Cureus
Part of SPRINGER NATURE

Open Access Review Article

Published via Dr. Kiran C. Patel College of Osteopathic Medicine (KPCOM)

Skin Meets Brain: The Neuropsychiatric Burden of Inflammatory Skin Diseases

Archana Banda [1], Alena Bashinskaya [2]

1. Osteopathic Medicine, Nova Southeastern University Dr. Kiran C. Patel College of Osteopathic Medicine, Clearwater, USA 2. Dermatology, St. John's Episcopal Dermatology Residency, New York, USA

Corresponding author: Alena Bashinskaya, alenabashin@gmail.com

Received 09/07/2026
Review began 10/21/2026
Review ended 11/01/2026
Published 11/18/2026

© Copyright 2026
Banda et al. This is an open access article distributed under the terms of the Creative Commons Attribution License CC-BY 4.0., which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

DOI: 10.7759/cureus.119872

Abstract

Chronic inflammatory skin diseases, including psoriasis, atopic dermatitis, hidradenitis suppurativa, and systemic lupus erythematosus, are associated with substantial psychological and neuropsychiatric burden, including depression, anxiety, stress, cognitive dysfunction, suicidality, and sleep disturbances. The emerging skin-brain axis highlights bidirectional communication between the skin, immune system, and nervous system. This narrative review explores inflammatory and immune-mediated pathways underlying these interactions and examines their implications for disease burden, quality of life, and long-term outcomes. Understanding the skin-brain axis may support integrated dermatologic and psychological care aimed at interrupting the bidirectional cycles between psychological distress and chronic inflammation and at providing more personalized and systemic treatment approaches. Recent studies have shown the efficacy of a multidisciplinary approach that incorporates cognitive-behavioral treatment and other psychological interventions alongside standard dermatological treatments to reduce the severity of these skin conditions. Specific biologics that significantly decrease depression and anxiety and improve patients' quality of life have also been explored. This therapeutic landscape continues to reveal novel opportunities to target cutaneous inflammation while mitigating psychological symptoms.

Categories: Psychiatry, Psychology, Dermatology
Keywords: hormones, hpa axis, neuroimmunomodulation, psych-dermatological approach, psychological distress, psychoneuroimmunology, psychosocial interventions, skin barrier, skin-brain axis, skin diseases

Introduction And Background

Chronic inflammatory skin conditions such as psoriasis, atopic dermatitis, hidradenitis suppurativa, and lupus tend to be associated with significant psychological burden. We chose these four conditions because they share distinct yet overlapping cytokine pathways, providing a broad framework for understanding the relationship between cutaneous inflammation and psychological well-being. Each disease adds to the skin-brain axis discussion in different ways. Psoriasis causes systemic inflammation, atopic dermatitis causes barrier dysfunction and neuroimmune signaling, and hidradenitis suppurativa causes chronic pain. Although lupus is not traditionally classified as a primary chronic inflammatory dermatosis, it was included to highlight how systemic autoimmune diseases also contribute to the bidirectional skin-brain axis. This literature review adds to the emerging research on this axis by expanding on how chronic inflammatory and autoimmune diseases have distinct inflammatory and clinical characteristics, detailing the biologic mechanisms behind the neuropsychiatric manifestations, and highlighting the need for integrated, multidisciplinary patient care.

Given their multisystem impact, patients often experience various symptoms for prolonged periods, affecting many aspects of their life. Historically, these conditions have been associated with higher rates of depression, anxiety, stress, cognitive dysfunction, suicidality, and sleep disturbances [1]. The skin-brain axis is an evolving framework that integrates psychiatry and dermatology to support more comprehensive, integrated approaches for patients with complex inflammatory conditions [1]. Since the neural crest, brain, and epidermis are all derived from the ectoderm, their interconnectedness is inherent. This narrative review aims to explore the bidirectional skin-brain axis and discuss immune-mediated pathways. Also, the implications of the existing skin-brain axis in these inflammatory skin conditions will be explored to understand how it affects long-term prognosis and to develop integrated dermatologic and psychiatric care to break psychosomatic and somatopsychic vicious cycles.

There is a bidirectional communication between the skin and brain. Skin is no longer conceptualized as a separate entity; rather, it is interconnected with many axes such as the brain and gut shown in Figure *1*.

How to cite this article
Banda A, Bashinskaya A (November 18, 2026) Skin Meets Brain: The Neuropsychiatric Burden of Inflammatory Skin Diseases. Cureus 18(11): e119872. DOI 10.7759/cureus.119872

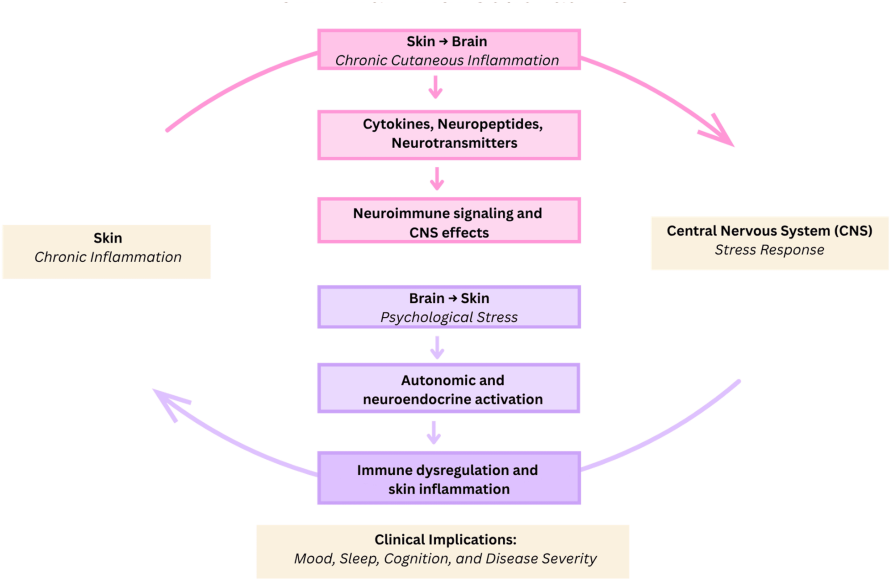


FIGURE 1: Skin-brain bidirectional axis

This axis has therapeutic implications in both directions: treating inflammation may improve psychological symptoms while psychological distress, pain, and itch may improve dermatologic outcomes. These findings illustrate the importance of an integrated biopsychosocial approach to chronic skin disease. Comprehensive dermatologic care, specifically psychological interventions can be a useful adjunct to dermatologic treatment, by improving both skin health and emotional well-being.

Image credit: Created by the authors using Canva (Canva Pty Ltd., Sydney, Australia).

The skin is recognized as a neuroendocrine organ that communicates with the central nervous system [2]. This is due to its ability to secrete hormones, signal cytokines, and modulate neurotransmitters [2]. Notably, there is emerging research at the intersection of neuroscience, dermatology and psychodermatology. Studying these areas allows for a deeper understanding of the skin-brain axis, ultimately improving skin and emotional health [3]. Skin disease can cause psychological distress which can worsen the condition, making it critical to explore the role of adjunctive psychological interventions in the comprehensive management of these diseases [4].

Review

Methods

This narrative review aims to gain a clearer understanding of the skin-brain axis, specifically in psoriasis, atopic dermatitis, hidradenitis suppurativa, and lupus. This was conducted using PubMed and Google Scholar with these search terms and Boolean operators: ("psoriasis" OR "eczema" OR "atopic dermatitis" OR "hidradenitis suppurativa" OR "systemic lupus erythematosus" OR "chronic inflammatory skin disease" OR "chronic inflammatory dermatoses") AND ("skin-brain axis" OR "neuropsychiatric" OR "mental health" OR "psychological stress" OR "anxiety" OR "depression" OR "psychodermatology"). This narrative review includes relevant articles from 1978 to 2026 and only articles written in English.

