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The Self-Concept and Feelings of Guilt and Shame in Patients with Psoriatic Arthritis: A Literature Review

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Abstract

Background: Psoriatic arthritis (PsA) is a chronic, systemic autoimmune disease that severely compromises patients' physical and psychosocial well-being. This literature review aims to synthesise contemporary scientific data to examine the multi-layered self-concept in PsA patients and evaluate the bidirectional, stress-mediated relationships between psychological attributes and somatic disease progression from a psychotherapeutic perspective.

Methods: A systematic literature search was conducted across international and national electronic databases (PubMed, ScienceDirect, ResearchGate, eLibrary, CyberLeninka) and finalized in January 2026. The search targeted peer-reviewed clinical, observational, qualitative studies, and meta-analyses published between 2015 and 2025, focusing on psychosomatic manifestations, illness representations, and emotional constructs in PsA.

Results: The findings demonstrate that the self-concept in PsA patients operates across three hierarchical levels: somatic, evaluative, and cognitive-behavioural. At the somatic level, visible skin lesions and chronic pain catalyze body dissatisfaction and skin-related shame. At the evaluative level, shame and unconscious racket guilt, examined through transactional analysis, operate as destructive internalized auto-aggression. Recent neuroimmune data confirm that depression in PsA serves as a true somatic comorbidity driven by the CRH-MC-Th17 axis. At the cognitive-behavioural level, patients exhibit severe cognitive uncertainty across Leventhal's Common-Sense Model, while alexithymia acts as a key predictor of disease exacerbation.

Conclusion: The review concludes that the psychological burden of PsA is intricately linked to its pathogenetic mechanisms, with unconscious racket guilt and alexithymia representing key predictors of disease exacerbation. Consequently, there is an urgent need to validate specific psychometric tools for guilt in PsA and to integrate specialised psychotherapy alongside psychopharmacotherapy into standard clinical guidelines to improve remission quality and reduce the healthcare system burden.

Keywords: Psoriatic arthritis; self-concept; guilt; shame; alexithymia; depression; Common-Sense Model; psychosomatic medicine; anxiety; stress; illness representations

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Introduction

Psoriatic arthritis (PsA) is a chronic, autoimmune, multifactorial, and multi-target inflammatory disease that affects the skin, joints, and internal organs (including the cardiovascular system, liver, kidneys, and gastrointestinal tract). In cases of systemic manifestations, the condition is often referred to as "psoriatic disease". The complex interaction of genetic predisposition, immune dysregulation, and environmental factors drives the development and progression of PsA, complicating therapeutic approaches and necessitating a comprehensive, multidisciplinary management strategy. Furthermore, the disease exerts a profound impact on both the psychological and psychosocial well-being of affected individuals [1].

According to various sources, the prevalence of PsA ranges from 6% to 42% among patients with psoriasis, with psoriasis itself affecting up to 11.43% of the general population in certain geographic regions [2]. This high prevalence poses a significant challenge to both healthcare systems and society as a whole. The International Federation of Psoriasis Associations (IFPA) reports that approximately 125 million people globally are estimated to live with psoriasis, implying that PsA may be diagnosed in up to 52 million individuals. The disease affects males and females with equal frequency; however, a hereditary predisposition is observed in 30% of cases, predominantly among male individuals. Conversely, psoriasis is rare among Japanese, Inuit, West African populations, and Native North and South American Indians [3, 4].

This review aims to identify the primary psychological characteristics of patients with PsA, delineate the interrelationships between these characteristics, evaluate the bidirectional influence of psychological factors on the clinical manifestations of PsA, and investigate the cognitive and emotional illness representations (the illness perception).

Methods

A database-driven literature search was conducted across several electronic databases, including PubMed, eLibrary, ScienceDirect, ResearchGate (using DOI identifiers), and CyberLeninka. The search was systematically conducted and finalized in January 2026, targeting scientific literature published in English and Russian over the last 10 years (between 2015 and 2025). The following keywords were utilized: "psoriatic arthritis", "psychosomatic manifestations", "anxiety", "stress", "anger", "guilt", "shame", "psychological states", "internal picture of the disease", and "illness representations". The search strategy encompassed clinical studies, observational studies, interview-based research, clinical trials, case reports, reviews, and meta-analyses, as well as psychometric tools validated for psychosomatic and rheumatic conditions (such as IPQ-R and HADS). This narrative biomedical review was structured in accordance with previously published guidelines for preparing descriptive reviews [5].

Historical Aspects

Psoriatic arthritis and psoriasis are traditionally categorized within the classic group of psychosomatic disorders due to the prominent role that psycho-emotional factors play in their onset and exacerbation. The concept of PsA as a psychosomatic illness is relatively modern and has evolved through several distinct historical phases:

1. **Conceptual Formulation (Early 19th to Mid-20th Century):** The term "psychosomatics" was introduced by J. Heinroth in 1818. Later, in the mid-20th century, F. Alexander formulated the "Holy Seven" classic psychosomatic disorders. Although psoriasis and PsA were not directly included in this list, they began to be viewed through the prism of this theory as conditions triggered by psychological stress and emotional suppression [6].

