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SKIN-BRAIN AXIS - A NARRATIVE REVIEW

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PSORIASIS AND PSYCHIATRIC DISORDERS: CURRENT EVIDENCE ON DEPRESSION, ANXIETY, PSYCHOSOCIAL BURDEN, AND THE SKIN-BRAIN AXIS - A NARRATIVE REVIEW

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ABSTRACT

Background: Psoriasis is a chronic immune-mediated inflammatory disease associated with substantial physical, psychological, and social burden. Beyond cutaneous manifestations, increasing evidence suggests a close relationship between psoriasis and psychiatric disorders, particularly depression and anxiety. Although psychosocial consequences such as stigmatization, social withdrawal, impaired body image, and reduced quality of life contribute significantly to mental distress, contemporary research also indicates that shared biological mechanisms may underlie this association.

Objective: This narrative review aims to summarize current evidence regarding the relationship between psoriasis and psychiatric disorders, with particular emphasis on depression, anxiety, psychosocial burden, stigma, and biological mechanisms involved in the skin-brain axis.

Methods: A narrative review was conducted based exclusively on twenty-nine peer-reviewed publications provided for this manuscript. The included literature comprised systematic reviews, meta-analyses, cross-sectional studies, observational investigations, neuroimaging studies, psychophysiological research, and psychodermatology reviews published between 2013 and 2026. Findings were synthesized qualitatively and organized into thematic domains.

Results: The reviewed literature consistently demonstrates increased prevalence of depression, anxiety symptoms, social anxiety, emotional dysregulation, self-stigmatization, and psychological distress among individuals with psoriasis. Psychosocial mechanisms include visible lesions, shame, perceived rejection, social avoidance, impaired sexual and occupational functioning, and reduced quality of life. At the same time, shared biological pathways involving TNF-alpha, IL-6, IL-17, IL-23, HPA-axis dysregulation, neuropeptides, neurotransmitter alterations, neurotrophins, and neuroimmune signaling provide a plausible mechanistic link between psoriasis and psychiatric symptoms.

Conclusions: The relationship between psoriasis and psychiatric disorders extends beyond psychosocial burden alone. Current evidence supports a bidirectional interaction involving inflammatory, neuroendocrine, neuroimmune, psychological, and social mechanisms. Routine screening for psychiatric symptoms and integration of psychodermatological care into psoriasis management may improve both dermatological and mental health outcomes.

KEYWORDS

Psoriasis, Depression, Anxiety, Psychodermatology, Skin-Brain Axis, Quality of Life

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1. Introduction

Psoriasis is a chronic, immune-mediated inflammatory skin disease characterized by recurrent erythematous plaques covered with silvery scales, epidermal hyperproliferation, and sustained immune activation. Although historically considered primarily a dermatological disorder, psoriasis is now increasingly understood as a systemic inflammatory condition associated with multiple comorbidities, including metabolic, cardiovascular, rheumatological, gastrointestinal, behavioral, and psychiatric disorders (Blackstone et al., 2022; Branisteanu et al., 2025; Dymek et al., 2025). The disease affects not only the skin but also the patient's emotional well-being, interpersonal functioning, occupational productivity, and overall quality of life.

The clinical burden of psoriasis is multidimensional. Physical symptoms such as pruritus, pain, scaling, bleeding, sleep disturbance, and joint involvement may directly impair daily functioning. However, the psychological and social consequences of psoriasis are often equally important. Visible lesions may lead to embarrassment, shame, avoidance of social situations, impaired body image, low self-esteem, and fear of negative evaluation. As a result, psoriasis can influence intimate relationships, work performance, leisure activities, social participation, and self-identity (Moon et al., 2013; O'Leary, 2025; Sahi et al., 2020).

Psychiatric disorders are among the most clinically relevant comorbidities associated with psoriasis. Depression and anxiety are consistently reported at higher rates among patients with psoriasis than in the general population (Dowlatshahi et al., 2014; Jalenques et al., 2022; Luna et al., 2023). Importantly, psychiatric

symptoms may not always correlate strongly with objective dermatological severity. Some patients with limited skin involvement may experience severe psychological distress, while others with extensive disease may maintain relatively preserved emotional functioning. This discrepancy suggests that clinical severity indices such as the Psoriasis Area and Severity Index may not fully capture patient suffering (Samela et al., 2025; Yildirim et al., 2020).

Historically, depression and anxiety in psoriasis were largely interpreted as psychological reactions to visible skin disease. From this perspective, psychiatric symptoms arise because patients experience stigma, social exclusion, discrimination, low self-confidence, and impaired quality of life. This model remains highly relevant. Stigmatization, particularly when internalized as self-stigma, has been shown to predict depression, anxiety, and reduced quality of life in patients with chronic inflammatory skin diseases (Schlachter et al., 2023). Social anxiety may also occur even when objective disease severity is not high, suggesting that perceived visibility and fear of social evaluation can be more important than the extent of skin involvement (Yildirim et al., 2020).

However, psychosocial explanations alone do not fully account for the relationship between psoriasis and psychiatric disorders. Increasing evidence suggests that psoriasis, depression, and anxiety may share inflammatory, neuroendocrine, and neuroimmune mechanisms (Fabrazzo et al., 2021; Maqbool et al., 2021; Tang et al., 2025). Cytokines central to psoriasis pathogenesis, including tumor necrosis factor- α , interleukin-6, interleukin-17, and interleukin-23, have also been implicated in mood and anxiety disorders. These mediators may influence neurotransmitter metabolism, blood-brain barrier function, microglial activation, neuroplasticity, stress responsiveness, and emotional regulation.

This evolving perspective has contributed to the development of the skin-brain axis concept. The skin-brain axis describes bidirectional communication between the skin, central nervous system, immune system, endocrine pathways, autonomic nervous system, and microbiome-related mechanisms (Mar & Rivers, 2023; Ramirez-Posada & Guillen-Burgos, 2026; Tang et al., 2025). Within this framework, psychological stress can influence skin inflammation through HPA-axis activation, sympathetic nervous system activity, neuropeptide release, and cytokine production. Conversely, inflammatory signals originating in the skin can affect brain function and contribute to psychiatric symptoms.

The hypothalamic-pituitary-adrenal axis appears particularly important. Psychological stress may trigger corticotropin-releasing hormone, adrenocorticotrophic hormone, and cortisol responses, while chronic stress may lead to dysregulated feedback mechanisms and persistent immune activation. The skin itself also contains peripheral HPA-like components, allowing local stress responses to influence keratinocyte behavior and cutaneous inflammation (Mar & Rivers, 2023; Tang et al., 2025). This provides a biological explanation for the clinical observation that stress may both trigger psoriasis exacerbations and worsen psychiatric symptoms.

