific movements they believe will harm them, reducing pain-related fear in patients with kinesiophobia.[41-43] Notably, emerging research suggests that exercise protocols which induce mild and controlled pain may be superior to completely pain-free protocols in reducing fear-avoidance; by permitting movement into mild pain, patients learn that pain does not equate to harm.[44] This approach to exercise transforms the patient into an active experimenter, using their own body to test and refute the hypothesis that leads to kinesiophobia. When a patient performs a feared movement (conditioned stimulus) and the catastrophic outcome (unconditioned stimulus) fails to occur, the brain generates a prediction error that update the internal model.[45]

Exercise as attentional training

Individuals with chronic pain frequently demonstrate a maladaptive attentional bias toward pain-related stimuli, characterized by hypervigilance to somatic sensations and difficulty in disengaging attention from nociceptive input.[46] Meta-analyses using dot-probe and visual-probe tasks show a small to moderate attentional bias toward sensory pain words and pictures in chronic pain patients compared to healthy controls.[47] This automatic orientation of attention is a primary driver of pain salience, and has been shown to be a strong predictor of the transition from acute to chronic pain, significantly modulating functional interference. A longitudinal study of patients with subacute back pain found that hypervigilance was the strongest predictor of pain severity and pain-related interference even after adjusting for baseline pain levels.[48] Some authors highlight its role in central sensitization processes, due to altered brain activity in regions such as the anterior cingulate cortex.[49-50]

Exercise interventions in chronic pain patients have been shown to improve activity in the Dorsolateral Prefrontal Cortex (DLPFC), a brain area crucial for top-down attentional control, the ability to voluntarily direct focus rather than having it automatically controlled by sensory stimuli like pain.[51,52] By improving cerebral perfusion and providing neurotrophic support to the DLPFC, exercise strengthens the cognitive control mechanisms required to inhibit irrelevant sensory input and disengage attention from pain, training the brain to filter nociceptive signals.[53] Furthermore, by engaging patients in sensorimotor training, attentional resources are shifted away from pain processing and redirected toward motor execution, through a mechanism commonly referred to as attentional gating.[54-55] Some exercise modalities utilize a distinct form of attentional engagement that amplifies focus on somatic sensation rather than diverting it; disciplines like Yoga and Tai Chi integrate low-intensity movement with focused interoception, sharing mechanisms with Mindfulness-Based Interventions (MBIs).[56-58] In mindful exercise, motor activity is associated with training in non-reactive attention: patients learn to observe somatic sensations without the automatic catastrophic response, inhibiting safety behaviors and reducing disability. Patients learn to broaden their field of attention to include neutral or positive bodily sensations (e.g., muscle contraction), reducing the dominance of the pain signal within the conscious experience. Evidence suggests that these interventions are generally safe and feasible, though more high-quality trials are needed to confirm long-term benefits.[59]

Exercise as committed action

Chronic pain is accompanied by maladaptive changes in mesolimbic pathways, with alterations on Behavioral Inhibition System (BIS) and Behavioral Activation System (BAS). The BIS, which regulates sensitivity to threat and punishment, is characteristically overactive in chronic pain states, driving hypervigilance and kinesiophobia.[60] Conversely, the BAS, which regulates motivation and goal-directed behavior, is frequently suppressed, correlating with depression, anhedonia, and reduced physical activity.[61] Emerging consensus suggests that effective rehabilitation must not only mitigate the BIS but actively stimulate the BAS, to increase patients engagement to daily activities, as described by the Motivation-Decision Model.[62,63] This neurobiological model posits that the mesocorticolimbic circuitry (the nucleus accumbens and dopamine pathways) continuously weights the salience of pain against the value of competing goals. In chronic pain, this valuation circuitry becomes dysfunctional and the negative value of nociception consistently outweighs the positive value of motor activity, leading to anhedonia and behavioral withdrawal.[64]

