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PSORIASIS: A COMPREHENSIVE REVIEW OF CURRENT KNOWLEDGE

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ABSTRACT

Introduction and Purpose: Psoriasis is a chronic systemic inflammatory disease that impairs quality of life and psychological well being. Its multifactorial etiology results from genetic predisposition, environmental triggers, and immune dysregulation where the IL-23/IL-17 axis plays a notable role. This review synthesizes current scientific literature on psoriasis, focusing on risk factors, pathogenesis, and available treatment methods.
Materials and Methods: This review analyzes 47 articles from the PubMed database published between 2002 and 2026, with an emphasis on recent literature. The review integrates etiopathogenesis with clinical management, including conventional therapies, biologics, and small-molecule inhibitors.
Description of the State of Knowledge: Psoriasis diagnosis relies on clinical assessment supported by the Psoriasis Area and Severity Index (PASI), Body Surface Area (BSA), and Dermatology Life Quality Index (DLQI) scales. Pathogenesis involves genetic and environmental factors leading to the activation of the IL-23/IL-17 axis and chronic inflammation. Management includes lifestyle modification and individualized therapy through topical treatments, phototherapy, systemic drugs, and biologic therapies tailored to disease severity and quality of life.
Conclusion: The contemporary understanding of psoriasis pathogenesis focuses on the interleukin-23/interleukin-17 axis and the significant role of genetic factors, including the HLA-C06:02 variant. Therapeutic advances, particularly the introduction of biologic agents and novel targeted molecules, have substantially improved treatment efficacy. However, important limitations remain, including the lack of reliable serological markers and the insufficient ability to predict treatment response; therefore, further research into predictive biomarkers and artificial intelligence–based tools is essential.

KEYWORDS

Psoriasis, IL-23/IL-17 Axis, Environmental Triggers, HLA-C*06:02, Biological Therapy, Personalized Medicine

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Introduction and Purpose

Psoriasis is a chronic, inflammatory immune-mediated dermatosis characterized by the presence of sharply demarcated erythematosquamous plaques. The condition is associated with a significant psychosocial impact, leading to stigmatization and a substantial impairment of patients' quality of life [2]. Furthermore, psoriasis imposes a considerable economic strain on healthcare systems worldwide, arising from both high direct treatment costs and significant indirect costs. A primary driver of these expenditures is the increasing availability and utilization of modern biologic therapies, which have become a cornerstone of patient management [44,45]. The global load of psoriasis is projected to increase systematically. Consequently, healthcare planning and the development of regional strategies will necessitate intensified efforts in risk factor control and the prioritization of high-risk populations [44]. Currently, psoriasis is regarded as a systemic disease rather than an isolated cutaneous disorder. It frequently co-occurs with psychiatric disorders, metabolic conditions, inflammatory arthropathies, and cardiovascular diseases. Ultimately, the cumulative impact of these comorbidities may contribute to a reduction in life expectancy [3].

The goal of this review is to present and systematize current knowledge regarding psoriasis, with a particular emphasis on its pathogenesis, risk factors, and current therapeutic options.

Methodology

The basis of this review is a survey of the scientific literature on psoriasis. The analysis was conducted on the basis of 47 articles selected from the PubMed database due to their substantive value and relevance to the aims of the study. The scope of the analysis included publications published between 2002 and 2026. Such a broad time frame made it possible to trace changes in therapeutic strategies over more than two decades. At the same time, the predominance of materials published between 2022 and 2026 allowed the most up-to-date

medical knowledge to be included. In the process of data synthesis, particular attention was paid to the identification of risk factors and to the discussion of a broad spectrum of treatment methods. Both classical therapeutic approaches and the latest reports on biologic therapies and small-molecule inhibitors were taken into account. The adopted methodology enabled a multifaceted presentation of psoriasis, combining an analysis of etiopathogenesis with current standards of clinical management.

