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Review Article

Herbal Approaches to Psoriasis: Insights into Medicinal Plants and Their Mechanism of Action

Shital Shinde*, Pranit Sabale, Anisha Ghadage, Samiksha Patil, Shardul Kulkarni

Annasaheb Dange College of B Pharmacy, Ashta, Sangli, Maharashtra, India

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ABSTRACT

Excessive keratinocyte proliferation and the development of erythematous plaques with silvery scales are the hallmarks of psoriasis, a chronic immune-mediated inflammatory dermatological disorder, and associated systemic inflammation. The disorder affects nearly 2–3% of the worldwide population and arises from a complex interplay of genetic predisposition and environmental triggers. Its pathophysiology is primarily driven by immune system dysfunction, involving the activation of T helper cell subsets such as Th1, Th17, and Th22. The activation of immune pathways causes elevated secretion of pro-inflammatory cytokines like IL-17, IL-23, and TNF- α . Conventional treatment modalities—such as topical corticosteroids, vitamin D analogues, retinoids, systemic immunosuppressive agents, biologic therapies, and phototherapy—are effective in controlling disease symptoms; however, their clinical utility is frequently constrained by adverse effects, high costs, and the requirement for prolonged therapy. These limitations have prompted growing interest in herbal and naturally derived therapeutic options that possess anti-inflammatory, antioxidant, and antiproliferative activities, often with superior safety profiles. Several medicinal plants, including Aloe vera, Curcuma longa (turmeric), Azadirachta indica (neem), Capsicum annum (capsaicin), and Mahonia aquifolium (Oregon grape), have demonstrated beneficial effects in attenuating psoriatic inflammation and lesion severity. Novel nano-herbal drug delivery systems like liposomes, ethosomes, and nanostructured lipid carriers have shown promise in enhancing bioavailability, improving site-specific delivery, and decreasing systemic toxicity. The convergence of traditional herbal medicine with contemporary nanotechnological approaches represents a novel, patient-centric strategy for psoriasis management. Nonetheless, comprehensive clinical evaluations, formulation standardization, and rigorous regulatory approval are required to establish therapeutic efficacy, safety, and reproducibility, thereby facilitating the integration of evidence-based herbal interventions into mainstream psoriasis treatment.

***Corresponding Author:** Shital Shinde

Address: Annasaheb Dange College of B Pharmacy, Ashta, Sangli, Maharashtra, India

Email ✉: shinde.shitaladcbp@gmail.com

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INTRODUCTION

Psoriasis is a persistent autoimmune skin disorder that predominantly manifests as erythematous, scaly plaques, excessive keratinocyte development, and severe inflammation.^[1] Psoriasis can occasionally coexist with metabolic syndromes, which are characterized by elevated blood pressure, elevated cholesterol, problems from diabetes, and weight gain.^[2] These conditions increase the risk of atherosclerosis and cardiovascular mortality in people with psoriasis. Psoriasis can be made worse by infection, trauma, irritation (cuts, burns, rashes, or insect bites), and autoimmune conditions like rheumatoid arthritis.^[3]

Psoriasis affects 2% to 3% of people worldwide, though it varies by race, area, and environmental factors.^[4] More than 80 susceptibility loci have been found, most notably involving genes of the major histocompatibility complex (MHC), particularly HLA-Cw6.^[5] Environmental factors that can exacerbate the illness include infections, stress, trauma, and certain drugs, suggesting a multifactorial root cause.^[6] From a pathophysiological perspective, immune system dysregulation—more precisely, the overactivation of T-helper 1 (Th1), Th17, and Th22 cells—causes psoriasis. These cells produce Cytokines involved in inflammation include IL-17, IL-23, and TNF- α .^[7] These cytokines promote keratinocyte hyperproliferation and sustain chronic inflammation. Despite numerous improvements in topical, systemic, and biologic therapy, psoriasis is still incurable and sometimes requires long-term care. Current research focuses on new therapeutic approaches, including biologics, small-molecule inhibitors, and traditional herbal therapies, to achieve more efficacy with fewer side effects.^[8] Therefore, developing more effective and individualized treatment plans requires an

understanding of the intricate immunopathology and therapeutic modalities of psoriasis.

Although psoriasis can develop anywhere, it is most frequently found on the scalp, the sacral region, the extensor sides of the extremities, and the dorsum of the elbows and knees. On both sides, it is symmetrical. Psoriasis frequently also affects the areas around the fingernails and toenails. Histopathologically, psoriasis lesions show increased angiogenesis, rete ridges, immune-cell infiltration in the dermis, and epidermal acanthosis.^[9]

1.1 Types of Psoriasis [10]

1) Plaque Psoriasis :

The most frequently observed form of psoriasis is plaque psoriasis, which affects nearly 80–90% of cases. It is characterized by sharply bordered red lesions with silvery-white scales resulting from keratinocyte accumulation. These lesions most frequently occur on the elbows, knees, scalp, and lumbosacral region and are often associated with pruritus.

