



“From Genes to Treatment: An Overview of Psoriasis — Epidemiology, Pathophysiology, and Emerging Therapies”

Samiksha Ravindra Bhatkar ¹, Mr. Ashish Umale ², Dr. Swati Deshmukh³

1. Student, Shraddha Institute Of Pharmacy, Washim , Maharashtra, India.

2. Lecturer, Department of Pharmacy, Shraddha Institute Of Pharmacy, Washim, Maharashtra, India.

3. Professor, Department of Pharmacology, Shraddha Institute Of Pharmacy, Washim, Maharashtra, India.

Abstract

Psoriasis is a chronic, immune-mediated inflammatory skin disorder characterized by hyperproliferation of keratinocytes, dysregulated cytokine signaling, and recurrent erythematous, scaly plaques. This review provides a comprehensive overview of the disease, covering its etiology, immunopathogenesis, clinical features, diagnostic approaches, and current therapeutic strategies. Genetic susceptibility, environmental triggers, immune dysregulation—particularly involving the IL-23/IL-17 axis—and lifestyle factors collectively contribute to disease onset and progression. Clinical manifestations range from localized plaque lesions to severe systemic involvement, including psoriatic arthritis and metabolic syndrome. Standard treatments include topical agents, phototherapy, systemic drugs, and targeted biologics, each offering varying degrees of efficacy depending on disease severity. The review also highlights the growing role of herbal and natural ingredients with anti-inflammatory, antioxidant, and immunomodulatory properties as complementary therapies. Recent advances such as biologics, small-molecule inhibitors, nanotechnology-based delivery systems, gene-modulating therapies, and AI-driven personalized management are transforming therapeutic outcomes and represent promising future directions. Overall, psoriasis management is shifting toward integrative, personalized, and mechanism-based approaches aimed at achieving long-term remission and improving patient quality of life.

Keywords

Psoriasis; immune-mediated disorder; keratinocyte hyperproliferation; cytokines; IL-17; IL-23; biologics; nanotechnology; herbal therapy; phototherapy; systemic therapy; psoriatic arthritis; personalized medicine.

1.INTRODUCTION

Psoriasis is a chronic, immune-mediated inflammatory disorder characterized by rapid epidermal proliferation and the formation of red, scaly plaques. Although non-contagious, it is lifelong with alternating phases of remission and relapse. It is widely regarded as an autoimmune condition influenced by genetic susceptibility and environmental triggers. Psoriasis also affects emotional and social well-being, as visible lesions may cause embarrassment, reduced confidence, and social withdrawal, making it a significant public health concern in dermatology [1].

➤Structure and Function of the Skin

The skin is the largest organ of the body and provides protection against external hazards, microorganisms, and ultraviolet radiation. It also maintains internal balance by regulating temperature and fluid levels. Structurally, the skin consists of three main layers: the epidermis, dermis, and hypodermis, each supporting barrier function, sensation, and overall physiological stability [2].

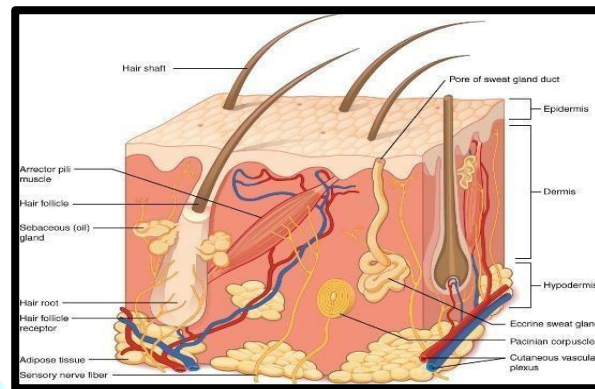


Fig.1.1: Structure of the human skin

➤Epidermis

The epidermis is the outermost layer of the skin and is mainly made of keratinocytes that produce keratin for strength and protection. Normally, keratinocytes mature and shed in about 28 days, but in psoriasis this cycle shortens to 3–5 days. This rapid turnover leads to the accumulation of immature cells, forming thick, scaly plaques that represent the early visible changes of psoriasis [3].

➤Dermis

The dermis lies beneath the epidermis and provides strength, elasticity, and nourishment to the skin. It contains blood vessels, lymphatic channels, glands, and sensory nerves. Fibroblasts in this layer produce collagen and elastin, which support healing and structural stability. Dermal involvement is essential in maintaining skin function and contributes to inflammation and vascular changes seen in psoriasis [4].

➤Hypodermis

The hypodermis, or subcutaneous layer, is composed mainly of adipose tissue. It acts as a protective cushion, stores energy, and provides insulation to regulate body temperature. Although not directly altered in psoriasis, this layer supports overall skin integrity.

➤Significance in Psoriasis

Healthy skin depends on proper coordination among the epidermis, dermis, and hypodermis. In psoriasis, immune-driven dysregulation disrupts keratinocyte production, leading to excessive cell buildup. This imbalance results in inflamed, itchy, and scaly plaques that define the disease [5].

➤Immunological Basis and Mechanism

Psoriasis is a T-cell-mediated autoimmune condition involving abnormal activation of Th1 and Th17 cells. These cells mistakenly attack healthy skin, releasing pro-inflammatory cytokines such as TNF- α , IL-17, and IL-23, which accelerate keratinocyte proliferation and initiate angiogenesis in the dermis, producing redness and scaling [6]. Genetic factors like HLA-Cw6, IL-23R, and TNFAIP3 increase susceptibility, and environmental triggers such as infection, stress, and trauma may initiate flare-ups.

➤ Clinical and Epidemiological Aspects

Psoriasis affects 2–3% of the global population, with no major gender differences. It commonly appears between 15 and 40 years and follows a chronic relapsing pattern influenced by stress, infections, and climate. Typical lesions are sharply demarcated erythematous plaques with silvery scales, often on the scalp, elbows, knees, and lower back. Nail involvement and psoriatic arthritis may also occur. Because the disease is highly visible, many patients experience psychological distress and social stigma.

➤ Immunological Mechanism of Psoriasis

Psoriasis involves both innate and adaptive immune responses, with T-cells, dendritic cells, and cytokines playing key roles [7]. Dendritic cells present self-antigens to naïve T-cells, activating Th1, Th17, and Th22 subsets, which migrate into the skin and release inflammatory mediators that drive keratinocyte hyperproliferation [8].