Recent neuroimaging and psychophysiological studies further support a biological link between psoriasis and psychiatric morbidity. Altered functional connectivity involving brain regions responsible for emotional regulation has been reported in patients with psoriasis and depression (Liu et al., 2026). Psychophysiological evidence also suggests right-prefrontal hemispheric dominance and increased sympathetic activity among patients with plaque psoriasis (Bozsanyi et al., 2023). These findings indicate that psychiatric symptoms in psoriasis may be associated with measurable neural and autonomic alterations rather than being purely subjective psychological reactions.

The clinical implications are substantial. Depression and anxiety may worsen treatment adherence, increase perceived disease burden, reduce quality of life, and contribute to poor dermatological outcomes (Blackstone et al., 2022; O'Leary, 2025). Conversely, insufficient control of skin inflammation may perpetuate psychological distress through ongoing symptoms, stigma, sleep disturbance, and social impairment. Therefore, comprehensive psoriasis care requires not only effective anti-inflammatory treatment but also routine assessment of psychological well-being, stigma, coping, sleep, social functioning, and psychiatric symptoms.

Psychodermatology has emerged as an interdisciplinary field addressing these interactions between skin disease and mental health. This approach emphasizes integrated care involving dermatologists, psychologists, psychiatrists, nurses, dietitians, and other healthcare professionals when needed (Baskaran et al., 2025; Blackstone et al., 2022; Dymek et al., 2025). Such models are particularly relevant for patients with psoriasis, whose disease burden often reflects a combination of inflammation, visible symptoms, emotional distress, lifestyle factors, and social consequences.

Given the growing body of evidence regarding the complex relationship between psoriasis and psychiatric disorders, a narrative synthesis is warranted. The aim of this review is to summarize current evidence on depression, anxiety, psychosocial burden, stigma, shared inflammatory pathways, neurobiological mechanisms, and the skin-brain axis in psoriasis. Particular attention is given to the bidirectional nature of these relationships and to their implications for clinical practice.

2. Methodology

This study was designed as a narrative review focusing on the relationship between psoriasis and psychiatric disorders, particularly depression and anxiety. A narrative approach was selected because the topic encompasses diverse domains, including epidemiology, psychosocial burden, stigma, quality of life, inflammatory mechanisms, neuroendocrine pathways, neuroimaging, psychophysiology, and clinical management. This design allowed integration of evidence from systematic reviews, meta-analyses, cross-sectional studies, observational investigations, neuroimaging studies, psychophysiological research, and conceptual psychodermatology reviews.

Unlike a systematic review, the aim was not to perform a quantitative synthesis or meta-analysis. Instead, the objective was to provide a comprehensive qualitative synthesis of major findings and conceptual developments relevant to the association between psoriasis and psychiatric morbidity.

The review was based exclusively on twenty-nine peer-reviewed scientific publications provided for this manuscript. No additional external sources, guidelines, textbooks, databases, reports, or unpublished materials were included. This restriction was applied to maintain a clearly defined evidence base and ensure that all conclusions were derived only from the supplied literature.

The included publications were published between 2013 and 2026 and addressed one or more of the following themes: depression in psoriasis, anxiety disorders in psoriasis, psychosocial burden, quality of life, social anxiety, self-stigmatization, emotional dysregulation, resilience, shared inflammatory mechanisms, skin-brain axis communication, neuroimaging findings, psychophysiological correlates, psychopharmacology, lifestyle interventions, and multidisciplinary psychodermatological care.

Publications were included if they examined psoriasis in relation to depression, anxiety, psychological distress, psychiatric comorbidity, psychosocial functioning, quality of life, stigma, neurobiological mechanisms, inflammatory pathways, or psychodermatological management; they were peer-reviewed; they were available in full-text PDF format; and they formed part of the twenty-nine-publication source set selected for this review.

Studies and reviews were excluded from consideration if they focused exclusively on dermatological manifestations of psoriasis without addressing psychiatric, psychosocial, inflammatory, or neurobiological aspects relevant to the review question. No external publications were added during manuscript preparation.

Data were extracted qualitatively from each publication. The extracted information included study design, population characteristics when available, psychiatric outcomes, depression and anxiety measures, quality-of-life findings, psychosocial determinants, stigma-related findings, inflammatory or neurobiological mechanisms, neuroimaging or psychophysiological results, and therapeutic implications. For systematic reviews and meta-analyses, emphasis was placed on pooled prevalence estimates, odds ratios, methodological heterogeneity, and major conclusions. For original studies, emphasis was placed on sample characteristics, main results, correlations, and clinical interpretation.

Because of heterogeneity across study designs, populations, psychiatric instruments, dermatological severity measures, and outcome definitions, quantitative pooling was not performed. Instead, findings were organized thematically into the following domains: depression in psoriasis; anxiety disorders and anxiety symptoms in psoriasis; psychosocial burden and quality of life; stigma, self-stigma, and social functioning; shared inflammatory and neurobiological mechanisms; the skin-brain axis; neuroimaging and psychophysiological evidence; and therapeutic implications and multidisciplinary management.

Several methodological limitations should be acknowledged. First, this review was based only on the twenty-nine provided publications and therefore may not include all available literature on psoriasis and psychiatric disorders. Second, the included studies were heterogeneous in terms of design, sample size, diagnostic definitions, psychometric instruments, psoriasis severity assessment, and follow-up duration. Third, many original studies were cross-sectional, limiting causal inference. Fourth, no formal risk-of-bias assessment was performed because the manuscript was designed as a narrative rather than systematic review. Despite these limitations, the selected publications provide a broad and contemporary evidence base covering epidemiological, psychosocial, inflammatory, neurobiological, neuroimaging, and therapeutic aspects of the relationship between psoriasis and psychiatric disorders.

3. Results

3.1. Depression in Psoriasis

Depression is one of the most extensively documented psychiatric comorbidities in psoriasis. Across the reviewed literature, patients with psoriasis consistently demonstrated higher levels of depressive symptoms and a greater likelihood of clinical depression compared with individuals without psoriasis. Although prevalence estimates vary widely depending on methodology, population characteristics, and diagnostic tools, the overall direction of evidence is consistent: psoriasis is associated with substantial depressive burden.