2. **Conceptual Development (Late 20th Century to 2015):** During this period, the psychosomatic component of the disease was formally recognized within evidence-based medicine. The necessity of an interdisciplinary treatment approach was established, emphasizing the detection of anxiety-depressive states and the integration of antidepressants, anxiolytics, and psychotherapy into standard care. Research into the psychosomatic aspects of PsA focused predominantly on the impact of chronic psychosocial stress on disease exacerbations, identifying it as a primary mechanism triggering autoimmune responses. Specific personality traits—such as high anxiety, self-criticism, and anger suppression—were found to correlate with disease duration and severity. Depression and anxiety were characterized as psychological responses to the illness itself, driven primarily by skin lesions, joint pain, and the threat of permanent disability. The primary focus of researchers remained heavily centered on cutaneous symptoms [7].

3. **The OMERACT 2016 Plenary Session (Core Outcome Measures in Rheumatology) (Fig. 1):** At this international expert consensus meeting, a mandatory core domain set for assessing PsA was officially approved. This milestone officially consolidated the integration of psychological and quality-of-life aspects into the mandatory assessment framework, placing pain, fatigue, patient global assessment of disease activity, physical function, and health-related quality of life into the "inner circle" of clinical assessment. The "middle circle" (important but optional domains) included emotional well-being and social participation (social role, work). The domains requiring further validation before core integration included depression and anxiety (as specific psychological comorbidities), sleep disturbances, independence, and treatment burden [8 - 10].

OMERACT CORE DOMAIN SET FOR PSORIATIC ARTHRITIS

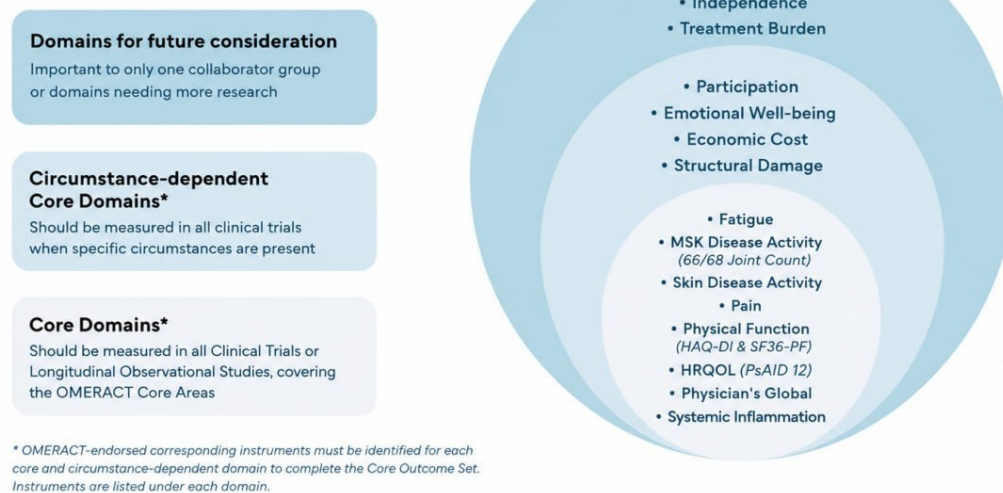


Figure 1. OMERACT core domain set for psoriatic arthritis.

A review of the current literature reveals a critical paucity of data regarding the psychological dimensions of psoriatic arthritis (PsA) and patient illness perceptions, both globally and domestically. Existing research remains predominantly constrained to the cutaneous manifestations of psoriasis and their psychosocial ramifications. Clinicians and researchers frequently underscore the complexity of delineating causal versus consequential psychological factors in disease onset and progression, highlighting a bidirectional synergy between somatic and psychiatric symptoms. Furthermore, while articular and extra-articular manifestations are routinely managed through multi-modal pharmacological and non-pharmacological strategies—such as physical therapy and nutritional interventions—the psychological etiology of PsA and its subsequent burden on mental health remain profoundly underestimated in clinical practice. This neglect persists despite established evidence that psychological well-being is as crucial to patient outcomes as somatic stability.

Additionally, a distinct lack of psychological rapport and insufficient informational or emotional support within the patient-physician dyad compromises therapeutic efficacy and exacerbates psychological distress. Patients frequently report a profound communication deficit, noting that clinicians focus almost exclusively on objective physical indicators while neglecting mental health. This clinical oversight induces feelings of isolation and significantly hinders effective patient self-management.

