MEDWIN PUBLISHERS
Committed to Create Value for Researchers

Clinical Dermatology Open Access Journal
ISSN: 2574-7800

Recent Advances in the Pathogenesis and Management of Psoriasis: A Mini Review

Connie Catherine* and Andrea Leslie

Department of Dermatology, Harvard Medical School, USA

***Corresponding author:** Connie Catherine, Department of Dermatology, Harvard Medical School, USA.

Mini Review

Volume 11 Issue 2

Received Date: May 06, 2026

Published Date: May 25, 2026

Abstract

Psoriasis is a chronic, immune-mediated inflammatory skin disorder that affects approximately 2–3% of the global population and significantly impairs patients' quality of life. The disease is characterized by erythematous, well-demarcated plaques covered with silvery scales and is associated with systemic comorbidities, including psoriatic arthritis, cardiovascular disease, metabolic syndrome, and psychological disorders. Over the past decade, substantial advances have been made in understanding the molecular and immunological mechanisms underlying psoriasis, particularly the central role of the interleukin (IL)-23/IL-17 axis and tumor necrosis factor-alpha (TNF-α) in disease pathogenesis. These discoveries have facilitated the development of targeted biologic therapies and novel small-molecule agents, resulting in improved clinical outcomes and enhanced patient safety. Despite these therapeutic advances, challenges remain in achieving sustained disease control, preventing relapse, and implementing personalized treatment strategies. This mini review summarizes current insights into the pathogenesis of psoriasis, highlights recent developments in therapeutic management, and discusses future directions that may further optimize patient care and improve long-term clinical outcomes.

Keywords: Psoriasis; Pathogenesis; IL-17; IL-23; Biologic therapy; TYK2 inhibitors; Precision medicine; Dermatology

Introduction

Psoriasis is a chronic, recurrent, immune-mediated inflammatory disorder that primarily affects the skin but also has significant systemic implications [1,2]. It is estimated to affect approximately 2–3% of the world's population, making it one of the most common chronic dermatological diseases [1,2]. Although psoriasis can develop at any age, it most frequently presents between the second and fourth decades of life and affects both males and females equally [1]. Clinically, the disease is characterized by sharply demarcated erythematous plaques covered with silvery-white scales, commonly involving the scalp, elbows, knees, lumbosacral region, and extensor surfaces [1,2]. The clinical presentation may vary considerably depending on disease subtype, severity, and individual patient characteristics.

The pathogenesis of psoriasis is multifactorial and results from a complex interaction between genetic susceptibility, environmental triggers, and immune dysregulation [2,3]. Genome-wide association studies have identified numerous susceptibility loci, including the human leukocyte antigen (HLA)-C*06:02 allele, which is strongly associated with early-onset psoriasis [2,3]. Environmental factors such as infections, psychological stress, obesity, smoking, alcohol consumption, trauma to the skin (Koebner phenomenon), and certain medications may initiate or exacerbate disease activity in genetically predisposed individuals [1-3]. Current evidence indicates that dysregulation of the innate and adaptive immune systems leads to excessive production of pro-inflammatory cytokines, particularly TNF-α, IL-17, and IL-23, which drive chronic inflammation and abnormal keratinocyte proliferation [2-4].

CC BY

Beyond its cutaneous manifestations, psoriasis is increasingly recognized as a systemic inflammatory disease associated with multiple comorbidities, including psoriatic arthritis, cardiovascular disease, metabolic syndrome, obesity, diabetes mellitus, inflammatory bowel disease, and depression [1,2,5]. These associated conditions contribute substantially to disease burden and emphasize the importance of comprehensive patient assessment and multidisciplinary management. In recent years, remarkable advances in molecular biology have transformed the understanding of psoriasis pathogenesis, leading to the development of highly targeted biologic therapies and novel small-molecule drugs that have significantly improved treatment outcomes [2,4,6]. This mini review summarizes the current understanding of psoriasis pathogenesis and highlights recent therapeutic advances that continue to shape modern clinical practice

Pathogenesis of Psoriasis

Psoriasis is a multifactorial, chronic inflammatory skin disease that develops through a complex interplay of genetic susceptibility, environmental triggers, and immune dysregulation. Although the precise cause of psoriasis remains incompletely understood, substantial progress over the past two decades has revealed that the disease is primarily driven by an aberrant immune response that promotes persistent inflammation and excessive proliferation of keratinocytes. This evolving understanding has transformed psoriasis from being considered solely a disorder of epidermal hyperproliferation to a systemic immune-mediated disease involving both innate and adaptive immune pathways [2,3].

