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# IMMUNOLOGICAL CHARACTERISTICS OF PSORIASIS

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**Abstract**
This article provides a comprehensive analysis of the immunopathogenesis, epidemiology, etiologic factors, and clinical manifestations of psoriasis. The central role of the IL-23/IL-17 axis and the interaction between keratinocytes and Th17 cells are substantiated. Diagnostic approaches, including the application of PASI, BSA, and DLQI indices, are highlighted. The systemic nature of psoriasis, its association with comorbid conditions, and the efficacy of biologic therapy are discussed based on scientific literature, emphasizing the importance of individualized treatment strategies. The results underscore the significance of early diagnosis and monitoring in disease management and provide a basis for developing preventive measures.

**Keywords:** Psoriasis, immunology, IL-17, IL-23, Th17 cells, pathogenesis, keratinocytes, biologic therapy.


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## Introduction

Psoriasis is a chronic, systemic, immune-mediated inflammatory disease primarily characterized by damage to the skin and joints (1). National clinical protocols approved by the Ministry of Health of the Republic of Uzbekistan emphasize the importance of diagnosis and treatment of this disease, highlighting its impact on public health (2). The pathogenesis of the disease is based on a complex interaction between genetic predisposition, environmental triggers, and immune system dysfunction (2). Recent studies indicate that psoriasis is a "mixed" type of immunological condition involving both autoimmune and autoinflammatory components (3).

In the initial stage of pathogenesis, antimicrobial peptides (AMPs) produced by keratinocytes, specifically LL-37, play a central role in activating the innate immune system (4). This process leads to the activation of dendritic cells and, consequently, the production of the cytokine interleukin (IL)-23 (5). The IL-23/IL-17 axis constitutes the immunological basis of psoriasis, facilitating the differentiation of T-helper 17 (Th17) cells and the production of IL-17 (6). Cytokines secreted by Th17 and Th1 cells lead to keratinocyte hyperproliferation and the formation of characteristic erythematosquamous lesions on the skin (7). In modern medicine, understanding these immunological mechanisms has enabled the development of highly effective selective biologic therapy methods for disease management (8).

## Results

Epidemiology of Psoriasis. Psoriasis is a prevalent chronic disease worldwide, the prevalence rate of which varies significantly depending on geographic location, ethnicity, and environmental factors (9). Globally, approximately 2-3% of the population is affected by this disease, which accounts for more than 125 million

individuals (10). The prevalence of the disease increases with distance from the equator; for instance, while rates in Northern Europe and Scandinavian countries can reach 5-11%, this indicator is significantly lower (0.1-0.5%) among the populations of Asia and Africa (8). In the Republic of Uzbekistan, psoriasis also occupies one of the leading positions among dermatological diseases, and national clinical protocols are aimed at reducing the socio-economic burden of this disease (11).

The age of disease onset typically exhibits a bimodal distribution, corresponding to two primary peaks: the first occurs between the ages of 15-25 (early-onset or Type I), and the second is observed between the ages of 50-60 (late-onset or Type II) (12). The incidence rate is relatively equal between men and women; however, the disease tends to onset at an earlier age in women (13). Epidemiological studies indicate that comorbid conditions such as metabolic syndrome, obesity, arterial hypertension, and diabetes mellitus occur more frequently in patients with psoriasis compared to the general population (14). Furthermore, smoking, alcohol consumption, and chronic psychosocial stress are considered major modifiable risk factors that influence the development and exacerbation of the disease (15).

Etiology. Psoriasis is a multifactorial disease, the development of which is the result of a complex interaction among genetic predisposition, immune system dysfunction, and environmental triggers (2). Hereditary factors occupy a central position in the etiopathogenesis of the disease, as evidenced by familial cases and twin studies (16).

