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Review

From Pain Catastrophizing to Hopelessness: Neurobiological Mechanisms, Causes, and Evidence-Based Implications for Pain and Outcomes in Rheumatic Diseases

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Abstract

Pain catastrophizing (PC) and hopelessness are increasingly recognized as central determinants of pain severity, disability, and treatment response in individuals with rheumatic and immune-mediated diseases. Traditionally conceptualized as secondary emotional reactions to pain, these cognitive-affective constructs instead represent active mechanisms that shape symptom perception, behavioral responses, and long-term outcomes. In this review, we synthesize evidence across neurobiological, psychological, and clinical domains to elucidate the pathways linking PC and hopelessness to maladaptive coping, kinesiophobia, and functional decline. Early life stress, trauma, and maladaptive cognitive schemas emerge as upstream vulnerability factors that prime heightened emotional reactivity and reduced prefrontal regulatory control, facilitating amplified pain signaling and fear-based avoidance behaviors. Avoidance and inactivity foster physical deconditioning, fatigue, and higher perceived disability, creating a vicious circle that sustains distress and poor quality of life. Moreover, inactivity-related metabolic dysfunction and weight gain may contribute to low-grade inflammation, particularly in conditions such as psoriatic arthritis, thereby intersecting with biological disease pathways. Importantly, these psychological processes identify a distinct patient subgroup for whom further escalation of immunosuppressive therapy provides limited benefit. Instead, integrated psychological approaches—including cognitive behavioral therapy, acceptance and commitment therapy, and coping-skills training—demonstrate meaningful effects on catastrophizing, agency, and functional recovery. We emphasize the need for routine screening to detect patients with maladaptive cognitive–emotional profiles and propose a stratified care model prioritizing targeted psychological interventions alongside standard rheumatologic therapy. Future research should refine phenotyping strategies, clarify neuroimmune links, and develop scalable intervention models to break the avoidance cycle and improve patient-centered outcomes.

Keywords: pain catastrophizing; hopelessness; autoimmunity; quality of life; cognitive behavioral treatment; pain; fibromyalgia; psychotherapy

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1. Introduction

The experience of pain in rheumatic disease is not simply a matter of peripheral inflammation or joint damage. Central neural systems, cognitive–emotional processes and behavioral responses all interact to shape individual variance in pain intensity, physical disability, and, thus, treatment outcomes [1–3]. Within this context, pain catastrophizing (PC) and hopelessness have consistently emerged as key determinants of pain experience and patient outcomes in these conditions.

PC in rheumatic disease has been defined as a persistent tendency to magnify the threat of pain, ruminate on pain sensations, and feel helpless in the presence of pain [4]. In its entirety, it is not simply a reaction to severe pain but it is independently associated with higher pain intensity, disability, poorer physical function, and reduced treatment response, even when inflammatory markers are controlled for [5,6]. Closely linked to catastrophizing is the psychological construct of hopelessness, which reflects a broader sense of negative future expectancy and the belief that improvement or control over pain is unlikely.

Crucially, both constructs are modifiable. Psychological interventions that are tailored to rheumatic disease, such as pain coping skills training, cognitive behavioral therapy, acceptance and commitment therapy, and behavioral activation, have been shown to reduce catastrophizing and improve patient outcomes by restoring a sense of agency and adaptive engagement. These therapies likely work in part by strengthening prefrontal regulation networks: thus, restoring positive expectancy pathways. This will then counteract the neurobiological patterns associated with pain distress.

Here, we synthesize all available evidence on hopelessness and PC in rheumatic diseases and outline a coordinated treatment model aimed at reducing both constructs in parallel to improve pain, functioning, and long-term outcomes.

We used a pragmatic narrative search strategy to identify the relevant literature on pain catastrophizing and hopelessness in rheumatic diseases. We prioritized studies published within the last 5–10 years and included earlier seminal contributions when conceptually important. Evidence was gathered through targeted searches in PubMed and PsycInfo, complemented by recent systematic reviews, meta-analyses, and updated rheumatology guidelines. Search terms included combinations of “pain catastrophizing”, “hopelessness”, “rheumatic disease”, “autoimmune disease”, “chronic pain”, and disease-specific terms. We gave priority to high-quality observational studies, mechanistic neurobiological work, and psychological intervention trials. Titles and abstracts were screened for conceptual relevance, with the exclusion of studies that were unrelated to rheumatic or chronic pain conditions.

2. Neurobiological Connection Between Pain Catastrophizing and Hopelessness

PC and hopelessness are associated with distinct yet overlapping neurobiological patterns that influence pain modulation. While most available evidence is correlational and derived from cross-sectional neuroimaging, laboratory pain studies, and psycho-physiological paradigms, the existing findings collectively support several hypothesized mechanisms through which these constructs may shape pain experience in rheumatic disease.