Genetic predisposition plays a fundamental role in the development of psoriasis. Family and twin studies have demonstrated a strong hereditary component, with numerous susceptibility loci identified through genome-wide association studies. Among these, the human leukocyte antigen (HLA)-C*06:02 allele, located within the psoriasis susceptibility locus (PSORS1), is recognized as the strongest genetic risk factor, particularly for early-onset plaque psoriasis. Additional genetic variants affecting cytokine signaling, antigen presentation, and immune regulation further contribute to disease susceptibility and influence clinical presentation [2,3].

Environmental factors frequently act as triggers in genetically predisposed individuals. Common precipitating factors include streptococcal infections, psychological stress, obesity, smoking, excessive alcohol consumption, skin trauma (Koebner phenomenon), and certain medications such as lithium, beta-blockers, antimalarial agents, and interferons. These factors activate innate immune responses within the skin, leading to the release of inflammatory mediators and the recruitment of immune cells that initiate disease progression [1–3].

The immunopathogenesis of psoriasis involves complex interactions among keratinocytes, dendritic cells, macrophages, neutrophils, and T lymphocytes. Activated dendritic cells produce cytokines such as interleukin (IL)-12 and IL-23, which promote the differentiation and activation of T-helper (Th) cells. IL-12 primarily stimulates Th1 cells, whereas IL-23 is essential for the expansion and maintenance of Th17 cells. The IL-23/Th17 pathway is now recognized as the central immunological mechanism responsible for sustaining chronic inflammation in psoriasis (Figure 1).

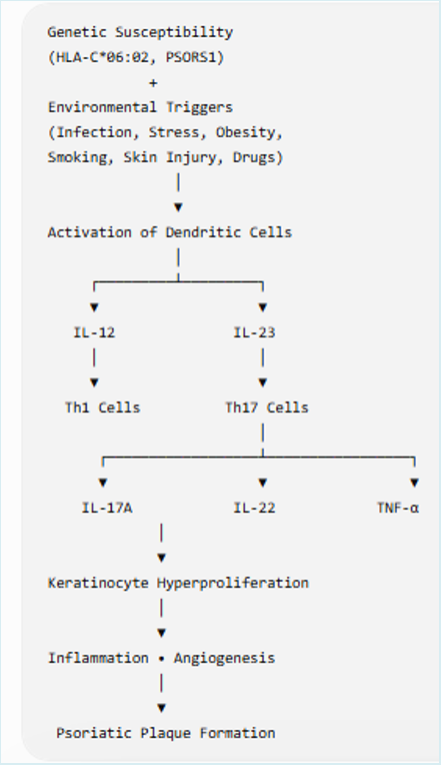


Figure 1: Immunopathogenesis of Psoriasis.

Activated Th17 cells secrete several pro-inflammatory cytokines, including IL-17A, IL-17F, IL-22, and IL-21, which stimulate keratinocyte proliferation and amplify inflammatory responses. Keratinocytes, once considered passive targets of inflammation, actively participate in disease pathogenesis by producing cytokines, chemokines, and antimicrobial peptides that further recruit immune cells into the skin. This continuous interaction between immune cells and keratinocytes creates a self-perpetuating inflammatory cycle responsible for the persistent erythematous and scaly plaques characteristic of psoriasis [2,4,7].

Another key mediator is tumor necrosis factor-alpha (TNF-α), which acts synergistically with IL-17 and IL-23

Copyright© Connie Catherine and Andrea Leslie.

to enhance inflammatory signaling, endothelial activation, leukocyte migration, and epidermal hyperplasia. The identification of these cytokine pathways has revolutionized psoriasis management by providing highly specific therapeutic targets for biologic agents that interrupt disease progression [1,2,6].

Recent research has also highlighted the contribution of the skin microbiome, epigenetic regulation, oxidative stress, and metabolic inflammation to psoriasis pathogenesis. Alterations in microbial diversity may influence immune activation, whereas epigenetic modifications such as DNA methylation and microRNA dysregulation affect the expression of genes involved in inflammation and keratinocyte differentiation. In addition, obesity and metabolic syndrome contribute to chronic low-grade systemic inflammation through adipokine production, potentially exacerbating disease severity and reducing treatment response [2,8,9].