Therapeutic potential of mental health interventions in skin diseases

As explained previously, the skin-brain axis is bidirectional. There is growing research centered on relaxation and other psychological interventions helping these chronic inflammatory skin disorders. For instance, in a review of 20 randomized controlled trials, the researchers found that psychological interventions, specifically habit reversal training (HRT), were more effective than educational interventions in improving atopic dermatitis severity [5]. This highlights the importance of clinicians prioritizing habit reversal, which directly targets behaviors that worsen the condition, alongside education on triggers and skin care. For instance, HRT can be utilized to offset maladaptive habits in which patients suffer from chronic pruritus leading to scratching and picking, resulting in increased skin inflammation and epidermal barrier damage. This vicious cycle, which generates more psychological stress leading to more scratching, can be effectively terminated with psychological interventions.

Another study that elaborated on the importance of psychotherapy interventions involved four group treatments for atopic dermatitis over a 12-week intervention period: dermatological education program

(DE), autogenic training (AT), cognitive behavioral therapy (BT), and combined education and behavioral therapy group (DEBT). Autogenic training is a relaxation-based psychological intervention, while behavioral therapy is a cognitive-behavioral intervention incorporating relaxation techniques, self-control of scratching, and stress management. This was a randomized controlled trial evaluating the effects of psychological and dermatologic interventions on atopic dermatitis severity. Notably, at the one-year follow-up, patients who had the psychological treatments (AT, BT, DEBT) demonstrated improvements in dermatologist-assessed skin severity, including erythema, excoriations, and dryness, with mean reductions of 13.9 points for AT, 10.3 points for BT, and 9.6 points for combined therapy, compared with a 0.7-point reduction following dermatological education alone [6]. This allowed participants to achieve significant reductions in the topical steroids used in their management. These studies demonstrate the role of addressing psychological and behavioral contributors alongside first-line dermatologic management. The benefits of psychotherapy extend into other chronic inflammatory conditions such as psoriasis.

After patients with psoriasis participated in seven psychotherapy sessions over 12 weeks, there was a significant reduction in total sign score (TSS) and laser Doppler skin blood flow (LDBF) [7]. This study emphasizes the role of stress management, guided imagery, and relaxation in managing psoriasis to improve skin health and quality of life [8]. The effectiveness of cognitive behavioral therapy has also been shown to be valuable alongside standard psoriasis treatment, especially for patients with psychological distress, ultimately leading to better skin health outcomes [9]. Other promising interventions for patients with psoriasis include mindfulness-based therapy, motivational interviewing, and education and interdisciplinary interventions [4,10]. Given the psychiatric comorbidities associated with these skin conditions, patients can experience reduced disease severity, as evidenced by statistically significant differences in the Psoriasis Area and Severity Index (PASI), Dermatology Life Quality Index (DLQI), and Hospital Anxiety and Depression Scale (HADS) when clinicians incorporate relaxation therapy into standard dermatology treatment [11-13]. The use of these objective scores provides dermatologists with a multidimensional view of treatment response. As shown, there is an inherent interconnected nature between the physical and psychosocial burden of psoriasis, which mandates appropriate treatment of both.

Mechanisms of the skin-brain axis

Autonomic Nervous System (ANS) & Systemic Inflammation

The ANS is interconnected with skin conditions. For instance, when patients have sepsis, pathogens activate the immune system, leading to the release of inflammatory cytokines such as IL-1, IL-6, and TNF-a, which activate the ANS. At this point, patients will have decreased blood flow, inflammation, and increased susceptibility to infection [14]. The skin's lack of perfusion becomes evident with the appearance of mottling and pallor. Also, the link between the skin and brain is supported by the inflammatory markers and lactate levels, which correlate with psychiatric symptoms seen in sepsis such as anxiety, sleep disturbances, and poor mood [14].

Neuroimmune signaling & Hypothalamus-Pituitary-Adrenal (HPA) axis

Psychological stress activates the brain, which impacts the development of various skin diseases [15]. From here, the HPA axis and the sympathetic nervous system influence the skin, as evidenced by changes in skin barrier function and inflammation. Stress-induced alterations in the immune system disrupt immune cells and inflammatory mediators in the skin, causing physical and psychological stress and increasing susceptibility to skin diseases [15]. Both types of stress also exacerbate existing skin diseases including psoriasis, atopic dermatitis, acne and urticaria [15]. The immune cells in the skin can lead to the triad of inflammatory cytokines mentioned above, as well as to activation of the peripheral corticotropin-releasing hormone (CRH)-proopiomelanocortin (POMC)-adrenocorticotropic hormone (ACTH)-corticosteroids axis, resulting in acute/chronic secretion of corticosteroids in the skin [16].

Psychoneuroimmunology of Chronic Skin Disease

Skin inflammation often leads to systemic inflammation, ultimately resulting in neuroinflammation. This impacts patients' mood, cognitive function, and sleep quality. This is a bidirectional relationship, meaning that stress & psychological dysfunction lead to autonomic activation, ultimately immune dysregulation and worsening skin inflammation.

Skin cells, specifically keratinocytes and fibroblasts, have receptors that allow them to respond to corticotropin-releasing hormones released by the hypothalamus. This initiates the downstream pathway discussed above, involving POMC, ACTH, and corticosteroid release. However, the skin has a localized HPA axis, allowing it to respond to essential neurotransmitters such as adrenaline, dopamine, acetylcholine, and histamine, as well as neuropeptides such as Substance P and Nerve Growth Factor [16]. The interconnected nature of the nervous system and skin cells is critical to understanding how the skin reacts to stress, itch, pain, and inflammation. For instance, the known cycle between skin inflammation and scratching is connected to the skin-brain axis. In atopic dermatitis, known as the itch that rashes, there are cerebral mechanisms of the itch that are being studied. However, there is limited clinical attention paid to targeting

pruritus with the current non-invasive treatments, including psychological interventions and transcranial direct current stimulation [17]. Understanding how neurosignaling affects inflammatory conditions such as atopic dermatitis enables comprehensive treatment alongside conventional topical or systemic anti-inflammatory medications [18].

Neuropsychological and psychosocial impact of psoriasis

Overview (Cause and Treatment)

Psoriasis is a common chronic immune-mediated inflammatory skin disease affecting 2-3% of the global population and presents as red, scaly patches of varying body surface area [19,20]. Existing literature supports a connection between metabolic comorbidities, specifically obesity and psoriasis [20]. Its multifactorial etiology includes genetic, immune, and environmental factors with systemic complication of psoriatic arthritis, cardiovascular disease, and metabolic syndrome [21]. These complications are associated with systemic inflammation, endothelial dysfunction, and atherosclerotic plaques [22]. Obesity is associated with psoriasis, doubles disease risk and has been shown to impact treatment outcomes. Higher BMI may also increase the risk of liver and renal toxicity from methotrexate and cyclosporine in patients with psoriatic arthritis [23,24]. Management therefore includes weight management, topical corticosteroids, and biologic therapies targeting tumor necrosis factor (TNF), IL-12, IL-17, and IL-23 based on disease severity [25]. A multidisciplinary approach is essential in addressing skin inflammation, comorbidities, lifestyle factors, and quality of life.

Hormonal Influences

Dysregulated sex hormones and psychological stress may contribute to psoriasis [26]. Patients with psoriasis had significantly lower morning cortisol levels than the control group, supporting an impaired stress-response system [23]. Altered cortisol response results in immune dysregulation leading to psoriasis inflammation, while visible lesions, pruritus, and fear of stigma can further reinforce psychological stress. Clinicians should therefore investigate underlying intrinsic and extrinsic triggers for psoriasis.