Finally, systemic barriers further impede optimal care; these include limited access to mental health resources, provider-level stigma, and the poor integration of psychological support into routine rheumatological care. Consequently, the necessity for highly personalized care—dictated by the heterogeneity of PsA phenotypes and diverse patient needs—is frequently overlooked, even within institutions maintaining formal hospital support programs [10, 11].

This descriptive review examines the self-concept of patients with psoriatic arthritis (PsA), focusing specifically on the manifestations of guilt and shame within its structure. It further delineates the intricate relationship between psychological status and PsA, highlighting the profound impact of psychological dimensions on disease course and clinical outcomes. Through a comprehensive literature review spanning the past decade, this paper aims to evaluate the prevalence of psychological comorbidities in PsA, as well as the bidirectional mechanisms underlying this interplay.

The Self-Concept

The self-concept (Self-Schema) (Figure 2) in chronically ill patients represents a consolidated system of internal representations regarding one's own physical and psychological attributes. Under the influence of chronic disease, this system undergoes profound alterations, fundamentally redefining how the patient perceives themselves, their illness, and their position in the social world. The structure of self-perception operates across three hierarchical levels:

- The Cognitive-Behavioral Level: Encompasses the individual's ideas regarding their physical characteristics, mental capabilities, personality traits, and characteristic behavioral responses that they consider inherent to themselves.
- The Evaluative Level: Involves self-esteem and the degree of self-acceptance or self-rejection in the context of chronic illness.
- The Somatic Level: Comprises the mental representation and awareness of one's own body, which in chronic illness may be perceived as "treacherous," "alienated," or "aesthetically compromised".

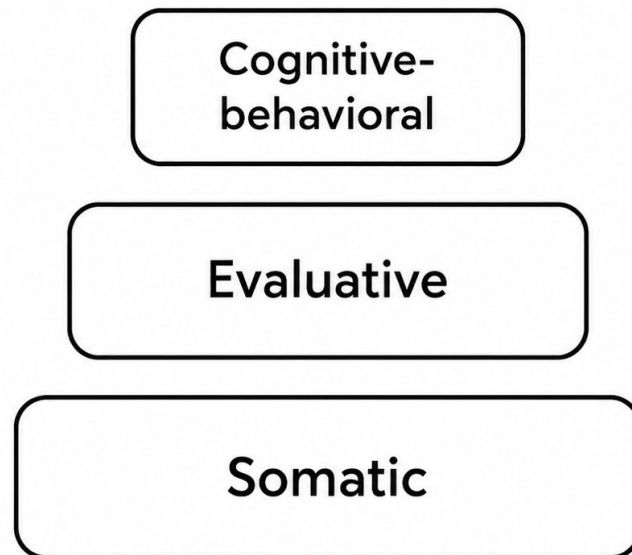


Figure 2. Self-concept structure.

The Somatic Level of Self-Perception

The self-concept of patients with PsA exhibits highly specific features, characterized by a disproportionate emphasis on the somatic level of self-perception. This is driven by highly disruptive symptoms: cutaneous manifestations (affecting 91.1% of patients), chronic pain (76.8%), profound fatigue (57%), mobility restrictions and joint dysfunction (60%), sexual dysfunction, and a high burden of comorbid conditions. More than 50% of patients present with multiorgan involvement, including obesity, cardiovascular, hepatic, and renal impairment, and malignancies [12].

Cutaneous manifestations receive substantial attention in the literature as the most prominent catalyst for skin-related shame. Studies comparing levels of shame between patients with PsA and rheumatoid arthritis (RA) demonstrate that individuals with PsA experience significantly higher levels of skin-related shame, as well as higher global and internalized shame [13]. Shame, in turn, is intricately linked to body image dissatisfaction, anxiety, loss of self-confidence, anger, rejection, and guilt, culminating in increased disease severity and a marked decline in quality of life.

Crucially, skin-related shame does not correlate directly with the objective Psoriasis Area and Severity Index (PASI). No statistically significant differences in skin-shame scores have been found between psoriasis patients with PsA and those without joint involvement. However, a powerful correlation exists between skin-related shame and perceived stigmatization (reported by 73% of patients) and embarrassment, serving as a reliable predictor of depressive disorders. While clinical severity (PASI) does not dictate the extent of perceived stigma, it significantly intensifies subjective bodily shame [13, 14].

Skin-related shame and social stigma reinforce social avoidance behaviors (e.g., avoiding public facilities, hair salons, swimming pools, and spas). This phenomenon is predominantly characteristic of younger female patients and can severely exacerbate sexual dysfunction, particularly when lesions are localized in the genital region [12].