Neurobiologically, PC has been linked to heightened responsivity in the salience network: particularly the insula and anterior cingulate cortex (ACC) (Figure 1), which function to assign emotional significance to sensory input [7]. Increased activation in these regions reflects greater pain-related threat appraisal, and sustained hypervigilance. Alongside this, catastrophizing is associated with reduced engagement of the prefrontal regulatory regions, including the dorsolateral and ventromedial prefrontal cortex (PFC), which ordinarily dampen emotional distress and support cognitive reappraisal [8].

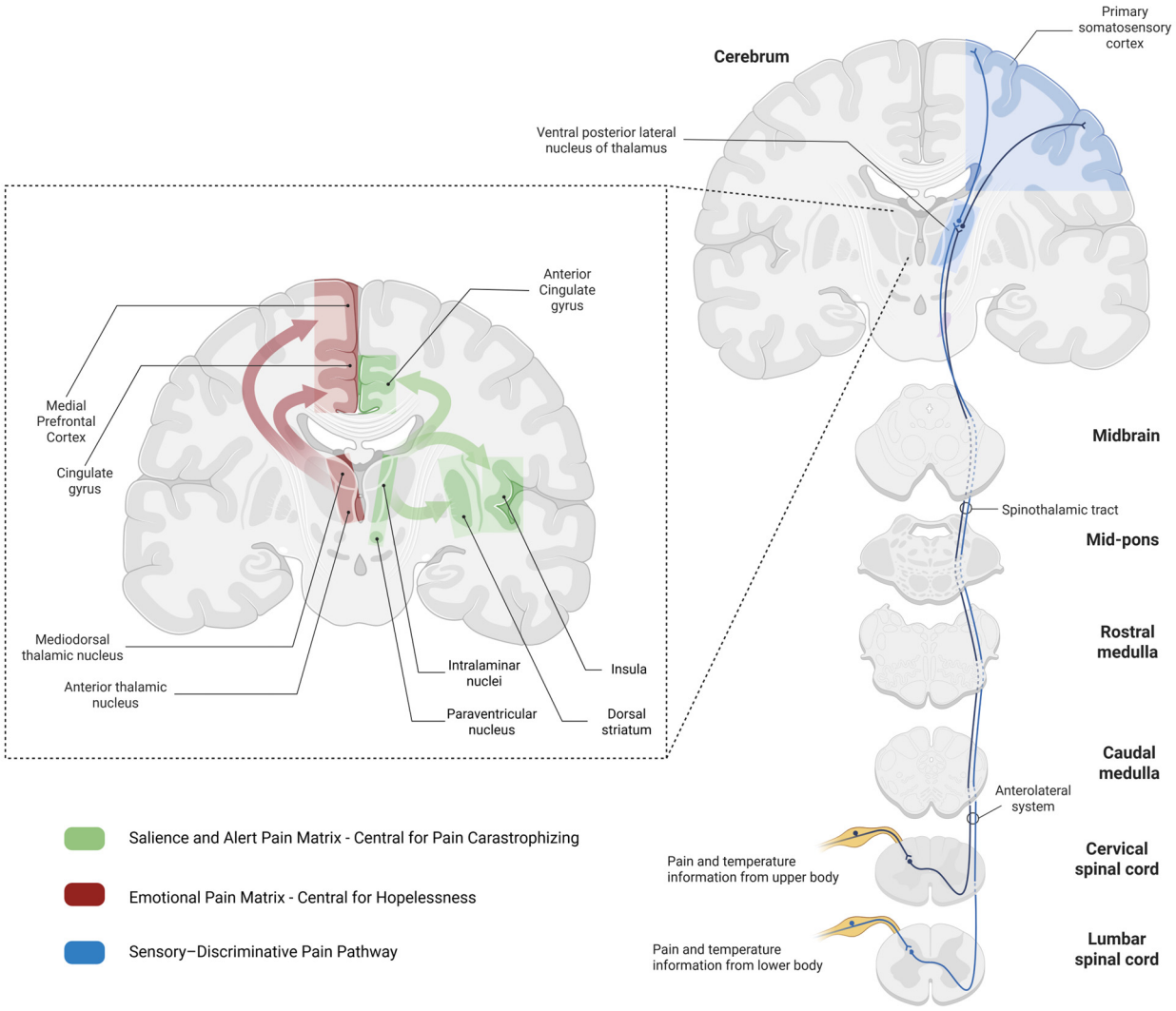


Figure 1. Ascending nociceptive input integration systems via different thalamic nuclei distribution of the signal.

Consistent with these mechanisms, resting-state functional MRI studies in chronic pain populations show altered connectivity between the amygdala and large-scale brain networks: most notably, the default mode network (DMN) and the central executive network (CEN). Higher levels of PC are associated with stronger coupling between the amygdala and the CEN, alongside a change in the typical amygdala–DMN connectivity pattern. In addition, individual dimensions of catastrophizing, such as rumination, magnification, and helplessness, have been linked to distinct connectivity changes across networks involved in self-referential processing, salience detection, sensory integration, and attentional control [9,10]. This pattern of strong emotional amplification with weakened cognitive regulation offers a biological pathway explaining why patients with rheumatic disease may report severe pain even during periods of minimal inflammatory activity.