Stigma and Body Image

Psoriasis is associated with increased anticipated and perceived stigma, including concerns that others may perceive patients as contagious [27]. Social embarrassment and stigma may further worsen disease burden [28]. In a survey of 106 patients, those with severe psoriasis were significantly more likely than those with mild disease to anticipate judgment, avoid social interactions, and feel stigmatized [27]. This demonstrates the need for comprehensive care which addresses the effects of psoriasis on identity, relationships, and belonging.

Cognitive Dysfunction & Sleep Disturbances

Among chronic inflammatory skin diseases, psoriasis has been shown to have the strongest overlap with dementia-related genetics, potentially due to shared loci and transcriptional regulators affecting IL-17/TNF signaling [29]. A meta-analysis, involving 1,274 patients, found that psoriasis was associated with poorer sleep quality, higher insomnia and higher depression scores, and 6.64 times higher odds of sleep disturbance [30].

Potential Immunologic Mechanisms

Psoriasis involves activation of IL-23/IL-17, TNF-a, IL-6, contributing to systemic inflammation, neuroimmune signaling and neuroinflammation [31]. IL-23 promotes T-helper (Th)17-cell activation and IL-17 mediated inflammation, while circulating inflammatory mediators may affect the nervous system. Notably, IL-6 influences neural signaling and has been associated with fatigue, sleep disturbances, depression, and cognitive changes [24].

Neuropsychological and psychosocial impact of atopic dermatitis

Overview (Cause and Treatment)

Atopic dermatitis, also known as atopic eczema, is a multifactorial etiology affecting one in ten people in their lifetime [32]. It is linked to barrier dysfunction, changes in cell-mediated immune responses, IgE-mediated hypersensitivity, and environmental factors [33]. Specific chemicals such as detergents, fragrances, and preservatives can also be triggers. In severe atopic dermatitis, loss-of-function mutations in epidermal barrier components, such as filaggrin, remain a common cause of skin barrier dysfunction [34]. Treatment includes emollients, topical corticosteroids, and topical calcineurin inhibitors, which serve as first-line options. If refractory to these agents, ultraviolet phototherapy is the next-preferred treatment [32].

Psychodermatological Impact of Atopic Dermatitis

Since atopic dermatitis can cause visible redness, scaling, skin thickening known as lichenification, many patients experience the fear of being judged, embarrassment, negative body image, social avoidance, and embarrassment. Given the chronic duration of this condition, many patients may experience exacerbations, specifically psychological stress. This activates the HPA axis, leading to increased stress hormones such as cortisol. Notably, stress further worsens skin conditions, particularly during a flare, due to changes in the epidermal barrier, leading to increased itch, sleep loss, and distress. These stress hormones create numerous changes in the stratum corneum, including decreased epidermal lipids and structural proteins and reduced stratum corneum hydration, leading to increased transepidermal water loss [35]. As depicted, this is a vicious cycle most patients with atopic dermatitis experience, creating more stress and vulnerable skin susceptible to bacterial infections. In addition to increased levels of stress, there is also a positive association with depression, anxiety, and suicidal ideation [36]. This makes it essential for clinicians to routinely ask patients how their skin condition is impacting their stress levels and overall mood.

Sleep Dysfunction Due to Chronic Pruritus

Many patients with atopic dermatitis struggle with intense nocturnal itching and scratching, which leads to sleep fragmentation, resulting in fatigue, irritability, anxiety, and depression [37]. This affects the quality of life of patients and their family members [38]. Sleep dysfunction observed in children and adults. It increases with disease severity, making it critical for clinicians to routinely ask general questions about the impact on lifestyle and sleep quality [39]. The relationship remains bidirectional between psoriasis and depression and anxiety, ultimately decreasing quality of life [40].

Immune & Neuroinflammatory Pathways

In atopic dermatitis, an imbalance between Th2 and Th1 cytokines, alter cell-mediated immune responses and promote IgE-mediated hypersensitivity [33]. Recent studies show that stress impairs skin barrier function, resulting in a shift in immunity toward helper type 2 cells or an allergic response [38]. When there is psychological stress, there is a release of neuromediators in the brain, endocrine organs, and the peripheral nervous system, all of which impact immune and resident cells in the skin [41]. The key Th2 cytokines in atopic dermatitis are IL-4, IL-13, and IL-31, which work together, leading to immune and barrier abnormalities [42].

Neuropsychological and psychosocial impact of hidradenitis suppurativa

Overview (Cause and Treatment)

Hidradenitis suppurativa is a chronic inflammatory skin condition. The key clinical presentation includes painful nodules, abscesses, and fistulae in intertriginous areas such as the armpit region [43]. The etiology is unclear; however, genetic mutations affecting keratinocyte function and the microbiome contribute to the pathogenesis [44]. Existing literature supports smoking, alcohol, obesity, and sleep disorders as being associated with increased inflammatory responses, disease severity, and recurrence rate [45]. To decrease disease burden, clinicians must address these modifiable lifestyle factors and encourage foods that promote the gut microbiome, such as those rich in short-chain fatty acids, omega-3 fatty acids, and vitamin D [45]. The first-line treatment for hidradenitis suppurativa depends on severity: topical antibiotics are used for their anti-inflammatory effects, and biologics, such as adalimumab, are used for moderate-to-severe and refractory cases [46,47].

Hormonal Influences

There is a significant association between hidradenitis suppurativa and polycystic ovarian syndrome (PCOS) [48]. Notably, a cross-sectional analysis of 22,990 patients, showed a 9% prevalence of PCOS among those with hidradenitis suppurativa compared with those without it, a clinically significant difference [49]. This known linkage makes it critical for patients with hidradenitis suppurativa who have signs of hyperandrogenism such as hirsutism, irregular periods, infertility, and virilization to be screened for PCOS [48].

Cognitive Dysfunction Impact & Quality of Life

Among patients with hidradenitis suppurativa, depression was present in 26.5%, and anxiety was present in 18.1%, demonstrating a statistically significant difference between the control group [50]. This leads to higher usage of antidepressants, anxiolytic use, and suicidality [50]. Due to the nonreversible scarring components of hidradenitis suppurativa leading to disfigurement, many patients develop negative body image, which might exacerbate depression and anxiety [46,51]. As shown, this condition has a major impact on patient lives due to the pain and suppuration [43]. These characteristics also impact sleep quality. Systematic review and meta-analysis demonstrate the higher risk of several psychiatric diseases in patients

with hidradenitis suppurativa, making it essential for clinicians who routinely see these patients to focus on comprehensive medical and early emotional support [52].

Immune & Neuroinflammatory Pathways

In hidradenitis suppurativa, the major inflammatory pathways include TNF-a, IL-1, IL-17, and IL-23 [53]. Among the listed cytokines, IL-17 has been shown to make the most significant contribution to the chronic inflammatory cascade [54]. This is due to IL-1β which activates IL-6, resulting in upregulation of T17 cells and IL-17 A/F, ultimately in chronic inflammation [55]. Also, IL-6 is correlated with disease severity and quality-of-life impairment, whereas IL-17A is associated with a longer duration of hidradenitis suppurativa [56]. Elevated inflammatory cytokines and high-sensitivity C-reactive protein in serum support the systemic inflammatory nature of hidradenitis suppurativa [57].

Neuropsychological and psychosocial impact of systemic lupus erythematosus

Overview (Cause and Treatment)

Systemic lupus erythematosus is a multifactorial and multisystemic autoimmune disorder. The etiology is also complex, including genetic susceptibility, hormonal, epigenetic, and environmental triggers [58]. Importantly, patients with systemic lupus erythematosus develop cardiovascular disease due to the promotion of atherosclerosis from chronic inflammation and immune system activation and antiphospholipid antibodies, which increase blood clots [59]. The existing treatment options include cytotoxic and immunosuppressive drugs, antimalarials such as hydroxychloroquine, glucocorticoids, and non-steroidal anti-inflammatory drugs (NSAIDs), which aim to reduce disease-related effects [60].

