KACALAK, Mateusz. Interactions Between Psychiatric Disorders and Inflammatory Skin Diseases - systematic review. Quality in Sport. 2025;42:60530. eISSN 2450-3118.
https://doi.org/10.12775/QS.2025.42.60530
https://apcz.umk.pl/QS/article/view/60530

The journal has been awarded 20 points in the parametric evaluation by the Ministry of Higher Education and Science of Poland. This is according to the Annex to the announcement of the Minister of Higher Education and Science dated 05.01.2024, No. 32553. The journal has a Unique Identifier: 201398. Scientific disciplines assigned: Economics and Finance (Field of Social Sciences); Management and Quality Sciences (Field of Social Sciences).
Punkty Ministerialne z 2019 - aktualny rok 20 punktów. Załącznik do komunikatu Ministra Szkolnictwa Wyższego i Nauki z dnia 05.01.2024 Lp. 32553. Posiada Unikatowy Identyfikator Czasopisma: 201398. Przypisane dyscypliny naukowe: Ekonomia i finanse (Dziedzina nauk społecznych); Nauki o zarządzaniu i jakości (Dziedzina nauk społecznych).
The authors declare that there is no conflict of interest regarding the publication of this paper.
Received: 24.04.2025. Revised: 30.04.2025. Accepted: 11.06.2025. Published: 14.06.2025.

Interactions Between Psychiatric Disorders and Inflammatory Skin Diseases - systematic review

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Abstract

Introduction:

Depression and anxiety are prevalent mental disorders often co-occurring with chronic inflammatory conditions such as psoriasis and atopic dermatitis. These associations suggest shared biological mechanisms, including immune dysregulation and persistent inflammation. Understanding these links is essential for developing integrated treatments addressing both psychiatric and dermatological symptoms.

Aim of Study:

This study explores shared pathomechanisms between depression, anxiety disorders, psoriasis, and atopic dermatitis.

Materials and Methods:

A systematic literature review was conducted using PubMed and Google Scholar. Articles published between 2019 and 2025 were analyzed to identify biological mechanisms linking psychiatric disorders with inflammatory skin diseases. Keywords included: "depression," "anxiety disorders," "psoriasis," "atopic dermatitis," "pathomechanism," and "inflammation."

Conclusion:

Findings underscore the interconnectedness of psychiatric and inflammatory skin disorders via common pathophysiological pathways. Targeted biological therapies and psychological interventions show promise in managing these comorbidities. Further research is required to enhance integrated care for affected patients.

Keywords:

Depression, Anxiety Disorders, Psoriasis, Atopic Dermatitis

Introduction

Depression and anxiety disorders are among the most prevalent mental health conditions worldwide, affecting a significant proportion of the population and contributing to substantial morbidity and mortality. These psychiatric disorders frequently co-occur with various chronic inflammatory diseases, including psoriasis and atopic dermatitis [5][25]. Psoriasis and atopic dermatitis are not only dermatological conditions but systemic chronic inflammatory diseases that have been linked to psychiatric comorbidities such as depression and anxiety through shared biological mechanisms [14][32].

The co-occurrence of psychiatric disorders and chronic inflammatory skin conditions can be attributed to overlapping pathophysiological pathways, including dysregulation of the immune system, chronic inflammation, and alterations in the hypothalamic-pituitary-adrenal (HPA) axis [33]. Inflammatory cytokines, such as tumor necrosis factor-alpha (TNF-α) and interleukins (IL), play a crucial role in both dermatological and psychiatric manifestations, influencing brain function and behavior [1][13].

Moreover, the psychological burden of visible skin conditions, including stigma, social isolation, and diminished self-esteem, exacerbates mental health issues [8]. This bidirectional relationship underscores the importance of an integrated approach to treatment that addresses both the physical and psychological aspects of these diseases. Understanding the common etiological factors between psychiatric and dermatological conditions could pave the way for targeted therapies that can effectively manage both sets of symptoms, thereby improving the overall quality of life for affected individuals [16].

Advances in biologic therapies, targeting specific inflammatory pathways, have demonstrated promise in not only alleviating skin symptoms but also in reducing the psychiatric burden associated with chronic inflammatory diseases [12]. These therapies, combined with psychological interventions, offer a comprehensive strategy to manage the multifaceted impact of these comorbid conditions [26].