On the other hand, hopelessness has been associated with reduced activity in the reward and motivational circuits, including the subgenual ACC, ventromedial PFC, and ventral striatum (Figure 1), which are central to the generation of positive expectancy and goal directed engagement [11,12]. When these circuits are underactive, patients may struggle to maintain motivation or belief in the possibility of improvement. This reinforces withdrawal, inactivity, and reduced coping, all of which are patterns frequently observed in chronic rheumatic disease [1].

Noticeably, although PC and hopelessness frequently co-occur and are strongly correlated, they represent partially distinct cognitive-affective constructs rather than different expressions of the same process. PC primarily operates at the level of threat appraisal and attentional bias toward pain, whereas hopelessness reflects a broader disruption of positive expectancy, agency, and motivational drive. A comparative table between PC and hopelessness to help clarify the two constructs is presented (Table 1).

Table 1. Conceptual and mechanistic comparison between pain catastrophizing and hopelessness in rheumatic diseases.

Domain	Pain Catastrophizing	Hopelessness
Core definition	Maladaptive cognitive–emotional response characterized by magnification of pain threat, rumination on pain sensations, and perceived helplessness in managing pain	Negative future expectancy characterized by loss of belief in improvement, diminished agency, and reduced motivation to engage in goal-directed behavior
Primary cognitive focus	Pain-centered appraisal ("this pain is unbearable, dangerous, uncontrollable")	Outcome-centered expectancy ("nothing will help", "there is no point in trying")
Temporal orientation	Present-oriented and stimulus-driven	Future-oriented and expectancy-driven
Dominant emotional tone	Anxiety, fear, distress	Demoralization, resignation, emotional blunting
Behavioral expression	Hypervigilance to pain, avoidance of movement, safety behaviors, reassurance seeking	Withdrawal, disengagement from treatment, reduced adherence, inactivity
Role in pain processing	Amplifies nociceptive input and central sensitization through threat appraisal and attentional bias	Sustains pain persistence by reducing motivation for coping, rehabilitation, and behavioral engagement
Key neurobiological correlates	Hyperactivation of salience and affective pain networks (insula, anterior cingulate cortex); reduced prefrontal inhibitory control (dlPFC, vmPFC)	Reduced activity in reward, motivation, and expectancy circuits (ventral striatum, subgenual ACC, vmPFC); impaired positive expectancy signaling
Relationship with inflammation	Inconsistent association; often dissociates pain severity from inflammatory activity	Indirect; associated with worse outcomes despite controlled inflammation, via behavioral and motivational pathways
Interaction with fear-avoidance	Central driver of kinesiophobia and movement avoidance	Maintains avoidance by suppressing perceived value of effort or recovery
Clinical phenotype	"Amplified pain" phenotype with high pain intensity and disability despite modest disease activity	"Disengaged" phenotype with poor adherence, functional decline, and low response to escalation of pharmacological therapy
Therapeutic targets	Cognitive restructuring, threat reappraisal, pain coping skills, exposure-based strategies	Restoration of agency, goal setting, motivational enhancement, behavioral activation
Responsiveness to psychological interventions	Highly responsive to CBT, pain coping skills training, ACT	Responsive to ACT, behavioral activation, hope-enhancing and values-based interventions
Role in disease course	Early amplifier of pain perception and disability	Later-stage perpetuator of chronicity and functional decline

It is likely that PC and hopelessness interact in a dynamic cycle. Heightened distress and perceived lack of control in the fact of PC can, over time, lead to loss of hope when the pain then persists despite treatment efforts. In turn, hopelessness reduces motivation to engage in adaptive strategies, which may worsen pain, increase disability, and reinforce catastrophizing. Currado et al. (2023) demonstrated this interaction in psoriatic arthritis (PsA) and axial spondylarthritis (axSpA), showing that higher catastrophizing was associated with higher disease activity and disability, alongside lower levels of psychological flexibility and hope [5].

Importantly, both constructs are modifiable and clinically actionable. Psychological interventions targeting threat reappraisal, coping skills, and emotional regulation may better suit PC strategies, whereas interventions enhancing agency, values-based engagement, and behavioral activation can be relevant for addressing hopelessness. Also, recognizing their differences has direct therapeutic implications, supporting tailored, mechanism-informed psychological interventions alongside standard rheumatologic care.

This cycle has direct implications for clinical outcomes. Patients with high levels of catastrophizing and hopelessness tend to report greater pain and fatigue even during controlled inflammation [4,6], experience reduced function and quality of life [5], respond less effectively to pharmacological therapy alone [1], and require a higher use of painkiller medications [13], and may benefit from integrated psychosocial–medical management approaches [14].