Collectively, these findings demonstrate that psoriasis is a complex systemic inflammatory disorder rather than a disease confined to the skin. A deeper understanding of its molecular and immunological mechanisms has not only improved knowledge of disease pathogenesis but has also paved the way for precision medicine and the development of highly targeted therapeutic strategies aimed at achieving sustained disease control and improving patient outcomes (Table 1).

Cytokine	Source	Biological Function	Clinical Significance
IL-23	Dendritic cells	Activates and maintains Th17 cells	Central driver of psoriasis
IL-17A	Th17 cells	Stimulates keratinocyte proliferation	Major therapeutic target
IL-22	Th17 cells	Epidermal hyperplasia	Thickened plaques
TNF-α	Macrophages, T cells	Amplifies inflammation	Target of biologic therapy
IL-12	Dendritic cells	Activates Th1 cells	Contributes to immune activation

Table 1: Major Cytokines and Their Roles in Psoriasis

Recent Advances in Management

The management of psoriasis has evolved remarkably over the past decade, driven by a deeper understanding of its immunopathogenesis and the development of targeted therapeutic strategies. The primary goals of treatment are to reduce inflammation, control disease activity, relieve symptoms, prevent relapses, improve quality of life, and minimize long-term complications. Treatment selection is individualized based on disease severity, extent of skin involvement, presence of psoriatic arthritis, associated comorbidities, previous treatment response, and patient preferences [1,6].

For patients with mild psoriasis, topical therapies remain the first-line treatment. Corticosteroids, vitamin D analogs such as calcipotriol, topical retinoids, salicylic acid, and calcineurin inhibitors are widely used to reduce inflammation, normalize keratinocyte proliferation, and alleviate scaling and pruritus. Fixed-dose combinations of corticosteroids and vitamin D analogs have demonstrated improved efficacy, better patient adherence, and reduced adverse effects compared with monotherapy. Appropriate patient education regarding the correct application of topical medications is essential to maximize therapeutic outcomes [6,10].

Phototherapy continues to be an effective option for patients with moderate psoriasis or those who do not achieve adequate disease control with topical agents. Narrowband ultraviolet B (NB-UVB) phototherapy remains the preferred modality because of its favorable efficacy and safety profile. Psoralen plus ultraviolet A (PUVA) therapy is also effective in selected patients but is used less frequently because of its association with long-term adverse effects, including photoaging and an increased risk of skin malignancies following prolonged exposure [6,10].

Conventional systemic therapies remain valuable treatment options, particularly in resource-limited settings. Methotrexate, cyclosporine, acitretin, and apremilast continue to play important roles in managing moderate-to-severe psoriasis. Methotrexate possesses anti-inflammatory and immunomodulatory properties and is particularly beneficial in patients with concomitant psoriatic arthritis. Cyclosporine provides rapid disease control in severe or unstable psoriasis but requires careful monitoring because of potential nephrotoxicity and hypertension. Acitretin is especially useful for pustular and erythrodermic psoriasis, whereas apremilast, an oral phosphodiesterase-4 (PDE-4) inhibitor, offers a convenient therapeutic option with a favorable safety profile for selected patients [6,11].

One of the most significant advances in psoriasis management has been the introduction of biologic therapies that selectively target key inflammatory cytokines involved in disease pathogenesis. Tumor necrosis factor-alpha (TNF-α) inhibitors, including infliximab, adalimumab, etanercept,

Copyright© Connie Catherine and Andrea Leslie.

and certolizumab pegol, were among the first biologic agents to demonstrate substantial clinical benefit. More recently, biologics targeting the IL-17 and IL-23 pathways have shown superior efficacy, rapid onset of action, and prolonged disease control. IL-17 inhibitors such as secukinumab, ixekizumab, and brodalumab effectively suppress keratinocyte activation and inflammatory signaling, while IL-23 inhibitors, including guselkumab, risankizumab, and tildrakizumab, provide sustained clinical responses with less frequent dosing schedules. These targeted therapies have transformed the management of moderate-to-severe psoriasis by enabling many patients to achieve near-complete or complete skin clearance (Figure 2).