One of the most influential syntheses of this relationship was a systematic review and meta-analysis showing that depressive symptoms were reported in approximately 28% of patients when assessed using questionnaires, while clinical depression was identified in a smaller but still clinically relevant proportion depending on diagnostic definitions (Dowlatshahi et al., 2014). Population-based studies indicated that patients with psoriasis were approximately 1.5 times more likely to have depression than controls (Dowlatshahi et al., 2014). The same review highlighted that prevalence estimates differed markedly according to whether depression was measured using self-report scales, diagnostic interviews, International Classification of Diseases codes, or antidepressant prescription data.

A later systematic literature review confirmed the substantial psychosocial burden of depression in psoriasis (Luna et al., 2023). Depression prevalence ranged widely, from very low estimates in some administrative datasets to markedly higher rates in studies using symptom-based screening tools. This variability reflects methodological heterogeneity rather than absence of association. Importantly, depression was generally more common in patients with more severe psoriasis and was also associated with female sex, comorbidities, sleep problems, sexual dysfunction, addictions, and poorer subjective well-being (Luna et al., 2023).

Original clinical studies further support these findings. High rates of depression among patients with psoriasis have been reported in cross-sectional research, with significant correlations between psoriasis severity, disease duration, perceived stress, anxiety, depression, and reduced quality of life (Lakshmy et al., 2015). Depressive symptoms have also been strongly associated with lower resilience, suggesting that psychological resources may influence the degree to which patients develop depressive morbidity in response to chronic inflammatory skin disease (Mitsiou et al., 2022).

Depression in psoriasis appears to be multifactorial. Psychosocial mechanisms are clearly important. Visible lesions may produce shame, embarrassment, low self-esteem, social withdrawal, impaired sexual confidence, and fear of rejection. Psoriasis affects personal, social, occupational, and sexual domains, thereby creating conditions that facilitate depressive symptoms (Sahi et al., 2020). Psoriasis has also been described as a disease with profound life impact, frequently underestimated in routine dermatological practice (Moon et al., 2013).

Stigma is an especially important contributor. Patients who perceive themselves as socially undesirable or who internalize negative societal beliefs about visible skin disease may develop chronic feelings of shame and inadequacy. Self-stigma significantly predicts depression, anxiety, and quality-of-life impairment in patients with psoriasis and atopic dermatitis (Schlachter et al., 2023). In psoriasis, younger age, current symptom burden, and lack of close social relationships were associated with greater self-stigmatization (Schlachter et al., 2023). These findings suggest that depression may arise not only from objective disease severity but also from the patient's subjective and social experience of the disease.

However, depression in psoriasis cannot be explained solely by visible lesions or psychosocial adversity. Several reviewed publications emphasized shared inflammatory and neurobiological mechanisms. Overlapping inflammatory mediators in psoriasis and depression include TNF-alpha, IL-6, IL-17, IL-23, IL-1 beta, and related immune pathways (Maqbool et al., 2021). A broader mechanistic model has also been proposed in which cytokines, neuroendocrine pathways, neuropeptides, HPA-axis dysregulation, and microbiome-related mechanisms contribute to psoriasis-associated depression (Tang et al., 2025).

The role of IL-17 is particularly notable. Psoriasis is strongly driven by the IL-23/Th17/IL-17 axis, and emerging evidence suggests that IL-17 may also influence blood-brain barrier integrity, microglial activation, neuroinflammation, and emotional regulation (Tang et al., 2025). Neuroimaging evidence supports this concept. In a resting-state functional MRI study, patients with psoriasis and depression showed altered functional connectivity involving the subgenual anterior cingulate cortex, a region strongly implicated in mood regulation (Liu et al., 2026). Connectivity changes correlated with depressive symptom severity and with IL-17 levels, suggesting a potential inflammatory-neural pathway linking psoriatic inflammation to depressive symptoms (Liu et al., 2026).

The relationship between psoriasis and depression is likely bidirectional. Depression may worsen treatment adherence, reduce motivation for self-care, intensify perceived disease burden, promote social withdrawal, and increase maladaptive coping behaviors such as alcohol use, smoking, and inactivity. Conversely, active psoriasis may worsen depression through pruritus, pain, sleep disturbance, visible lesions, stigma, and inflammatory signaling. This reciprocal interaction helps explain why psychological distress and skin symptoms may reinforce one another over time.

Overall, the reviewed evidence indicates that depression in psoriasis should be understood as a complex biopsychosocial phenomenon. It arises from the interaction of inflammatory pathways, neuroendocrine dysregulation, stigma, quality-of-life impairment, body image disturbance, social withdrawal, reduced resilience, and emotional dysregulation. Consequently, depression screening should be considered an essential component of psoriasis care.

3.2. Anxiety Disorders in Psoriasis

Anxiety disorders and anxiety symptoms are also highly prevalent among patients with psoriasis. Anxiety in psoriasis is heterogeneous and may include generalized anxiety, social anxiety, anticipatory fear, avoidance behavior, stress-related anxiety, emotional dysregulation, and subclinical anxious distress. Like depression, anxiety may be driven by both psychosocial and biological mechanisms.

A systematic review and meta-analysis specifically examining anxiety disorders and anxiety symptoms in psoriasis reported that anxiety symptoms were highly prevalent, with a pooled prevalence of approximately 34% (Jalenques et al., 2022). Social anxiety disorder, generalized anxiety disorder, and unspecified anxiety disorder were also common. Case-control analyses demonstrated that patients with psoriasis had increased odds of anxiety symptoms and unspecified anxiety disorder compared with controls (Jalenques et al., 2022).

This distinction between symptoms and disorders is clinically relevant. Anxiety symptoms may cause substantial impairment even when diagnostic criteria for an anxiety disorder are not fully met. Patients may experience chronic worrying, embarrassment, hypervigilance, fear of rejection, and avoidance of situations in which their skin might be visible. Such symptoms may progress over time if unrecognized. Conversely, established anxiety disorders require psychiatric assessment and targeted treatment.

High rates of anxiety symptoms among patients with psoriasis have been reported, with significant associations between anxiety, depression, stress, psoriasis severity, and disease duration (Lakshmy et al., 2015). These findings suggest that anxiety may increase with cumulative disease burden. However, other studies indicate that anxiety may occur independently of objective disease severity, especially when social evaluation is involved.