3. Pain Catastrophizing in Rheumatic Diseases

PC has been investigated more extensively than hopelessness across rheumatic diseases, largely because pain represents a more immediate, salient, and measurable dimension of disease burden than future-oriented expectancies. As a result, catastrophizing has been more readily incorporated into clinical and mechanistic studies, albeit with considerable heterogeneity in study design, populations, and outcomes. Nevertheless, despite heterogeneity, the available evidence across disease contexts suggests that PC captures a core cognitive-affective process, through which pain is amplified and maintained, providing a useful entry point for understanding maladaptive pain processing in rheumatic disease.

3.1. Osteoarthritis

PC plays a central role in osteoarthritis (OA), influencing pain intensity, physical function, and postoperative recovery. In patients with hip OA, those presenting higher levels of catastrophizing consistently report worse symptoms and poorer quality of life (QoL), even when controlling for structural severity or objective clinical metrics. In a multicenter study of 160 hip OA patients, higher PC scores independently predicted lower physical ($\beta = -0.28$, $p < 0.05$) and mental ($\beta = -0.29$, $p < 0.05$) QoL indices after adjusting for pain intensity, BMI, and range of motion, underscoring the independent contribution of cognitive-affective processes to disability in OA [15]. Similarly, preoperative catastrophizing has been associated with inferior postoperative outcomes in hip OA: patients with elevated PCS prior to total hip arthroplasty reported greater pain and functional limitations at twelve months, independent of the baseline characteristics, suggesting that catastrophizing not only reflects distress but can modulate recovery trajectories, probably through facilitating movement-avoidant behaviors [16–19].

In a study assessing fear-avoidance and PC in hip OA, both constructs showed significant associations with reduced QoL and functional limitation, reinforcing their intertwined role in pain-related outcomes [16]. In this sense, catastrophizing and kinesiophobia frequently co-occur, suggesting shared mechanisms such as hypervigilance toward movement-related pain, anticipatory threat appraisal, and reduced willingness to

engage in physical activity: behaviors that may accelerate deconditioning and disability. Evidence in knee OA mirrors hip OA findings. A prospective study demonstrated that higher preoperative PCS scores predicted worse pain and poorer function following total knee arthroplasty, with PC emerging as a stronger determinant of outcomes than radiographic severity [20]. In this patient population, the cognitive–emotional burden mediated through PC appears to interact with central pain mechanisms. In another investigation, applying transcranial direct current stimulation as a neuro-modulatory approach in 68 knee OA patients, a greater number of stimulation sessions correlated with reduced pain ($r = -0.42$, $p = 0.002$) and improved function ($r = 0.39$, $p = 0.004$), and PC mediated these associations, highlighting the mechanistic interplay between cortical modulation and catastrophizing tendencies [21]. Functional performance data further emphasize the behavioral impact of catastrophizing. High-catastrophizing knee OA patients demonstrate a slower gait speed and poorer chair-rise performance, indicating that catastrophizing contributes to measurable physical impairment beyond subjective pain reports [20]. Beyond the lower limb, PC is even markedly elevated in hand OA patients versus controls (mean difference ~10.8 points on PCS) [22], and catastrophizing has been shown to impair post-operative outcomes even in shoulder OA patients [19].

Notably, PC is clinically relevant, even in patients with modest disease burden. A cross-sectional study of both autoimmune and mechanic musculoskeletal diseases found that higher PC scores were associated with failure to achieve low disease activity, even in lower inflammatory states [23]. Evidence across OA consistently shows that catastrophizing contributes to heightened pain perception, functional impairment, diminished QoL, and poorer surgical recovery.

Although hopelessness has rarely been assessed in OA, one study in 104 older adults with chronic OA pain interpreted depressive symptomatology through Abramson's hopelessness model, despite not using any validated hopelessness scale [24]. In that work, cognitive behavioral variables such as PC, passive coping, and pain-related beliefs differentiated individuals with versus without depressive symptoms; the authors supported the relevance of hopelessness processes in OA chronic pain.

3.2. Inflammatory Arthritis

In spondyloarthritis (SpA), PC is increasingly recognized as a key contributor to pain burden, treatment outcomes, and perceived disease activity. In a recent Italian prospective multicenter cohort including PsA and axSpA patients, the higher baseline PC predicted treatment discontinuation over two years [25]. The effect was consistent across univariate analyses, where each unit increase in the PC scores on the helplessness or rumination subscales was associated with a higher risk of drug withdrawal. Importantly, the helplessness domain of the PC scores showed the strongest signal. Although multivariable confirmation for axSpA was limited by low event numbers, these findings suggest that cognitive-affective dysregulation contributes to poorer treatment persistence, likely via reduced perceived benefit and lower adherence.