➤ Role of the Immune System

In healthy conditions, immune cells protect the body, but in psoriasis they overreact to harmless signals. Overactivated T-cells accumulate in the dermis and epidermis, releasing cytokines that maintain chronic inflammation. This cycle accelerates epidermal turnover and sustains plaque formation [8].

➤ Cytokine Pathways

Key cytokines—TNF- α , IL-17, and IL-23—are significantly elevated in psoriasis. TNF- α amplifies inflammation, while IL-17 and IL-23 maintain the psoriatic cycle. These cytokines stimulate rapid keratinocyte growth and promote further immune activation, creating a persistent inflammatory loop [9].

➤ Cellular Changes in the Skin

Continuous cytokine release leads to structural alterations, including thickened epidermis, increased vascular dilation in the dermis, and infiltration of inflammatory cells such as lymphocytes, macrophages, and neutrophils. These changes contribute to the persistent redness, scaling, and chronicity of psoriatic plaques [10]

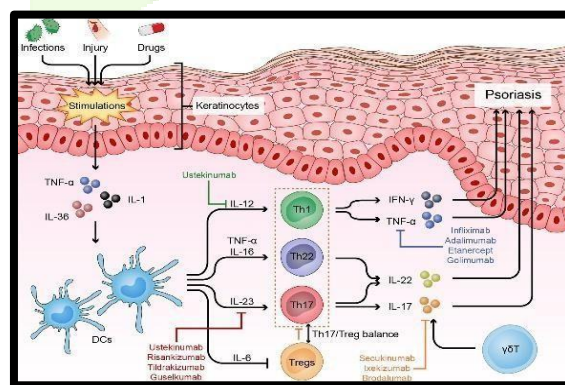


Fig.1.2: Immunopathogenic mechanism of psoriasis showing T-cell activation and cytokine signaling pathways.

2. CAUSES OF PSORIASIS

Psoriasis results from a complex interaction of genetic, immunological, environmental, and lifestyle factors. No single cause is responsible; instead, multiple triggers influence disease onset and flare-ups. Understanding these contributors helps in effective management and prevention strategies [11].

➤ Genetic Factors

Genetic predisposition is a major contributor, with about one-third of patients having an affected family member. Studies show significantly higher risk among individuals with psoriasis in their family. Several genes related to immune regulation and skin barrier stability—such as HLA-Cw6, IL-23R, TNFAIP3, and LCE3B/3C—are implicated. HLA-Cw6 is strongly linked to early-onset and guttate psoriasis. These genetic factors influence susceptibility, clinical type, and disease severity.

➤ Immune System Dysregulation

Psoriasis is strongly driven by immune malfunction, where the immune system mistakenly targets healthy skin cells, triggering an autoimmune response. This activates Th1 and Th17 T-cells, which release TNF- α , IL-17, and IL-23. These cytokines accelerate keratinocyte growth and maintain chronic inflammation, resulting in the formation of thick, scaly plaques. Once initiated, this inflammatory cycle tends to persist without appropriate treatment [12].

➤ Environmental Triggers

External factors often trigger or worsen psoriasis in genetically predisposed individuals. Streptococcal infections, particularly *Streptococcus pyogenes*, are known to induce guttate psoriasis. Climate also plays an important role—cold, dry weather can aggravate symptoms, while warm and humid conditions often improve them. Skin trauma may cause new lesions through the Koebner phenomenon. Other triggers include pollution, ultraviolet exposure, and certain chemical irritants [13].

➤ Medications

Several medications are associated with psoriasis onset or worsening. These include beta-blockers, lithium, antimalarials like chloroquine, and NSAIDs. Such drugs may disrupt immune function or alter the skin cell cycle, precipitating flare-ups. Patients with psoriasis should always seek medical guidance before starting new medications to avoid possible exacerbation [14].

➤ Lifestyle and Psychological Factors

Lifestyle habits significantly influence disease severity. Smoking increases oxidative stress and inflammation, while alcohol consumption weakens immune responses and affects treatment effectiveness. Obesity contributes to systemic inflammation through adipokines, increasing disease activity. Psychological stress is a recognized trigger, activating hormonal pathways that release inflammatory mediators. Stress-reduction techniques such as yoga, counseling, and relaxation therapy are shown to help control flare-ups [15].

➤ Hormonal and Metabolic Influences

fluctuations may alter psoriasis activity. Periods such as puberty, pregnancy, and menopause can influence symptoms. Many women experience improvement during pregnancy due to higher estrogen levels, while postpartum worsening may occur. These hormonal effects highlight the role of metabolic and endocrine balance in disease modulation.

Fig. 2.1: Major factors contributing to the development and aggravation of psoriasis

3. SYMPTOMS OF PSORIASIS

Psoriasis exhibits diverse clinical symptoms that vary in severity, appearance, and body distribution. Lesions may occur anywhere, but the scalp, elbows, knees, and lower back are most commonly affected. Symptoms usually develop gradually and understanding them is essential for accurate diagnosis and management [16,17].

➤ Skin Lesions

The hallmark sign of psoriasis is well-defined red plaques covered with silvery-white scales. These lesions result from rapid accumulation of immature keratinocytes. The underlying skin appears erythematous due to increased blood flow and inflammation. Lesions range from small papules to large plaques and may cause itching, pain, cracking, or bleeding in severe cases [18].

➤ Itching and Burning Sensation

Pruritus is one of the most frequent complaints among patients. The itching may be mild or severe and is often accompanied by burning or stinging sensations. Persistent scratching can worsen inflammation, damage the skin, or lead to secondary infections. These sensations arise from inflammatory cytokines and heightened nerve sensitivity.

➤ Dryness and Cracking

Psoriatic skin becomes extremely dry due to impaired barrier function and increased water loss. Thick scaling prevents adequate moisturization, causing fissures and painful cracks that may bleed. This dryness increases irritation and makes the skin more vulnerable to external triggers.

➤ Nail Changes

Nail psoriasis affects nearly half of all patients and may even precede skin symptoms. Common features include pitting, onycholysis, discoloration, and nail thickening or brittleness. These changes reflect the same hyperproliferative process occurring in the skin and are often associated with psoriatic arthritis [19].