In this review, we explore the shared pathomechanisms underlying depression, anxiety, psoriasis, and atopic dermatitis, examining how these intersecting pathways contribute to the development and progression of both psychiatric and dermatological disorders. By elucidating these connections, we aim to highlight potential therapeutic targets and strategies for holistic patient care, which integrates dermatological and mental health management [3][24].

Depression and Anxiety Disorders

Depression is the most common mental disorder globally. According to WHO data, it affects approximately 3.8% of the world's population. It is a life-threatening condition, with around 700,000 people committing suicide each year [31]. Furthermore, depression often co-occurs with other mental disorders [5] and somatic diseases [25]. Similar to other psychiatric disorders, depression affects women more frequently than men [14].

Depressive disorders are characterized by at least two weeks of low mood, loss of interest, and pleasure, significantly reducing the patient's quality of life. Other symptoms include memory and concentration disturbances, low self-esteem, feelings of helplessness and hopelessness, and suicidal thoughts. Patients often report somatic symptoms such as appetite disturbances, sleep disorders, decreased sexual drive, headaches, muscle pains, and increased fatigue [32].

Depression frequently co-occurs with anxiety disorders. It is estimated that over 70% of individuals with depression experience anxiety symptoms, and 40-70% meet the criteria for at least one type of anxiety disorder [33]. Anxiety disorders represent a diverse group of conditions characterized by excessive fear and worry, significantly impairing daily functioning in personal, familial, social, educational, and professional domains [1]. Physical symptoms may include irritability, fatigue, concentration difficulties, headaches, heart palpitations, chest pain, nausea, abdominal pain, and sweating [13].

The pathomechanism of depression is complex, involving dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis, leading to excessive cortisol production and chronic stress, which affect neurotransmitters such as serotonin, dopamine, and norepinephrine [8]. Reduced hippocampal volume and neuroplasticity disturbances are also critical for the development of depression [16]. Inflammation plays a significant role in the pathogenesis of depression, as evidenced by elevated levels of pro-inflammatory cytokines such as IL-6 and TNF-α [12].

Anxiety disorders are associated with hyperactivity of the amygdala and dysfunctions in the prefrontal cortex, disrupting the response to stress stimuli. Dysfunctions in the GABAergic and serotonergic systems contribute to excessive anxiety responses [26]. Studies also highlight the importance of the gut-brain axis, where an abnormal gut microbiota can influence HPA axis functioning and immune system modulation, exacerbating symptoms of anxiety and depression [3].

Depression and anxiety disorders are primarily treated with psychotherapy and pharmacotherapy. Contemporary therapeutic approaches focus on optimizing these methods, considering the diverse biological mechanisms underlying these disorders [24].

Psoriasis and Atopic Dermatitis

Psoriasis is a chronic dermatosis, characterized by reddish-brown papules covered with silvery-white scales. In 2016, the WHO recognized psoriasis as a serious global health issue. The highest prevalence is observed in Norway at 8%, with a prevalence rate of 2.5% among the Caucasian population [11]. The disease affects both sexes equally.

Psoriasis is characterized by a polygenic and multifactorial inheritance pattern. The first and later most significant allele associated with psoriasis susceptibility was HLA-Cw*06, located on chromosome 6 within the PSORS1 region [18]. The formation of primary lesions is caused by an abnormal immune response mediated by T lymphocytes, particularly Th17 and Th1 subpopulations. Psoriasis onset is strongly associated with external factors such as infections, stress, or mechanical trauma. T. Henseler and E. Christophers were the first to distinguish two types of psoriasis. Type 1 is much more common, manifests early (before the age of 40), and is often associated with a family history of the disease [23]. Type 2 usually manifests between the ages of 50 and 59.

Diagnosis is based on a thorough physical examination, during which a physician examines the skin lesions. Histopathological examination is sometimes performed. Psoriasis patients have an increased risk of venous and arterial vascular diseases, as described by McDonald [9], as well as joint [28] and inflammatory bowel diseases. Severe cases of psoriasis are associated with increased mortality [20]. Patients with visible lesions have a lower quality of life index (LQI) and often face stigma due to their appearance and the public's lack of education, leading to a higher incidence of mental health issues such as depression and anxiety disorders [10].

Atopic dermatitis (AD) is a chronic inflammatory disease characterized by a recurrent and varied course. AD often coexists with other IgE-mediated atopic diseases such as allergic rhinitis and asthma [6]. The disease typically manifests in early childhood, with approximately 80% of cases starting before the age of five [30].