Additional evidence comes from a large real-world cohort across multiple autoimmune diseases (n = 1229), where PC showed a disease-specific impact in axSpA (n = 255). Specifically, a higher PCS significantly reduced the likelihood of achieving low disease activity (OR 0.33, 95% CI 0.12–0.88) [26]. These results underscore a particular vulnerability of axial disease to cognitive amplification of pain and support the concept of a "residual pain" phenotype, where symptoms persist despite adequate inflammatory control. Neurophysiological data further delineate this phenotype [26]. In a quantitative sensory testing-based profiling study, patients with PsA demonstrated lower pressure pain thresholds and greater temporal summation compared with healthy controls, along with significantly higher

catastrophizing scores, amplifying PC and sensory hypersensitivity metrics. These findings reinforce that PC is closely intertwined with central pain sensitization rather than simply responding to the nociceptive drive from inflammation [22]. Younger SpA cohorts show aligned trends, wherein associations between PC, higher BASDAI scores, and kinesiophobia frequently fall in the low-to-moderate range ($r \approx 0.25$–0.40), indicating that catastrophizing co-evolves with movement-related fear and avoidance early in the disease course [27]. These interactions may partially explain why younger patients with limited structural damage can still report substantial disability and pain.

Beyond SpA, PC also represents a key modulator of pain severity, emotional distress, and physical functioning for rheumatoid arthritis (RA), where it often exceeds the explanatory power of inflammatory markers alone. In a cohort of 220 RA patients, 61.8% reported high levels of PC, which showed strong inverse associations with both cognitive flexibility ($\beta = -0.34$, $p < 0.001$) and self-efficacy ($\beta = -0.53$, $p < 0.001$), together accounting for 51% of the variance in catastrophizing [28]. These data underscore the importance of cognitive-affective mechanisms in shaping pain experience and coping capacity in RA. Catastrophizing has been consistently linked to greater pain intensity, increased disease impact, and heightened psychological burden, independent of disease activity [1,29]. Importantly, PC in inflammatory arthritis is closely tied to fear-avoidance behaviors, where fear of movement and anticipation of pain drive the avoidance of physical activity [30,31].

Regarding hopelessness, qualitative evidence from the Swedish TIRA cohort shows that this psychological construct is a recurring emotional reaction in early RA, emerging in the response to restrictions such as difficulties dressing, working, or performing basic daily tasks, severely affecting QoL [32]. Furthermore, a study in juvenile idiopathic arthritis (JIA) used the Hopelessness Scale for Children to examine its relation to pain. Hopelessness showed correlations with pain levels and accounted for a proportion of variance in pain severity [33]. Importantly, in those studies, hopelessness is described as a situational emotional consequence rather than a stable cognitive trait, and no standardized hopelessness instrument was used. This emphasizes the near absence of structured hopelessness assessment in rheumatic disease studies.

3.3. Connective Tissue Diseases

In connective tissue diseases (CTDs), PC plays a central role in modulating pain perception, disability, and emotional burden, frequently exceeding the influence of humoral disease activity biomarkers. In systemic sclerosis (SSc), PC and hopelessness are highly prevalent and clinically meaningful. In one cohort of SSc patients, high catastrophizing was significantly associated with worse physical functioning, higher fatigue, and greater emotional distress, independently of disease severity scores [34]. Furthermore, hopelessness was present in over 40% of patients and strongly correlated with higher pain ratings and disability indices, suggesting that maladaptive cognitive patterns contribute substantially to perceived disease burden and to amplified pain processing pathways [35]. These findings support a cognitive-affective amplification model layered onto the microvascular and neuropathic pain mechanisms that are typical of SSc.

In systemic lupus erythematosus (SLE), catastrophizing is a key determinant of pain and fatigue, despite weak or absent correlations between disease activity and patient-reported pain severity. In a study including SLE patients, high PC scores were associated with greater pain, stiffness, lower positive mood, and significantly worse QoL, while disease activity did not correlate with psychological distress or pain intensity [36]. Importantly, in a study of 104 women with SLE, kinesiophobia, depression, body awareness, and BMI explained 52% of the variance in PC scores, highlighting the central role of psychological and perceptual factors over biological ones [37]. Moreover, 63% of SLE

patients exhibited clinically significant kinesiophobia, which was directly associated with fatigue, poor function, and reduced quality of life, despite minimal association with inflammatory markers or physician global scores [38]. This underscores a predominant fear-avoidance behavioral phenotype, reinforcing physical inactivity and perpetuating central sensitization and disability.

In primary Sjögren's syndrome (pSS), PC emerges as one of the strongest predictors of pain outcomes. In a cohort of 92 pSS patients, catastrophizing, along with fibromyalgia comorbidity and negative illness beliefs, explained 55% of the variance in pain severity [39]. Compared to depression and anxiety measures, catastrophizing was a stronger predictor of pain intensity, suggesting that cognitive–emotional mechanisms may outweigh affective symptoms in driving pain amplification in pSS. These findings align with the high prevalence of neuropathic-like pain, fatigue, and centralized pain features observed in the disease [40].