➤ Scalp and Hair Symptoms

Scalp psoriasis presents as thick, crusted plaques with prominent scaling. Patients often report severe itching and flaking that is more inflammatory than dandruff. Persistent scratching or inflammation may lead to temporary hair loss, which typically resolves once the condition is treated [20].

➤ Joint and Musculoskeletal Symptoms

Psoriatic arthritis occurs in some patients and involves painful inflammation of joints such as the fingers, toes, wrists, and knees. Symptoms include stiffness, swelling, and reduced mobility. If untreated, joint damage may become irreversible. Fatigue is also common due to the systemic inflammatory nature of the disease [21].

➤ Psychological and Emotional Effects

Psoriasis heavily impacts psychological well-being. Visible lesions often cause embarrassment, reduced confidence, and avoidance of social situations. This emotional burden may contribute to anxiety, depression, and low self-esteem. The chronic, visible, and stigmatizing nature of the disease affects both personal and professional life, adding to overall disease burden [22].



Fig. 3.1: Clinical presentation of psoriasis showing scaly red plaques on elbows and knees.

4. TYPES OF PSORIASIS

Psoriasis presents in several clinical forms that differ in appearance, severity, and distribution, although all share the same immune-mediated origin. Identifying the correct type is essential for proper diagnosis and treatment planning.

➤ **Plaque Psoriasis (Psoriasis Vulgaris)**

Plaque psoriasis is the most common variant, representing 80–90% of cases. It presents as raised, red, inflamed plaques covered with silvery-white scales, typically on the scalp, elbows, knees, and lower back. The lesions are sharply demarcated and often symmetrical. Patients commonly report itching, burning, and tightness of the skin [23].

➤ **Guttate Psoriasis**

Guttate psoriasis appears as numerous small, drop-shaped red papules, mostly on the trunk and limbs. It often develops suddenly after upper respiratory infections, especially streptococcal throat infection, and is frequently seen in children and young adults.

➤ **Inverse Psoriasis**

Inverse psoriasis affects skin folds such as the groin, armpits, under the breasts, and between the buttocks. Lesions appear smooth, shiny, and bright red due to the moist environment, with minimal scaling compared to plaque psoriasis [24].

➤ **Pustular Psoriasis**

Pustular psoriasis is marked by sterile pustules surrounded by red, inflamed skin. It may be localized—commonly on the hands and feet—or generalized, covering larger areas. During flare-ups, patients may experience fever, chills, and fatigue due to systemic inflammation.

➤ **Erythrodermic Psoriasis**

Erythrodermic psoriasis is a rare but severe form characterized by widespread redness, inflammation, and extensive shedding of skin. It may arise from unstable plaque psoriasis or be triggered by infections, severe stress, or sudden withdrawal of corticosteroids [25].

➤ **Nail Psoriasis**

Nail psoriasis affects both fingernails and toenails, causing pitting, discoloration, thickening, and separation of the nail plate from the nail bed. These changes result from inflammation of the nail matrix and are commonly seen in patients with psoriatic arthritis.

➤ Scalp Psoriasis

Scalp psoriasis appears as thick, scaly patches on the scalp and may extend to the forehead, neck, or ears. It is often mistaken for severe dandruff but is more inflammatory. Patients experience itching, flaking, and tightness, and temporary hair loss may occur due to scratching. Treatment often includes medicated shampoos, topical steroids, and vitamin D analogues [26]

➤ Psoriatic Arthritis

Psoriatic arthritis affects about 10–30% of psoriasis patients. It involves inflammation of joints, tendons, and surrounding tissues, leading to pain, stiffness, and swelling. Without timely treatment, permanent joint deformities may occur. Nail changes and severe skin involvement are frequently associated with this subtype [27].



Fig. 4.1: Major clinical types of psoriasis showing variation in lesion pattern and affected body areas.

5. PATHOPHYSIOLOGY OF PSORIASIS

Psoriasis is a chronic inflammatory condition resulting from the interplay of genetic susceptibility, immune dysfunction, and environmental triggers. It is now recognized as a systemic autoimmune disease rather than only a disorder of keratinocyte proliferation. The disease involves immune-cell activation, release of inflammatory cytokines, and accelerated epidermal turnover, producing thickened and scaly plaques.

➤ Initiation of the Inflammatory Process

The psoriatic cascade starts when triggers such as infection, trauma, or stress activate immune cells in predisposed individuals. Dendritic cells in the skin release IL-12 and IL-23, which stimulate naïve T-cells to differentiate into Th1 and Th17 subsets. These activated T-cells migrate to the skin and release cytokines that initiate inflammation and drive lesion development [28].

➤ Role of T-Cells and Cytokines

Th1, Th17, and Th22 cells are central to disease progression. Th1 cells secrete IFN- γ and TNF- α , amplifying inflammatory responses, while Th17 cells produce IL-17 and IL-22, which stimulate keratinocyte proliferation and attract neutrophils. These cytokines form a self-sustaining inflammatory loop that maintains chronic disease activity. TNF- α , IL-17, and IL-23 are key cytokines responsible for persistent lesions and serve as major targets for current biologic therapies [29].

➤ Keratinocyte Hyperproliferation

Keratinocytes respond to inflammatory cytokines by proliferating rapidly and maturing incompletely. This shortens normal epidermal turnover from about 28 days to 3–5 days. The accumulation of immature keratinocytes forms thick, scaly plaques. The epidermis becomes hyperplastic, dermal papillae elongate, and neutrophil-filled microabscesses (Munro's microabscesses) may form.

➤ Vascular and Inflammatory Changes

Psoriasis also affects dermal vasculature. Cytokines promote angiogenesis, creating dilated, tortuous blood vessels that increase blood flow and contribute to erythema. Neutrophils, macrophages, and dendritic cells accumulate in the dermis, enhancing inflammation. Increased vascular permeability allows more immune cells to enter the skin. Because this inflammation is systemic, psoriasis is associated with metabolic syndrome, psoriatic arthritis, and cardiovascular disorders [30].