A controlled cross-sectional study demonstrated that patients with psoriasis had significantly higher social anxiety than healthy controls (Yildirim et al., 2020). Social fear and social avoidance were significantly associated with impaired quality of life, while PASI scores did not significantly correlate with social anxiety (Yildirim et al., 2020). This finding suggests that the psychological burden of psoriasis may depend more on lesion visibility, perceived stigma, fear of negative evaluation, and personality factors than on total body surface involvement.

Social anxiety is particularly relevant in psoriasis because visible lesions may provoke misunderstanding, rejection, or fear of contagion among others. Patients may avoid swimming pools, gyms, public changing rooms, social gatherings, dating, or physical intimacy. Avoidance may reduce short-term embarrassment but reinforce long-term anxiety by preventing corrective social experiences and increasing isolation.

Stigma and self-stigma play central roles in anxiety. Self-stigmatization significantly predicts anxiety and depression (Schlachter et al., 2023). Patients who internalize negative social beliefs may become hypervigilant in social situations and anticipate rejection even when others behave neutrally. This cognitive pattern resembles mechanisms observed in social anxiety disorder.

Emotional dysregulation may further increase vulnerability to anxiety. Psoriasis patients frequently exhibit emotional dysregulation and alexithymic features (Samela et al., 2025). Patients with alexithymia may struggle to identify and communicate emotional distress, resulting in somatic complaints, irritability, sleep disturbance, treatment dissatisfaction, or nonspecific distress. Such features may complicate recognition of anxiety in dermatology settings.

Stress is another key mechanism. Stress has been described as both a cause and consequence of psoriasis, producing a vicious circle (Foks-Ciekalska et al., 2023). Psychological stress may exacerbate inflammatory activity, while worsening skin symptoms generate further stress and anticipatory anxiety. Through HPA-axis

activation, sympathetic nervous system activity, neuropeptide release, and cytokine production, anxiety may influence psoriasis biologically rather than only behaviorally.

Anxiety also affects treatment outcomes. Patients with high anxiety may be more likely to perceive treatments as ineffective, discontinue therapy, miss appointments, or avoid discussing concerns. Anxiety may also intensify pruritus perception, sleep disturbance, and social withdrawal. Conversely, inadequate control of psoriasis may increase anxiety and hopelessness. This reciprocal relationship supports integrated dermatological and psychological care.

Overall, anxiety in psoriasis is common, multifactorial, and clinically significant. It reflects the interaction of visible disease, stigma, self-stigma, emotional dysregulation, stress sensitivity, inflammatory pathways, personality traits, and coping resources. Routine screening for anxiety symptoms should therefore be incorporated into psoriasis management.

3.3. Psychosocial Burden and Quality of Life

The psychosocial burden of psoriasis is one of the most consistent findings across the reviewed literature. Although psoriasis is not usually life-threatening, its impact on quality of life may be comparable to other chronic systemic diseases. The burden includes physical discomfort, emotional distress, social restriction, impaired work functioning, sexual difficulties, sleep disturbance, and reduced self-esteem.

Quality-of-life impairment in psoriasis is multidimensional. Physical symptoms such as pruritus, pain, scaling, bleeding, and discomfort may directly affect daily functioning. At the same time, the visible and chronic nature of lesions creates psychological and social difficulties. Patients may experience shame, frustration, irritability, helplessness, and fear of negative judgment. These reactions may contribute to depression, anxiety, social withdrawal, and avoidance.

Psoriasis severity correlates positively with depression, anxiety, and perceived stress, and negatively with social relationships and environmental domains of quality of life (Lakshmy et al., 2015). Patients with psychiatric morbidity have worse quality-of-life outcomes (Lakshmy et al., 2015). However, several publications emphasized that quality of life may not correlate perfectly with objective severity. PASI scores may fail to capture visibility, stigma, daily functioning, and emotional burden (Blackstone et al., 2022).

Lesion location is therefore highly important. Small lesions on the face, hands, scalp, nails, or genital area may cause severe distress, while larger lesions in covered areas may be less socially disabling. This discrepancy shows why clinicians should assess both objective severity and subjective burden. Patient-reported outcomes such as the Dermatology Life Quality Index may help identify distress not visible through clinical examination alone.

Social functioning is frequently impaired. Patients may avoid social contact because they fear being stared at, questioned, rejected, or misunderstood. Avoidance and concealment are common coping strategies in psoriasis (Blackstone et al., 2022). These behaviors may reduce immediate embarrassment but increase loneliness, reinforce shame, and reduce social support over time.

Social fear and avoidance correlate with impaired quality of life (Yildirim et al., 2020). This finding supports the view that psoriasis-related disability is not only physical but also interpersonal. The fear of being negatively evaluated may become a major determinant of quality of life, even when clinical severity is moderate.

Relationships and sexuality are also affected. Patients may feel unattractive, avoid intimacy, or fear rejection by partners. Genital involvement can directly impair sexual activity, but even non-genital lesions may reduce body confidence. These issues are often underreported because patients may feel embarrassed to discuss them during dermatology appointments.

Occupational functioning may also suffer. Psoriasis can reduce work productivity through physical symptoms, fatigue, treatment burden, stigma, and reduced confidence. Patients in public-facing roles may feel particularly vulnerable. Psoriasis can affect employment, relationships, social life, and leisure activities, reinforcing the need for holistic assessment (Moon et al., 2013).

Sleep disturbance represents another major contributor. Itching, pain, anxiety, and stress may disrupt sleep, while poor sleep worsens emotional regulation, fatigue, coping capacity, and inflammatory activity. Sleep optimization should be part of multidisciplinary psoriasis care because sleep disturbance may influence both mental health and disease progression (Dymek et al., 2025).

Substance use may reflect maladaptive coping. Psychiatric and behavioral comorbidities in patients with psoriasis include tobacco use disorder, alcohol use disorder, binge eating, anxiety, and depression (Branisteanu

et al., 2025). Smoking, alcohol use, overeating, and inactivity may worsen systemic inflammation, reduce treatment response, and increase psychiatric vulnerability.

The psychosocial burden of psoriasis is often underrecognized. Many patients experience emotional effects of skin disease, but few receive psychological support (Blackstone et al., 2022). Psychological distress is often unidentified and untreated despite its impact on quality of life, treatment outcomes, and healthcare delivery (O'Leary, 2025).

Overall, psoriasis profoundly affects quality of life across emotional, social, occupational, sexual, and behavioral domains. Its burden cannot be measured adequately by skin severity alone. Comprehensive care should therefore include patient-reported outcomes, psychological screening, stigma assessment, sleep evaluation, and discussion of daily functioning.