3.4. Fibromyalgia

Fibromyalgia (FM), the most comorbid condition in all autoimmune rheumatic diseases, represents a prototypical condition in which PC plays a central pathophysiological and clinical role. Across FM cohorts, catastrophizing consistently correlates with higher pain intensity, emotional distress, disability, and poorer treatment response. Evidence supports a bidirectional model in which catastrophizing not only amplifies nociceptive input and central sensitization, but is also reinforced by persistent pain and hypervigilance. A growing body of work highlights catastrophizing as a key mediator between psychological and clinical variables in FM. In a study of 105 FM patients, PC significantly mediated the relationship between pain self-efficacy and disease severity, indicating that catastrophizing independently predicted symptom burden and accounted for part of the effect of low self-efficacy on FM severity [41].

Cognitive-affective processes further contribute to disability in FM through repetitive negative thinking and rumination. In a cohort of 82 FM patients, the link between pain and anxious–depressive symptoms was doubly mediated by catastrophizing and RNT, suggesting a serial model where maladaptive cognitive loops exacerbate both the emotional burden and pain experience [42]. These findings position catastrophizing as part of a broader emotion-regulation deficit in FM. Treatment response data also underline the clinical implications of catastrophizing in FM beyond autoimmunity. A randomized controlled trial, assessing dry needling in 96 women with FM, reported significant improvements in pressure pain thresholds; however, individuals with a moderate baseline PC exhibited a diminished immediate post-intervention benefit [43]. This highlights catastrophizing as a moderator of therapy efficacy, reinforcing the need for integrated multimodal approaches. Finally, catastrophizing has been linked to suicidality in FM. In an observational study, higher catastrophizing scores were associated with elevated suicidal ideation, independent of depressive symptoms, underscoring its role as a critical psychological risk marker in FM [44].

4. Hopelessness in Rheumatic Diseases

Across rheumatic diseases, evidence for hopelessness' influence on disease outcomes remains extremely limited. In OA, hopelessness has only been examined in the context of depressive symptomatology [24]. In early RA, it was described qualitatively as a consequence of participation restrictions [32], and in JIA, it showed some association with pain [33]. Importantly, no study—except the recent prospective work from Bearzi et al. (2025)—has evaluated hopelessness as a prognostic factor or integrated it into routine rheumatologic assessment [35].

While research in rheumatic disease remains limited, findings from broader chronic pain population suggest that hopelessness exerts a significant influence on long-term pain outcomes and psychological adjustment. For instance, Serafini et al. demonstrated that individuals with chronic pain who reported higher levels of hopelessness experienced greater emotional distress, poorer functional outcomes, and reduced quality of life compared to those without hopelessness [45]. These patterns likely extend to rheumatic disease, where sustained pain and functional limitation can foster pessimistic expectations for recovery. Patients experiencing hopelessness are therefore more likely to disengage from self-management, physical activity or rehabilitation—behaviors that are known to alleviate pain and disability when maintained.

5. Psychological and Developmental Origins

PC does not arise in isolation; rather, it reflects an interaction between early environmental exposures, emotional learning, and cognitive–emotional vulnerability traits. A substantial body of evidence implicates adverse childhood experiences (ACEs), particularly emotional and physical abuse, as key precursors to catastrophizing tendencies later in life. In a chronic pain cohort, childhood trauma—particularly emotional abuse—significantly predicted higher PC, even after adjusting for depression and anxiety, whereas trauma exposure in adulthood did not exert the same effect [46]. These findings suggest that a sensitive developmental window exists, during which adverse social environments may shape maladaptive cognitive schemas about threat, control, and vulnerability.

Similarly, childhood trauma and abuse have been repeatedly linked to exaggerated threat perception, heightened emotional reactivity, and impaired stress-regulation circuits, including dysregulation of the hypothalamic–pituitary–adrenal axis and limbic responsivity. These alterations may foster hypervigilance to bodily sensations, persistent anticipatory anxiety about pain, and a reduced sense of coping capacity, providing a neurobiological substrate for catastrophizing to emerge as a default cognitive style.

In FM populations specifically, childhood abuse is highly prevalent and has been associated with greater bodily distress, worse physical functioning, and elevated catastrophizing [47]. While some samples did not find a direct correlation between childhood abuse and pain severity, PC moderated the relationship between early trauma and clinical burden—suggesting that catastrophizing acts as a cognitive amplifier through which early adversity translates into vulnerability to pain as an adult [46]. In other words, individuals exposed to early life stress who also develop maladaptive pain cognitions exhibit worse pain interference and functional impairment than those without such cognitions.

More surprisingly, even across immune-mediated conditions, exposure to maltreatment and psychological trauma has been shown to predispose individuals to increased PC, greater emotional dysregulation, and poorer coping resources, reinforcing the notion that catastrophizing represents a learned cognitive and affective response that is rooted in early adversity and reinforced by subsequent pain experiences [48]. This framework positions PC not simply as a mood-related epiphenomenon, but as a developmentally acquired vulnerability that is embedded in stress neurobiology and emotional learning systems.