Another critical determinant of the somatic level of self-perception is the individual's perception of pain. Pain is defined as an individual sensory and emotional experience modulated by psychological factors [12,13]. Chronic pain syndromes are invariably driven by a combination of distinct pathogenetic mechanisms (nociceptive, neuropathic, and psychogenic). It is well established that PsA, alongside other rheumatic conditions and comorbid anxiety-depressive disorders, is accompanied by an elevated production of pro-inflammatory cytokines. These cytokines activate the hypothalamic-pituitary-adrenal (HPA) axis; under the influence of stressors, a stress-induced activation

of the HPA axis triggers a subsequent cascade of pro-inflammatory cytokines, leading to central and peripheral sensitization, which severely restricts the body's adaptive capacity [16]. Consequently, the anticipation of pain activates anterior cortical regions of the brain, which can independently provoke a painful episode [12]. Consequently, the anticipation of pain activates anterior brain regions, potentially provoking an independent painful episode [12].

PsA is frequently accompanied by autonomic dysfunction; comorbid psycho-autonomic disturbances amplify the severity of the pain syndrome by lowering the pain threshold. This threshold reduction is linked to altered substance P metabolism, disruptions in the equilibrium between glutamatergic and monoaminergic pathways, and deficiencies within the central serotonergic and noradrenergic systems. Patients with PsA and concurrent autonomic dysfunction exhibit significantly higher scores not only in overall pain intensity but also in the affective distress associated with chronic pain, alongside elevated rates of anxiety and depressive disorders.

This phenomenon of pain catastrophization—defined as a negative cognitive-affective response to anticipated or actual pain—is robustly linked to poor outcomes and therapy discontinuation [16, 17]. The cognitive interpretation of a painful stimulus radically alters its subjective experience; evaluating pain as "unbearable" intrinsically amplifies its intensity. Pain in PsA frequently acquires a neuropathic character, presenting as burning or tingling sensations, which can cause the patient to subjectively overestimate the clinical activity of the disease [18].

Pain catastrophization is more pronounced in patients with anxiety and depression, highlighting a profound bidirectional relationship in PsA. Pain and depression serve as more powerful predictors of functional disability than objective disease activity (PASI) or radiographic joint damage, resulting in a blunted therapeutic response and a reduced drug retention rate. Furthermore, specific personality traits, such as perfectionism and cognitive rigidity, can further complicate the management of chronic pain, as these individuals struggle to deploy flexible coping strategies, precipitating the chronicization of the process. Comorbid personality disorders, such as borderline or obsessive-compulsive personality disorders, also severely complicate pain management [12].

It is equally critical to highlight the cascading impact of pain on fatigue, physical deconditioning, and social withdrawal. This induces functional disability, which manifests as an unwillingness to attend events or interact with loved ones due to joint stiffness and prolonged preparation times. This sequela reinforces feelings of internalized guilt and shame toward others, driven by the subjective perception of being a physical and emotional burden to family members [19].

Fatigue represents one of the most debilitating yet systematically underestimated variables within the somatic self-perception of PsA patients, though it rarely becomes the primary subject of research interest [20, 21, 22]. Chronic fatigue is identifiable in 25% of patients, with 30% categorising it as severe [20]. Patients frequently report fatigue as a more disruptive and distressing symptom than objective joint pain or cutaneous lesions. Fatigue displays a clear gender bias, affecting females significantly more than males (54.2% versus 21.4%, respectively) [19]. While fatigue correlates closely with the composite DAPSA score, it frequently exhibits independence from isolated laboratory inflammatory biomarkers (such as ESR or C-reactive protein) when analyzed outside the index framework [22].

The onset of fatigue is closely linked to anxiety, concurrent depressive symptoms, pruritus (itching), and persistent nociceptive inputs that cause sleep fragmentation [21]. At the molecular level, fatigue is driven by chronic systemic inflammation mediated by the overproduction of pro-inflammatory cytokines (such as IL-6 and IL-1RA); however, the exact mechanisms governing fatigue in PsA warrant deeper investigation [20]. Fatigue promotes withdrawal from habitual activities, social isolation, and occupational disability. This sequela reinforces feelings of internalized guilt and shame toward oneself and family members, while precipitating anxiety-depressive episodes [19, 21]. Furthermore, numerous studies confirm a bidirectional relationship between regular episodes of insomnia and heightened individual pain sensitivity [12, 23].

The high prevalence of comorbidities further complicates the somatic self-perception profile in PsA. Prominent among these are cardiovascular diseases, which represent the leading cause of mortality in this cohort (accounting for 20-56% of total deaths) [12], metabolic syndrome, malignancies, ophthalmic pathologies, hepatic and renal diseases, osteoporosis, inflammatory bowel diseases, and autonomic dysfunction [12, 16, 21]. This vast clinical burden heavily compromises body image and overall quality of life [12]. Metabolic disturbances occur with significantly higher frequency in PsA patients than in the general population, manifesting as impairments in carbohydrate and lipid metabolism (dyslipidemia and obesity).