The pathogenesis of atopic dermatitis is complex. Environmental factors play a significant role in disease development [30]. The most important gene associated with AD susceptibility is FLG, which encodes filaggrin [28]. Mutations in this gene increase the risk of AD fourfold. Patients exhibit increased IgE production and decreased suppressor T lymphocyte activity. They also have an epidermal barrier defect, primarily due to abnormal skin structure (previously mentioned filaggrin), leading to increased transepidermal water loss and greater susceptibility to pathogen penetration through the skin. Allergens play a significant role in childhood dermatosis by stimulating inflammation.

The clinical presentation varies with age. The location of lesions is characteristic of different age groups. In infants, lesions are located on the cheeks and extensor surfaces of the limbs and may appear on the abdomen, excluding the diaper area. In children (aged 2-12 years), skin lesions primarily affect flexural areas and the neck. In adults, the location is similar, but facial and hand areas are more frequently involved. Patients often complain of severe itching and insomnia, which lower their quality of life. Patients with AD are at increased risk of mental health disorders such as depression and anxiety due to the frequent lack of understanding and unjustified fear of contagion from visible skin changes. Approximately 9.3–44.3% of AD patients suffer from depression, while

3.31–26.2% exhibit anxiety disorders [20]. There is a correlation between the Patient-Oriented Eczema Severity Scoring (PO-SCORAD) scale and the severity of depressive symptoms. The diagnosis of AD is based on the Hanifin and Rajka criteria [10]. To establish a diagnosis, three major and three minor criteria must be identified. Physical examination and patient history are also important.

Common Pathomechanisms Linking Psoriasis, Atopic Dermatitis, Depression, and Anxiety Disorders

Chronic inflammatory skin diseases such as psoriasis and atopic dermatitis (AD) share fundamental immunological processes that not only perpetuate local inflammation in the skin but also contribute to systemic effects, including neuroinflammation and psychological disorders. These processes involve dysregulation of immune pathways and overproduction of proinflammatory cytokines, linking dermatological and psychiatric symptoms.

Psoriasis is predominantly a Th1/Th17-mediated inflammatory disease. Key cytokines driving the inflammatory response include tumor necrosis factor-alpha (TNF-α), interleukin-17 (IL-17), and interleukin-23 (IL-23). TNF-α increases vascular permeability, allowing immune cells to infiltrate the epidermis, while IL-17 stimulates keratinocyte proliferation, leading to the characteristic thickened plaques observed in psoriasis [35,7]. IL-23 acts as an upstream driver, maintaining the Th17 cell population and perpetuating the chronic inflammatory state. Beyond the skin, TNF-α and IL-17 can cross the blood-brain barrier (BBB), where they activate microglia. Microglial activation triggers the release of reactive oxygen species (ROS) and additional cytokines, contributing to neuroinflammation and cognitive impairments [19].

AD, in contrast, is characterized by a Th2-mediated immune response, with interleukins IL-4, IL-13, and IL-31 playing pivotal roles. IL-4 and IL-13 impair the skin barrier by downregulating the expression of filaggrin and other structural proteins, increasing transepidermal water loss and susceptibility to allergens and pathogens [2]. IL-31, known as the “itch cytokine,” activates sensory nerves in the skin, causing persistent pruritus. Chronic scratching exacerbates skin damage, leading to secondary infections and further inflammation [4]. Elevated levels of these cytokines in circulation also affect the central nervous system, promoting neuroinflammation and heightening the risk of anxiety and depressive disorders [17].

Both psoriasis and AD involve a breakdown in the balance between proinflammatory and regulatory cytokines. Regulatory T cells (Tregs), which suppress excessive immune responses, are functionally impaired in these conditions. This loss of regulation leads to the unchecked activity of effector T cells (Th1, Th2, and Th17), resulting in sustained inflammation and tissue damage [27].

Inflammatory mediators also influence neurotransmitter systems. Proinflammatory cytokines such as IL-6 and TNF-α activate the kynurenine pathway, diverting tryptophan metabolism away from serotonin synthesis. This leads to reduced serotonin levels, a well-established mechanism contributing to depressive symptoms in chronic

inflammatory states [21]. Elevated levels of kynurenine metabolites, such as quinolinic acid, are neurotoxic and further exacerbate neuronal damage, particularly in regions such as the hippocampus and prefrontal cortex [22].

The systemic nature of inflammation in these diseases underscores the need for treatments targeting both local and systemic pathways. Biologic therapies, such as TNF-α inhibitors (e.g., infliximab) and IL-17 inhibitors (e.g., secukinumab), have demonstrated efficacy in reducing skin symptoms and improving psychological outcomes. These therapies modulate systemic cytokine levels, alleviating both physical and mental health burdens [21].