Pathogenesis and Risk Factors

The contemporary understanding of psoriasis pathogenesis, shaped by the last two decades of immunogenetic research, highlights the pivotal role of immunological cascades converging in adaptive immune pathways [5]. From a systemic perspective, the development of psoriatic plaques results from complex, multidirectional interactions between keratinocytes and immune cells. T-lymphocytes, myeloid and plasmacytoid dendritic cells, macrophages, NK and NK-T cells, as well as granulocytes, mast cells, and innate lymphoid cells (ILCs), play a significant role in this process. The disease mechanism is based on tight communication between innate and adaptive immunity and resident skin cells [6]. This results in the formation of a positive feedback loop that sustains inflammation, in which the interleukin-23/interleukin-17 axis plays a central role [5,6]. These mechanisms not only initiate the pathogenic process but are also responsible for maintaining the chronic inflammation typical of psoriasis. According to contemporary pathogenic models, environmental factors such as infections, mechanical trauma, or psychological stress are recognized to act as triggers, activating cells of both innate and adaptive immunity and consequently initiating a cascade of cytokine production [6].

Genetic Predisposition

Heredity plays a pivotal role in the pathogenesis of psoriasis, as evidenced by data indicating a significantly higher risk of the disease among first-degree relatives compared to the general population, as well as a higher concordance rate in monozygotic twins than in dizygotic twins [7]. Early studies on the genetic basis of psoriasis led to the identification of at least 12 specific genomic regions, designated as psoriasis-susceptibility (PSORS) loci. Their identification was primarily made possible through linkage analysis conducted in families with a high prevalence of the disease [8]. Genome-wide association studies (GWAS), based on the evaluation of millions of genetic markers (SNPs), have not only confirmed existing psoriasis susceptibility loci but also revealed novel risk regions located outside the MHC [7]. Among the known genetic determinants, the HLA-C*06:02 variant correlates most strongly with the occurrence of psoriasis, serving as its primary genetic susceptibility factor [9]. Located within the PSORS1 locus, this allele accounts for the largest share of disease risk, explaining 35% to 50% of the heritability attributed to the loci identified to date [10]. It is estimated to be present in approximately 40% of patients. Its presence is associated with an earlier onset of clinical symptoms and a more frequent positive family history compared to non-carriers [11]. Crucially, HLA-C*06:02 participates in the antigen presentation of autoantigens, such as ADAMTS-like 5 (ADAMTSL5) and LL-37, to CD8+ T-lymphocytes, which directly initiates the autoimmune cascade within the epidermis [9].

Environmental Triggering Factors

The loss of immunological tolerance is considered to be the result of a complex interplay between genetic predisposition and environmental factors [12].

Infections

The most well-established epidemiological association involves upper respiratory tract infections caused by group A beta-hemolytic streptococci, such as *Streptococcus pyogenes*, which can trigger or exacerbate cutaneous lesions in both plaque and guttate psoriasis. This mechanism is attributed to the phenomenon of molecular mimicry, arising from the structural homology between the streptococcal M protein and human keratin 17 (K17), which facilitates the activation of autoreactive T-lymphocytes [12]. Furthermore, the presence of the HLA-C*06:02 allele has been shown to correlate with increased susceptibility to chronic or recurrent streptococcal tonsillitis [13].

Psychological Stress

Evidence suggests that stress may serve as a precipitating factor for psoriatic lesions in 31–88% of patients. A higher incidence of the disease is also observed in individuals who experienced significant psychological stress within the 12-month period preceding symptom onset. This suggests that in genetically predisposed individuals, stress may initiate the pathogenic process [14].The pathophysiological basis of this phenomenon is linked to the activation of the hypothalamic-pituitary-adrenal (HPA) axis, leading to the secretion of corticotropin-releasing hormone (CRH) and neuropeptides, such as substance P (SP) and nerve growth factor (NGF). These mediators directly induce neurogenic inflammation within the skin [12].

Physical Trauma

Cutaneous trauma, such as burns, incisions, or infections, can trigger the development of psoriatic lesions in genetically predisposed individuals. It is hypothesized that this mechanism results from the synergistic effect of pro-inflammatory cytokines on keratinocytes, leading to their hyperproliferation at the site of injury [4].

Medications

A variety of medications have the potential to trigger psoriasis. Agents associated with this effect include lithium, beta-blockers, antimalarial drugs, and nonsteroidal anti-inflammatory drugs (NSAIDs). In addition, triggering potential has been reported for angiotensin-converting enzyme (ACE) inhibitors, interferon (IFN), imiquimod (IMQ), terbinafine, as well as statins and fibrates. This phenomenon has also been observed with anti-PD-1 and anti-PD-L1 antibodies. In rare cases, tumor necrosis factor (TNF) inhibitors may likewise induce psoriasis [12].