Patients with concurrent PsA and metabolic syndrome characteristically present with abdominal obesity accompanied by elevated serum leptin levels; notably, leptin concentrations correlate with cutaneous disease activity (PASI) and can act as a direct pro-inflammatory trigger [24, 25]. Consequently, in patients with PsA and metabolic syndrome, a distinct association has been identified between abdominal obesity and polyarticular involvement. This somatic burden is accompanied not only by heightened bodily shame and guilt over physical inactivity but also by sharp increases in trait/state anxiety, depression, and pain catastrophizing levels [24 - 27].

The Evaluative Level of Self-Perception

The evaluative domain of the self-concept in PsA is predominantly populated by the interconnected affects of shame and guilt. While shame has been the subject of extensive investigation, guilt remains profoundly under-researched.

Shame and guilt are major determinants of the psychosocial status of these patients, exerting a more destructive impact on quality of life than in other inflammatory arthropathies, yet they are routinely overlooked in clinical practice.

Comparative analyses reveal that patients with PsA exhibit significantly higher scores across all dimensions of shame—including global trait, behavioral, and bodily shame—than patients with RA. This elevation is heavily tied to cutaneous symptoms, presenting predominantly as bodily shame in female cohorts and as characterological shame in younger demographics. It also correlates with objective disease activity (PASI), as skin lesions and functional articular impairments operate as mutually exacerbating stressors [14, 28].

Investigating the constructs of guilt has revealed a direct correlation between elevated levels of experienced guilt and overall disease activity. Female patients and unemployed individuals demonstrate a higher vulnerability to guilt, with a significant proportion of patients simultaneously experiencing secondary shame for their own self-blaming tendencies [15]. Crucially, guilt correlates less with the objective severity of PsA symptoms and more with psychosocial variables and social stigmatization (reported by 47% of patients). This stigma is rooted in the public's reaction to visible skin lesions, which are frequently misinterpreted as contagious or met with disgust. This triggers feelings of rejection, alienation, irritation, anger, apprehension, and embarrassment in the patient, culminating in profound social withdrawal (sometimes leading to forced resignation from work), psychosocial maladaptation, and an intensification of guilt toward loved ones due to a self-perceived status as a burden.

Patients with PsA frequently exhibit low self-esteem, characterized by a tendency to perceive themselves as unattractive and socially devalued. They experience pronounced difficulties in interpersonal interactions, struggle to establish positive self-presentation, and face obstacles in integrating into occupational collectives. Furthermore, patients tend to catastrophize interpersonal challenges [19]. The resulting chronic stress further drives disease activity, precipitating depressive and anxiety disorders, compounding shame and guilt, compromising treatment compliance, and increasing the incidence of suicidal ideation (affecting up to 10% of patients aged 18-34) [12]. Prolonged, relapsing disease courses ultimately forge specific personality adaptations, characterized by an obsessive fixation on physical appearance and hypersensitivity within interpersonal relationships [29].

As delineated above, chronic systemic inflammation and persistent pain alter the psychological sphere, compromising social adaptation. These factors lower the individual's threshold for psycho-emotional stress and can distort ego defense mechanisms. In this scenario, psychological defenses intended to stabilize the ego yield maladaptive outcomes, specifically giving rise to alexithymia. Alexithymia is a personality construct characterized by a profound deficit in the cognitive processing and regulation of affects, involving a structural dissociation between emotional experiences and physical somatic responses to life events [30].

Alexithymia is observable in 24,8% of patients with PsA, with an elevated prevalence among females due to their higher susceptibility to mood fluctuations [31 - 33]. The biological substrate of alexithymia in PsA involves elevated levels of pro-inflammatory cytokines, tumor necrosis factor (TNF), and increased laboratory markers such as CRP and ESR. A positive correlation has been established between alexithymia scores, functional impairment indices, and pain scales [34]. Alexithymic patients are highly prone to pain catastrophization due to an inability to differentiate internal emotional states from somatic sensations; conversely, they paradoxically minimize overt reports of physical and emotional distress [31], which can mislead clinicians into underestimating the true severity of the patient's condition.

Thus, alexithymia serves as a somatizing mediator between internalized distress and cutaneous-articular exacerbations, operating via the Hypothalamic-pituitary-gonadal axis (HPA axis). Given their impaired emotional regulation, alexithymic individuals are highly vulnerable to depression and anxiety.

Drawing upon classic concepts in Transactional Analysis, it can be hypothesized that racket guilt—a learned, non-authentic emotion internalized in childhood to substitute for primary, authentic emotions (such as anger or fear) prohibited by parental figures—is extensively deployed by these patients. Because guilt represents a socially acceptable response to distress, the presence of high alexithymia creates a psychological environment where racked guilt dominates the emotional landscape, effectively masking the patient's authentic emotional reactions and core psychological needs [35, 36].