2) Inverse Psoriasis:

Inverse psoriasis primarily affects intertriginous areas, including the axillae, umbilical region, and inframammary folds. It presents as smooth, erythematous lesions with minimal or absent scaling due to the moist environment of skin folds.

3) Guttate Psoriasis:

Guttate psoriasis commonly develops following streptococcal infections, particularly of the upper respiratory tract. This condition is characterized by the presence of multiple small, erythematous, teardrop-shaped scaly lesions and is predominantly observed in children and young adults.



4) Pustular Psoriasis:

This form of psoriasis is an immune-mediated inflammatory disease in which sterile yellowish pustules develop over erythematous plaques. It is considered a variant of psoriasis vulgaris and may present in localized or generalized forms, reflecting varying clinical patterns and distribution.

5) Erythrodermic Psoriasis:

Erythrodermic psoriasis is a rare but severe form that involves more than 90% of the body surface area. It is characterized by widespread erythema, extensive desquamation, and significant disruption of normal skin function.

6) Sebopsoriasis:

Sebopsoriasis predominantly affects the scalp and facial regions. Clinically, it manifests as erythematous plaques with greasy, yellowish scales and exhibits overlapping features of both psoriasis and seborrheic dermatitis.

7) Nail Psoriasis:

Nail psoriasis results in various nail abnormalities, including discoloration and structural changes affecting both fingernails and toenails.

Body Sites Commonly Affected by Psoriasis ^[11]

Psoriatic lesions may occur on any area of the body. However, commonly affected sites include the elbows, knees, scalp, lower back, facial region, oral mucosa, fingernails and toenails, as well as the palms and soles. In the majority of patients, the disease remains localized to limited areas of skin.



Elbows and knees



Facial area and within the oral cavity.



Scalp



Fingernails and toenails

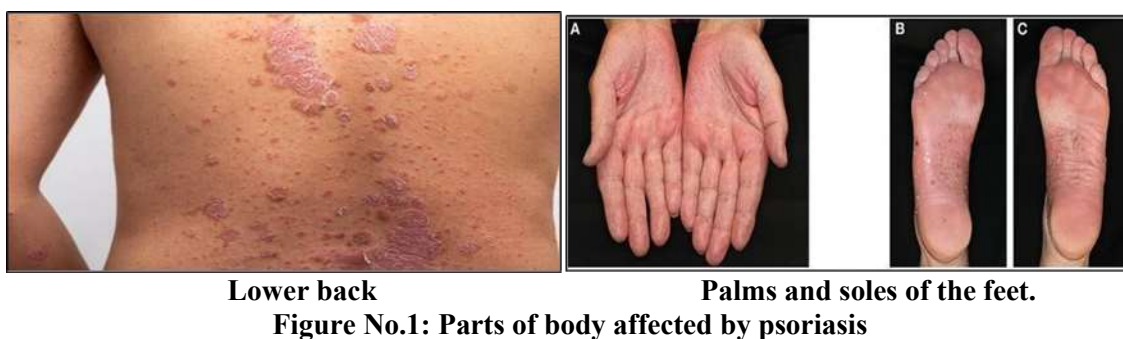


Figure No.1: Parts of body affected by psoriasis

1.2 Psoriasis Symptoms

Typical signs and symptoms include:

1. Red areas of skin that have thick, silvery scales covering them.
2. Tiny scaling areas.
3. Dry, cracked skin that could itch or bleed.
4. Soreness, burning, or itching

2. Pathophysiology Of Psoriasis

Psoriasis is characterized by immune system dysregulation involving aberrant activation of T lymphocytes, particularly Th1 and Th17 subsets. Activated immune cells release high levels of Pro-inflammatory cytokines such as IL-17, TNF- α , and IFN- γ induce keratinocyte hyperactivation, resulting in increased production of chemokines and adhesion molecules that facilitate the

migration of inflammatory cells, including neutrophils and dendritic cells, into the epidermis and dermis.