➤ Maintenance of the Psoriatic Cycle

Continuous interaction between immune cells and keratinocytes sustains the disease. Activated keratinocytes release chemokines that attract more immune cells, while infiltrating immune cells produce cytokines that further stimulate keratinocyte proliferation. Persistence of TNF- α , IL-17, and IL-23 keeps this cycle active, making psoriasis long-lasting and relapsing.

➤ Molecular Pathways and Targeted Therapy

Key molecular pathways involved in psoriasis include the NF- κ B pathway, which regulates transcription of inflammatory genes, and the JAK-STAT pathway, which mediates cytokine signaling between immune cells and keratinocytes. Dysregulation of these pathways leads to chronic activation of T-cells and overproduction of TNF- α , IL-17, and IL-23. Advances in understanding these mechanisms have enabled the development of biologic therapies that block specific immune signals and interrupt the inflammatory cascade.

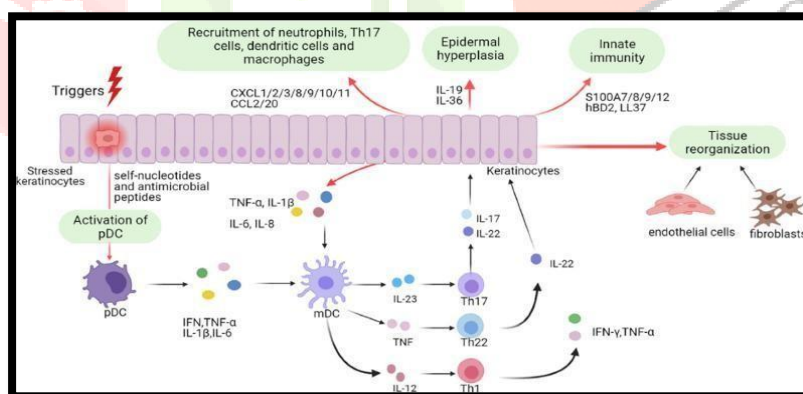


Fig. 5.1: Pathophysiological mechanism of psoriasis showing T-cell activation, cytokine release, and keratinocyte hyperproliferation.

➤ Psoriatic Arthritis

Psoriatic arthritis (PsA) is a major complication of psoriasis, affecting about 10–30% of patients. It is characterized by inflammation of joints, tendons, and periarticular tissues, leading to pain, stiffness, swelling, and reduced mobility, commonly in fingers, toes, and knees. Chronic inflammation may cause bone erosion, joint deformity, and disability if not treated early. Nail changes such as pitting and onycholysis are frequently associated with PsA. Prompt diagnosis using clinical assessment and imaging is essential to prevent irreversible damage [32].

➤ **Cardiovascular Disorders**

Chronic systemic inflammation in psoriasis is associated with an increased risk of cardiovascular disease. Elevated TNF- α , IL-6, and IL-17 contribute to endothelial dysfunction, promoting hypertension, atherosclerosis, myocardial infarction, stroke, and other vascular events. Patients with severe psoriasis show higher cardiovascular morbidity than the general population, emphasizing the need for active management of risk factors such as obesity, smoking, dyslipidemia, and physical inactivity.

➤ **Metabolic Syndrome**

Metabolic syndrome, comprising obesity, insulin resistance, dyslipidemia, and hypertension, frequently coexists with psoriasis. Pro-inflammatory cytokines alter lipid and glucose metabolism, increasing the risk of type 2 diabetes mellitus. Adipose tissue in obese individuals releases additional inflammatory mediators, creating a vicious cycle that aggravates both psoriasis and metabolic dysfunction. Lifestyle interventions—healthy diet, weight control, and regular exercise—are crucial for reducing these complications [33].

Psychological and Emotional Effects

Psoriasis exerts a substantial psychological burden. Visible lesions often cause embarrassment, social withdrawal, and experiences of stigma, leading to anxiety, depression, and low self-esteem. Sleep disruption due to itching and pain further worsens emotional health. The chronic, unpredictable nature of the disease can generate feelings of frustration and hopelessness. Psychological counseling, patient education, and support groups are important components of holistic management.

Infectious and Treatment-Related Complications

Systemic and immunosuppressive therapies, such as methotrexate and cyclosporine, increase susceptibility to bacterial, viral, and fungal infections. These medications may also cause liver, kidney, or hematological toxicity. Biologic agents, despite their specificity, can predispose to opportunistic infections if not carefully monitored. Regular clinical review and laboratory investigations are therefore essential to ensure safe long-term treatment.

Other Systemic Associations

Psoriasis is associated with a higher incidence of autoimmune diseases such as Crohn's disease, ulcerative colitis, and autoimmune thyroid disorders. Chronic inflammation may also affect the eyes (uveitis) and internal organs such as the liver and kidneys, contributing to broader systemic morbidity [34].

6. DIAGNOSIS AND LABORATORY EVALUATION OF PSORIASIS

Psoriasis is primarily a clinical diagnosis, supported by history, histopathology, and laboratory tests when needed. Because it can resemble conditions like eczema, seborrheic dermatitis, or fungal infections, accurate differentiation is important for appropriate management.

➤ **Clinical Diagnosis**

Diagnosis is mainly based on the appearance and distribution of characteristic plaques, as well as history of triggers such as infections, drugs, or stress. Features like well-demarcated erythematous plaques with silvery scales on typical sites and nail changes (pitting, onycholysis) support the diagnosis, particularly when psoriatic arthritis is suspected [35].

➤ **Histopathological Examination**

In doubtful cases, a skin biopsy confirms the diagnosis. Typical findings include hyperkeratosis, parakeratosis, acanthosis, elongated dermal papillae, and dilated capillaries, explaining the clinical redness. Neutrophilic microabscesses in the stratum corneum (Munro's microabscesses) are characteristic and help distinguish psoriasis from other dermatoses.

➤ **Laboratory Investigations**

There is no specific blood test for psoriasis, but investigations help assess systemic involvement and monitor therapy. CBC, liver and kidney function tests, and lipid profile are routinely used, especially in patients on systemic drugs. Fasting blood glucose and lipid profile are important due to the association with metabolic syndrome and dyslipidemia. Elevated ESR and CRP indicate systemic inflammation or active psoriatic arthritis and are useful for disease monitoring [36].