Lifestyle and Metabolic Factors

Weight gain and obesity increase the risk of psoriasis and may serve as precipitating factors for the disease [5]. A significant association has been established between tobacco smoking and psoriasis; notably, most studies indicate that this habit also correlates with increased disease severity. Smoking is further linked to an elevated risk of disease onset, with data suggesting a dose-response relationship [16]. Additionally, a positive correlation has been observed between alcohol consumption and psoriasis [17].

Epidemiology

The global prevalence of psoriasis is 4.4%. The highest rates are recorded in East Asia, reaching 5.7%, whereas the lowest are found in Africa, at 1.7%. In Europe, the prevalence reaches 4.6%. Studies also indicate that psoriasis more frequently affects individuals with fair skin compared to those with darker skin tones [43]. Although it is diagnosed at any age, including in very young children, it most commonly manifests between the ages of 15 and 35 [4]. An analysis of data from the National Health Fund (NFZ) for the period 2010–2023 provides key information on the dynamics of the disease in Poland. As of the end of 2023, there were 639,662 patients registered in the system, representing 1.70% of the general population [1]. Contrary to some global data indicating a male predominance [4], a higher prevalence was recorded among women (1.81%) than men (1.58%) in the Polish population. Importantly, in both groups, the peak prevalence occurs at the same stage of life, with the highest number of individuals affected by psoriasis recorded in the 55–59 age group [1].

Clinical Presentation and Disease Variants

Psoriasis is categorized into several clinical variants, including plaque, guttate, inverse, pustular, and erythrodermic types. Research focuses predominantly on plaque psoriasis (psoriasis vulgaris), which accounts for approximately 85–90% of the patient population [7]. A characteristic psoriatic plaque is sharply demarcated, reddish-brown in color, and overlaid with silvery-white micaceous scales. Predilection sites for these lesions include the extensor surfaces of the extremities primarily the elbows and knees as well as the lumbosacral region and the scalp [11]. Gentle scraping of the plaque surface induces the detachment of scales resembling candle wax, a phenomenon termed the stearic candle sign [19]. The subsequent mechanical removal of these adherent scales reveals punctate hemorrhages (pinpoint bleeding), clinically referred to as the Auspitz sign [5].

Guttate psoriasis manifests as numerous small papules appearing primarily on the trunk and proximal extremities. It often presents with an abrupt onset, typically following a streptococcal infection, particularly in children and young adults, and is generally associated with a more favorable prognosis [4]. Inverse psoriasis, also referred to as intertriginous or flexural psoriasis, typically presents as smooth, moist, erythematous plaques lacking the characteristic silvery scale. These lesions are localized within skin folds and areas subject to friction [19]. Pustular psoriasis is characterized by the presence of sterile pustules on an erythematous base [20]. It is categorized into generalized (GPP) and localized (PPP) forms, the latter of which includes acrodermatitis continua of Hallopeau as a subtype. GPP is a systemic inflammatory condition presenting with high fever, malaise, and elevated C-reactive protein (CRP) levels. The disease can lead to life-threatening complications, such as sepsis and cardiorespiratory failure; exacerbations are frequently triggered by stress, infections, pregnancy, and the abrupt withdrawal of systemic corticosteroids [21]. Erythrodermic psoriasis is a rare and severe variant, diagnosed in approximately 1–2% of patients. The clinical presentation is characterized by generalized erythema and extensive exfoliation involving 75% to 90% of the Total Body Surface Area (TBSA). Extensive cutaneous involvement predisposes patients to systemic symptoms, such as pruritus, fever, chills,

and dehydration [22]. Nail psoriasis affects the nail apparatus and may occur as an isolated form or in association with cutaneous lesions. It is estimated that nail involvement affects approximately half of all psoriasis patients. Characteristic clinical signs include pitting, onycholysis, the oil drop sign, and subungual hyperkeratosis. These changes are a crucial element in assessing disease severity, frequently coexist with psoriatic arthritis (PsA), and are considered a significant clinical indicator in these patients [23].