A clinical model that specifically targets this dysfunctional mechanism is Acceptance and Commitment Therapy (ACT), which centers on the construct of psychological flexibility, the capacity to persist in value-based behavior despite the presence of interfering psychological or physical difficulties (e.g. pain). ACT directly addresses the motivational and behavioral deficits commonly observed in chronic pain by encouraging individuals to engage in meaningful activities despite the persistence of pain, reducing experiential avoidance, inactivity, and loss of function.[65] Within this framework, exercise serves as a vehicle for Committed Action, described in ACT theory as an action guided by long-term values rather than immediate sensory relief or symptom avoidance.[66] Exercise as Committed Action teaches patients that the goal is not necessarily to wait until pain disappears before moving, but to learn that movement can still be safe, purposeful, and worthwhile even when some pain persists. This shift is particularly important in chronic pain, where behavior is often organized around short-term control strategies aimed at minimizing pain, even when such strategies ultimately reinforce inactivity, disability, and psychological distress. Trials of combined ACT and physical exercise programs report improvements in disability and physical function, with benefits maintained at follow-ups up to one year.[67] In conclusion, committed exercise disrupts pain-contingent behavior (*i.e.,* stopping movement the moment pain arises) by establishing value-contingent behavior, helping patients to engage in physical activity to facilitate meaningful life roles.[68,69]

Exercise and metacognition: from content to process

Metacognition refers to the processes controlling how an individual relates to their own thoughts and sensations; in chronic pain it involves how individuals relate to their thoughts and sensations, particularly through beliefs about the controllability and usefulness of worry and rumination.[70] Negative metacognitive beliefs, such as viewing thoughts as uncontrollable or dangerous, are associated with higher levels of pain catastrophizing and emotional distress, and show small-to-moderate associations with somatic symptom burden, supporting the view that rumination can amplify symptom perception and pain intensity.[71,72] Additionally, metacognitions influence health anxiety and pain-related disability, indicating their role in broader emotional and functional outcomes in chronic pain populations.[73] Emerging evidence shows that interventions that address both cognitive content and metacognitive processes may be more effective than those focusing solely on thought content to reduce suffering and improve coping in chronic pain patients.[74]

Physical exercise can act as a powerful metacognitive intervention by redirecting attentional and physiological resources away from ruminative thought patterns toward goal-directed tasks.[75] Preliminary evidence shows that during complex movement the motor cortex engages neural resources that would otherwise be allocated to the Default Mode Network, which is associated with rumination, providing experiential evidence that worry can be controlled.[76] Therefore, physical exercise provides a practical mechanism for the chronic pain patients to disrupt perseverative thinking by engaging brain networks that compete with rumination processes.[77] Furthermore, mind-body exercises (e.g., Yoga, Tai Chi) cultivate metacognitive awareness, the ability to view thoughts and somatic sensations as transient mental events rather than objective facts, increasing acceptance of pain.[78] This process, often called cognitive defusion or decentering, helps patients step back from the pain experience and adopt a non-reactive attitude toward nociceptive signals, reducing emotional suffering and disability linked to pain.[79] This shift involves improving interoceptive accuracy and metacognitive awareness of interoception, the ability to correctly identify bodily signals, often compromised in chronic pain patients.[80] For example, in patients with fibromyalgia altered interoceptive sensibility correlates with pain severity, fatigue, and anxiety, suggesting that dysfunctional interoception contributes to symptom expression.[81] Mindful physical exercises are associated with improvements in self-reported interoceptive awareness and objective measures like heartbeat counting, helping patients develop a non-reactive stance toward nociceptive signals.[82-83]