7. TREATMENT APPROACHES FOR PSORIASIS

Management aims to reduce inflammation, normalize epidermal turnover, alleviate symptoms, and improve quality of life. Therapy is tailored to disease severity, lesion distribution, and patient-specific factors, often combining topical, phototherapeutic, and systemic modalities.

➤ **Topical Therapy**

Topical agents are first-line in mild to moderate disease. Corticosteroids reduce inflammation and pruritus and are used in varying potencies depending on site and plaque thickness. Vitamin D analogues (calcipotriol, calcitriol) regulate keratinocyte proliferation and are often combined with steroids for better efficacy. Coal tar, salicylic acid, and keratolytics help remove scales, while emollients restore barrier function and relieve dryness [37].

➤ **Phototherapy**

Phototherapy is indicated in moderate to severe psoriasis or when topical therapy is insufficient. UVB (broadband or narrow-band 311 nm) and PUVA (psoralen plus UVA) slow keratinocyte proliferation and suppress immune activity. Narrow-band UVB is preferred for its efficacy and safety. PUVA is effective but requires caution due to potential photoaging and increased skin cancer risk [38].

➤ **Systemic Therapy**

Systemic agents are used in extensive or refractory disease. Methotrexate inhibits DNA synthesis and T-cell activity, improving both skin lesions and psoriatic arthritis but requiring liver function monitoring. Cyclosporine provides rapid control by inhibiting T-lymphocyte activation, but long-term use is limited by nephrotoxicity and hypertension. Retinoids such as acitretin normalize keratinocyte differentiation and are useful in pustular and erythrodermic psoriasis but are teratogenic and contraindicated in pregnancy [39].

➤ **Biologic Therapy**

Biologic drugs target specific cytokines involved in psoriasis pathogenesis, such as TNF- α , IL-17, and IL-23. Agents like adalimumab, etanercept, infliximab, secukinumab, and ustekinumab provide substantial lesion clearance and prolonged remission, with additional benefits in psoriatic arthritis. Pre-treatment screening for latent infections, especially tuberculosis, is essential [40].

➤ **Lifestyle and Supportive Measures**

Lifestyle modification complements pharmacologic therapy. Avoiding smoking, alcohol, and stress, along with maintaining a healthy diet and body weight, helps reduce flares and comorbid risks. Gentle skin care, regular moisturization, and psychosocial support further enhance treatment outcomes.

8. PREVENTIVE AND LIFESTYLE MEASURES

Although psoriasis cannot be cured permanently, its severity and relapse frequency can be reduced through targeted preventive and lifestyle strategies. These aim to control triggers, support immune balance, and maintain healthy skin.

➤ **Avoidance of Triggers**

Recognizing and avoiding triggers such as infections, trauma, cold weather, stress, and certain medications is crucial. Streptococcal throat infections are strongly linked with guttate psoriasis, and Koebner phenomenon explains lesion development after skin injury. Drugs like beta-blockers, lithium, and antimalarials may precipitate flares. Gentle hygiene with mild, fragrance-free cleansers, lukewarm baths, and avoidance of harsh irritants is recommended. Patients should also avoid excessive alcohol and smoking due to their pro-inflammatory effects [41].

➤ **Diet and Nutrition**

Diet plays an important role in modulating inflammation. A balanced diet rich in omega-3 fatty acids, vitamins A, C, E, zinc, selenium, whole grains, fruits, vegetables, nuts, and fatty fish helps reduce oxidative stress and systemic inflammation. Limiting refined sugars, saturated fats, processed meats, and alcohol can prevent exacerbations and prolong remission. Maintaining healthy weight and gut microbiome has been associated with better treatment response and lower inflammatory burden [42].

➤ **Stress Management**

Psychological stress is a well-recognized trigger that exacerbates psoriasis via activation of the hypothalamic-pituitary-adrenal axis and inflammatory pathways. Relaxation techniques such as meditation, yoga, deep breathing, and mindfulness help break the stress-flare cycle. Counseling, support groups, and patient education programs further improve coping skills and adherence to therapy. Regular monitoring is especially important for those on systemic drugs like methotrexate, cyclosporine, or acitretin due to potential hepatic or renal toxicity [44].

➤ **Skincare and Sun Exposure**

Routine use of moisturizers restores the skin barrier, reduces dryness, and minimizes scaling. Products containing glycerin, aloe vera, or urea are particularly helpful. Patients should avoid scratching to prevent infection and Koebnerization. Controlled sun exposure can benefit psoriasis by slowing cell turnover, but excessive exposure and sunburn should be avoided to prevent worsening of lesions [45].

➤ **Regular Exercise and Sleep**

Moderate physical activity improves cardiovascular health, supports weight control, and reduces systemic inflammation. Activities like walking, yoga, swimming, and cycling are suitable and joint-friendly. Exercise also reduces stress, anxiety, and depression. Adequate, good-quality sleep is essential for immune regulation and tissue repair; poor sleep increases inflammatory markers and stress hormones, worsening disease. Good

sleep hygiene and relaxation practices before bedtime (meditation, deep breathing, warm bath) support overall healing [46].

9. HERBAL AND NATURAL INGREDIENTS IN PSORIASIS MANAGEMENT

Herbal and natural agents are increasingly used due to their favorable safety profile and multitargeted actions. They exert anti-inflammatory, antioxidant, antimicrobial, and wound-healing effects, supporting skin barrier restoration and reducing recurrence of flares. Traditional systems such as Ayurveda, Unani, and Siddha have long employed herbs for chronic skin disorders. Today, these ingredients are formulated into ointments, gels, creams, lotions, and oils, alone or in combination with conventional drugs to relieve itching, scaling, redness, and dryness [47].

➤ Mechanisms of Herbal Action

Many herbal agents inhibit pro-inflammatory cytokines (TNF-α, IL-6, IL-17), reduce oxidative stress, and suppress keratinocyte hyperproliferation. They can enhance collagen synthesis, improve barrier function, and modulate immune responses by downregulating Th1/Th17 pathways while promoting IL-10 and other anti-inflammatory mediators. These combined actions help normalize epidermal turnover and support long-term remission with minimal systemic toxicity [48].

