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Psoriasis: A Comprehensive Review of Pathophysiology, Clinical Manifestations, and Therapeutic Advancements

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Abstract: *Psoriasis is a chronic, immune-mediated inflammatory dermatosis characterized by keratinocyte hyperproliferation and altered differentiation. Affecting approximately 2–3% of the global population, it is no longer viewed solely as a skin condition but as a systemic inflammatory disease with significant comorbidities, including psoriatic arthritis, cardiovascular disease, and metabolic syndrome. This review explores the complex interplay between genetic predisposition and environmental triggers, emphasizing the central role of the IL-23/Th17 signaling axis in disease pathogenesis. We further examine clinical subtypes, current diagnostic challenges, and the evolution of treatment modalities from traditional topicals to highly targeted biologic therapies.*

Keywords: Psoriasis, IL-23/IL-17 Axis, Biologics, Keratinocyte Hyperproliferation, Systemic Inflammation, Psoriatic Arthritis

I. INTRODUCTION

Psoriasis is a non-contagious, inflammatory dermatosis that has challenged the medical community for centuries. Once erroneously grouped with leprosy, it is now understood as a sophisticated immunological disorder. The clinical hallmark of the disease is the "psoriatic plaque"—a well-demarcated, erythematous area covered with silvery, micaceous scales. These lesions are the result of a massive acceleration in the skin cell life cycle. While healthy skin cells mature and shed over nearly a month, psoriatic keratinocytes reach the surface in just a few days, leading to the accumulation of immature cells (parakeratosis) and visible scaling.

The Genetic Landscape

The predisposition to psoriasis is heavily hereditary. Research into the human genome has identified the *PSORS1* locus on chromosome 6p21 as the most significant genetic determinant, specifically the HLA-Cw6 allele. Individuals carrying this allele are more likely to develop Type I psoriasis, which appears before age 40 and often presents with more severe skin involvement. Other genetic factors involve mutations in the *IL23R* gene and genes regulating the NF-\kappaB pathway, suggesting that the body's innate ability to regulate inflammation is fundamentally compromised in these patients.

Pathophysiological Mechanisms

The transition from healthy skin to a psoriatic lesion is often triggered by an external stressor—trauma (the Koebner phenomenon), infection (notably Streptococcal pharyngitis), or even psychological stress. This stress causes keratinocytes to release self-DNA and antimicrobial peptides like LL-37. These molecules act as "danger signals," activating plasmacytoid dendritic cells (pDCs).

The activated pDCs secrete Interferon-alpha, which stimulates myeloid dendritic cells to produce Interleukin-23 (IL-23). IL-23 is the "master switch" that promotes the survival and expansion of Th17 and Th22 cells. These T-cells, in turn, produce IL-17A, IL-17F, and IL-22. These cytokines act directly on the epidermis, causing the rapid proliferation of keratinocytes and the recruitment of neutrophils, which form Munro's microabscesses within the stratum corneum.

The Systemic "Psoriatic March"

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Modern dermatology views psoriasis through the lens of systemic inflammation. The inflammatory cytokines produced in the skin do not remain localized; they enter the systemic circulation, contributing to insulin resistance, endothelial dysfunction, and atherosclerosis. This "psoriatic march" explains why patients with severe psoriasis have a significantly higher risk of cardiovascular events, such as myocardial infarction and stroke, compared to the general population. Metabolic syndrome, characterized by obesity, hypertension, and dyslipidemia, is also frequently comorbid.

Clinical Phenotypes and Diagnosis

While Chronic Plaque Psoriasis (*Psoriasis Vulgaris*) accounts for nearly 90% of cases, other variants present unique challenges:

Guttate Psoriasis: Small, drop-like lesions often following a viral or bacterial infection.

Inverse Psoriasis: Found in skin folds; lacks the typical scale due to moisture and friction.

Pustular Psoriasis: A potentially life-threatening variant characterized by sterile white pustules.

Erythrodermic Psoriasis: Involving over 90% of the body surface, leading to severe thermoregulation and fluid balance issues.

The diagnosis is primarily clinical, often utilizing the "Auspitz sign"—where peeling back a scale reveals pinpoint bleeding points due to the close proximity of dilated dermal capillaries to the surface.

Psoriasis is a multifaceted, lifelong inflammatory disease that presents a significant challenge to global public health. While its most visible manifestations occur on the skin, modern medical science recognizes psoriasis as a systemic condition driven by an overactive immune system. The disease is characterized by the rapid turnover of skin cells; where normal skin cells mature and shed in approximately 28–30 days, psoriatic skin cells complete this process in only 3–5 days. This acceleration leads to the characteristic buildup of silvery-white scales (micaceous scales) on an erythematous base.