Exercise and emotional-affective outcomes

Approximately 40% of adults with chronic pain experience clinically significant symptoms of depression and anxiety with higher prevalence in those with nociplastic pain.[84] This comorbidity is so prevalent that recent literature conceptualizes the condition as a pain-depression dyad, a bidirectional relationship where pain precipitates affective distress through functional limitation and neural atrophy, while depression and anxiety lower pain thresholds and impair coping mechanisms.[85] Recent studies confirmed that in chronic pain depression is associated with greater pain intensity, increased interference in daily activities, and worse physical and emotional functioning.[86,87] This overlap is supported by the Common Soil hypothesis, which attributes both conditions to shared neurobiological dysfunctions including monoaminergic dysregulation and maladaptation of the hypothalamic-pituitary-adrenal axis.[88] Neuroplasticity impairments in key brain regions such as the hippocampus and prefrontal cortex further contribute to this comorbidity by disrupting emotional regulation and cognitive function.[89]

Therapeutic exercise functions by upregulating key neurotrophic factors, most notably Brain-Derived Neurotrophic Factor (BDNF) and restoring the balance of neurotransmitters such as serotonin and dopamine.[90] BDNF facilitates neurogenesis in the hippocampus, a region that often undergoes atrophy in chronic pain states; exercise-induced BDNF release can reverse this atrophy, improving both mood and pain modulation.[91] Additionally, exercise modulates purinergic receptors, which regulate BDNF release and may underlie its neuroprotective effects against pain and depression.[92] Beyond these generalized neuroprotective effects, emerging evidence suggests that specific exercise modalities can modulate affective regulation. Resistance Training (RT) has demonstrated significant efficacy in reducing depressive symptoms, with meta-analyses showing consistent improvements across diverse populations, including older adults and young adults with mild to moderate depression.[93] It is hypothesized that the sensation of muscle tension and the objective progression of load fosters a sense of embodied competence that directly counters the feelings of physical fragility and learned helplessness that characterize the depressive symptoms.[94] Conversely, anxiety-related conditions, driven by sympathetic hyperarousal and the fear-avoidance cycle, respond to aerobic and mind-body interventions. These modalities operate primarily through autonomic modulation, enhancing vagal tone and reducing the generalized arousal that sensitizes nociceptors and exacerbates the perception of pain.[95] Regular aerobic exercise also increases serotonin release in the ACC, a key brain region for processing pain and anxiety, which improves synaptic plasticity and alleviates both

symptoms.[96] Furthermore, structured exercise disrupts the inertia and withdrawal behaviors inherent to chronic pain and depression, replacing avoidance with goal-directed, reinforcing activities that reduce the depressed mood.[97-98] Overall, exercise promotes emotional regulation and psychological resilience through both neurobiological mechanisms and psychosocial pathways involving increased positive affect, autonomic modulation and neuroplasticity.[99]

Discussion

Chronic pain remains one of the most pervasive global health challenges, affecting a substantial proportion of the adult population and contributing significantly to long-term disability, psychological distress, and reduced quality of life.[100] Modern biopsychosocial models of pain recognize chronic pain as a complex, multidimensional experience influenced by biological, psychological, and social factors that interact dynamically.[101] This complexity calls for therapeutic approaches capable of addressing the full biopsychosocial architecture of pain rather than focusing exclusively on tissue-level dysfunction. Within this framework, physical exercise should no longer be viewed merely as a tool for restoring musculoskeletal function, but as a multidimensional intervention capable of acting on the broader mechanisms that sustain chronic pain over time. The evidence synthesized in this review suggests that the therapeutic effects of exercise extend well beyond improvements in strength, endurance, flexibility, or joint mobility; although these physical effects remain clinically relevant, exercise also appears to function as a neurobehavioral intervention that modulates pain processing.[102] Physical activity may counteract central sensitization, enhance synaptic homeostasis, and support functional changes in brain systems involved in pain modulation; these neurobiological effects are paralleled by clinically meaningful changes in cognitive, behavioral, attentional, motivational, metacognitive, and emotional-affective domains.[103] Exercise can challenge maladaptive pain predictions and dysfunctional beliefs, strengthen pain self-efficacy and restore a sense of agency, which is often profoundly disrupted in chronic pain.[104] At the same time, exercise acts as a form of attentional training, helping patients disengage from hypervigilance toward pain-related signals and foster adaptive metacognitive processes, such as decentering and non-reactive awareness of bodily sensations.[105]