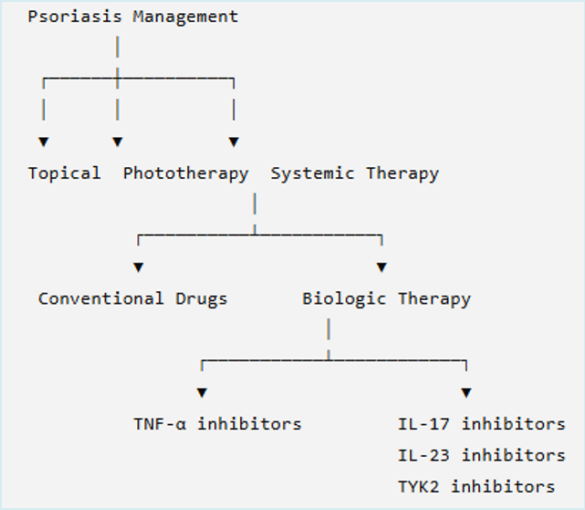


Figure 2: Current Therapeutic Strategies for Psoriasis.

In addition to biologic agents, the emergence of novel oral small-molecule therapies has expanded the therapeutic landscape. Tyrosine kinase 2 (TYK2) inhibitors represent a promising advancement because they selectively inhibit intracellular signaling pathways involved in cytokine-mediated inflammation. Deucravacitinib, the first approved TYK2 inhibitor for plaque psoriasis, has demonstrated superior efficacy compared with placebo and apremilast in clinical trials while maintaining a favorable safety profile. Ongoing research is evaluating additional TYK2 inhibitors and other targeted oral therapies that may further improve treatment options [2,4,6,12].

Contemporary psoriasis management also emphasizes the importance of holistic and multidisciplinary care. Patients should be routinely assessed for cardiovascular disease, obesity, diabetes mellitus, hypertension, dyslipidemia, inflammatory bowel disease, depression, anxiety, and psoriatic arthritis, as these comorbidities substantially influence disease burden and therapeutic decision-making. Lifestyle modifications, including weight management, smoking cessation, regular physical activity, stress reduction, and moderation of alcohol consumption, complement pharmacological therapy and may improve both treatment response and overall health [12,13].

Recent advances in digital health are further enhancing psoriasis management. Teledermatology has improved access to specialist care, particularly for patients in remote or underserved areas, while artificial intelligence (AI)-based image analysis and machine learning algorithms are being investigated for disease detection, severity assessment, and treatment monitoring. These technologies have the potential to support clinical decision-making, facilitate early intervention, and optimize long-term disease management [1,2,5,14,15].

Overall, the integration of targeted biologic therapies, innovative oral agents, comprehensive patient assessment, and emerging digital technologies has significantly improved the management of psoriasis. Continued advances in translational research and personalized medicine are expected to further refine therapeutic strategies, allowing clinicians to achieve durable disease control while minimizing treatment-related adverse effects and improving patients' quality of life (Table 2) [6,12,15].

Therapy	Target	Examples	Clinical Benefit
Topical corticosteroids	Inflammation	Clobetasol	Mild disease
Vitamin D analogues	Keratinocyte proliferation	Calcipotriol	Plaque reduction
Methotrexate	T-cell proliferation	Methotrexate	Moderate-to-severe psoriasis
Cyclosporine	Calcineurin pathway	Cyclosporine	Rapid disease control
TNF-α inhibitors	TNF-α	Adalimumab, Etanercept	High efficacy
IL-17 inhibitors	IL-17	Secukinumab, Ixekizumab	Rapid skin clearance
IL-23 inhibitors	IL-23	Guselkumab, Risankizumab	Long-term remission
TYK2 inhibitor	TYK2	Deucravacitinib	Oral targeted therapy

Table 2: Recent Therapeutic Advances in Psoriasis.

Copyright© Connie Catherine and Andrea Leslie.

Future Perspectives

Despite remarkable progress in understanding and treating psoriasis, several challenges remain in achieving sustained disease remission and individualized patient care. Future research should focus on identifying reliable biomarkers that can predict disease progression, treatment response, and the risk of relapse. Such biomarkers would enable clinicians to select the most appropriate therapy for each patient, thereby improving treatment outcomes while minimizing unnecessary drug exposure.

Advances in precision medicine are expected to further transform psoriasis management by integrating genetic, immunological, and clinical data to develop personalized therapeutic strategies. The continued development of highly selective biologic agents and novel oral small-molecule therapies, including next-generation TYK2 inhibitors, may provide safer, more effective, and longer-lasting disease control. Furthermore, combination therapies targeting multiple inflammatory pathways may offer additional benefits for patients with refractory or severe disease.