Historical Context and Evolution of Understanding

Historically, psoriasis was often confused with leprosy or other contagious infections, leading to social ostracization of patients. It was only in the mid-19th century that Ferdinand von Hebra clearly distinguished psoriasis from leprosy. Throughout the 20th century, the focus shifted from a primary epidermal "skin-only" disorder to a T-cell-mediated autoimmune condition. Today, we understand that the cross-talk between the innate and adaptive immune systems, specifically involving dendritic cells, T-lymphocytes, and keratinocytes, creates a "feed-forward" loop of inflammation that sustains the disease.

Epidemiology and Global Burden

The prevalence of psoriasis varies significantly across the globe. It is most common in northern European populations and those of European descent, with prevalence rates reaching 3% to 4.5% in countries like Norway and the United States. Conversely, it is less common in Asian and African populations, and nearly absent in certain indigenous South American groups. The disease exhibits a bimodal age of onset: Type I (early-onset), which peaks between ages 20–30 and is often associated with a strong family history and HLA-Cw6 genetic linkage, and Type II (late-onset), which peaks between ages 50–60.

The Pathophysiological Framework

The hallmark of psoriasis is the "psoriatic plaque." Under the microscope, these plaques show striking features:

* Acanthosis: Thickening of the epidermis.
* Parakeratosis: Retention of nuclei in the stratum corneum, indicating incomplete maturation.
* Munro's Microabscesses: Accumulations of neutrophils within the skin layers.

The immunological trigger often begins when stressed keratinocytes release self-DNA/RNA. These form complexes with antimicrobial peptides (like LL-37), which activate plasmacytoid dendritic cells (pDCs). These cells then produce Interferon-alpha, pushing myeloid dendritic cells to migrate to lymph nodes and stimulate the differentiation of Th17 and Th1 cells. The subsequent release of Interleukin-17 (IL-17) and Tumor Necrosis Factor (TNF-alpha) acts directly

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on keratinocytes, causing them to proliferate and produce even more inflammatory cytokines, thus perpetuating the cycle.

Psychosocial and Systemic Impact

The "heartbreak of psoriasis" is not just a metaphor. Patients often suffer from profound psychological distress, including anxiety, depression, and social withdrawal. Furthermore, the systemic nature of the inflammation means that patients with severe psoriasis have a higher risk of "the psoriatic march"—a progression from skin inflammation to systemic vascular inflammation, leading to an increased risk of myocardial infarction and stroke.

Psoriasis is a chronic, non-contagious, immune-mediated inflammatory disease that primarily affects the skin and joints but carries significant systemic implications. For decades, psoriasis was viewed narrowly as a primary disorder of epidermal keratinocytes. However, revolutionary advancements in molecular biology and immunology have redefined it as a complex, multi-organ inflammatory syndrome. Characterized by the rapid overproduction of skin cells, the disease manifests as well-demarcated, erythematous plaques covered with silvery scales. Beyond the visible cutaneous lesions, psoriasis is deeply intertwined with systemic comorbidities, including psoriatic arthritis, cardiovascular disease, and metabolic syndrome, making it a profound challenge for both patients and healthcare systems worldwide.

Historical Evolution of Psoriasis

The history of psoriasis is marked by centuries of social stigma and medical misunderstanding. In ancient times, psoriasis was frequently confused with leprosy (*tsaraat* in biblical texts), leading to the tragic ostracization of patients. It was Ferdinand von Hebra, the founder of the Vienna School of Dermatology, who in 1841 officially separated psoriasis from leprosy and provided the first clinical description that mirrors our modern understanding.

During the early 20th century, researchers focused on the "hyperkeratosis" aspect—the thickening of the skin—viewing it as a structural skin defect. By the 1970s and 80s, the focus shifted toward the immune system when clinicians observed that immunosuppressive drugs like cyclosporine, used for organ transplants, led to the dramatic clearance of psoriatic plaques. This serendipitous discovery confirmed that psoriasis was not just a skin "growth" problem but an "immune" problem.

Epidemiology and Global Impact

Psoriasis affects approximately 2% to 3% of the global population, which translates to over 125 million individuals. However, its distribution is not uniform. The highest prevalence is observed in northern European countries (reaching up to 8% in some Norwegian populations) and North America. Conversely, it is less common in East Asian and African populations and is virtually non-existent in certain indigenous South American groups.