3.4. Stigma, Self-Stigma, and Social Functioning

Stigmatization is one of the most important psychosocial mechanisms linking psoriasis with psychiatric morbidity. Because psoriasis is visible and often misunderstood, patients may encounter staring, avoidance, intrusive questions, rejection, discrimination, and misconceptions about contagion. These experiences may alter self-perception and social behavior even when disease severity is limited.

Stigma operates at several levels. External stigma refers to negative reactions from others. Perceived stigma reflects the patient's belief that others judge them negatively. Self-stigma occurs when patients internalize negative social attitudes and incorporate them into their own identity. Self-stigma may lead to shame, guilt, low self-worth, helplessness, and avoidance.

Stigmatization has been described as a central aspect of psoriasis-related distress (Moon et al., 2013). Patients may experience public rejection, humiliation, and disgust reactions. Patients often feel unattractive, ashamed, and socially rejected, especially when lesions affect visible areas (Sahi et al., 2020).

A cross-sectional study provided particularly strong evidence regarding self-stigma (Schlachter et al., 2023). Self-stigmatization significantly predicted depression, anxiety, and quality-of-life impairment in patients with psoriasis and atopic dermatitis (Schlachter et al., 2023). In psoriasis, self-stigma was associated with current symptoms, younger age, and absence of close social relationships. These findings suggest that self-stigma is not merely a subjective discomfort but a measurable predictor of psychiatric burden.

Younger patients may be especially vulnerable because appearance, social acceptance, dating, sexuality, and identity formation are particularly salient. Lack of close relationships may reduce protection against negative self-perception. Social support may therefore buffer the psychological consequences of stigma.

Stigma often leads to concealment and avoidance. Patients may cover lesions, avoid exposing skin, withdraw from social activities, or avoid intimacy. These strategies may reduce immediate discomfort but maintain anxiety and isolation. Concealment and avoidance are common coping responses associated with embarrassment, shame, anxiety, depression, and perceived stigmatization (Blackstone et al., 2022).

Social anxiety is one of the clearest clinical expressions of stigma-related distress. Elevated social anxiety has been observed in psoriasis patients, with social fear and avoidance linked to quality-of-life impairment (Yildirim et al., 2020). Personality traits also influence social anxiety: neuroticism increases risk, while extraversion appears protective (Yildirim et al., 2020). This suggests that individual psychological characteristics shape how patients interpret and cope with psoriasis-related social experiences.

Psychological distress may be underestimated, especially when patients struggle to describe emotional experiences (O'Leary, 2025). Direct questioning about social avoidance, shame, relationships, work, body image, and support needs is therefore necessary. Patients may not spontaneously report stigma because doing so can itself feel embarrassing.

Stigma may also interact with biological mechanisms. Chronic social stress can activate the HPA axis and sympathetic nervous system, potentially increasing inflammatory activity. Thus, stigma may not only result from psoriasis but may also contribute indirectly to disease persistence through stress-mediated pathways.

Anti-stigma interventions should therefore be part of psychodermatological care. Psychoeducation, cognitive behavioral therapy, social skills support, self-esteem work, body image interventions, and resilience-building strategies may help reduce internalized stigma. Clinicians should explicitly communicate that psoriasis is not contagious and that psychological suffering is a legitimate part of disease burden.

Overall, stigma and self-stigma occupy a central position in the relationship between psoriasis and psychiatric disorders. They influence self-esteem, social participation, emotional well-being, adherence, and quality of life. Assessment of stigma should therefore be incorporated into comprehensive psoriasis management.

3.5. Shared Inflammatory and Neurobiological Mechanisms

Although psychosocial burden is highly relevant, psoriasis-associated depression and anxiety cannot be explained by psychosocial factors alone. The reviewed literature increasingly supports shared inflammatory and neurobiological mechanisms linking psoriasis with psychiatric symptoms.

Psoriasis is characterized by immune activation involving dendritic cells, T lymphocytes, keratinocytes, macrophages, neutrophils, mast cells, and cytokine networks. Key mediators include TNF-alpha, IL-1 beta, IL-6, IL-17, IL-22, and IL-23. These cytokines drive keratinocyte proliferation, immune-cell recruitment, and chronic cutaneous inflammation. Many of the same mediators have been implicated in depression and anxiety.

The neuro-dermatological association between psoriasis and depression has been described as an immune-mediated inflammatory process (Maqbool et al., 2021). TNF-alpha, IL-6, IL-17, IL-23, IL-1 beta, and related pathways may influence neurotransmitter metabolism, serotonin transporter activity, neuroplasticity, and stress-system regulation. This supports the view that depression in psoriasis may partly reflect systemic inflammatory burden.

A systematic review of shared biological mechanisms in chronic inflammatory skin diseases and mental disorders identified BDNF/TrkB signaling and NF-kappa B/p38 MAPK pathways as potentially relevant in psoriasis-related models (Fabrazzo et al., 2021). Clinical evidence suggested that IL-6, IL-18, and IL-17A may act as inflammatory mediators in psoriasis with comorbid depression (Fabrazzo et al., 2021).

The IL-17/IL-23 axis is especially important. In psoriasis, IL-23 promotes Th17 activation and IL-17A production. IL-17 may also contribute to neuroinflammation, blood-brain barrier dysfunction, glial activation, and emotional dysregulation (Tang et al., 2025). These mechanisms provide a plausible molecular bridge between psoriatic inflammation and psychiatric symptoms.

Microglial activation is another key pathway. Peripheral inflammatory cytokines may influence the brain by crossing or signaling through the blood-brain barrier, activating microglia, altering synaptic function, and promoting neuroinflammatory responses. Activated microglia may influence tryptophan metabolism through the kynurenine pathway, reducing serotonin availability and generating neuroactive metabolites. This may contribute to depressive symptoms, fatigue, cognitive impairment, and altered stress responsiveness.

TNF-alpha may also influence serotonergic signaling. In psoriasis, TNF-alpha contributes to immune activation and plaque formation. In depression, TNF-alpha has been associated with altered serotonin transport, glutamatergic signaling, and reduced neuroplasticity. IL-6 may similarly influence HPA-axis activation, fatigue, sleep disturbance, and mood symptoms.

BDNF is relevant because of its role in neuronal survival, synaptic plasticity, and antidepressant response. Reduced BDNF signaling has been implicated in depression, and altered BDNF/TrkB signaling has been discussed as a potential pathway in psoriasis-associated depressive symptoms (Tang et al., 2025). This suggests that neurotrophic mechanisms may link skin inflammation with mood regulation.