1. Genetic Factors. The primary genetic locus associated with psoriasis is PSORS1 (Psoriasis Susceptibility 1), located on chromosome 6 (5). The HLA-Cw*06:02 allele, situated in this region, is the strongest genetic marker for disease susceptibility and is detected in over 60% of patients with early-onset (Type I)




psoriasis (11). Additionally, polymorphisms in the IL-23 receptor (IL23R) and IL-12B genes also increase the risk of disease development (3).

2. Environmental and Triggering Factors. In individuals with a genetic predisposition, the manifestation of the disease often requires an external "trigger" or precipitating factor:

Infections: Streptococcal infections of the upper respiratory tract, in particular, cause the development of acute guttate psoriasis (4).

Physical Trauma (Koebner Phenomenon): The appearance of psoriatic lesions at the site of mechanical skin injury, sunburn, or postoperative scars (11).

Psychosocial Stress: Severe emotional strain can trigger or exacerbate the disease by disrupting the neuroendocrine regulation of the immune system (15).

Medications: The abrupt discontinuation of beta-blockers, lithium preparations, antimalarial drugs, and systemic corticosteroids leads to the exacerbation of psoriasis (17).

3. Lifestyle and Metabolic Factors. Contemporary epidemiological data indicate that obesity (visceral fat accumulation) is an independent risk factor for the development of psoriasis, as adipose tissue produces pro-inflammatory cytokines (adipokines) (18). Furthermore, smoking and alcohol consumption are also noted as factors that increase the severity of the disease and reduce treatment efficacy (14).

The pathogenesis of psoriasis is a multi-step process resulting from complex interactions between cells of the innate and adaptive immune systems, keratinocytes, and inflammatory cytokines (16). The main stages of this process are described below:

Pathogenesis of Psoriasis: Immunological Mechanisms

Initialization Stage. The initial stage of disease development begins with keratinocyte injury induced by external triggers (mechanical trauma, infection, or

medications) (4). Injured keratinocytes secrete antimicrobial peptides (AMPs), specifically LL-37 (cathelicidin). LL-37 forms complexes with self-DNA and RNA molecules, activating plasmacytoid dendritic cells (pDCs) (16). Consequently, these cells produce large amounts of interferon-alpha, which initiates the inflammatory cascade (3).

Role of Antigen-Presenting Cells and IL-23. Under the influence of interferon-alpha and other initial signals, myeloid dendritic cells are activated and migrate to the lymph nodes. There, they present antigens to naive T-lymphocytes and produce the cytokine interleukin-23 (IL-23) (5). IL-23 is considered the "central driver" of psoriasis pathogenesis and is responsible for the differentiation, survival, and proliferation of T-helper 17 (Th17) cells (2).

The IL-23/IL-17 Axis and Effector Phase. Th17 cells migrate to the dermal layer of the skin, where they secrete cytokines IL-17A, IL-17F, and IL-22, which induce the primary pathological hallmarks of psoriasis (3). IL-17A binds to receptors on the surface of keratinocytes, stimulating the following processes:

Keratinocyte proliferation: Cell division accelerates, resulting in epidermal thickening (acanthosis) and the emergence of incompletely matured stratum corneum cells (parakeratosis) (11).

Neutrophil infiltration: Chemokines (CXCL1, CXCL8) secreted by keratinocytes attract neutrophils to the epidermis, forming Munro's microabscesses (16).

Chronic Inflammatory Cycle (Positive Feedback Loop). Activated keratinocytes not only proliferate but also begin to produce additional inflammatory factors (TNF, IL-1, IL-6) and chemokines themselves (2). This leads to the accumulation of even more immune cells in the affected area, creating a chronic, self-sustaining inflammatory loop (3).

Vascular and Dermal Changes. The inflammatory process causes the dilation of blood vessels in the dermis and the enhancement of angiogenesis (the formation of

new blood vessels). Clinically, this process determines the bright red color of psoriatic plaques and the shedding of silvery scales from them (11).