➤ Important Herbal and Natural Ingredients

Commonly used herbs include aloe vera, turmeric, neem, tulsi, camphor, calendula, green tea, chamomile, tea tree oil, coconut oil, manjistha, and licorice. Their phytochemicals—flavonoids, terpenoids, alkaloids, and phenolics—reduce inflammation, scaling, microbial growth, and oxidative stress. For example, camphor relieves itching, calendula promotes wound healing, and green tea polyphenols like EGCG modulate keratinocyte proliferation [49].

Table 1: Key Herbal and Natural Ingredients Used in Psoriasis Management[50]

Sr. No.	Herbal Ingredient (Botanical Name)	Major Active Constituents	Mechanism of Action	Therapeutic Benefits in Psoriasis
1	Aloe vera (Aloe barbadensis Miller)	Polysaccharides, vitamins, amino acids	Anti-inflammatory, moisturizing, promotes wound healing	Reduces itching, redness, and scaling
2	Turmeric (Curcuma longa)	Curcumin	Inhibits TNF-α, IL-17, and NF-κB; antioxidant effect	Decreases plaque thickness and inflammation
3	Neem (Azadirachta indica)	Azadirachtin, nimbin, quercetin	Antibacterial, antifungal, and anti-inflammatory	Prevents infection and soothes skin
4	Tulsi (Ocimum sanctum)	Eugenol, ursolic acid	Immunomodulatory, antioxidant, antimicrobial	Reduces oxidative stress and irritation
5	Tea Tree Oil (Melaleuca alternifolia)	Terpinen-4-ol, cineole	Antimicrobial and anti-inflammatory; relieves itching	Prevents infection and inflammation

6	Coconut Oil (<i>Cocos nucifera</i>)	Lauric acid, polyphenols	Moisturizing, emollient, barrier-restoring	Reduces dryness and improves texture
7	Green Tea (<i>Camellia sinensis</i>)	Epigallocatechin gallate (EGCG)	Antioxidant, anti-proliferative, inhibits keratinocyte hyperactivity	Reduces erythema and scaling
8	Calendula (<i>Calendula officinalis</i>)	Flavonoids, triterpenoids	Anti-inflammatory, promotes epithelial regeneration	Enhances healing and reduces scaling
9	Manjistha (<i>Rubia cordifolia</i>)	Anthraquinones, purpurin	Detoxifying, antioxidant, improves microcirculation	Aids in clearing lesions and supports healing
10	Licorice (<i>Glycyrrhiza glabra</i>)	Glycyrrhizin, liquiritin	Corticosteroid-like anti-inflammatory action	Reduces redness, swelling, and irritation

10. RECENT ADVANCES AND RESEARCH IN PSORIASIS MANAGEMENT

Progress in understanding the immunopathology of psoriasis has led to targeted therapies focusing on cytokine modulation, signaling pathways, and advanced delivery systems. Current research integrates biotechnology, pharmacogenomics, and nanotechnology to achieve safer and more durable control.

➤ Targeted Biologic Therapies

Biologic agents—monoclonal antibodies and fusion proteins—specifically inhibit cytokines such as TNF- α , IL-17, and IL-23, thereby disrupting the psoriatic inflammatory cycle. Drugs like adalimumab, etanercept, infliximab, ustekinumab, secukinumab, and guselkumab achieve rapid lesion clearance and sustained remission, and are effective in psoriatic arthritis as well [51]. Newer biologics such as bimekizumab (dual IL-17A/F inhibitor) and risankizumab (IL-23 inhibitor) show promising efficacy with improved safety profiles; biosimilars are under development to enhance accessibility.

➤ Small Molecule and Oral Therapies

Oral small molecules target intracellular signaling. Apremilast, a PDE-4 inhibitor, modulates cytokine production with relatively mild immunosuppression. JAK inhibitors such as tofacitinib and upadacitinib act on the JAK-STAT pathway involved in immune activation and cytokine signaling [52]. Ongoing research aims to increase selectivity and reduce risks such as infection or organ toxicity; combination regimens with topical or biologic agents are being explored.

➤ Nanotechnology and Advanced Drug Delivery Systems

Nanocarriers (liposomes, niosomes, solid lipid nanoparticles, polymeric nanogels) enhance penetration through thick psoriatic plaques and provide controlled drug release, improving bioavailability and reducing side effects. Herbal-based nanoformulations containing curcumin, aloe vera, or neem show strong anti-inflammatory and wound-healing activity. Transdermal patches and microneedle arrays are being developed to deliver biologics and herbal extracts directly into dermal layers, increasing patient adherence [53].

➤ **Gene and Stem Cell Therapy**

Gene-silencing strategies using siRNA and miRNA are under investigation to downregulate overexpressed inflammatory genes (e.g., IL-17, TNF- α). Mesenchymal stem cells (MSCs) offer immunomodulatory and regenerative potential, with early clinical studies suggesting benefit in restoring immune balance and tissue repair.

➤ **Artificial Intelligence and Personalized Medicine**

AI tools and machine learning models assist in image-based diagnosis, objective severity scoring, and prediction of treatment response. Pharmacogenomic data support individualized therapy selection, optimizing efficacy while minimizing adverse effects and cost [54].

➤ **Integrative and Preventive Research**

Recent work underscores the importance of diet, gut microbiome modulation, and stress management in reducing disease activity. Nutrient-rich, anti-inflammatory diets and psychological interventions (counseling, behavioral therapy) enhance quality of life, adherence, and clinical outcomes when combined with standard treatments [55].

10. FUTURE PROSPECTS IN PSORIASIS MANAGEMENT

Future strategies focus on more precise, durable, and patient-specific interventions. Gene-based therapies, including CRISPR-Cas9 and RNA interference, aim to silence key inflammatory genes such as IL-17, TNF- α , and IL-23, while modulation of microRNAs may help restore immune equilibrium and normal keratinocyte behavior [57]. Mesenchymal stem cell therapy and tissue engineering hold promise for regenerating damaged skin and providing long-term immune rebalancing.

Nanotechnology will enable smart carriers that release drugs in response to local stimuli (pH, temperature), enhancing targeted delivery and minimizing systemic toxicity [58]. AI-driven decision tools and digital dermatology platforms will support precise diagnosis, monitoring, and personalized treatment planning. Overall, psoriasis care is expected to evolve into an integrative, personalized model combining advanced biotechnology with lifestyle and preventive strategies to achieve sustained remission and improved quality of life [59].