Altogether, these findings underscore that PC often reflects developmentally ingrained cognitive heuristics shaped by trauma, threat exposure, and early stress environments. Recognizing these origins highlights the importance of trauma-informed approaches and targeted interventions (e.g., cognitive restructuring, resilience-building, mindfulness-based strategies) aimed at interrupting maladaptive appraisal cycles and restoring a sense of agency in patients with chronic pain.

6. Clinical Implications and Future Directions

The reviewed evidence indicates that PC and hopelessness are not epiphenomena of disease burden but mechanistic drivers of pain amplification, disability, and suboptimal treatment responses in rheumatic diseases [29,49]. Clinically, this supports a paradigm shift away from viewing persistent pain solely through an inflammatory lens, toward recognizing cognitive-affective and behavioral processes as central therapeutic targets.

One key mechanism linking these psychological traits to disability is the fear-avoidance cycle, in which catastrophizing and kinesiophobia fuel movement avoidance, leading to reduced physical activity, progressive deconditioning, and greater disability (Figure 2) [30,50]. Over time, physical inactivity can promote weight gain and metabolic dysfunction, which are particularly relevant in arthritis and other immune-mediated diseases, where excess adiposity contributes to low-grade inflammation and worsened disease control [51–55]. This creates a self-perpetuating cycle of pain sensitization, disability, emotional distress, and heightened inflammatory burden. Breaking this cycle requires timely recognition and targeted intervention.

From a treatment standpoint, it is critical to identify patients in whom psychological drivers outweigh inflammatory activity. In these individuals, escalation of immunosuppressive therapy may offer a limited benefit and can expose patients to unnecessary risk and healthcare burdens. Conversely, appropriately delivered psychological and behavioral interventions—including cognitive behavioral therapy, pain-coping skills training, acceptance and commitment therapy, motivational interviewing, and behavioral activation—may yield the greatest therapeutic return. These approaches can reduce PC, restore a sense of agency, and support gradual re-engagement in valued physical and social activities [43,56–58]. Integration with exercise-based rehabilitation, graded activity programs, and structured physical therapy may further enhance outcomes by addressing both the cognitive–emotional and motor–behavioral components of pain maintenance [59].

Future care models should therefore incorporate systematic screening for catastrophizing, hopelessness, and fear-avoidance profiles early in the disease course, ideally embedded within routine rheumatology pathways. Stratified approaches will allow clinicians to allocate patients to precision psychosocial interventions when these are most needed, rather than reflexively escalating pharmacotherapy. Emerging digital therapeutics, remote behavioral coaching, and blended care models (combining brief clinician-guided sessions with app-based support) may facilitate scalable delivery of these interventions across diverse healthcare systems.

Further research is needed to refine these stratification strategies, define the neurobiological substrates linking cognitive-affective traits and inflammation, and evaluate combined immunomodulatory–psychological pathways as synergistic, rather than competing, treatment modalities. Ultimately, recognizing and treating the psychological architecture of pain alongside immunological drivers represents a key frontier in improving outcomes for patients with rheumatic disease.

The sensory-discriminative component of pain (blue pathway) is transmitted via the ventral posterolateral and ventral posteromedial thalamic nuclei to the primary and secondary somatosensory cortices, supporting localization and intensity processing.

The affective-motivational component of pain (red pathway) is mediated by the mediodorsal thalamic nucleus, which projects to the medial prefrontal and cingulate cortices, integrating pain with emotional evaluation, meaning, and behavioral relevance.

The salience and arousal system (green pathway) involves the paraventricular and intralaminar thalamic nuclei, including the centromedian–parafascicular complex, projecting to the anterior cingulate cortex, striatum, and insular cortex. This network modulates the vigilance, autonomic reactivity, and detection of behaviorally relevant nociceptive signals.