The clinical picture of psoriasis is diverse, manifesting as lesions on various areas of the skin, nails, and joints (16). The clinical course of the disease is chronic, alternating periodically between phases of exacerbation (relapse) and subsidence (remission) (11).

**Clinical Manifestations of Psoriasis**

1. Plaque Psoriasis (Psoriasis vulgaris). This is the most common form of the disease (approximately 80-90%), characterized by the appearance of sharply demarcated, bright pink or red papules and plaques on the skin (2). The surface of these lesions is covered with easily detachable silvery-white scales. The eruptions are most often symmetrically located on the extensor surfaces of the elbow and knee joints, the scalp, and the lumbar region (11).

2. Diagnostic Psoriatic Phenomena (Psoriatic Triad). During the process of scraping (grattage) psoriatic lesions, the following pathognomonic signs are observed sequentially: Stearin spot phenomenon: When the scales are scraped, a whitish spot resembling stearin shavings is formed; Terminal film phenomenon: After the complete removal of scales, a shiny, moist, and thin surface is revealed Blood dew (Auspitz) phenomenon: Upon further scraping of the terminal film, punctate bleeding (pinpoint blood drops) appears (11).

3. Koebner Phenomenon (Isomorphic Response). In the progressive (active) stage of the disease, the appearance of new psoriatic eruptions is observed within 7-14 days at the sites of mechanical skin injury (scratches, burns, or cuts) (2).

4. Nail Psoriasis (Onychodystrophy). Damage to the nail plates is observed in nearly 50% of patients with psoriasis. The clinical manifestations include the following: Pitting (thimble-like punctations): The appearance of small depressions

on the nail surface; "Oil drop" sign: The appearance of yellowish-brown spots beneath the nail plate; Onycholysis: The detachment of the nail from the distal nail bed (16).

5. Other Clinical Forms. Guttate psoriasis: Small, drop-like eruptions that typically appear suddenly on the body following a streptococcal infection (4). Exudative psoriasis: The formation of yellowish crusts (exudates) in place of scales, frequently encountered in patients with obesity or diabetes mellitus (11); Pustular psoriasis: A severe form characterized by the formation of purulent blisters (pustules) on the skin; Psoriatic erythroderma: A life-threatening condition characterized by the erythema and inflammation of almost the entire body surface (more than 90%) (2).

6. Psoriatic Arthritis. Joint inflammation is observed in approximately 20-30% of patients suffering from psoriasis (2). This predominantly manifests as pain, swelling, and morning stiffness in the distal joints of the fingers; if prolonged, it can lead to joint deformity and disability (11).

The diagnosis of psoriasis primarily relies on the clinical examination of the patient, the study of the morphological elements of the skin pathology, and anamnestic data (11). Due to the presence of specific clinical signs of the disease, additional instrumental investigations are not required for diagnosis in most cases (2).

Diagnosis of Psoriasis: Key Stages

Clinical Diagnosis and the "Psoriatic Triad." The foundation of diagnosis is the identification of pathognomonic signs through the scraping (grattage) of skin eruptions. This sequentially reveals the stearin spot, the terminal film, and the Auspitz phenomenon (punctate bleeding) (11). Furthermore, the appearance of new eruptions at sites of mechanical skin injury during the active phase of the disease, known as the Koebner phenomenon, is of diagnostic significance (16).

Assessment of Disease Severity. To determine the severity of psoriasis and the need for systemic therapy, the PASI (Psoriasis Area and Severity Index) is utilized, which evaluates the erythema, infiltration, and desquamation of lesions on a 72-point scale (12). The BSA (Body Surface Area) index is applied to calculate the affected skin area, wherein the surface of the patient's palm is considered equivalent to 1% of the total body surface area (19). To assess the patient's psychosocial status, the DLQI (Dermatology Life Quality Index) questionnaire is administered (19).