Taken together, these findings support the interpretation of exercise as an active experiential therapy; its therapeutic value lies not only in improving biomechanical capacity, but in enabling patients to

generate corrective evidence about the meaning of pain, the safety of movement, and their own functional abilities. From a clinical perspective, these considerations suggest that exercise prescription in chronic pain should move beyond a purely biomedical rationale and become increasingly psychologically informed. Exercise programs should be tailored not only to physical impairments, but also to the dominant cognitive and emotional maintaining factors present in each patient. However, although exercise-based interventions are generally associated with beneficial effects on pain, function, and psychological well-being, the literature also reports heterogeneous and sometimes conflicting findings. Importantly, exercise interventions may not be universally tolerated, as some patients can experience pain exacerbation, fatigue, adherence difficulties, or limited feasibility due to comorbidities and functional impairments. Future research should now move beyond the broad question of whether exercise is beneficial on cognitive, metacognitive and emotional processes and instead determine more precisely for whom, under which conditions, and through which mechanisms specific exercise approaches exert their effects (Table 2). Studies should adopt clearer phenotyping strategies, distinguishing between nociceptive, neuropathic, and nociplastic presentations, while also identifying clinically relevant subgroups characterized by high catastrophizing, marked fear of movement, attentional hypervigilance, depressive symptoms, or reduced psychological flexibility. Furthermore, in addition to conventional outcomes such as pain intensity and disability, research should more consistently incorporate measures targeting relevant psychological and neurocognitive processes; this would allow to move beyond simple efficacy testing and begin to identify the active therapeutic ingredients of exercise interventions. A further priority is to clarify whether different exercise modalities preferentially target distinct biopsychosocial domains: resistance training may be particularly relevant for improving self-efficacy, perceived competence, and depressive symptoms through mastery experiences and progressive load tolerance; aerobic exercise may exert stronger effects on affect regulation, autonomic balance, and generalized distress through cardiorespiratory, neurochemical, and behavioral activation mechanisms; mind-body modalities may be especially suited to targeting attentional control, body awareness, interoception, and metacognitive regulation, particularly in patients with pronounced hypervigilance or maladaptive symptom monitoring; and graded exposure approaches may be most appropriate in patients whose disability is strongly maintained by fear-avoidance beliefs and movement-related threat appraisal. Ultimately, a more mechanism-based understanding of exercise could support the development of personalized rehabilitation models that tailor exercise according also to the psychosocial processes associated with chronic pain in each patient, optimizing outcomes such as pain reduction and functional recovery.

Conclusions

The evidence synthesized in this narrative review suggests that physical exercise should no longer be viewed solely as a means of improving physical function in chronic pain. Rather, it may be more appropriately conceptualized as a multidimensional intervention capable of acting on the cognitive, behavioral, attentional, emotional, and metacognitive processes that contribute to pain persistence and disability. Although this review aimed to provide a comprehensive and multidimensional overview of the cognitive and emotional mechanisms underlying exercise effects in chronic pain, some limitations should be acknowledged. As a narrative (non-systematic) review, the study may be subject to selection bias and methodological heterogeneity among included studies. Furthermore, several findings derive from heterogeneous clinical populations and interventions, while the strength of evidence varies across domains; further high-quality studies are needed to clarify for whom, under which conditions, and through which mechanisms specific exercise modalities exert their effects, as well as to identify the psychological and neurocognitive processes that mediate clinical benefit in real-world settings. A more precise understanding of these pathways may support the development of personalized, psychologically informed exercise prescriptions according to each patient's specific biopsychosocial profile and maintaining factors.