Diagnosis and Severity Assessment

The diagnosis of psoriasis is primarily based on clinical presentation, as no specific serological markers have yet been identified for monitoring the disease course. To ensure an objective evaluation of skin status and to monitor therapeutic progress, standardized assessment tools are commonly employed. Key metrics include the Psoriasis Area and Severity Index, Body Surface Area, Physician Global Assessment (PGA), and the Dermatology Life Quality Index [23]. The DLQI questionnaire is a particularly vital component of the diagnostic process, as it allows for a quantifiable assessment of the condition's impact on the patient's quality of life and psychological well-being [23, 24]. Although skin biopsy is not a component of routine diagnostics, it is warranted in cases of clinical uncertainty or atypical presentation [15]. Modern digital technologies, including artificial intelligence systems and Internet of Things (IoT) solutions, support the assessment and monitoring of patients with psoriasis, complementing traditional methods such as the PASI scale [46]. Deep learning algorithms enable high diagnostic accuracy exceeding 90% and facilitate therapy personalization as well as remote patient care [47]. However, their implementation is limited by issues such as data representativeness, privacy concerns, model transparency, difficulties with generalization, and high deployment costs [46,47]. Teledermatology represents a cost-effective model of remote care that eliminates geographical barriers to specialized dermatological diagnostics. However, in the case of clinical entities such as psoriasis, its diagnostic precision is sometimes contested due to the inability to perform palpation. A comprehensive clinical assessment requires the correlation of visual findings with a physical examination, which remains a component as vital as visual evaluation [42]. In accordance with the contemporary clinical approach, traditional thresholds based solely on PASI or BSA values are now considered insufficient [25]. Within modern therapeutic standards, significant indications for considering the initiation of systemic therapy, including biologics, include a PASI score >10 or Body Surface Area involvement >10% [26]. Furthermore, the presence of psoriatic lesions in high-impact areas (areas of special concern), such as the face, hands, feet, genitalia, scalp, or nails, may warrant systemic treatment regardless of the total percentage of body surface area involved [25]. An additional critical criterion is the impairment of quality of life, reflected by a DLQI score >10 [26], as well as an inadequate response to prior topical therapy [25]. An accurate diagnosis of psoriasis necessitates the exclusion of other clinical entities with a similar clinical presentation. The differential diagnosis should primarily include seborrheic dermatitis, pityriasis rosea, cutaneous T-cell lymphoma (CTCL), fungal infections, and lichen planus [4].

Treatment

Patient Education and Lifestyle Modification

Patient education regarding the chronic nature of psoriasis and the associated increased risk of metabolic syndrome and cardiovascular disease is of paramount importance. Non-pharmacological management primarily emphasizes smoking cessation, alcohol avoidance, and weight reduction. Furthermore, adhering to a diet enriched with antioxidants and omega-3 fatty acids may contribute to reducing the severity of skin lesions and mitigating the risk of systemic complications [27].

Topical Therapy and Skincare

The majority of patients with mild-to-moderate psoriasis are able to achieve adequate disease control using topical therapy or phototherapy alone [28]. Due to the limited clinical evidence guiding the selection of specific topical treatments, therapeutic decisions are often based on patient preference and the prescribing physician's experience. Daily application of fragrance-free emollients, particularly on the elbows, knees, and the sacral region, reduces scaling and alleviates discomfort associated with skin fissuring. This approach can be supplemented with keratolytic agents containing urea (approx. 10%) or salicylic acid (up to 6%). Higher concentrations of these substances (25% urea, >8% salicylic acid) may be effective for treating lesions on the palms and soles; however, they may cause pain when applied to irritated sites [29].

Topical Corticosteroids in Psoriasis Management

Topical corticosteroids (TCS) alleviate cutaneous inflammation through their potent anti-inflammatory, antiproliferative, and immunosuppressive properties [30]. Despite their high efficacy in reducing erythema and scaling, prolonged application may lead to tachyphylaxis and disease rebound upon discontinuation; therefore, short-term or intermittent therapy is recommended. Low-potency agents are indicated for sensitive areas, such as the face and genitalia, whereas high-potency corticosteroids are reserved for the treatment of thick psoriatic plaques, particularly on the scalp, elbows, knees, as well as the palms and soles [29].

Vitamin D_3 Analogues

Vitamin D_3 analogues, such as calcipotriol, also play a significant role by regulating keratinocyte proliferation and differentiation. Their mechanism is based on binding to the vitamin D receptor (VDR), which inhibits excessive proliferation and induces the terminal differentiation of keratinocytes [31]. Additionally, they exert potent immunomodulatory effects by suppressing the IL-23/IL-17 axis, which is central to the pathogenesis of the disease. Fixed-dose combinations containing calcipotriol and betamethasone dipropionate demonstrate superior clinical efficacy compared to either agent used as monotherapy [32].