The disease typically follows a bimodal distribution in age of onset. **Type I Psoriasis** (early onset) appears before age 40, usually peaking between 20 and 30 years. This form is strongly associated with a positive family history and the presence of the HLA-Cw6 genetic marker. **Type II Psoriasis** (late onset) occurs after age 40, typically peaking between 50 and 60 years; it is generally milder and less dependent on genetic predisposition.

The Pathophysiological Framework: The IL-23/IL-17 Axis

To understand the 2000-word scope of psoriatic pathology, one must examine the "cross-talk" between the innate and adaptive immune systems. The process begins when a genetically predisposed individual encounters an environmental trigger. This trigger causes keratinocytes (the primary skin cells) to undergo stress and release self-nucleic acids.

These nucleic acids form complexes with antimicrobial peptides (such as LL-37), which act as "danger signals." These complexes activate **Plasmacytoid Dendritic Cells (pDCs)**. The activated pDCs release Interferon-alpha, which stimulates **Myeloid Dendritic Cells**. These cells then migrate to the local lymph nodes and secrete **Interleukin-23 (IL-23)**.

IL-23 is often referred to as the "master regulator." It drives the differentiation and survival of **Th17 cells**. These T-cells then travel back to the skin and release a cascade of pro-inflammatory cytokines, most notably **IL-17A, IL-17F, and IL-22**. These cytokines bind to receptors on keratinocytes, triggering a "feed-forward" loop. The keratinocytes respond

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by proliferating wildly (acanthosis) and failing to mature properly (parakeratosis), while also producing more chemokines that attract even more immune cells—specifically neutrophils—into the skin.

Clinical Manifestations and Subtypes

The clinical presentation of psoriasis is remarkably diverse. While the disease is unified by its inflammatory origin, it manifests in several distinct phenotypes:

Plaque Psoriasis (Psoriasis Vulgaris): The most common form (90% of cases). It typically appears on the extensor surfaces like elbows and knees, the scalp, and the lumbosacral region.

Guttate Psoriasis: Characterized by small, "drop-like" lesions. It often appears suddenly in children or young adults following a Streptococcal throat infection.

Inverse Psoriasis: Also known as flexural psoriasis, it occurs in skin folds (axilla, groin, inframammary). Because of the moisture in these areas, the typical silvery scale is absent, leaving shiny, red, well-demarcated patches.

Pustular Psoriasis: A severe variant where sterile, white pustules appear on red skin. It can be localized (palms and soles) or generalized (Von Zumbusch type), the latter being a medical emergency due to systemic toxicity.

Erythrodermic Psoriasis: The most inflammatory form, involving over 90% of the body surface. It compromises the skin's barrier function, leading to hypothermia, fluid loss, and potential heart failure.

The Systemic Burden: More Than Skin Deep

One of the most critical shifts in the last decade of psoriasis research is the concept of **Systemic Inflammation**. The cytokines (IL-17, TNF-alpha) produced in psoriatic skin do not stay there; they leak into the bloodstream. This leads to a state of chronic, low-grade systemic inflammation known as the "Psoriatic March."

Psoriatic Arthritis (PsA): Up to 30% of psoriasis patients develop PsA. It is an inflammatory joint disease that can lead to permanent joint destruction if not treated early. It typically presents as dactylitis ("sausage digits") and enthesitis (inflammation where tendons meet bone).

Cardiovascular Disease: Patients with severe psoriasis have a significantly higher risk of myocardial infarction and stroke. The same inflammation that causes skin plaques also causes "plaques" in the arteries (atherosclerosis).

Metabolic Syndrome: There is a high prevalence of obesity, Type 2 diabetes, and hypertension among psoriasis patients. Interestingly, adipose tissue (body fat) also produces pro-inflammatory cytokines, creating a vicious cycle where obesity worsens psoriasis and psoriasis promotes metabolic dysfunction.

Psychosocial Impact and Quality of Life

The visibility of psoriasis causes profound psychological distress. Patients often report feelings of shame, stigmatization, and social withdrawal. Studies have shown that the impact of psoriasis on "Quality of Life" (QoL) is comparable to that of cancer, heart disease, or chronic obstructive pulmonary disease. The prevalence of depression and anxiety is significantly higher in this population, and the "itch" (pruritus) associated with the lesions often leads to sleep deprivation and further mental health decline.

The Therapeutic Revolution

The treatment landscape for psoriasis has undergone a paradigm shift. We have moved from "Treating the Surface" to "Targeting the Source."

Topical Therapies: For mild cases, corticosteroids, Vitamin D analogues, and tar-based preparations remain the first line.

Phototherapy: Narrowband Ultraviolet B (NB-UVB) remains a highly effective and safe option, particularly for widespread plaque or guttate psoriasis.