The HPA axis provides another major mechanism. Chronic stress may lead to dysregulated cortisol signaling, glucocorticoid resistance, and persistent inflammation. The skin also contains peripheral HPA-like structures capable of producing CRH, ACTH, and cortisol. CRH expression in psoriatic lesions may promote cytokine release, keratinocyte activation, and neuroinflammatory signaling (Tang et al., 2025).

Serotonin, melatonin, and neuropeptides may also participate. Altered serotonin transporter activity and reduced serotonergic signaling may contribute to depression in psoriasis (Maqbool et al., 2021). Reduced melatonin levels have been described in both psoriasis and depression and may contribute to sleep disturbance, inflammation, oxidative stress, and mood symptoms (Maqbool et al., 2021; Sahi et al., 2020). Substance P and other neuropeptides may promote neurogenic inflammation, itch, mast-cell activation, and anxiety.

Metabolic inflammation may amplify these mechanisms. Inflammatory biomarkers in obesity-associated psoriasis include leptin, resistin, IL-6, TNF-alpha, and adipokine dysregulation (Rodriguez-Cerdeira et al., 2019). Because obesity, psoriasis, depression, and metabolic syndrome frequently coexist, metabolic inflammation may intensify both dermatological and psychiatric risk.

Overall, shared inflammatory and neurobiological mechanisms provide a strong biological foundation for psoriasis-associated psychiatric morbidity. These mechanisms do not replace psychosocial explanations but complement them. Psoriasis-associated depression and anxiety should therefore be understood as outcomes of interacting immune, neural, endocrine, metabolic, psychological, and social pathways.

3.6. The Skin-Brain Axis

The skin-brain axis provides a unifying framework for understanding the relationship between psoriasis and psychiatric disorders. This model describes bidirectional communication between the skin, immune system, endocrine system, peripheral nervous system, central nervous system, and microbiome-related mechanisms.

Psoriasis has been described as a psychodermatological disorder involving the brain-skin connection, psychological distress, stress physiology, and quality-of-life impairment (Moon et al., 2013). The mind-body connection in dermatological conditions has also been framed in terms of neuroimmune and neuroendocrine mechanisms relevant to depression and anxiety (Mar & Rivers, 2023). More recently, the skin-brain axis has been conceptualized as a network involving neuroimmune, neuroendocrine, microbial, neurotransmitter, and neurotrophic pathways (Ramirez-Posada & Guillen-Burgos, 2026).

The skin is highly innervated, immunologically active, hormonally responsive, and exposed to environmental and social stressors. Keratinocytes can produce cytokines, neuropeptides, and stress-related mediators. Immune cells in the skin communicate with peripheral nerves, while systemic inflammatory signals can influence brain function. Conversely, psychological stress can alter skin inflammation through HPA-axis activation, sympathetic activity, neuropeptide release, and immune modulation.

Stress is central to this model. Stress has been described as both a cause and consequence of psoriasis (Foks-Ciekalska et al., 2023). Stress may trigger psoriasis flares, while worsening skin symptoms increase distress. This cycle may continue through endocrine, immune, behavioral, and psychosocial pathways.

The HPA axis links stress with inflammation. Acute HPA activation is adaptive, but chronic activation may lead to dysregulated cortisol signaling and persistent immune activation. The skin itself may generate local stress responses through CRH, ACTH, and cortisol-like pathways. Local CRH signaling in psoriatic lesions may promote inflammation and interact with central stress pathways (Tang et al., 2025).

Neuropeptides are another important component. Substance P, calcitonin gene-related peptide, neuropeptide Y, and nerve growth factor can influence inflammation, itch, vascular regulation, pain, and emotional responses. Substance P is particularly relevant because it is released during stress and can contribute to mast-cell activation and neurogenic inflammation.

Inflammatory cytokines produced in psoriatic skin may influence the brain. TNF-alpha, IL-1 beta, IL-6, IL-17, IL-23, and related mediators may affect neurotransmitters, microglia, blood-brain barrier function, synaptic plasticity, and stress responsiveness. Through these pathways, psoriasis may contribute biologically to depression, anxiety, fatigue, and cognitive symptoms.

The skin-brain axis also helps explain why psychological symptoms may not correlate perfectly with PASI. PASI measures skin extent and severity, but does not capture lesion visibility, stigma, sleep disturbance, systemic inflammation, neuroimmune signaling, emotional regulation, or patient perception. Therefore, psychiatric symptoms may persist despite dermatological improvement if neural, behavioral, or cognitive patterns have become established.

Overall, the skin-brain axis integrates biological and psychosocial explanations. Psoriasis is visible and stigmatizing, but it is also inflammatory and systemic. Depression and anxiety may emerge through both social suffering and immune-neural mechanisms. This framework supports integrated psychodermatological care.

3.7. Neuroimaging and Psychophysiological Evidence

Neuroimaging and psychophysiological studies provide objective support for the biological relationship between psoriasis and psychiatric morbidity. A resting-state functional MRI study in psoriasis patients with depression and matched controls reported altered functional connectivity involving the subgenual anterior cingulate cortex, a key region in emotional regulation (Liu et al., 2026).

Specifically, increased sgACC connectivity with default mode network regions, including the posterior cingulate cortex and angular gyrus, was observed (Liu et al., 2026). Altered connectivity with executive network regions, including the superior and middle frontal gyri, was also reported. sgACC-angular gyrus connectivity correlated with depressive symptom severity, while sgACC-frontal connectivity correlated with IL-17 levels (Liu et al., 2026). These findings support a dual-circuit model in which depressive symptoms and inflammatory activity relate to distinct but interacting neural pathways.

This evidence is important because it connects psoriasis-associated inflammation with measurable brain changes. The default mode network is involved in self-referential processing and rumination, while prefrontal executive regions regulate emotion and cognition. Altered connectivity in these networks may contribute to depressive symptoms in psoriasis.

Frontal hemispheric lateralization in plaque psoriasis and atopic dermatitis has been assessed using EEG, heart rate variability, and psychological tests (Bozsanyi et al., 2023). Patients with psoriasis demonstrated significant right-prefrontal lateralization compared with healthy controls. Right-prefrontal dominance has been associated with negative affect, behavioral inhibition, stress sensitivity, and vulnerability to anxiety and depression. Elevated sympathetic nervous system activity was also observed in psoriasis (Bozsanyi et al., 2023).