The hypothalamic-pituitary-adrenal (HPA) axis is the body's central stress-response system, responsible for maintaining homeostasis during physiological and psychological stress. It integrates signals from the central nervous system (CNS) and peripheral immune responses to regulate the production of cortisol, a glucocorticoid hormone with potent anti-inflammatory effects. Dysregulation of the HPA axis is a hallmark of chronic inflammatory skin diseases such as psoriasis and atopic dermatitis (AD), and it has profound implications for the progression of these conditions as well as their associated psychiatric comorbidities [35,7].

Under normal conditions, the HPA axis operates as a tightly regulated feedback loop. Stress activates the hypothalamus to secrete corticotropin-releasing hormone (CRH), which in turn stimulates the anterior pituitary gland to release adrenocorticotropic hormone (ACTH). ACTH signals the adrenal glands to produce cortisol, which then exerts its effects systemically. Cortisol downregulates proinflammatory cytokines, such as TNF-α and IL-6, and upregulates anti-inflammatory mediators, restoring immune balance [19].

In psoriasis, the HPA axis often exhibits a blunted response to stress. Reduced cortisol levels fail to adequately suppress the production of proinflammatory cytokines, including IL-17, IL-23, and TNF-α, which are central to the disease's pathogenesis. This cortisol insufficiency perpetuates systemic inflammation, exacerbates skin lesions, and contributes to neuroinflammation in the brain [22]. Blunted HPA axis activity also affects the serotonergic system, disrupting pathways involved in mood regulation. This dysfunction has been linked to the heightened prevalence of depression and anxiety in individuals with psoriasis [15].

In AD, the HPA axis follows a different pattern of dysregulation, often characterized by initial hyperactivity. Chronic pruritus acts as a persistent stressor, driving excessive CRH release. CRH not only stimulates the release of ACTH and subsequent cortisol production but also directly activates mast cells, leading to histamine release. This exacerbates itching and inflammation, creating a vicious cycle [2]. Over time, the chronic overactivation of the HPA axis can deplete adrenal reserves, resulting in reduced cortisol production in later disease stages. This paradoxical shift—from hyperactivity to eventual exhaustion—contributes to the persistence of inflammation and impairs the skin barrier's ability to repair itself [17].

Systemic inflammation in both psoriasis and AD disrupts the normal feedback regulation of the HPA axis. Elevated circulating levels of cytokines, such as IL-6 and TNF-α, interfere with glucocorticoid receptor signaling in the hypothalamus and pituitary gland, reducing their sensitivity to cortisol. This desensitization amplifies the inflammatory response and further destabilizes HPA axis function, creating a self-perpetuating cycle [21].

The interaction between the HPA axis and systemic inflammation also extends to the brain, where neuroinflammation contributes to cognitive and emotional dysfunction. Proinflammatory cytokines activated by HPA axis dysregulation can cross the blood-brain barrier and activate microglial cells, leading to neuronal

damage in regions such as the hippocampus and prefrontal cortex. These changes are associated with symptoms of depression, anxiety, and impaired stress resilience commonly observed in patients with psoriasis and AD [4].

Addressing HPA axis dysfunction in these diseases requires integrated therapeutic strategies. Biologic therapies targeting cytokines such as TNF-α and IL-17 have demonstrated efficacy in reducing systemic inflammation and normalizing cortisol levels, indirectly improving HPA axis regulation. Additionally, interventions such as mindfulness-based stress reduction (MBSR) and cognitive-behavioral therapy (CBT) can mitigate stress and promote HPA axis recovery, providing comprehensive benefits for both physical and mental health [27,34].

Psychological Consequences of Chronic Skin Diseases

Chronic skin diseases also impose profound psychological consequences on patients. Visible skin lesions in psoriasis often lead to stigmatization, triggering feelings of shame, social isolation, and diminished self-esteem. Patients may avoid social interactions due to fear of judgment or rejection, which exacerbates depressive symptoms and reduces access to supportive relationships [36]. This cycle of social withdrawal and emotional distress perpetuates the psychological impact of the disease. In AD, chronic itching disrupts sleep, leading to fatigue, irritability, and cognitive impairments. Sleep deprivation further amplifies systemic inflammation, creating a feedback loop where physical symptoms and mental health deteriorate in tandem. Persistent pruritus also sensitizes central nervous system pathways, contributing to hypervigilance and heightened stress responses [37].