Depression and anxiety are well-documented yet insufficiently managed comorbidities in PsA [37]. Across various data sources, the prevalence of depression ranges from 20% to 50% (with 20% classified as mild and 14% as moderate), while anxiety affects 15-30% of the PsA population. These disorders are more prevalent among female cohorts, where they frequently run a subclinical course [19]. The concurrent presence of disfiguring skin lesions and painful joint destruction in PsA accounts for the elevated rate of depressive comorbidity. Depression in PsA is a

multidimensional, multi-phenotypic state that frequently includes cognitive deficits (up to 44% of cases, typically manifesting as executive dysfunction, planning difficulties, word retrieval problems, decreased concentration, and slowed information processing speed), alexithymia, and anhedonia [19]. Anxiety in PsA typically manifests as elevated generalized anxiety, specific phobias, trait anxiety, and persistent worry, whereas interpersonal anxiety per se is less pronounced [38, 39].

Recent years have witnessed a surge in research examining the pathogenetic links between systemic inflammation and depression [40 - 42]. Inflammatory pathways are now recognized as a shared pathogenetic denominator for both PsA and depressive disorders, playing a definitive role in neurogenesis and mood regulation. Depression in PsA is robustly associated with higher tender joint counts, poorer global health assessments, and elevated laboratory inflammatory markers. While it maintains a tight, bidirectional relationship with chronic pain intensity, it does not demonstrate a direct correlation with objective joint swelling. Crucially, the presence of depression is associated with a significantly reduced probability of achieving clinical remission over a two-year period [43].

The complex pathophysiological mechanisms bridging inflammation and depression include the following pathways: HPA axis hyperactivity mediates the link between the stress response and the inflammatory immune response, activating cell-mediated autoimmunity and sustaining chronic systemic inflammation. This process involves the hypersecretion of neuroendocrine hormones and neurotransmitters engaged in neuroimmune cross-talk, including corticotropin-releasing hormone (CRH), adrenocorticotropic hormone (ACTH), and cortisol. These molecules act directly upon key immune effectors, specifically mast cells (MCs) and T-helper 17 (Th17) cells, which release an array of pro-inflammatory cytokines, including tumor necrosis factor (TNF), interleukin-1 (IL-1), IL-6, and IL-17.

These circulating cytokines activate the kynurenine pathway of tryptophan degradation, which simultaneously depletes central serotonin levels and generates neurotoxic metabolites. These cytokines breach the blood-brain barrier (BBB) and activate microglia, culminating in localized neuroinflammation and disrupted synaptic plasticity within the hippocampus and prefrontal cortex—the neuroanatomical regions responsible for affect regulation, which serves as a canonical pathway for depressive disorders, thereby aggravating psoriatic arthritis symptoms [44, 45].

Thus, the neuroimmune response mediated by the CRH-MC-Th17 axis represents the foundational mechanism linking the inflammatory pain syndrome and depression. These states mutually reinforce one another, allowing inflammation to be viewed as a primary pathogenetic predictor of depression, effectively transforming depression from an isolated psychiatric entity into a true somatic comorbidity.

The scheme (Fig.3) demonstrates profound, bidirectional, stress-mediated relationships between somatic and emotional manifestations and comorbid conditions, illustrating the complexity of the etiological and pathogenetic mechanisms of PsA [46].