These findings suggest that psoriasis may be associated with autonomic and cortical patterns relevant to emotional regulation. Increased sympathetic activity may reflect chronic stress burden and may also contribute to inflammatory dysregulation. Right-prefrontal dominance may indicate heightened vulnerability to negative affective processing.

Emotional dysregulation and alexithymia in psoriasis provide additional evidence of altered emotional processing (Samela et al., 2025). Emotional dysregulation may impair coping, increase stress reactivity, and contribute to depression and anxiety. Alexithymia may also make distress more difficult to recognize clinically because patients may struggle to describe emotions.

Resilience has been identified as a protective factor. Patients with more severe depressive symptoms had lower resilience, and resilience explained a substantial proportion of variance in depression scores (Mitsiou et al., 2022). This suggests that individual psychological resources influence whether chronic inflammatory and social stressors translate into psychiatric symptoms.

Dermatological treatment alone may be insufficient to resolve psychopathological symptoms in chronic skin disease (Costeris & Petridou, 2026). Although psoriasis was grouped with eczema in that study, the findings support the broader principle that psychological distress should be assessed directly rather than assumed to improve automatically with dermatological treatment.

Together, neuroimaging, EEG, autonomic, emotional regulation, and resilience studies support a multidimensional model of psoriasis-associated psychiatric morbidity. Psychiatric symptoms are associated with measurable changes in neural connectivity, hemispheric activity, autonomic regulation, emotional processing, and psychological adaptation.

3.8. Therapeutic Implications and Multidisciplinary Management

The reviewed evidence has important implications for clinical practice. Psoriasis management should not be limited to lesion clearance or reduction of inflammatory severity. It should also include assessment of depression, anxiety, distress, self-stigma, social functioning, sleep, coping, substance use, and quality of life.

Routine screening is essential. Clinicians should not assume that psychological burden correlates with PASI (Blackstone et al., 2022). Psychological distress is common but often underrecognized and untreated (O'Leary, 2025). Standardized tools such as PHQ-9, GAD-7, HADS, DLQI, and distress screening instruments may help identify patients requiring further support.

Clinical conversation is equally important. Patients may not spontaneously disclose distress, stigma, sexual difficulties, suicidal ideation, or avoidance behaviors. Direct but sensitive questions about mood, anxiety, sleep, social participation, relationships, work, body image, coping, and support needs should be incorporated into consultations.

Psychological interventions may improve outcomes. Cognitive behavioral therapy can address shame, avoidance, catastrophizing, hopelessness, and maladaptive beliefs. Mindfulness-based interventions may reduce stress reactivity and rumination. Compassion-focused approaches may help patients with self-stigma and body image distress. Psychoeducation may reduce misconceptions and improve self-management.

Resilience-building interventions may reduce depressive symptoms (Mitsiou et al., 2022). Addressing self-stigma through counseling, coping strategies, self-esteem enhancement, psychoeducation, and cognitive behavioral therapy may also improve psychological functioning (Schlachter et al., 2023). These approaches may remain useful even when skin disease persists.

Psychopharmacology may be necessary for clinically significant psychiatric disorders. Psychopharmacological approaches in dermatology include antidepressants, anxiolytics, and antipsychotics (Katamanin et al., 2026). Psychotropic treatment should be individualized and coordinated with dermatological care, especially because some medications may affect skin disease.

Biologic therapies may also influence psychiatric symptoms. Anti-inflammatory biologic therapies may reduce depressive symptoms in psoriasis (Holsken et al., 2021). Biologics targeting inflammatory pathways may also improve psychological outcomes (Blackstone et al., 2022). However, it remains unclear whether

these benefits result from direct reduction of systemic inflammation, improved skin clearance, reduced stigma, better sleep, or combined mechanisms.

Lifestyle interventions are also relevant. A multidisciplinary model integrating diet, physical activity, psychological support, and sleep interventions has been proposed for psoriasis management (Dymek et al., 2025). Weight management may reduce metabolic inflammation and improve treatment response. Physical activity may improve mood, sleep, cardiovascular risk, and inflammatory balance. Sleep optimization may improve both emotional regulation and disease burden.

Multidisciplinary psychodermatology clinics may be ideal for complex cases. Such services can integrate dermatologists, psychiatrists, psychologists, nurses, dietitians, and physiotherapists. This model recognizes that psoriasis affects the whole person and that optimal care requires attention to skin inflammation, mental health, lifestyle, social functioning, and long-term coping.

Overall, effective psoriasis care should aim not only for dermatological improvement but also for improved emotional well-being, reduced stigma, better sleep, stronger resilience, social participation, and quality of life.

4. Discussion

This narrative review synthesized evidence from twenty-nine publications examining the association between psoriasis and psychiatric disorders, with particular emphasis on depression, anxiety, psychosocial burden, shared inflammatory mechanisms, neuroimaging findings, and the skin-brain axis. Across diverse study designs, the reviewed evidence consistently supports a close relationship between psoriasis and psychiatric morbidity.

Depression appears to be one of the most prevalent psychiatric comorbidities in psoriasis. Increased prevalence and odds of depressive symptoms and clinical depression have been demonstrated in systematic review and meta-analytic evidence (Dowlatsahi et al., 2014), while a broad systematic literature review confirmed substantial psychosocial burden across patient populations (Luna et al., 2023). Original studies further show that depressive symptoms are associated with disease severity, stress, reduced quality of life, and low resilience (Lakshmy et al., 2015; Mitsiou et al., 2022).

Anxiety is similarly important. Anxiety symptoms and anxiety disorders are more common in psoriasis (Jalenques et al., 2022), while increased social anxiety and avoidance have also been reported (Yildirim et al., 2020). Importantly, social anxiety may not correlate with PASI, suggesting that objective severity is insufficient to estimate psychological burden (Yildirim et al., 2020). This finding has major clinical implications because patients with apparently mild disease may still experience severe distress.

A recurring theme is the gap between clinical severity and patient experience. Traditional dermatological measures focus on lesion extent, erythema, scaling, and thickness. They do not adequately capture visibility, stigma, itch, sleep disturbance, treatment burden, body image, social avoidance, sexual difficulties, or emotional meaning. Therefore, psoriasis assessment should include patient-reported outcomes and psychological screening.

Stigma and self-stigma emerged as central psychosocial mechanisms. Self-stigmatization significantly predicts depression, anxiety, and quality-of-life impairment (Schlachter et al., 2023). Shame, concealment, avoidance, and fear of rejection have also been repeatedly emphasized in the psychodermatological literature (Blackstone et al., 2022; Moon et al., 2013; O'Leary, 2025; Sahi et al., 2020). These mechanisms help explain why visible skin disease can become a chronic psychiatric burden.