Emerging technologies such as artificial intelligence (AI), machine learning, and digital dermatology are likely to play an increasingly important role in psoriasis care. AI-assisted image analysis, teledermatology, and wearable digital health technologies may facilitate early diagnosis, objective disease severity assessment, remote monitoring, and personalized treatment recommendations. In parallel, greater emphasis on multidisciplinary collaboration among dermatologists, rheumatologists, cardiologists, psychologists, and primary care physicians will be essential for addressing the systemic nature of psoriasis and its associated comorbidities. Continued translational research will further enhance our understanding of disease mechanisms and contribute to the development of innovative therapeutic approaches that improve long-term patient outcomes.

Conclusion

Psoriasis is a complex, chronic immune-mediated inflammatory disease that extends beyond the skin and is increasingly recognized as a systemic disorder associated with multiple comorbidities. Significant advances in the understanding of its immunopathogenesis, particularly the pivotal roles of the IL-23/IL-17 axis and TNF-α, have revolutionized therapeutic strategies and led to the development of highly effective targeted biologic agents and novel oral therapies. These advances have substantially improved disease control, patient quality of life, and long-term clinical outcomes. Nevertheless, challenges such as treatment resistance, disease recurrence, and variability in therapeutic response continue to exist. Future progress in precision medicine, biomarker discovery, and artificial intelligence-assisted clinical care is expected to further optimize individualized treatment strategies. A comprehensive, patient-centered, and multidisciplinary approach remains fundamental to achieving sustained disease control and improving the overall well-being of individuals living with psoriasis.

References

1. Armstrong AW, Read C (2020) Pathophysiology, Clinical Presentation, and Treatment of Psoriasis: A Review. JAMA 323(19): 1945-1960.

2. Griffiths CEM, Armstrong AW, Gudjonsson JE, Barker JNWN (2021) Psoriasis. Lancet 397(10281): 1301-1315.

3. Nast A, Smith C, Spuls PI (2024) EuroGuiDerm Guideline on the Systemic Treatment of Psoriasis Vulgaris – Update 2024. J Eur Acad Dermatol Venereol.

4. Elmets CA, Leonardi CL, Davis DMR (2020) Joint American Academy of Dermatology–National Psoriasis Foundation Guidelines of Care for the Management of Psoriasis. J Am Acad Dermatol.

5. Menter A, Strober BE, Kaplan DH (2019) Joint AAD–NPF Guidelines of Care for the Management and Treatment of Psoriasis. J Am Acad Dermatol.

6. Warren RB, Blauvelt A, Bagel J (2023) Advances in Biologic Therapy for Moderate-to-Severe Psoriasis. J Eur Acad Dermatol Venereol.

7. Armstrong AW, Gooderham M, Warren RB (2022) Deucravacitinib in Moderate-to-Severe Plaque Psoriasis: Clinical Efficacy and Safety. N Engl J Med.

8. Gisondi P, Bellinato F, Targher G (2023) Comorbidities Associated with Psoriasis: Current Evidence and Clinical Implications. Clin Rev Allergy Immunol.

9. Boehncke WH, Schon MP (2025) Psoriasis. Lancet.

10. Rendon A, Schakel K (2023) Psoriasis Pathogenesis and Emerging Therapeutic Targets. Clin Cosmet Investig Dermatol.

11. Lowes MA, Suarez-Farinas M, Krueger JG (2024) Immunology of Psoriasis. Annu Rev Immunol.

12. Lebwohl MG, Mehta NN (2024) Psoriasis and Cardiometabolic Disease: Recent Advances and Future Directions. J Am Acad Dermatol.

13. Gordon KB, Strober B, Lebwohl M (2024) Long-Term Safety and Effectiveness of IL-23 Inhibitors in Plaque

Copyright© Connie Catherine and Andrea Leslie.

Psoriasis. Br J Dermatol.

14. Blauvelt A, Papp KA, Griffiths CEM (2025) Precision Medicine and Future Therapeutic Strategies in Psoriasis. Nat Rev Rheumatol.

15. (2016) Global Report on Psoriasis. World Health Organization.

Copyright© Connie Catherine and Andrea Leslie.