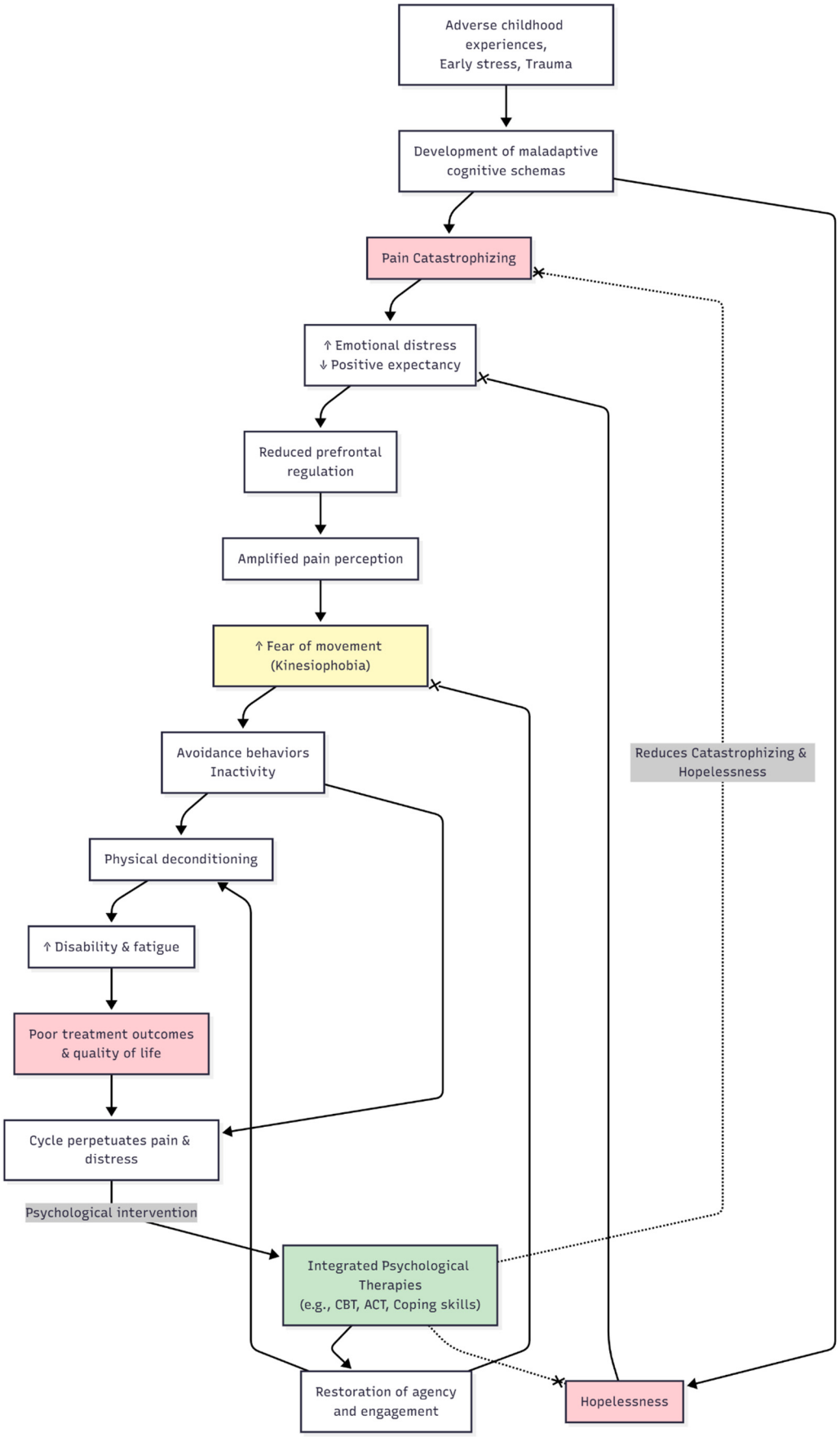


Figure 2. Clinical implications of pain catastrophizing and hopelessness and potential interventions.

Together, these thalamo-cortical circuits account for the sensory, emotional, and salience-driven dimensions of pain experience. Dysregulation of affective-motivational and salience pathways contributes to the heightened threat appraisal, hypervigilance, and pain persistence that is typical of chronic rheumatic disease.

The conceptual model illustrates the developmental and behavioral pathways linking early-life stress to pain catastrophizing, hopelessness, and disability in rheumatic disease. Early adverse experiences foster maladaptive cognitive schemas, predisposing people to pain catastrophizing, heightened emotional distress, and impaired prefrontal regulatory control. This amplifies pain perception and increases kinesiophobia, leading to avoidance behaviors, physical inactivity, and deconditioning, ultimately worsening disability and quality of life in a self-reinforcing cycle. Integrated psychological therapies disrupt this cycle by restoring agency and reducing catastrophizing and hopelessness.

Arrow notation: The pointed arrow indicates promotion/causal progression and the x-ended arrow indicates reduction/inhibition.

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Abbreviations

The following abbreviations are used in this manuscript:

ACC	Anterior Cingulate Cortex
ACEs	Adverse Childhood Experiences
ACT	Acceptance and Commitment Therapy
axSpA	Axial Spondyloarthritis
BMI	Body Mass Index
CBT	Cognitive Behavioral Therapy
CEN	Central Executive Network
CTD	Connective Tissue Disease
dlPFC	Dorsolateral Prefrontal Cortex
DMN	Default Mode Network
FM	Fibromyalgia
HPA axis	Hypothalamic–Pituitary–Adrenal Axis
JIA	Juvenile Idiopathic Arthritis
MRI	Magnetic Resonance Imaging
OA	Osteoarthritis
OR	Odds Ratio
PC	Pain Catastrophizing
PCS	Pain Catastrophizing Scale
PFC	Prefrontal Cortex
pSS	Primary Sjögren's Syndrome
PsA	Psoriatic Arthritis

QST	Quantitative Sensory Testing
QoL	Quality of Life
RA	Rheumatoid Arthritis
RNT	Repetitive Negative Thinking
SLE	Systemic Lupus Erythematosus
SpA	Spondyloarthritis
SSc	Systemic Sclerosis
tDCS	Transcranial Direct Current Stimulation
vmPFC	Ventromedial Prefrontal Cortex

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