However, psychosocial mechanisms are only part of the explanation. The reviewed literature strongly supports biological overlap between psoriasis, depression, and anxiety. Common inflammatory pathways involve TNF-alpha, IL-6, IL-17, IL-23, IL-1 beta, IL-18, BDNF/TrkB signaling, and NF-kappa B/p38 MAPK pathways (Fabrazzo et al., 2021; Maqbool et al., 2021). Hormones, neuroendocrine pathways, neuropeptides, the microbiome, and HPA-axis dysregulation have also been implicated (Tang et al., 2025).

The IL-17 pathway is especially important because it links core psoriasis immunology with potential neuropsychiatric mechanisms. Direct evidence that IL-17 levels correlate with altered brain connectivity in psoriasis patients with depression suggests that psoriatic inflammation may influence emotional regulation networks and supports the concept of inflammation-associated depression (Liu et al., 2026).

The skin-brain axis offers the most comprehensive framework for integrating these findings. It explains how psychological stress may worsen psoriasis through HPA-axis activation, sympathetic activity, and neuroimmune signaling, and how cutaneous inflammation may influence brain function through cytokines,

neuropeptides, neurotransmitters, and microglial activation (Mar & Rivers, 2023; Ramirez-Posada & Guillen-Burgos, 2026; Tang et al., 2025). This model avoids a false dichotomy between psychological and biological explanations. In psoriasis, psychiatric symptoms may be simultaneously social, emotional, inflammatory, endocrine, and neural.

Neuroimaging and psychophysiological evidence strengthen this paradigm. Altered sgACC connectivity in psoriasis with depression has been reported (Liu et al., 2026). Right-prefrontal lateralization and sympathetic activation have also been demonstrated in plaque psoriasis (Bozsanyi et al., 2023). Emotional dysregulation has been identified as a psychodermatological feature (Samela et al., 2025), while resilience appears protective against depressive symptoms (Mitsiou et al., 2022). Together, these findings indicate that psoriasis-associated psychiatric morbidity involves measurable brain, autonomic, and emotional regulation patterns.

The clinical implications are substantial. Dermatologists should not rely only on PASI or lesion clearance when evaluating treatment success. Patients may require psychological support even when skin symptoms improve. Screening for depression, anxiety, distress, stigma, sleep problems, substance use, and suicidal ideation should become routine, especially in patients with visible lesions, poor quality of life, high stress, or treatment dissatisfaction.

Multidisciplinary care is strongly supported. Integrated dermatological and mental health care has been emphasized across several reviews and clinical discussions (Baskaran et al., 2025; Blackstone et al., 2022; Dymek et al., 2025; Katamanin et al., 2026; O'Leary, 2025). Psychodermatology clinics may provide the most appropriate setting for complex patients because they address both inflammatory disease and psychological suffering.

Future research should prioritize longitudinal studies. Many available studies are cross-sectional, limiting causal inference. It remains unclear whether inflammation precedes psychiatric symptoms, whether psychiatric symptoms worsen inflammation, or whether both processes reinforce each other in cycles. Studies measuring cytokines, neuroimaging outcomes, HPA-axis markers, sleep, quality of life, stigma, and psychiatric symptoms over time would help clarify causality.

Future trials of biologic therapies should include standardized psychiatric outcomes. If anti-inflammatory treatments improve depression and anxiety, researchers should determine whether this occurs through skin clearance, reduced systemic inflammation, improved sleep, reduced stigma, or direct neuroimmune modulation. Such work could help develop personalized treatment strategies for patients with inflammation-associated psychiatric symptoms.

There is also a need for psoriasis-specific psychological assessment tools. Generic scales may miss disease-specific distress related to visibility, concealment, itch, treatment burden, sexuality, social stigma, and body image. A validated psoriasis distress instrument could improve both clinical practice and research comparability.

This review has limitations. It was conducted as a narrative review and did not include formal risk-of-bias assessment or quantitative synthesis. The included studies varied in design, population, diagnostic tools, and outcome measures. The review was also based exclusively on twenty-nine provided publications, which strengthens source consistency but may limit comprehensiveness. Nevertheless, the selected literature provides a broad evidence base covering epidemiology, psychosocial burden, stigma, inflammation, neurobiology, neuroimaging, psychophysiology, and treatment.

Overall, the evidence supports a paradigm shift. Psoriasis should not be understood solely as a skin disease with occasional psychological consequences. Rather, it should be recognized as a systemic psychodermatological condition involving bidirectional interactions between skin inflammation, brain function, immune signaling, stress physiology, social experience, and mental health.

5. Conclusions

Psoriasis is strongly associated with psychiatric disorders, particularly depression and anxiety. The reviewed evidence demonstrates elevated rates of depressive symptoms, clinical depression, generalized anxiety, social anxiety, emotional dysregulation, sleep disturbance, stigma, self-stigma, impaired quality of life, and reduced social functioning among patients with psoriasis.

The relationship between psoriasis and psychiatric morbidity is multifactorial. Psychosocial mechanisms such as visible lesions, shame, perceived rejection, concealment, impaired body image, sexual difficulties, occupational burden, and social withdrawal play a major role. At the same time, biological mechanisms involving TNF-alpha, IL-6, IL-17, IL-23, HPA-axis dysregulation, serotonergic changes,

melatonin abnormalities, neuropeptides, BDNF-related pathways, microglial activation, and metabolic inflammation provide a plausible pathophysiological link.

The skin-brain axis offers a comprehensive model for understanding these interactions. It explains how psychological distress may influence inflammatory skin disease and how cutaneous inflammation may affect emotional regulation and brain function. Neuroimaging and psychophysiological studies support the existence of measurable neural and autonomic alterations in psoriasis patients with psychiatric symptoms.

Clinical management should therefore extend beyond lesion clearance. Routine screening for depression, anxiety, distress, stigma, sleep disturbance, substance use, and quality-of-life impairment should be incorporated into psoriasis care. Patients with significant psychiatric symptoms should have access to psychological support, psychiatric treatment when indicated, and multidisciplinary psychodermatology services.

Future research should prioritize longitudinal designs, standardized psychiatric measures, psoriasis-specific distress tools, biomarker discovery, and trials assessing whether anti-inflammatory therapies directly improve psychiatric outcomes. A more integrated understanding of psoriasis as a systemic psychodermatological disorder may improve both dermatological and mental health outcomes.

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