Traditional Systemics: Methotrexate and Cyclosporine are still used globally due to their low cost, though they require rigorous monitoring of the liver and kidneys.

Biologic Therapies: This is the "Golden Age" of psoriasis treatment. Monoclonal antibodies targeting TNF-alpha (Adalimumab), IL-12/23 (Ustekinumab), IL-17 (Secukinumab, Ixekizumab), and IL-23 (Risankizumab, Guselkumab) have made "PASI 100" (100% skin clearance) a realistic goal for many patients.

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Conclusion of the Introduction

In summary, psoriasis is a complex, multi-systemic disease that requires a multidisciplinary approach. While we have mastered the ability to clear the skin through targeted biologics, the challenge for the next decade lies in early intervention—treating the skin early enough to prevent the "Psoriatic March" and the development of irreversible joint and vascular damage. As our understanding of the genetic and environmental triggers continues to grow, the move toward personalized medicine offers hope for a future where psoriasis is not just managed, but potentially cured.

Aim and Objectives

Aim

The primary aim of this review is to synthesize the molecular, clinical, and therapeutic data surrounding psoriasis to provide a holistic view of the disease management in the 21st century.

Objectives

To analyze the role of the IL-23/IL-17 cytokine axis in disease pathogenesis.

To evaluate the efficacy and safety profiles of current systemic and biologic therapies.

To examine the impact of psoriasis on systemic health, specifically cardiovascular and metabolic comorbidities.

To identify gaps in current treatment strategies, such as the management of pediatric and geriatric populations.

To summarize the genetic underpinnings and environmental triggers that contribute to disease flares.

II. LITERATURE REVIEW

Greb et al. (2016): Confirmed the **IL-23/Th17 axis** as the primary driver of psoriatic inflammation, leading to the development of highly specific IL-23 and IL-17 inhibitors.

Gelfand et al. (2025 Meta-analysis): A recent systematic review establishing that patients with severe psoriasis have a **1.54x higher risk of all-cause mortality**, largely driven by cardiovascular disease and systemic inflammation.

Nestle et al. (2005): Demonstrated that **Plasmacytoid Dendritic Cells (pDCs)** are the "initiators" of the psoriatic cascade, producing Interferon-alpha to activate the immune response.

IPC Consensus (2024/2025): The International Psoriasis Council redefined disease severity, shifting from simple surface area measurements to a "Candidate for Systemic Therapy" model based on **high-impact sites** (face, scalp, genitals).

Papp et al. (2017): Head-to-head trials showed that **IL-17 inhibitors** (Secukinumab) provided significantly faster skin clearance compared to older TNF-inhibitors like Etanercept.

Gisondi et al. (2025): Confirmed that the **Mediterranean Diet** significantly reduces PASI scores in mild-to-moderate patients, regardless of weight loss, due to its anti-inflammatory properties.

Armstrong & Read (2020): A seminal review highlighting the "Psoriatic March," where skin inflammation leads to **endothelial dysfunction** and subclinical atherosclerosis.

Blauvelt et al. (2024 Study): Phase 3 trials for **Guselkumab (Tremfya)** showed superior efficacy in clearing scalp psoriasis across all skin tones (Fitzpatrick types I-VI).

Mease et al. (2024): Research into the **synovium-enthesis axis** revealed how joint cytokines drive abnormal tissue accumulation, explaining the progression to Psoriatic Arthritis.

Henseler & Christophers (1985): The foundational study classifying psoriasis into **Type I (early onset, HLA-Cw6 positive)** and **Type II (late onset)** phenotypes.

1. Parisi et al. reported that psoriasis affects around 2–3% of the global population

Parisi et al. highlighted that psoriasis prevalence varies widely between regions, being more common in Western countries than in parts of Asia and Africa. Their work emphasized that psoriasis is a major worldwide public-health concern, affecting millions of individuals and contributing significantly to disease burden due to chronicity and systemic associations.

2. Nestle et al. described psoriasis as an immune-mediated inflammatory disorder

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Nestle et al. explained that psoriasis is driven by T-cell activation and cytokine release, particularly involving TNF-α, IL-17, and IL-23 pathways. This shifted the previous thinking of psoriasis being purely a keratinocyte disorder to recognizing it as a complex immune-mediated disease.

3. Lowes et al. demonstrated the key role of Th17 cells in the pathogenesis of psoriasis

Lowes et al. found that Th17 cells and their cytokines, especially IL-17, are central mediators of inflammation in psoriatic lesions. Their findings helped form the scientific basis for newer biologic therapies targeting IL-17 and IL-23.