Cognitive Dysfunction & Quality of Life

Systemic lupus erythematosus has a major cognitive impact, specifically lower quality of life due to depression, cognitive dysfunction, and increased disease activity, further supporting the need for clinicians to evaluate cognitive function and rehabilitation when indicated [61,62]. This disease commonly leads to psychosis or mood disorders, impaired attention, working memory, and processing speed [62]. Also, meta-analyses have shown a moderate relationship between depression and poor sleep quality in patients with systemic lupus erythematosus, further worsening daytime dysfunction [63]. Polysomnography showed that patients with systemic lupus erythematosus experience increased stage 1 sleep, periodic limb movement disorder, and reduced stages 2 and 4 slow-wave sleep [64]. As with atopic dermatitis and psoriasis, clinicians must inquire about how this condition affects sleep quality and daily responsibilities.

Autoantibodies and Neuroinflammation

Blood-brain barrier (BBB) disruption allows immune factors to access the central nervous system, leading to neuroinflammation, characteristic of neuropsychiatric systemic lupus erythematosus [65]. This is supported by the presence of anti-N-methyl-D-aspartate (NMDA) antibodies, which facilitate BBB disruption, resulting in neuronal damage and memory deficits [66]. Also, IL-6 and complement C3 were present in the cerebrospinal fluid (CSF) of patients with neuropsychiatric systemic lupus erythematosus during acute confusion [67].

Shared inflammatory mechanisms & shared neuropsychiatric manifestations

Psoriasis, atopic dermatitis, hidradenitis suppurativa, and lupus share similarities, specifically TNF-a and IL-6. These chronic inflammatory skin conditions share similar manifestations of depression, anxiety, cognitive dysfunction, and sleep disturbances. Despite these shared manifestations, each disease has specific mechanisms. IL-17 & IL-23 remain critical in psoriasis. TH2, IL-4, IL-13, and IL-31 are central to atopic dermatitis. Also, in hidradenitis suppurativa, systemic inflammation is central whereas autoantibody-mediated CNS predominates in lupus. Also, chronic inflammatory skin diseases such as psoriasis, rosacea, atopic dermatitis, and bullous pemphigoid may be biologically connected to neurodegenerative diseases such as Alzheimer's and Parkinson's [29]. This is due to the shared genetic and immune pathways as well as underlying inflammation present in the skin, which is connected to cognitive health and cognition changes, as illustrated in Figure *2* [29].

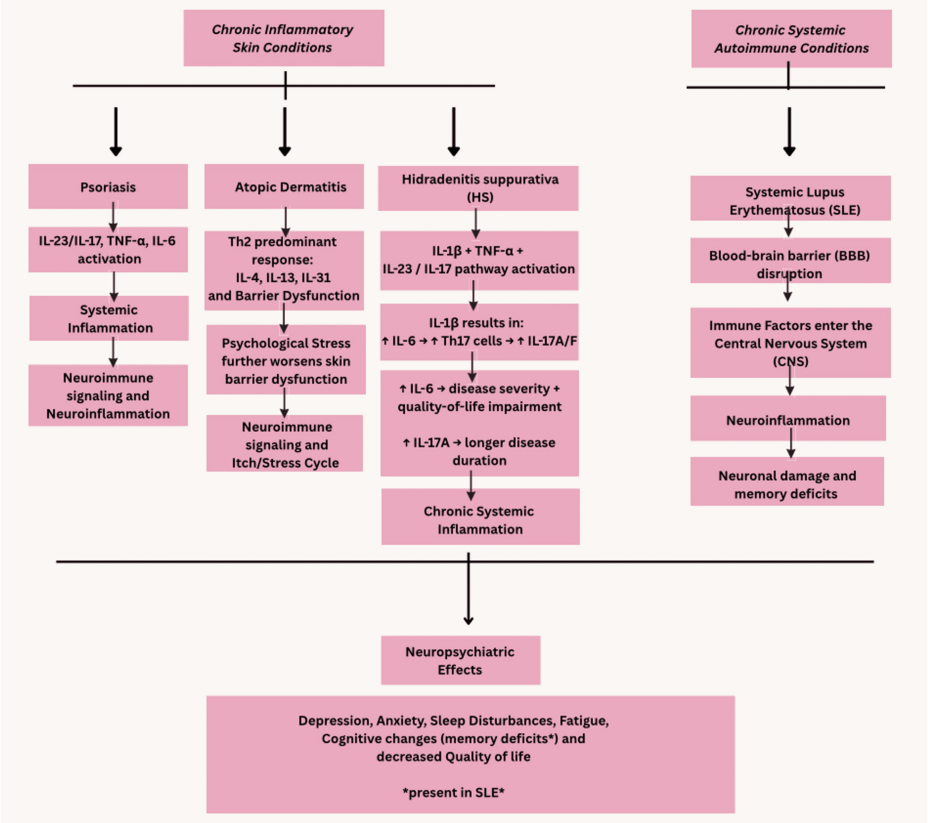


FIGURE 2: Flowchart of the cytokine/neuroimmune pathway

This diagram above illustrates the unique mechanisms of chronic inflammatory skin conditions (psoriasis, atopic dermatitis, hidradenitis suppurativa) and chronic systemic autoimmune conditions such as systemic lupus erythematosus. It also emphasizes the shared neuropsychiatric effects present across these conditions, emphasizing the intersection of psychiatry and dermatology.

Image credit: Created by the authors using Canva (Canva Pty Ltd., Sydney, Australia).

Sleep-inflammation-mental health feedback loop

Inflammatory skin diseases lead to elevated cytokine levels, pruritus, and pain. These result in sleep disruptions leading to depression, anxiety, and cognitive dysfunction. Ultimately, this leads to HPA-axis/autonomic dysregulation and increased inflammation, which exacerbates skin disease. Inflammation in the skin is also linked to neuroinflammation, contributing to cognitive decline, which is notably evident in the aging population. This necessitates a greater focus on improving the health and function of the skin barrier to potentially alleviate age-related cognitive decline [68]. Figure 3 illustrates the feedback loop between sleep, inflammation, and mental health.