11. SUMMARY AND CONCLUSION

Psoriasis is a chronic, immune-mediated inflammatory disorder of the skin, nails, and joints, characterized by excessive keratinocyte proliferation and erythematous, scaly plaques. Its pathogenesis reflects a complex interplay between genetic factors, environmental triggers, and immune dysregulation, particularly involving overactivation of T-cells and cytokines such as TNF- α , IL-17, and IL-23. Clinical presentation ranges from localized plaque lesions to severe erythrodermic forms, often with nail involvement and psoriatic arthritis. Diagnosis is largely clinical, supported by histopathology and laboratory tests when necessary.

Although no definitive cure exists, multiple therapeutic options—topical agents, phototherapy, systemic drugs, and biologics—can effectively control symptoms and reduce systemic inflammation. Modern biologics and small-molecule inhibitors provide targeted modulation of cytokine pathways, improving efficacy and safety compared to traditional immunosuppressants. Alongside pharmacologic approaches, lifestyle

modification (diet, exercise, stress management, sleep) and psychosocial support are essential for optimal long-term outcomes.

Herbal and natural ingredients with anti-inflammatory, antioxidant, and immunomodulatory properties offer promising adjuncts or alternatives, especially for maintenance therapy. With ongoing advances in molecular biology, nanotechnology, gene therapy, and personalized medicine, future management strategies aim not only to clear lesions but also to correct underlying immune imbalance, minimize comorbidities, and significantly enhance quality of life for individuals living with psoriasis.

12. REFERENCES

1. Parisi R., Symmons D.P., Griffiths C.E.M., Ashcroft D.M. (2013). Global epidemiology of psoriasis: a systematic review of incidence and prevalence. *Journal of Investigative Dermatology*, 133(2): 377–385.
 2. Yousef H. (2024). Anatomy, Skin (Integument), Epidermis. In: *StatPearls [Internet]*. Treasure Island (FL): StatPearls Publishing.
 3. Kim J.Y., Kim J., Lopez-Ojeda W. (2023). Physiology, Integument (Skin). In: *StatPearls [Internet]*. Treasure Island (FL): StatPearls Publishing.
 4. Lopez-Ojeda W., Pandey S. (2023). Anatomy, Skin Dermis and Function. In: *StatPearls [Internet]*. Treasure Island (FL): StatPearls Publishing.
 5. Kim J.Y., Lopez-Ojeda W. (2023). Anatomy, Subcutaneous Tissue (Hypodermis). In: *StatPearls [Internet]*. Treasure Island (FL): StatPearls Publishing.
 6. Griffiths C.E.M., Armstrong A.W., Gudjonsson J.E., Barker J.N.W.N. (2017). Psoriasis. *The Lancet*, 390(10105): 1299–1312.
 7. Lowes M.A., Suarez-Fariñas M., Krueger J.G. (2014). Immunology of psoriasis. *Annual Review of Immunology*, 32: 227–255.
 8. Villanova F., Di Meglio P., Nestle F.O. (2013). Biomarkers in psoriasis and autoimmune diseases. *Frontiers in Immunology*, 4: 295.
 9. Potestio L., Raimondo A., Fabbrocini G., Megna M. (2024). The role of the interleukin- 23/17 axis in psoriasis management: a comprehensive review. *Clinical, Cosmetic and Investigational Dermatology*, 17: 1301–1319.
 10. Ortiz-López L.I., Domínguez-Cherit J. (2022). Psoriasis: cellular mechanisms and keratinocyte dysfunction. *International Journal of Molecular Sciences*, 23(17): 9874.
 11. Kamiya K., Kishimoto M., Sugai J., Komine M., Ohtsuki M. (2019). Risk factors for the development of psoriasis. *International Journal of Molecular Sciences*, 20(18): 4347.
 12. Chen L., Tsai T.F. (2018). HLA-Cw6 and psoriasis: clinical significance and genetic association. *Journal of Dermatology*, 45(6): 645–650.
 13. Di Meglio P., Villanova F., Nestle F.O. (2017). Psoriasis pathogenesis and immune regulation. *Frontiers in Immunology*, 8: 193.
 14. Gudjonsson J.E., Thorarinsson A.M., Sigurgeirsson B., Kristinsson K.G., Valdimarsson H. (2003). Streptococcal throat infections and exacerbation of chronic plaque psoriasis. *British Journal of Dermatology*, 149(3): 530–534.
-