List of Abbreviations

ACT, Acceptance and Commitment Therapy

ACC, Anterior Cingulate Cortex

BDNF, Brain-Derived Neurotrophic Factor

CRPS, Complex Regional Pain Syndrome

DLPFC, Dorsolateral Prefrontal Cortex

FAM, Fear-Avoidance Model

PAG, Periaqueductal Gray

PFC, Prefrontal Cortex

RT Resistance Training

VEGF, Vascular Endothelial Growth Factor

Corresponding Author

Gianluca Regazzo, Section of Rehabilitation, Department of Neuroscience-DNS, University of Padova, Italy.

gianluca.regazzo@unipd.it

0009-0004-6277-4199

Daniele Coraci

0000-0002-7019-9006

daniele.coraci@unipd.it

Stefano Masiero

0000-0002-0361-4898

stef.masiero@unipd.it

Contributions:

Gianluca Regazzo, conceptualization, methodology, investigation, writing—original draft preparation; Stefano Masiero, Daniele Coraci, writing—review and editing. All authors have read and agreed to the published version of the manuscript.

Conflicts of Interest

The authors declare no conflicts of interest.

Ethics approval

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Table 1. Summary of the main mechanisms through which physical exercise may influence cognitive, behavioral, attentional, metacognitive, and emotional-affective dimensions of chronic pain (Strong evidence: RCT's, systematic reviews, or meta-analyses with consistent results. Moderate evidence: findings supported by controlled or observational studies but limited consistency or replication. Preliminary evidence: exploratory, theoretical, mechanistic, or emerging findings.)

Exercise mechanism	**Main evidence**
1. Exercise as cognitive restructuring	Strong evidence: exercise may promote cognitive restructuring in chronic pain by reducing catastrophizing, modifying maladaptive pain beliefs, and strengthening pain self-efficacy.[17-32]
2. Exercise as a behavioral experiment	Strong evidence: exercise can act as a graded exposure strategy, helping patients disconfirm catastrophic expectations related to movement. This process may reduce kinesiophobia, weaken avoidance behaviors, and improve disability.[33-45]
3. Exercise as attentional training	Moderate evidence: chronic pain is associated with hypervigilance and attentional bias toward pain-related stimuli. Exercise may counter this pattern by redistributing attentional resources and reducing the salience of nociceptive input.[46-59]
4. Exercise as committed action	Preliminary evidence: exercise may function as committed action by shifting behavior from pain-contingent avoidance to value-based engagement,

	supporting functional recovery, psychological flexibility, and sustained participation.[60-70]
5. Exercise and metacognition	Preliminary evidence: exercise may influence metacognitive processes (e.g., attentional monitoring, rumination, and cognitive control strategies) and contents (e.g., beliefs and interpretations about thoughts and symptoms) by reducing rumination, promoting decentering, and improving awareness of bodily sensations.[71-83]
6. Exercise and affective outcomes	Strong evidence: exercise improves emotional-affective outcomes in chronic pain by reducing depressive and anxious symptoms and by enhancing quality of life.[84-98]

Table 2. Key recommendations for future research on the psychosocial effects of exercise interventions in chronic pain.

Research topic	Recommendation	Rationale
Mechanistic understanding	Studies should clarify under which conditions and through which mechanisms exercise exerts its effects in chronic pain.	To move beyond general efficacy and identify the active therapeutic processes underlying exercise interventions.
Process-based outcomes	Studies should include psychological, attentional, metacognitive, and emotional outcomes in addition to pain and disability.	To better identify mediators of change and explain how exercise produces clinical benefit.
Modality-specific effects	Future research should determine whether different exercise modalities preferentially target distinct biopsychosocial domains.	To support a more targeted and mechanism-based selection of exercise approaches.

Personalized rehabilitation	Moderation analyses and stratified designs are needed to inform individualized, psychologically informed exercise programs.	To optimize treatment matching and improve clinical effectiveness in chronic pain populations.