The psychological burden of these conditions is further compounded by their economic and social ramifications. Frequent medical visits, high treatment costs, and reduced workplace productivity generate financial stress, while strained relationships with family and caregivers contribute to emotional distress. Caregivers often report feelings of helplessness and emotional exhaustion, particularly when managing the demands of a patient with severe symptoms. These dynamics create a reciprocal relationship between the patient and their caregivers, where the stress of one party amplifies the burden on the other [27]. Gender and age differences also play a role; younger individuals, especially adolescents, are particularly vulnerable to anxiety and social withdrawal due to heightened peer pressures and concerns about body image. Women with psoriasis are more likely to experience emotional distress and stigma, whereas men often report challenges in workplace interactions and social participation [34].

These conditions frequently coexist with systemic diseases such as metabolic syndrome, cardiovascular disorders, and autoimmune conditions. These comorbidities amplify the psychological burden by increasing the complexity of disease management. Patients often experience frustration and helplessness as they navigate multiple chronic illnesses, further elevating their risk of psychiatric disorders [7]. Addressing the dual burden of these skin conditions and their psychiatric comorbidities requires an integrated treatment approach.

Biologic therapies targeting cytokines such as TNF-α, IL-17, and IL-23 have revolutionized the management of psoriasis and AD. These treatments not only alleviate skin symptoms but also modulate systemic inflammation and neuroinflammatory pathways, improving mood and cognitive symptoms [38]. For example, TNF-α inhibitors like infliximab and IL-17 inhibitors like secukinumab have demonstrated efficacy in reducing depressive symptoms in addition to controlling dermatological manifestations [21]. Complementary

psychological interventions, such as cognitive-behavioral therapy (CBT) and mindfulness-based stress reduction (MBSR), further enhance treatment outcomes. These therapies equip patients with tools to manage emotional distress and develop adaptive coping strategies, reducing the impact of stigma and improving overall quality of life [22].

Relaxation techniques, including yoga, meditation, and biofeedback, also play a valuable role in managing stress and regulating the HPA axis. Educational and peer support programs empower patients with knowledge about their conditions and provide platforms for sharing experiences, reducing feelings of isolation [21]. By addressing both physical and psychological dimensions, integrated care approaches offer the best prospects for improving outcomes in individuals with chronic skin diseases.

Conclusion

Psoriasis and atopic dermatitis (AD) are not merely dermatological conditions but systemic diseases with profound implications for both physical and mental health. The shared pathomechanisms linking these diseases to psychiatric disorders such as depression and anxiety highlight the intricate interplay between immune dysregulation, neuroinflammation, and neuroendocrine dysfunction. Central to this connection is the role of proinflammatory cytokines, the hypothalamic-pituitary-adrenal (HPA) axis, and the subsequent impact on brain regions critical for emotional and cognitive functioning. The bidirectional relationship between systemic inflammation and neuropsychiatric symptoms creates a vicious cycle that perpetuates disease severity and diminishes quality of life. While inflammatory pathways such as the Th1/Th17 axis in psoriasis and the Th2 axis in AD drive local and systemic manifestations, the psychological burden—including stigma, social isolation, and financial stress—further exacerbates disease progression. The complexity of these intertwined mechanisms underscores the necessity of integrative treatment approaches. Advances in biologic therapies targeting specific cytokines have shown promise in mitigating systemic inflammation and its psychological effects. However, addressing the psychological dimensions of these diseases through interventions such as cognitive-behavioral therapy, mindfulness-based stress reduction, and social support programs remains equally critical. Future research should focus on elucidating the precise molecular mechanisms connecting skin inflammation to neuroinflammation and psychiatric outcomes. This knowledge will pave the way for more personalized therapies, targeting both the dermatological and psychological aspects of these chronic conditions. Ultimately, a holistic approach that bridges the gap between physical and mental health care is essential to improving patient outcomes and quality of life.

Disclosure:

Authors 'contribution:

Conceptualization: Mateusz Kacalak

Methodology: Mateusz Kacalak

Software: Mateusz Kacalak

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Formal Analysis: Mateusz Kacalak

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Writing-Review and Editing: Mateusz Kacalak

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Supervision: Mateusz Kacalak

Project Administration: Mateusz Kacalak

Funding Statement: The study did not receive special funding.

Institutional Review Board Statement: Not applicable.

Informed Consent Statement: Not applicable.

Data Availability Statement: Not applicable.

Conflict of Interest: The author declares no conflict of interest.

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