15. Raharja A., Muralidharan S., Burge S.M., Millington G.W. (2021). Drug-induced and drug- exacerbated psoriasis: a review. *Clinical Dermatology*, 39(2): 189–196.
 16. Masson W., Lobo F., Jaramillo A. (2020). Cardiometabolic comorbidities and lifestyle factors in psoriasis: a review. *Clinical Dermatology*, 38(2): 244–252.
 17. Armstrong A.W., Harskamp C.T., Armstrong E.J. (2012). The association between psoriasis and obesity: a systematic review and meta-analysis. *Journal of the American Academy of Dermatology*, 67(4): 654–659.
 18. Rendon A., Schäkel K. (2019). Psoriasis pathogenesis and treatment. *International Journal of Molecular Sciences*, 20(6): 1475.
 19. Tufnell A., Chi C.C., Fieldhouse R. (2021). Psoriasis: a concise overview of clinical presentation and management. *Dermatology Research and Practice*, 2021: 9930483.
 20. Schons K.R.R., Knob C.F., Murussi N., Beber A., Neumaier W., Monticelo O.A. (2014). Nail psoriasis: a review of the literature. *Anais Brasileiros de Dermatologia*, 89(3): 415– 421.
 21. Gladman D.D. (2005). Clinical features and frequency of psoriatic arthritis. *Annals of the Rheumatic Diseases*, 64(Suppl 2): ii56–ii59.
 22. Kimmel G., Lebwohl M.G. (2018). Psychological burden and quality of life in psoriasis. *Dermatologic Clinics*, 36(3): 335–345.
 23. Radtke M.A., Schafer I., Gajur A., Blome C., Augustin M. (2013). Clinical forms and classification of psoriasis: an overview. *Journal der Deutschen Dermatologischen Gesellschaft*, 11(5): 397–404.
 24. Balan R., Sivaramakrishnan A., Balasubramanian D. (2021). Pathophysiology and histopathology of psoriasis. *Clinical Dermatology and Pathology Review*, 6(1): 12–22.
 25. Boehncke W.H., Schön M.P. (2014). Comorbidities of psoriasis: an update. *Journal of the European Academy of Dermatology and Venereology*, 28(11): 1409–1420.
 26. Fernandez-Figueras M.T., Puig L. (2019). Histopathology of psoriasis: an update. *Clinical and Experimental Dermatology*, 44(3): 260–265.
 27. Elmetts C.A., Lim H.W., Stoff B. et al. (2019). Joint AAD–NPF guideline of care for psoriasis with phototherapy. *Journal of the American Academy of Dermatology*, 81(3): 775–804.
 28. Menter A., Strober B.E., Kaplan D.H. et al. (2020). Guidelines for the management of psoriasis with systemic therapy. *Journal of the American Academy of Dermatology*, 82(6): 1445–1486.
 29. Armstrong A.W., Puig L., Joshi A., Kaur M. (2018). Lifestyle modification and dietary management in psoriasis: review. *Open Dermatology Journal*, 12: 70–78.
 30. Thirumal D., Sankar V., D’Souza C. (2023). Pathology and treatment of psoriasis using nanoformulations: recent advances. *Biomedicines*, 11(6): 1589.
 31. Guo C., Li Y., Zhu S., Chen L. (2022). Role of cytokines and immune regulation in psoriasis pathogenesis. *Dermatology International*, 4(1): 1–10.
 32. Ogdie A., Weiss P. (2015). The epidemiology of psoriatic arthritis. *Rheumatic Disease Clinics of North America*, 41(4): 545–568.
 33. Boehncke W.H. (2018). Psoriasis and metabolic syndrome. *Clinical Dermatology*, 36(1): 21–28.
-

34. Coimbra S., Oliveira H., Reis F. (2020). Systemic implications of psoriasis. *Frontiers in Medicine*, 7: 170.
35. Griffiths C.E.M., Barker J.N.W.N. (2022). Pathogenesis and clinical diagnosis of psoriasis. *New England Journal of Medicine*, 386(9): 859–872.
36. Nast A., et al. (2020). Diagnostic methods in psoriasis. *British Journal of Dermatology*, 182(4): 1029–1039.
37. Lebwohl M. (2018). Topical therapy for psoriasis. *New England Journal of Medicine*, 379: 2313–2324.
38. Dawe R.S. (2021). Phototherapy for psoriasis: mechanisms and clinical applications. *Photodermatology, Photoimmunology & Photomedicine*, 37(1): 1–12.
39. Elmets C.A., et al. (2019). Systemic treatment of psoriasis: review of methotrexate and retinoids. *Journal of Dermatological Treatment*, 30(6): 556–567.
40. Mrowietz U., et al. (2020). Biologic therapies in psoriasis: efficacy and safety update. *British Journal of Dermatology*, 183(2): 189–202.
41. Dattola A., Silvestri M., Bennardo L. (2021). Psoriasis triggers and prevention strategies. *Dermatology Practical & Conceptual*, 11(2): e2021013.
42. Ford A.R., Siegel M., Bagel J., et al. (2018). Dietary influences in psoriasis. *Cutis*, 101(5): 353–358.
43. Wolters M. (2019). Nutrition and psoriasis: role of diet. *American Journal of Clinical Dermatology*, 20(5): 647–659.
44. Zachariae R. (2017). Psychoneuroimmunology of psoriasis. *Dermatology and Therapy*, 7(1): 1–15.
45. Vangipuram R., Feldman S.R. (2019). Daily skin care and sun exposure in psoriasis management. *Psoriasis: Targets and Therapy*, 9: 19–27.
46. Frankel H.C., Han J. (2018). Exercise and sleep in inflammatory skin diseases. *Journal of Dermatological Science*, 89(1): 77–85.
47. Choudhary N., et al. (2020). Herbal medicines in the treatment of psoriasis. *Journal of Ethnopharmacology*, 261: 113–231.
48. Panda S., Kar A. (2022). Phytochemicals with anti-psoriatic potential: a review. *Pharmacognosy Reviews*, 16(32): 124–138.
49. Sharma M., Gupta S. (2021). Natural remedies for psoriasis: an overview. *Journal of Ayurveda and Integrative Medicine*, 12(4): 567–579.
50. Jaiswal P., Mishra S. (2020). Key herbal and natural ingredients in psoriasis management. *Indian Journal of Pharmaceutical Sciences*, 82(6): 1032–1042.
51. Strober B.E., Ryan C., van de Kerkhof P. (2020). Biologic therapies for psoriasis: recent progress. *Dermatology and Therapy*, 10(1): 15–30.
52. Reich K., Papp K.A. (2022). Small molecule therapies in psoriasis. *Expert Opinion on Pharmacotherapy*, 23(3): 273–283.
53. Kaur G., Singh D. (2023). Nanoformulations for psoriasis treatment: a review. *International Journal of Nanomedicine*, 18: 2021–2038.
54. Zhou X., et al. (2024). Artificial intelligence and personalized dermatology in psoriasis. *Frontiers in Artificial Intelligence*, 7: 142–156.
-

55. Marie D., et al. (2023). Integrative approaches combining psychological and medical care in psoriasis management. *Journal of Integrative Dermatology*, 5(2): 145–160.
56. Ali F., Bano S. (2022). Emerging therapeutic strategies in psoriasis: biologics and nanomedicine. *Pharmaceutical Nanotechnology*, 10(2): 67–79.
57. Chen Y., et al. (2021). Gene editing and regenerative therapy for psoriasis. *Molecular Therapy*, 29(8): 2209–2223.
58. Patel R., et al. (2023). Smart nanocarriers in psoriasis treatment. *Drug Delivery Letters*, 13(3): 155–164.
59. Singh P., Gupta A. (2025). Future prospects in psoriasis management: holistic and molecular perspectives. *Journal of Advanced Clinical Research Insights*, 12(1): 45–62.