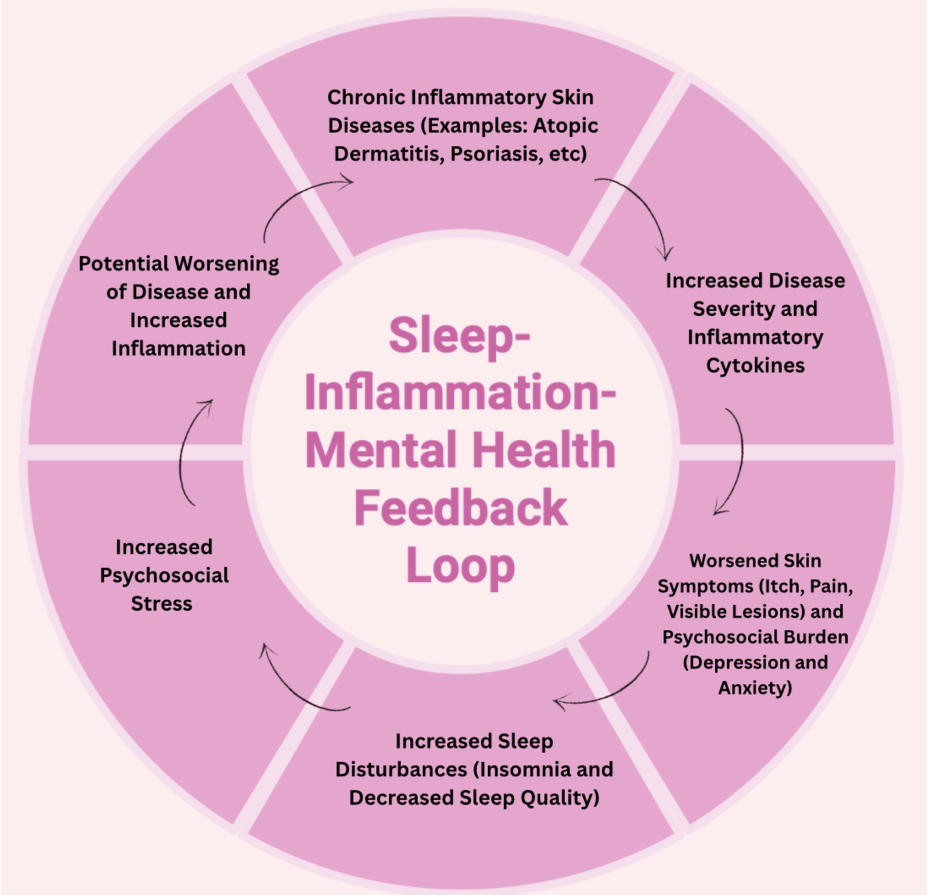


FIGURE 3: Sleep-inflammation-mental health feedback loop

This diagram illustrates the vicious cycle of chronic skin inflammatory diseases resulting in increased itch and pain, which causes sleep disturbances, depression, anxiety, psychosocial stress and inflammation. Thus, it is critical to understand how biologics can be used to decrease disease severity while also decreasing psychosocial burden.

Image credit: Created by the authors using Canva (Canva Pty Ltd., Sydney, Australia).

Skin-directed therapies as psychotropic interventions

In addition to treating these chronic inflammatory skin conditions, TNF, IL-17, and IL-23 inhibitors can improve depressive symptoms. This is especially significant for patients with psoriasis on biologics, because it can reduce disease severity and psychological comorbidity. There is a bidirectional relationship between disease severity, since increased severity necessitates biologics and is associated with worsened depression and anxiety symptoms [28, 69].

Impact of Biologics

In a study of 106 patients with psoriasis and an equal number of controls, after six months of biologic treatment, patients had significantly less severe psoriasis, better quality of life, and decreased levels of depression and anxiety [69]. Also, when psoriasis severity improved, depression and anxiety improved, while improvements in overall quality of life were not impacted in this study. These variables were measured using the PASI, which indicates the severity and extent of psoriasis, and the DLQI, which indicates how the skin disease is affecting someone's daily life. Specific biologics that have been effective in reducing anxiety, depression, and quality of life in patients with psoriasis are ustekinumab, secukinumab, and brodalumab [70-72]. Notably, after 48 weeks of treatment with secukinumab, patients had significantly improved skin clearance alongside improvements in anxiety and depression [71]. In a small study of 20 patients taking brodalumab after suboptimal response to TNF-a inhibitors, this biologic demonstrated positive effects on depression and anxiety, independent of the reduction of psoriatic lesions [72]. These demonstrate the possibility of targeting inflammatory cytokine pathways to address the significant psychological burden while decreasing psoriasis severity.

Impact of Selective Serotonin Reuptake Inhibitor (SSRIs) & Selective Norepinephrine Reuptake Inhibitor (SNRIs)

In hidradenitis suppurativa, many patients experience chronic pain and comorbid depression, making medications such as duloxetine targeting both symptoms essential in management [73]. Along with SNRIs, clinicians should reassure patients that these conditions are not due to poor hygiene and provide support options such as counseling. Clinicians routinely addressing how hidradenitis suppurativa impacts social interactions, work productivity, sexual dysfunction, and mood is critical. Notably, SSRIs such as escitalopram and dupilumab can be used in psychogenic pruritus to target the brain-skin/itch pathway [74]. Further research is needed to establish the efficacy of this treatment strategy; however, it offers a promising solution to other skin conditions with nocturnal pruritus, such as atopic dermatitis. Given the connection between the skin and psyche, it remains pertinent for clinicians to offer patients with psychopruritic disorders, such as excoriation disorder and prurigo nodularis, psychotherapy to enhance therapy efficacy [75].

Discussion

Integrated Dermatologic and Mental Health Care: Screening for Depression and Anxiety

As previously discussed, many chronic skin diseases contribute to psychological distress. This highlights the importance of routinely screening patients for depression and anxiety. Psychological distress can negatively impact medication adherence and long-term motivation for treatment, while also potentially worsening existing skin inflammation. Integrating mental health assessments into dermatologic care allows clinicians to address the dynamic skin-brain relationship and provide more comprehensive care. These assessments can improve quality of life by identifying patients who may benefit from additional psychological support. Given the chronicity of these conditions and the frequent interactions patients have with their dermatologists, clinicians need to ask how their patients' skin conditions are affecting mood and daily life.

Assessment of Sleep

Chronic pruritus, common in chronic inflammatory conditions such as atopic dermatitis and psoriasis, is associated with impaired sleep and decreased quality of life. This is significant, with approximately 1% of doctor visits due to patients concerned with chronic pruritus [76].However, before clinicians treat inflammatory chronic pruritus with topical steroids, it is critical to rule out systemic diseases that cause secondary itch. Therefore, the following tests should be considered: complete blood cell count, complete metabolic panel, and thyroid function testing to evaluate for hematologic malignancy, liver disease, kidney disease, or thyroid disease [76]. Once clinicians rule out systemic diseases, pruritus should be appropriately treated. Doing so is crucial because lack of sleep can impair neurocognitive functions, notably focus, sleep efficiency, and intelligence quotient (IQ) [77]. Consequently, dermatologists routinely ask patients with inflammatory skin conditions about their sleep quality, mood, and daily life, which is crucial.

Assessment of Pain and Functional Impairment

Skin pain occurs in many chronic inflammatory skin conditions discussed. This symptom is impactful because it increases patient discomfort, increases disease burden, and reduces quality of life [78]. Despite the current therapies for atopic dermatitis, such as topical calcineurin inhibitors and biologics addressing itch symptoms, skin pain remains a central symptom experienced by more than 40% of patients [79]. Existing literature supports differences between itch and pain signaling; however, neurons that respond to itch also respond to pain [79]. Notably, dupilumab, a biologic targeting IL-4 and IL-13, reduces disease severity, especially when used with a topical corticosteroid. However, there have been adverse effects such as headache, injection site reaction, conjunctivitis, nasopharyngitis, and herpes viral infections [80,81]. This necessitates increased attention to understand how skin pain and chronic itch both can be adequately addressed while being aware of the potential adverse effects of medications.

In addition to atopic dermatitis, skin pain is also prevalent in psoriasis, impacting various aspects of patient lives. Specifically, this symptom reduces level of physical activity, disrupts sleep, and decreases productivity, ultimately creating emotional distress in their lives [82]. Unfortunately, the pain experienced by patients with psoriasis often leads to maladaptive coping strategies and avoidance of painful activities, which isolates them further socially [82]. To alleviate skin pain in psoriasis, a novel aerosol foam formulation of calcipotriol plus betamethasone dipropionate improves skin lesions and relieves the cutaneous pain commonly experienced by patients [83]. In addition to atopic dermatitis and psoriasis, patients with hidradenitis supporativa also report pain as a significant symptom impacting their quality of life [84]. It is considered that there are hidradenitis supporativa therapies such as adalimumab that decrease the associated pain; however, it has been found to provide insufficient relief for many patients. Therefore, there remains an increased need for therapeutic solutions for skin pain and additional hidradenitis supporativa symptoms. Pain is also a predominant symptom in other chronic diseases such as systemic lupus erythematosus. It has been reported to interfere with patients' quality of life, resulting in decreased strength and struggles with daily living [85]. Notably, it has been found that patients with mild to moderate disease activity also have severe pain. As shown across these inflammatory conditions, identifying effective management strategies is critical in addressing the skin-brain axis to ensure patients' psychological and dermatological health is adequately supported.