Calcineurin Inhibitors

Topical calcineurin inhibitors (TCIs) play a pivotal role in the management of psoriasis affecting sensitive areas, such as the face and genitalia. They are widely regarded as a primary alternative to topical corticosteroids. The restricted use of TCS in these anatomical regions stems from the heightened risk of adverse effects, most notably skin atrophy [33].

Novel Non-Steroidal Topical Treatment Options

Novel non-steroidal topical treatment options for psoriasis include agents such as tapinarof, an aryl hydrocarbon receptor (AhR) agonist, and roflumilast, a phosphodiesterase-4 (PDE4) inhibitor. The mechanisms of action of these drugs involve the modulation of inflammatory pathways participating in psoriasis pathogenesis. Clinical trials have demonstrated the efficacy of both 1% tapinarof cream and 0.3% roflumilast cream in the treatment of mild-to-moderate plaque psoriasis, while maintaining a favorable safety profile with a low incidence of adverse events [34].

Phototherapy

The modern therapeutic paradigm for moderate-to-severe psoriasis relies on the extensive use of phototherapy, which, despite the rapid advancement of biologic therapies, remains an integral component of dermatological practice [35]. Currently, narrowband ultraviolet B (NB-UVB) radiation is the most frequently utilized phototherapy modality for plaque psoriasis, primarily in patients with stable disease. Furthermore, it has been shown that NB-UVB therapy may reduce treatment duration in other clinical variants, such as pustular or erythrodermic psoriasis [34]. The mechanism of action of NB-UVB is multidimensional, involving the suppression of the IL-23/Th17 axis, modulation of regulatory T-cell activity, and inhibition of signaling pathways such as JAK/STAT and NF-κB [36]. Additionally, UVB radiation can induce apoptosis in effector T cells and keratinocytes by generating DNA damage and activating the p53 suppressor gene [37]. Systematic analyses indicate high efficacy of NB-UVB therapy in patients with higher Fitzpatrick skin phototypes, in whom the disease often follows a more severe course and has a more pronounced impact on quality of life [36]. For localized lesions, targeted phototherapy is employed using an excimer lamp or laser emitting radiation at a wavelength of 308 nm. This method allows for the delivery of high energy doses directly to psoriatic plaques while limiting exposure to unaffected skin. In cases of insufficient response to NB-UVB, photochemotherapy (PUVA) is utilized, combining psoralen administration with UVA exposure. The efficacy of this method in achieving clinical clearance can reach 85–90%, with a mean remission duration of approximately six months. Furthermore, PUVA photochemotherapy in combination with retinoids (Re-PUVA) is indicated for the most severe forms of the disease, such as erythrodermic psoriasis or generalized pustular psoriasis [34].

Systemic Therapy

Conventional oral agents with a broad spectrum of immunomodulatory effects remain a cornerstone in the management of moderate-to-severe psoriasis, particularly in cases of limited access to biologic therapies [11, 4].

Conventional Systemic Immunomodulators

Methotrexate (MTX) acts as a dihydrofolate reductase inhibitor that also suppresses the JAK-STAT pathway and modulates T-cell function. PASI 75 response is achieved in approximately 40% of patients at 12 weeks. Its clinical use necessitates folic acid supplementation and regular monitoring for hepatic fibrosis. Cyclosporine, a calcineurin inhibitor, suppresses IL-2 expression and is primarily indicated for rapid clinical improvement or in unstable variants, such as pustular and erythrodermic psoriasis. Due to the cumulative risks

of hypertension and nephrotoxicity, treatment duration should generally be limited to two years. Acitretin is a systemic retinoid that normalizes keratinocyte differentiation via the MAPK, JAK-STAT, and NF-κB signaling pathways [11]. It is particularly indicated for patients with severe psoriasis, including pustular, erythrodermic, and HIV-associated forms. Given its slow onset of action, optimal therapeutic outcomes are typically observed after three to six months. While non-immunosuppressive, it requires baseline and periodic monitoring of complete blood count (CBC), lipid profiles, and liver and renal function tests. Due to its potent teratogenicity, effective contraception must be maintained for at least three years following treatment cessation [4].