4. Griffiths et al. discussed genetic susceptibility involving HLA-Cw6

Griffiths et al. showed that psoriasis has a strong heritable component, particularly linked to HLA-Cw6 and other PSORS gene regions. Individuals with affected first-degree relatives are at significantly higher risk, confirming familial clustering and genetic predisposition.

5. Mallbris et al. identified streptococcal infection as a trigger for guttate psoriasis

Mallbris et al. demonstrated that acute streptococcal throat infections are frequently associated with guttate psoriasis, especially in children and young adults. This suggests that immune cross-reactivity between bacterial antigens and skin may trigger disease onset.

6. Gudjonsson et al. reported accelerated keratinocyte proliferation in psoriatic lesions

Gudjonsson et al. documented that keratinocytes in psoriatic plaques divide much faster than in normal skin, leading to thickened epidermis and scaling. However, they emphasized that this process is driven by immune dysregulation rather than being the primary cause.

7. Boehncke et al. described psoriasis as a systemic inflammatory disorder

Boehncke et al. discussed that psoriasis is not limited to the skin but is associated with systemic inflammation affecting multiple organs. Their research highlighted links with metabolic syndrome, cardiovascular disease, and diabetes, shifting clinical focus toward holistic management.

8. Mease et al. found that up to 30% of patients develop psoriatic arthritis

Mease et al. reported that a significant proportion of psoriasis patients eventually develop psoriatic arthritis, characterized by joint pain, stiffness, and swelling. Early recognition is crucial because untreated disease can lead to irreversible joint damage and disability.

9. Gelfand et al. demonstrated increased cardiovascular risk in psoriasis patients

Gelfand et al. found that moderate-to-severe psoriasis is associated with a higher risk of myocardial infarction and cardiovascular mortality. They suggested that persistent systemic inflammation may accelerate atherosclerosis.

10. Kimball et al. highlighted the psychological burden of psoriasis

Kimball et al. reported that psoriasis is strongly associated with depression, anxiety, social stigma, and impaired quality of life. Visible lesions and chronic disease often lead to emotional distress, social withdrawal, and decreased work productivity.

11. Armstrong et al. linked psoriasis with obesity and metabolic syndrome

Armstrong et al. found a strong correlation between psoriasis and obesity, hypertension, insulin resistance, and dyslipidemia. They proposed that adipose-derived inflammatory mediators may worsen psoriasis severity, creating a vicious cycle.

12. Naldi et al. associated smoking and alcohol consumption with increased disease severity

Naldi et al. demonstrated that smoking and heavy alcohol intake worsen psoriasis severity and may trigger disease onset. They suggested a dose-dependent relationship, particularly in pustular and palmoplantar forms.

13. Honma et al. confirmed the effectiveness of narrow-band UVB therapy

Honma et al. reported that narrow-band UVB phototherapy significantly improves plaque psoriasis by suppressing skin inflammation and slowing keratinocyte proliferation, making it a safe and effective option for many patients.

14. Menter et al. established guidelines for systemic therapy

Menter et al. described the clinical use of systemic medications such as methotrexate, cyclosporine, and retinoids. These drugs are effective for severe disease but require careful monitoring due to hepatotoxicity, nephrotoxicity, and teratogenicity risks.

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15. Gordon et al. demonstrated high efficacy of TNF-α inhibitors in severe psoriasis
Gordon et al. showed that biologics targeting TNF-α, such as etanercept and infliximab, produce rapid and significant lesion improvement, particularly in patients unresponsive to conventional treatments.
16. Blauvelt et al. highlighted the success of IL-17 inhibitors
Blauvelt et al. reported that IL-17 inhibitors such as secukinumab provide superior clinical clearance rates, underlining the central role of IL-17 in disease mechanisms and offering new hope for resistant cases.
17. Langley et al. evaluated long-term safety of biologic therapy
Langley et al. demonstrated that biologics generally show favorable long-term safety profiles, although monitoring remains necessary due to infection and immune-related risks.
18. Bronckers et al. reviewed pediatric psoriasis management
Bronckers et al. noted that psoriasis in children requires special consideration for growth, psychological impact, and safety, as aggressive or inappropriate therapy may carry long-term consequences.
Finlay et al. developed quality-of-life assessment tools
Finlay et al. created the Dermatology Life Quality Index (DLQI), which is widely used to measure the psychosocial burden of psoriasis and assess treatment outcomes in clinical practice and research.
Fowler et al. emphasized the role of patient education and multidisciplinary care
Fowler et al. concluded that optimal psoriasis management requires patient involvement, lifestyle modification, psychological support, and coordinated care between dermatologists, rheumatologists, and primary physicians to address systemic risk factors.

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