Challenges Present in the Skin-Brain Bidirectional Relationship

Given the previous elaborated relationship between psychological stress aggravating skin inflammation which creates further psychological stress, it is pertinent to discuss the challenges of this bidirectional relationship. The primary challenge is understanding the primary event that led to increased skin disease inflammation especially since stress can precede a flare while worsened skin disease can also cause stress. This can make it difficult to establish a cause and effect. As explained previously, many of these conditions have a vicious itch-scratch cycle which can have a significant impact on patients' sleep quality. However, it can be difficult to elucidate whether itching/scratching or the effects of consecutive nights of poor sleep are damaging one's skin barrier.

Another challenge are the various inflammatory pathways present across the skin conditions such as atopic dermatitis and psoriasis. These differences translate to potentially needing different stress-management and psychological interventions to address every skin disease. Also, psychological stress is subjective to the patient, ever-changing, and difficult to measure, which makes it difficult for clinicians to correlate it with skin findings of visible inflammation. Lastly, it is important to account for confounding factors such as environmental exposures, genetics, and access to treatments which can influence stress levels and skin disease. Therefore, it can make this bidirectional relationship between the skin and brain difficult to predict.

Limitations

Currently, there remains a lack of standardized definitions of cognitive dysfunctions, making it difficult to standardize them across studies. Also, it is challenging to establish causality and temporal relationships between chronic skin conditions and mental health concerns at times. There is a continued need for prospective studies examining if controlling inflammation and specific biologics improves psychiatric outcomes. Exploring these concepts is critical given the psychological stress experienced by patients with chronic inflammatory dermatoses.

Conclusions

Overall, recognizing the role of the skin-brain axis may encourage more clinicians to conduct comprehensive screening and integrated management of patients with inflammatory skin conditions. There is an intricate balance between stress and skin diseases. Clinicians must understand the interconnected nature of psychology and dermatology to ensure that the underlying causes of skin disease and their exacerbations are appropriately and comprehensively addressed, rather than solely managing the symptoms. Collaboration between dermatologists, psychologists, and psychiatrists helps mitigate the vicious cycle between psychological distress and triggers of these skin conditions. The continuous communication among the brain, nerves, skin cells, and immune system underscores the skin's role as a dynamic organ and influences the diagnosis and treatment of numerous inflammatory dermatological conditions.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Archana Banda, Alena Bashinskaya

Acquisition, analysis, or interpretation of data: Archana Banda, Alena Bashinskaya

Drafting of the manuscript: Archana Banda, Alena Bashinskaya

Critical review of the manuscript for important intellectual content: Archana Banda, Alena Bashinskaya

Supervision: Alena Bashinskaya

Disclosures

Conflicts of interest: In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

References

1. Ramirez-Posada M, Guillen-Burgos HF: Skin-brain axis: biological foundations and clinical implications . J Acad Consult Liaison Psychiatry. 2026, 67:451-5. 10.1016/j.jaclp.2026.04.006
2. Yoon KN, Chung JH: Healthy skin, healthy brain. J Dermatol Sci. 2025, 119:47-52. 10.1016/j.jdermsci.2025.06.001
3. Haykal D, Berardesca E, Kabashima K, Dréno B: Beyond beauty: neurocosmetics, the skin-brain axis, and the future of emotionally intelligent skincare. Clin Dermatol. 2025, 43:523-7. 10.1016/j.clindermatol.2025.05.002
4. Rafidi B, Kondapi K, Beestrum M, Basra S, Lio P: Psychological therapies and mind-body techniques in the management of dermatologic diseases: a systematic review. Am J Clin Dermatol. 2022, 23:755-73. 10.1007/s40257-022-00714-y
5. Hua T, Kim LS, Yousaf M, et al.: Psychological interventions are more effective than educational interventions at improving atopic dermatitis severity: a systematic review. Dermatitis. 2023, 34:301-7. 10.1097/DER.0000000000000868
6. Ehlers A, Stangier U, Gieler U: Treatment of atopic dermatitis: a comparison of psychological and dermatological approaches to relapse prevention. J Consult Clin Psychol. 1995, 63:624-35. 10.1037//0022-006x.63.4.624
7. Zachariae R, Oster H, Bjerring P, Kragballe K: Effects of psychologic intervention on psoriasis: a preliminary report. J Am Acad Dermatol. 1996, 34:1008-15. 10.1016/s0190-9622(96)90280-7
8. Neerackal RJ, Abdul Latheef EN, Sukumarakurup S, Jafferany M: Relaxation therapy in the management of psoriasis. Dermatol Ther. 2020, 33:e14030. 10.1111/dth.14030
9. Sijercic I, Ennis N, Monson CM: A systematic review of cognitive and behavioral treatments for individuals with psoriasis. J Dermatolog Treat. 2020, 31:631-8. 10.1080/09546634.2019.1690625
10. Qureshi AA, Awosika O, Baruffi F, Rengifo-Pardo M, Ehrlich A: Psychological therapies in management of psoriatic skin disease: a systematic review. Am J Clin Dermatol. 2019, 20:607-24. 10.1007/s40257-019-00437-7
11. Fredriksson T, Pettersson U: Severe psoriasis-oral therapy with a new retinoid . Dermatologica. 1978, 157:238-44. 10.1159/000250839
12. Finlay AY, Khan GK: Dermatology Life Quality Index (DLQI)-a simple practical measure for routine clinical use. Clin Exp Dermatol. 1994, 19:210-6. 10.1111/j.1365-2230.1994.tb01167.x
13. Zigmond AS, Snaith RP: The hospital anxiety and depression scale . Acta Psychiatr Scand. 1983, 67:361-70. 10.1111/j.1600-0447.1983.tb09716.x
14. Nagamine T: Gut-skin-brain axis in people suffering from sepsis with acute skin failure . World J Psychiatry. 2025, 15:106062. 10.5498/wjp.v15.i7.106062
15. Zhang H, Wang M, Zhao X, Wang Y, Chen X, Su J: Role of stress in skin diseases: a neuroendocrine-immune interaction view. Brain Behav Immun. 2024, 116:286-302. 10.1016/j.bbi.2023.12.005
16. Pondeljak N, Lugović-Mihić L: Stress-induced interaction of skin immune cells, hormones, and neurotransmitters. Clin Ther. 2020, 42:757-70. 10.1016/j.clinthera.2020.03.008
17. Mochizuki H, Schut C, Nattkemper LA, Yosipovitch G: Brain mechanism of itch in atopic dermatitis and its possible alteration through non-invasive treatments. Allergol Int. 2017, 66:14-21. 10.1016/j.alit.2016.08.013
18. Yarbrough KB, Neuhaus KJ, Simpson EL: The effects of treatment on itch in atopic dermatitis . Dermatol Ther. 2013, 26:110-9. 10.1111/dth.12032
19. Kuang Y, Luo Y, Yi X, et al.: Prevalence and risk factors for cognitive impairment in patients with psoriasis . J Eur Acad Dermatol Venereol. 2022, 36:e152-5. 10.1111/jdv.17707
20. Mahé E: Childhood psoriasis. Eur J Dermatol. 2016, 26:537-48. 10.1684/ejd.2016.2932
21. Jensen P, Skov L: Psoriasis and obesity. Dermatology. 2016, 232:633-9. 10.1159/000455840
22. Grozdev I, Korman N, Tsankov N: Psoriasis as a systemic disease . Clin Dermatol. 2014, 32:343-50. 10.1016/j.clindermatol.2013.11.001
23. Gisondi P, Geat D, Bellinato F, et al.: Psychological stress and salivary cortisol levels in patients with plaque psoriasis. J Pers Med. 2021, 11:1069. 10.3390/jpm11111069
24. Mrowietz U, Sümbül M, Gerdes S: Depression, a major comorbidity of psoriatic disease, is caused by metabolic inflammation. J Eur Acad Dermatol Venereol. 2023, 37:1731-8. 10.1111/jdv.19192
25. Ighani A, Partridge AC, Shear NH, Lynde C, Gulliver WP, Sibbald C, Fleming P: Comparison of management guidelines for moderate-to-severe plaque psoriasis: a review of phototherapy, systemic therapies, and biologic agents. J Cutan Med Surg. 2019, 23:204-21. 10.1177/1203475418814234
26. Liu S, He M, Jiang J, et al.: Triggers for the onset and recurrence of psoriasis: a review and update . Cell Commun Signal. 2024, 22:108. 10.1186/s12964-023-01381-0
27. Wan MT, Pearl RL, Chiesa Fuxench ZC, Takeshita J, Gelfand JM: Anticipated and perceived stigma among patients with psoriasis. J Psoriasis Psoriatic Arthritis. 2020, 5:93-9. 10.1177/2475530320924009
28. Krstanović K, Kroflin K, Kroflin L, Kušević Z: Psoriasis and mental health: a psychodermatological approach with an emphasis on psychotherapy. Psychiatr Danub. 2024, 36:7-16. 10.24869/psyd.2024.7
29. Grech VS, Lotsaris K, Kefala V, Rallis E: From skin to brain: key genetic mediators associating cutaneous inflammation and neurodegenerative diseases. Genes (Basel). 2025, 16:1463. 10.3390/genes16121463
30. Guo M, Su J, Zheng S, Chen B: Sleep in psoriasis: a meta-analysis . J Psychosom Res. 2024, 176:111543. 10.1016/j.jpsychores.2023.111543
31. Tang J, Zhao S, Shi H, Li X, Ran L, Cao J, He Y: Effects on peripheral and central nervous system of key inflammatory intercellular signalling peptides and proteins in psoriasis. Exp Dermatol. 2024, 33:e15104. 10.1111/exd.15104
32. Frazier W, Bhardwaj N: Atopic dermatitis: diagnosis and treatment. Am Fam Physician. 2020, 101:590-8.
33. David Boothe W, Tarbox JA, Tarbox MB: Atopic dermatitis: pathophysiology. Adv Exp Med Biol. 2017, 1027:21-37. 10.1007/978-3-319-64804-0_3
34. Fujii M: Current understanding of pathophysiological mechanisms of atopic dermatitis: interactions among skin barrier dysfunction, immune abnormalities and pruritus. Biol Pharm Bull. 2020, 43:12-9. 10.1248/bpb.b19-00088
35. Maarouf M, Maarouf CL, Yosipovitch G, Shi VY: The impact of stress on epidermal barrier function: an