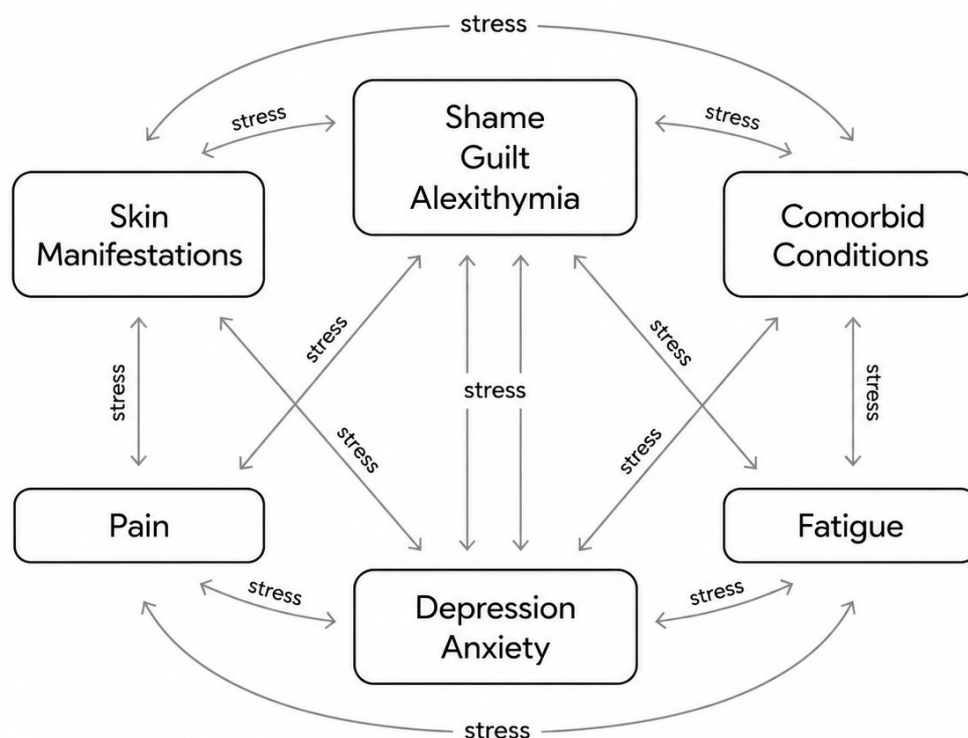


Figure 3. Interrelationship structure.

The Cognitive-Behavioral Level of Self-Perception

In accordance with Howard Leventhal's Common Sense Model (CSM), patients cognitively appraise and manage health threats by constructing illness representations across five primary domains: identity (symptom labeling), timeline (perceived chronicity or cyclicity), cause (perceived etiology), consequences (anticipated clinical and socioeconomic outcomes), and control (beliefs regarding treatability and personal manageability). Leventhal posited that encountering novel clinical manifestations or receiving a new diagnosis triggers the formulation of these subjective frameworks, which subsequently dictate emotional and behavioral coping strategies. Driven by heterogeneous sources and modulated by personality and contextual factors, these illness perceptions can undergo maladaptive shifts into cognitive distortions. Such distortions frequently precipitate dysfunctional coping mechanisms, including profound social withdrawal, substance abuse (e.g., alcohol dependency), and persistent psychological distress, ultimately acting as critical determinants of longitudinal clinical outcomes.

Dissecting these cognitive frameworks is paramount to elucidating how patients process a diagnosis of psoriatic arthritis (PsA) and approach shared therapeutic decision-making. Empirical evidence confirms that the cognitive landscape of patients with PsA aligns structurally with the CSM paradigm. Notably, due to the highly volatile and heterogeneous nature of PsA, patients frequently experience profound illness uncertainty across all five cognitive domains, which heavily exacerbates systemic distress and undermines treatment adherence [48]. Crucially, beyond these intrinsic cognitive representations, external socio-environmental macro-factors and structural determinants exert a profound influence on patients' health beliefs, continuously reshaping their overarching interpretation of the diagnosis and therapeutic trajectory.

- Cognitive uncertainty regarding disease identity is heavily driven by the profound diagnostic difficulties inherent to PsA and a severe lack of structured clinician-patient communication, creating massive barriers to effective self-management. Patients are also severely impacted by negative social comparisons, which force them to contrast themselves with healthy peers or other PsA patients, leading to feelings of inadequacy, defectiveness, and subsequent depressive episodes. Furthermore, the "invisible" nature of many symptoms induces a form of the "impostor phenomenon," where both patients and their immediate social circles underestimate the true disabling impact of the disease, severely compromising social and occupational adaptation [49, 50].
- Patients routinely encounter immense difficulties delineating the exact causes of their symptoms, frequently struggling to separate PsA manifestations from age-related changes, treatment side effects, or concurrent comorbidities. This diagnostic ambiguity is often mirrored by the attending physicians, further destabilizing the patient's cognitive baseline. Even patients who maintain rigorous healthy lifestyles express profound confusion regarding etiology, highlighting a deficit in accessible medical education. This systemic misunderstanding is exacerbated by public ignorance; patients frequently face hostile social encounters where visible skin lesions are misinterpreted as infectious, inducing trauma and social withdrawal [51, 52].
- While patients generally demonstrate a realistic awareness of the chronic, progressive nature of PsA, this exact structural certainty operates as a primary font of existential dread, treatment anxiety, and pessimistic forecasting. The highly unpredictable velocity and erratic nature of PsA flares generate acute cognitive helplessness. Patients frequently express acute guilt over being a progressive physical burden [53], noting that they would have made entirely different life and professional decisions had they accurately anticipated the disease's trajectory [49].
- Patients acknowledge that during the initial phases of the diagnostic journey, a profound deficit of clear clinical information led to highly counterproductive behavioral patterns. For example, individuals frequently continued high-impact occupational or leisure activities that significantly accelerated joint destruction. Furthermore, profound cognitive uncertainty regarding the long-term toxicity and side effects of systemic medications drives widespread medical non-compliance, medication apprehension, or total treatment refusal [49, 54].
- Because the prevailing biomedical framework focuses exclusively on pharmacological interventions, profound cognitive uncertainty regarding the exact mechanism and ultimate objective of systemic therapies severely compromises drug retention rates. A lack of clinician rapport is identified as the paramount structural barrier to disease control; patients are routinely left unguided regarding long-term symptom management. This care gap is particularly severe regarding psychological support, which is systematically ignored, leaving patients completely isolated, disoriented, and emotionally stranded [49, 55].