Prolonged keratinocyte activation, accompanied by the accumulation of immune cells, leads to the development of psoriatic plaques, which clinically present as thickened, erythematous, and scaly lesions. In addition to immune-mediated inflammation, psoriasis is associated with impaired skin barrier function, which enhances antigen penetration and further intensifies inflammatory responses. Moreover, disturbances in epidermal differentiation and proliferative signaling pathways result in abnormal keratinocyte maturation and excessive cornification. These pathological alterations collectively contribute to epidermal hyperplasia and the persistent formation of psoriatic plaques.[12]

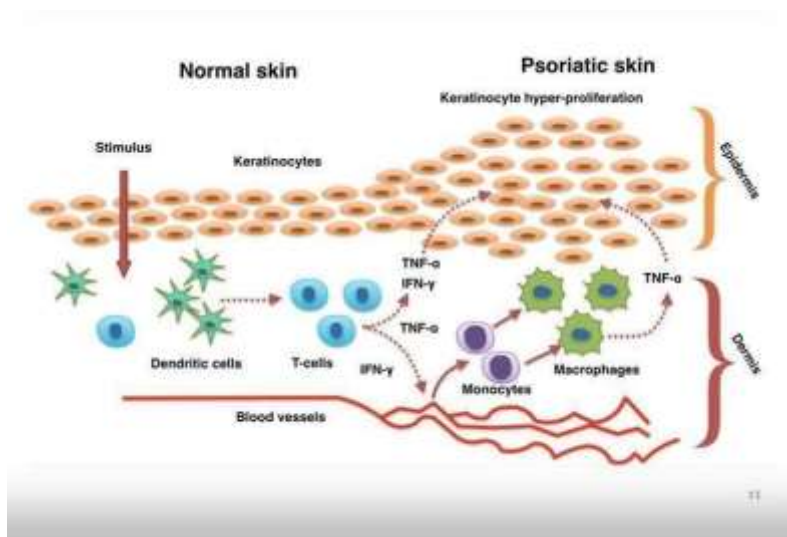


Figure no2: Pathophysiology of psoriasis

2.1 Current Treatment for Psoriasis [13]

Following the clinical diagnosis of psoriasis—primarily through physical examination and, in rare instances, confirmed by skin biopsy—therapeutic management is initiated based on disease severity.

Although Psoriasis is a long-lasting skin disease that cannot be completely cured. condition, it can be effectively controlled using appropriate therapeutic interventions. The primary treatment modalities include topical therapy, systemic therapy, and phototherapy, which aim to manage cutaneous manifestations and reduce disease progression.

2.1.1 Topical Treatment

Topical treatment is considered the first choice for managing mild to moderate psoriasis and includes the use of creams, ointments, gels, and lotions applied directly to the skin. Salicylic acid-based formulations are widely used to eliminate psoriatic scales by enhancing skin shedding, whereas topical corticosteroids are effective in relieving inflammation and pruritus. Coal tar preparations help suppress abnormal keratinocyte proliferation. During exacerbations of the disease, topical retinoids and vitamin D₃ analogues, including calcipotriene (Dovonex, Soriplus) and calcitriol (Vectical), are employed to reduce plaque thickness. Emollients and moisturizers are routinely advised to maintain skin hydration and strengthen the skin barrier. Nevertheless, in cases of moderate to severe psoriasis affecting more than 5–10% of the body surface area, topical treatment alone is generally insufficient.

2.1.2 Systemic Treatment

Systemic therapy is recommended for patients with moderate to severe psoriasis or those

unresponsive to topical treatment. Common systemic agents include retinoids, immunosuppressive drugs, phosphodiesterase-4 (PDE-4) inhibitors, and biologic agents. Retinoids such as acitretin decrease plaque formation and scaling by suppressing abnormal keratinocyte proliferation and differentiation. Immunosuppressants like methotrexate inhibit dihydrofolate reductase, exerting anti-inflammatory, antiproliferative, and immunomodulatory effects, whereas cyclosporine suppresses T-cell activation, thereby regulating cytokine production and keratinocyte growth. Apremilast (Otezla), a PDE-4 inhibitor, reduces inflammation by modulating cytokines such as tumour necrosis factor- α (TNF- α) and interleukin-23 (IL-23).

Biologic treatments are designed to specifically target immune mediators involved in the development of psoriasis. Drugs such as etanercept, infliximab, adalimumab, and certolizumab exert their effects by suppressing tumor necrosis factor-alpha (TNF- α). Other agents, including ustekinumab, guselkumab, and tildrakizumab, act by inhibiting interleukin-23 (IL-23), whereas secukinumab, ixekizumab, and brodalumab selectively block interleukin-17A (IL-17A). In situations where systemic therapies alone fail to provide sufficient disease control, phototherapy may be used as an additional treatment option.