evidence-based review. Br J Dermatol. 2019, 181:1129-37. 10.1111/bjd.17605
36. Almutawa YM, AlGhareeb M, Bhattarai E, Aljalahma J: Investigation of the impact of atopic dermatitis (AD) on stress, depression, anxiety, and suicidal ideation: a systematic review and meta-analysis. Cureus. 2024, 16:e63376. 10.7759/cureus.63376
37. Chang YS, Chou YT, Lee JH, et al.: Atopic dermatitis, melatonin, and sleep disturbance. Pediatrics. 2014, 134:e397-405. 10.1542/peds.2014-0376
38. Arndt J, Smith N, Tausk F: Stress and atopic dermatitis. Curr Allergy Asthma Rep. 2008, 8:312-7. 10.1007/s11882-008-0050-6
39. Jeon C, Yan D, Nakamura M, Sekhon S, Bhutani T, Berger T, Liao W: Frequency and management of sleep disturbance in adults with atopic dermatitis: a systematic review. Dermatol Ther (Heidelb). 2017, 7:349-64. 10.1007/s13555-017-0192-3
40. Hawro T, Hawro M, Zalewska-Janowska A, Weller K, Metz M, Maurer M: Pruritus and sleep disturbances in patients with psoriasis. Arch Dermatol Res. 2020, 312:103-11. 10.1007/s00403-019-01998-7
41. Suárez AL, Feramisco JD, Koo J, Steinhoff M: Psychoneuroimmunology of psychological stress and atopic dermatitis: pathophysiologic and therapeutic updates. Acta Derm Venereol. 2012, 92:7-15. 10.2340/00015555-1188
42. Dubin C, Del Duca E, Guttman-Yassky E: The IL-4, IL-13 and IL-31 pathways in atopic dermatitis . Expert Rev Clin Immunol. 2021, 17:835-52. 10.1080/1744666X.2021.1940962
43. McCarthy S: Hidradenitis suppurativa. Annu Rev Med. 2025, 76:69-80. 10.1146/annurev-med-051223-031234
44. Villani A: Hidradenitis suppurativa: pathogenesis . Eur J Dermatol. 2025, 35:4-7. 10.1684/ejd.2025.4860
45. Saito-Sasaki N, Sawada Y: Hidradenitis suppurativa and daily lifestyle . Front Immunol. 2025, 16:1627638. 10.3389/fimmu.2025.1627638
46. Tzellos T: Conventional treatment of hidradenitis suppurativa: serving the "window of opportunity" concept. Clin Dermatol. 2025, 43:485-9. 10.1016/j.clindermatol.2025.05.015
47. Alikhan A, Sayed C, Alavi A, et al.: North American clinical management guidelines for hidradenitis suppurativa: a publication from the United States and Canadian Hidradenitis Suppurativa Foundations: part II: topical, intralesional, and systemic medical management. J Am Acad Dermatol. 2019, 81:91-101. 10.1016/j.jaad.2019.02.068
48. Phan K, Charlton O, Smith SD: Hidradenitis suppurativa and polycystic ovarian syndrome: systematic review and meta-analysis. Australas J Dermatol. 2020, 61:e28-33. 10.1111/ajd.13110
49. Garg A, Neuren E, Strunk A: Hidradenitis suppurativa is associated with polycystic ovary syndrome: a population-based analysis in the United States. J Invest Dermatol. 2018, 138:1288-92. 10.1016/j.jid.2018.01.009
50. Patel KR, Lee HH, Rastogi S, et al.: Association between hidradenitis suppurativa, depression, anxiety, and suicidality: a systematic review and meta-analysis. J Am Acad Dermatol. 2020, 83:737-44. 10.1016/j.jaad.2019.11.068
51. Schneider-Burrus S, Jost A, Peters EM, Witte-Haendel E, Sterry W, Sabat R: Association of hidradenitis suppurativa with body image. JAMA Dermatol. 2018, 154:447-51. 10.1001/jamadermatol.2017.6058
52. Folkmann CL, Pinborg EH, Iversen M, et al.: The association between hidradenitis suppurativa and psychiatric disease: a systematic review and meta-analysis. Br J Dermatol. 2025, 193:212-20. 10.1093/bjd/ljaf151
53. Del Duca E, Morelli P, Bennardo L, Di Raimondo C, Nisticò SP: Cytokine pathways and investigational target therapies in hidradenitis suppurativa. Int J Mol Sci. 2020, 21:8436. 10.3390/ijms21228436
54. Frew JW, Hawkes JE, Krueger JG: A systematic review and critical evaluation of inflammatory cytokine associations in hidradenitis suppurativa. F1000Res. 2018, 7:1930. 10.12688/f1000research.17267.1
55. Kim J, Lee J, Li X, et al.: Single-cell transcriptomics suggest distinct upstream drivers of IL-17A/F in hidradenitis versus psoriasis. J Allergy Clin Immunol. 2023, 152:656-66. 10.1016/j.jaci.2023.05.012
56. Francke LS, Al-Bayatti A, Zhang H, Alimohammadi M, Törmä H, Sonkoly E: Proteomic analysis in hidradenitis suppurativa reveals systemic inflammation in all disease stages. J Eur Acad Dermatol Venereol. 2026, 1-10. 10.1111/jdv.70546
57. Saraç Öztürk G, Ergun T, Peker Eyüboğlu İ, Akkiprik M: Serum high-sensitivity C-reactive protein, tumor necrosis factor-α, interleukin (IL)-1β, IL-17A and IL-23 levels in patients with hidradenitis suppurativa. Cytokine. 2021, 144:155585. 10.1016/j.cyto.2021.155585
58. Dai X, Fan Y, Zhao X: Systemic lupus erythematosus: updated insights on the pathogenesis, diagnosis, prevention and therapeutics. Signal Transduct Target Ther. 2025, 10:102. 10.1038/s41392-025-02168-0
59. Frostegård J: Systemic lupus erythematosus and cardiovascular disease . J Intern Med. 2023, 293:48-62. 10.1111/joim.13557
60. Su X, Yu H, Lei Q, et al.: Systemic lupus erythematosus: pathogenesis and targeted therapy . Mol Biomed. 2024, 5:54. 10.1186/s43556-024-00217-8
61. Calderón J, Flores P, Aguirre JM, et al.: Impact of cognitive impairment, depression, disease activity, and disease damage on quality of life in women with systemic lupus erythematosus. Scand J Rheumatol. 2017, 46:273-80. 10.1080/03009742.2016.1206617
62. Seet D, Allameen NA, Tay SH, Cho J, Mak A: Cognitive dysfunction in systemic lupus erythematosus: immunopathology, clinical manifestations, neuroimaging and management. Rheumatol Ther. 2021, 8:651-79. 10.1007/s40744-021-00312-0
63. Yin R, Li L, Xu L, Sui W, Niu M, Xu R, Srirat C: Association between depression and sleep quality in patients with systemic lupus erythematosus: a systematic review and meta-analysis. Sleep Breath. 2022, 26:429-41. 10.1007/s11325-021-02405-0
64. Iaboni A, Ibanez D, Gladman DD, Urowitz MB, Moldofsky H: Fatigue in systemic lupus erythematosus: contributions of disordered sleep, sleepiness, and depression. J Rheumatol. 2006, 33:2453-57.
65. Duarte-Delgado NP, Vásquez G, Ortiz-Reyes BL: Blood-brain barrier disruption and neuroinflammation as pathophysiological mechanisms of the diffuse manifestations of neuropsychiatric systemic lupus erythematosus. Autoimmun Rev. 2019, 18:426-32. 10.1016/j.autrev.2018.12.004