Consequently, inadequate understanding and illness misperceptions can yield adverse health outcomes for patients with psoriatic arthritis (PsA). Maladaptive illness beliefs may fundamentally impair an individual's capacity to perceive and process medical information. Furthermore, patients frequently experience a profound degree of cognitive uncertainty across all Common-Sense Model (CSM) domains, with external factors—such as public perceptions—exerting a substantial influence on their illness representations. Both parameters compound the psychological burden of the disease and perpetuate psychological distress. Within this framework, illness uncertainty operates as an inability to grasp the clinical meaning of disease-related events or forecast outcomes, manifesting as ambiguity, complexity, information deficits, inconsistency, and unpredictability. High levels of

uncertainty, particularly when reinforced by an uninformed social environment, are robustly linked to maladaptive coping strategies. Crucially, the functional expectations of a misinformed social network routinely clash with patients' actual physical capacities. Raising illness awareness among both patients and their immediate social circles through structured patient education regarding disease assessment and symptom management significantly fosters adaptive coping mechanisms, enhances treatment compliance, dampens anxiety, and radically improves psychosocial outcomes and social interaction.

Conclusions

1. In patients with psoriatic arthritis (PsA), the somatic level of self-perception acts as the primary driver of the internal self-concept. This phenomenon is structurally mediated by highly visible cutaneous lesions and objective joint destruction, which systematically trigger body dissatisfaction, physical alienation, and skin-related shame.
2. Distinct, stress-mediated bidirectional relationships operate between somatic symptoms (chronic pain, severe fatigue, abdominal obesity) and internalized affects (guilt, shame, anxiety, depression). These psychological factors do not correlate linearly with clinical severity indexes (such as PASI) but depend heavily on individual psychosocial variables and perceived social stigmatization.
3. Based on contemporary neuroimmune data, depressive states in PsA represent an intrinsic somatic comorbidity rather than an isolated psychiatric reaction. This process is driven by chronic systemic inflammation, where the CRH-MC-Th17 axis induces microglial activation, central serotonin depletion, and blood-brain barrier disruption.
4. At the cognitive-behavioral level, PsA patients exhibit a high degree of cognitive uncertainty across all domains of Leventhal's Common-Sense Model of Self-Regulation (identity, etiology, timeline, consequences, and control). This lack of structured illness representation frequently forces individuals to adopt maladaptive coping mechanisms and triggers medical non-compliance.
5. The relationship between unconscious racket guilt, alexithymia, and psychosomatic exacerbations remains critically overlooked by attending clinicians. Alexithymia operates as a somatizing mediator that blunts overt emotional reporting while exacerbating central pain sensitization, leading to a systematic underestimation of the disease's true psychological burden. The study of guilt remains deeply compromised by a severe deficit in valid, specialized psychometric tools. Current clinical assessments are restricted to general scales, including the Ilyin Psychometric Test for assessing self-directed and outward aggressive tendencies [56], and specific subscales from the 4DSQ [57], the SCL-90 [58], and the MMPI [59] instrument framework.

Suggestions for Future Research

Future studies should investigate and delineate the role of unconscious guilt, conceptualized not merely as a secondary consequence of somatic psoriatic arthritis (PsA) manifestations, but as a robust predictive biomarker. Specifically, it may act as a socially sanctioned, "racket" emotional substitute for auto-aggression. The current lack of empirical research exploring the correlations between unconscious guilt, alexithymia, and PsA clinical profiles leads to a systemic underestimation by clinicians regarding the impact of psychological components on disease onset and exacerbation [60].

Consequently, integrating standardized psychotherapy into current clinical care guidelines for PsA is essential to reduce the escalating burden on healthcare infrastructure and mitigate disability-adjusted life years (DALYs). Implementing early psychological interventions is projected to decrease healthcare expenditure while significantly enhancing the depth and quality of clinical remission. Over the long term, the development and integration of structured psychoeducational and corrective programs are paramount to assisting PsA patients in modulating stress responses and resolving latent auto-aggressive mechanisms [61, 62].

Declarations

Author Contributions

Conceptualization, methodology, validation, formal analysis, literature search, screening, data extraction, writing—original draft preparation, writing—review and editing, V.V. Polenko. All authors have read and agreed to the published version of the manuscript.

Data Availability Statement

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Institutional Review Board Statement

Ethical review and approval were waived for this study because it is a literature review of previously published secondary data and did not involve any direct interventions with human participants or animals.

Informed Consent Statement

This study is a narrative literature review and does not include any original studies involving human participants or animals conducted by any of the authors. Ethical approval and informed consent were not required.

Conflicts of Interest

The author declares that there is no conflict of interest regarding the publication of this manuscript.

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