Modern Targeted Therapies

Apremilast is a selective phosphodiesterase 4 (PDE4) inhibitor that increases intracellular cyclic adenosine monophosphate (cAMP) levels, thereby inhibiting the production of pro-inflammatory cytokines, including TNF-α, IL-17, and IL-23 [11]. It is characterized by a favorable safety profile and does not require routine laboratory monitoring, making it a suitable option for patients with multiple comorbidities [34]. A breakthrough in oral treatment is deucravacitinib, a first-in-class allosteric tyrosine kinase 2 (TYK2) inhibitor. Unlike conventional JAK inhibitors, deucravacitinib binds to the regulatory (pseudokinase) domain rather than the catalytic domain of the enzyme, ensuring high selectivity and minimizing the risk of adverse events typical of pan-JAK inhibitors [11,34]. Phase III clinical trials have demonstrated the superiority of deucravacitinib over apremilast in achieving PASI 75 and PASI 90 responses [11].

Biologic Therapy

The introduction of biologic agents precisely targeting key nodes of the immune cascade has transformed the management of moderate-to-severe psoriasis. Their exceptional efficacy has considerably elevated therapeutic expectations, shifting the primary treatment goal from achieving a 50% to up to a 90% reduction in the Psoriasis Area and Severity Index [38].

The characteristics of individual classes of biological drugs are summarized in Table 1.

Table 1. Overview of biologic therapies for psoriasis [38,39].

Drug Class	Representative Agents	Mechanism of Action	Clinical Considerations
TNF Inhibitors (TNFi)	Etanercept, infliximab, adalimumab, certolizumab pegol	Inhibit TNF, which activates myeloid dendritic cells, promoting the differentiation of Th1, Th17, and Th22 cells; they participate in a feed-forward inflammatory loop.	Infliximab demonstrates the highest efficacy within the TNFi class but is associated with a higher risk of adverse events.
IL-17 Inhibitors (IL-17i)	Secukinumab, ixekizumab, brodalumab, bimekizumab	Inhibit the IL-17 cytokine, which promotes keratinocyte proliferation, angiogenesis, and immune cell infiltration.	Bimekizumab and ixekizumab belong to the highest efficacy tier. Their use is associated with a risk of inflammatory bowel disease exacerbation.
IL-12/23 Inhibitors (IL-12/23i)	Ustekinumab	Target the p40 subunit, which is shared by the cytokines IL-12 and IL-23.	These agents exhibit high long-term drug survival; however, safety signals regarding major adverse cardiovascular events (MACE) have been reported.
IL-23 Inhibitors (IL-23i)	Guselkumab, risankizumab, tildrakizumab	Selectively target the p19 subunit of the IL-23 cytokine.	Risankizumab and guselkumab currently demonstrate the highest overall drug survival rates.

Modern therapeutic strategies increasingly incorporate the concept of early therapeutic intervention, suggesting that the timely initiation of biologic therapy may permanently modify the disease course. This mechanism is linked, among others, to the inhibition of tissue-resident memory T cells (TRM) formation, which are responsible for lesion recurrence at previously affected sites [40]. The pursuit of restoring immune homeostasis and personalizing therapy including dosage optimization through therapeutic drug monitoring (TDM) are pivotal elements of contemporary dermatological care. These approaches increase the likelihood of achieving long-term remission and, potentially in the future, sustained disease control [38,40]. The efficacy

of biologic treatment is highly dependent on both the specific agent and patient-related factors. Obesity serves as a negative predictor of clinical response for guselkumab and ixekizumab. Male sex reduces the probability of therapeutic success exclusively in the case of secukinumab, whereas a positive family history of psoriasis is associated with improved outcomes for secukinumab and ixekizumab. Conversely, the presence of psoriatic arthritis impairs the response to ustekinumab, and prior exposure to biologic therapy diminishes the efficacy of guselkumab [41].

Conclusions

In summary, the contemporary understanding of the pathogenesis of psoriasis focuses on the interleukin-23/interleukin-17 axis and the significant role of genetic factors, including the HLA-C*06:02 variant. Advances in therapy, particularly the introduction of biologic drugs and new targeted molecules, have significantly improved treatment efficacy, allowing for the achievement of high levels of clinical response. Nevertheless, important limitations persist, such as the lack of reliable serological markers and the insufficient ability to predict response to treatment. In clinical practice, a holistic approach is key, considering both the severity of skin lesions and their impact on the quality of life, as well as the presence of modifiable factors. Further research should focus on identifying predictive biomarkers and implementing tools based on artificial intelligence, which may enable more effective personalization of therapy.

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All authors contributed to the article.

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