66. Kowal C, Degiorgio LA, Lee JY, Edgar MA, Huerta PT, Volpe BT, Diamond B: Human lupus autoantibodies against NMDA receptors mediate cognitive impairment. Proc Natl Acad Sci U S A. 2006, 103:19854-9. 10.1073/pnas.0608397104
67. Asano T, Ito H, Kariya Y, et al.: Evaluation of blood-brain barrier function by quotient alpha2 macroglobulin and its relationship with interleukin-6 and complement component 3 levels in neuropsychiatric systemic lupus erythematosus. PLoS One. 2017, 12:e0186414. 10.1371/journal.pone.0186414
68. Wen S, Elias PM, Wakefield JS, Mauro TM, Man MQ: The link between cutaneous inflammation and cognitive impairment. J Eur Acad Dermatol Venereol. 2022, 36:1705-12. 10.1111/jdv.18360
69. Timis TL, Beni L, Mocan T, Florian IA, Orasan RI: Biologic therapies decrease disease severity and improve depression and anxiety symptoms in psoriasis patients. Life (Basel). 2023, 13:1219. 10.3390/life13051219
70. Langley RG, Feldman SR, Han C, et al.: Ustekinumab significantly improves symptoms of anxiety, depression, and skin-related quality of life in patients with moderate-to-severe psoriasis: results from a randomized, double-blind, placebo-controlled phase III trial. J Am Acad Dermatol. 2010, 63:457-65. 10.1016/j.jaad.2009.09.014
71. Talamonti M, Malara G, Natalini Y, et al.: Secukinumab improves patient perception of anxiety and depression in patients with moderate to severe psoriasis: a post hoc analysis of the SUPREME study. Acta Derm Venereol. 2021, 101:adv00422. 10.2340/00015555-3712
72. Andersch-Björkman Y, Micu E, Seifert O, Lonne-Rahm SB, Gillstedt M, Osmancevic A: Effects of brodalumab on psoriasis and depressive symptoms in patients with insufficient response to TNF-α inhibitors. J Dermatol. 2023, 50:1401-14. 10.1111/1346-8138.16917
73. Ballard K, Sathe NC, Shuman VL: Hidradenitis suppurativa. StatPearls [Internet]. StatPearls Publishing, Treasure Island (FL); 2024.
74. Ghani H, Ghofrani S, Modanlo N, Cheedalla N, Jafferany M: Novel and existing therapeutic interventions in psychogenic pruritus. Clin Exp Dermatol. 2026, 51:951-7. 10.1093/ced/llag005
75. Lee HG, Stull C, Yosipovitch G: Psychiatric disorders and pruritus. Clin Dermatol. 2017, 35:273-80. 10.1016/j.clindermatol.2017.01.008
76. Butler DC, Berger T, Elmariah S, et al.: Chronic pruritus: a review. JAMA. 2024, 331:2114-24. 10.1001/jama.2024.4899
77. Khosravi A, Glińska J, Barańska-Rybak W: Sleep efficiency and neurocognitive decline in atopic dermatitis: a systematic review. Acta Derm Venereol. 2024, 104:adv40459. 10.2340/actadv.v104.40459
78. Ständer S, Simpson EL, Guttman-Yassky E, et al.: Clinical relevance of skin pain in atopic dermatitis . J Drugs Dermatol. 2020, 19:921-6. 10.36849/JDD.2020.5498
79. Yosipovitch G, Kim B, Luger T, et al.: Similarities and differences in peripheral itch and pain pathways in atopic dermatitis. J Allergy Clin Immunol. 2024, 153:904-12. 10.1016/j.jaci.2023.10.034
80. Narla S, Silverberg JI, Simpson EL: Management of inadequate response and adverse effects to dupilumab in atopic dermatitis. J Am Acad Dermatol. 2022, 86:628-36. 10.1016/j.jaad.2021.06.017
81. Tameez Ud Din A, Malik I, Arshad D, Tameez Ud Din A: Dupilumab for atopic dermatitis: the silver bullet we have been searching for?. Cureus. 2020, 12:e7565. 10.7759/cureus.7565
82. Ljosaa TM, Bondevik H, Halvorsen JA, Carr E, Wahl AK: The complex experience of psoriasis related skin pain: a qualitative study. Scand J Pain. 2020, 20:491-8. 10.1515/sjpain-2019-0158
83. Gallo L, Megna M, Cirillo T, et al.: Psoriasis and skin pain: real-life effectiveness of calcipotriol plus betamethasone dipropionate in aerosol foam formulation. J Eur Acad Dermatol Venereol. 2019, 33:1312-5. 10.1111/jdv.15488
84. Surapaneni V, Milosavljevic MV, Orenstein LA: Pain management in hidradenitis suppurativa . J Am Acad Dermatol. 2024, 91:S52-63. 10.1016/j.jaad.2024.09.006
85. Nichilatti LP, Fernandes JM, Marques CP: Physiopathology of pain in systemic erythematosus lupus . Lupus. 2020, 29:721-6. 10.1177/0961203320919872