Laboratory and Immunological Analyses. There are no specific laboratory markers for psoriasis; however, to determine the level of systemic inflammation, the erythrocyte sedimentation rate (ESR) in a complete blood count and the C-reactive protein (CRP) level in a biochemical analysis are evaluated (16). Immunologically, quantifying cytokines such as IL-17, IL-23, and TNF $\alpha$ assists in understanding the immunological activity of the disease (3). When joint pain is observed, it is necessary to rule out or confirm psoriatic arthritis by evaluating the rheumatoid factor and performing joint radiography (11).

Histopathological Examination. In cases where the clinical picture is ambiguous or rare forms (e.g., pustular psoriasis) are suspected, a skin biopsy is performed (2). Histological examination typically reveals acanthosis, parakeratosis, loss of the granular layer in the epidermis, and the presence of Munro's microabscesses formed by the accumulation of neutrophils, which are characteristic of psoriasis (16).

Differential Diagnosis. During the diagnostic process, psoriasis must be differentiated from diseases with similar clinical presentations, such as seborrheic dermatitis, lichen planus, pityriasis rosea (Gibert's disease), and atopic dermatitis (11).


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**Discussion**

The results of this study further confirm the complex immunological nature of psoriasis. The IL-23/IL-17 axis occupies a central position in the pathogenesis of the disease. This finding is consistent with contemporary scientific literature. In particular, (2) also notes that the activity of Th17 cells and the elevated levels of the IL-17 cytokine are the primary drivers of the disease in psoriasis. Furthermore, this work indicates that psoriasis is a "mixed" disease that combines both autoimmune and autoinflammatory components. This concept is supported by (3), who evaluate psoriasis as the product of an interaction between innate and adaptive immunity, distinguishing it from classical autoimmune diseases.

The conclusion that keratinocytes are not merely passive targets but active participants is also of significant importance. They have been shown to produce inflammatory mediators, a finding that is in complete agreement with the studies by (7). These authors emphasize that keratinocytes are crucial effector cells that amplify the immune response. From an epidemiological perspective, the 2–3% global prevalence rate presented in the article aligns with the reports by (10). Concurrently, the increasing role of factors such as metabolic syndrome, obesity, and stress in disease development has been noted in other studies as well (14). This highlights the necessity of considering psoriasis not solely as a dermatological condition, but as a systemic disease.

Additionally, the views expressed in the article regarding the possibilities of biologic therapy correspond with modern approaches. According to the data from (8), biologic agents targeting IL-17 and IL-23 demonstrate high efficacy in the treatment of psoriasis, initiating a new era in disease control. Overall, the results of this study are harmonious with international scientific data and serve to deepen the understanding of psoriasis pathogenesis. At the same time, the necessity of

further in-depth investigation of immunological markers to develop individualized (personalized) treatment methods in the future is identified.

## Conclusion

Psoriasis is not merely a skin disease, but a complex, systemic immunological dysfunction triggered by the interaction of genetic predisposition and environmental factors. At the core of the disease's pathogenesis lies the IL-23/IL-17 inflammatory axis, which leads to the pathological hyperproliferation of keratinocytes and the formation of a chronic inflammatory cycle. Epidemiological data demonstrate that this pathology represents a significant socio-economic burden on a global scale, particularly among the population of Uzbekistan.

The diagnostic process primarily relies on specific clinical signs and the phenomena of the "psoriatic triad"; however, the modern approach requires an objective assessment of disease severity using international indices such as the PASI and DLQI. A profound understanding of immunological mechanisms, especially the role of Th17 cells and their cytokines, has laid the groundwork for the development of selective biologic therapies in modern medicine.

In conclusion, the effective management of psoriasis must be directed not only at eliminating skin eruptions but also at controlling systemic inflammation, preventing comorbid conditions (such as metabolic syndrome and psoriatic arthritis), and improving the patients' quality of life. Future research will serve to facilitate the early detection of immunological markers of the disease and the further refinement of individualized (personalized) treatment strategies.


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