2.1.3 Phototherapy

Phototherapy, also referred to as light therapy, involves controlled exposure to ultraviolet radiation and remains an important treatment option for psoriasis. Ultraviolet B (UVB) therapy, often combined with topical agents such as coal tar or emollients, is widely used in clinical practice. Broadband UVB is considered one of the safer phototherapeutic options; however, it requires



frequent sessions—typically three times per week over several months—to achieve clinical improvement. Narrowband UVB therapy has demonstrated superior efficacy compared to broadband UVB.

An alternative phototherapy modality is psoralen plus ultraviolet A (PUVA), where psoralen administration is followed by controlled exposure

to UVA radiation. Psoralen may be administered orally or applied topically, including via water immersion containing 8-methoxypsoralen, before UVA exposure. Although both PUVA and UVB therapies are effective in managing psoriasis, practical limitations such as treatment frequency, duration, and patient convenience often restrict their widespread use.[14]

Table No: 1 Current treatment for psoriasis (Herbal)

Sr. No.	Plant	Major Active Constituents	Common Formulations
1	<i>Aloe vera</i>	Anthraquinones, salicylic acid, acemannan	Topical gels and creams
2	Neem (<i>Azadirachta indica</i>)	Azadirachtin, nimbin, salannin, nimbandiol	Oils, pastes, creams, medicated soaps (e.g., Vedicderm Psoriasis Cream, Psoronil)
3	Capsaicin (<i>Capsicum spp.</i>)	Capsaicin, capsaisin	Topical creams, lotions, patches
4	Turmeric (<i>Curcuma longa</i>)	Curcumin, demethoxycurcumin, bisdemethoxycurcumin, curcuminoids	Oral formulations (capsules, powders); topical pastes and creams (e.g., Psoro Care, Psoronil)
5	Oregon grape (<i>Mahonia aquifolium</i>)	Berberine, berbamine, oxyacanthine, alkaloids	Topical creams and ointments (commonly 10% extract formulations)

Table No:2 Current treatment for psoriasis (synthetic)

Sr. No.	Drug Name	Mechanism of Action	Dosage Form
1	Apremilast	Apremilast acts by inhibiting phosphodiesterase-4 (PDE-4), resulting in decreased production of pro-inflammatory mediators and modulation of inflammatory cytokine levels.	Oral tablet (Otezla)
2	Brodalumab	Brodalumab is a fully human monoclonal IgG2 antibody that selectively binds to interleukin-17 receptor A (IL- 17RA), thereby blocking its interaction with IL-17A, IL- 17F, IL-17C, IL-17A/F heterodimers, and IL-25.	Injectable formulation (Siliq)
3	Calcipotriol	Calcipotriol regulates keratinocyte proliferation and differentiation without exerting cytotoxic effects, thereby reducing the number of abnormal cells present in psoriatic lesions.	Topical ointment (Taclonex)
4	Tapinarof	Tapinarof functions as an aryl hydrocarbon receptor (AhR) modulating agent. By activating and binding to AhR, it influences gene transcription involved in immune regulation and keratinocyte function.	Topical cream (Vtama)
5	Secukinumab	Secukinumab selectively binds to interleukin-17A (IL-17A), preventing its biological activity and interrupting the inflammatory cascade responsible for psoriasis progression.	Injectable formulation (Cosentyx)

3. Potential of Herbal Medicines in the Treatment of Psoriasis.

1. Psoriasis is a long-standing chronic disorder that frequently necessitates continuous, lifelong



management. Consequently, therapeutic interventions must comply with stringent standards of quality, efficacy, and patient safety, as emphasized by the World Health Organization (2016). Current treatment strategies for psoriasis predominantly rely on synthetic pharmacological agents. While these therapies are effective in providing symptomatic relief, their long-term use is often associated with a range of adverse effects that may compromise patient safety and treatment adherence.

2. Prolonged administration of corticosteroids has been linked to complications such as gastric ulceration, thinning of the skin and bones, and the development of premature cataracts. Topical vitamin D analogues may cause local skin irritation, whereas extended use of salicylic acid on the scalp has been associated with hair loss. Additionally, coal tar-based formulations can lead to skin dryness and increased photosensitivity. Phototherapy, although effective, requires strict medical supervision due to potential adverse outcomes ranging from acute skin burns to an elevated risk of skin malignancies. Therefore, continuous safety monitoring remains a critical component of psoriasis management.

3. These limitations have driven the development of advanced drug delivery systems capable of sustained drug release and reduced dosing frequency, ultimately enhancing therapeutic efficacy and patient adherence. Alongside these innovations, herbal medicines have gained increasing acceptance among patients due to their perceived safety, widespread availability, cost-effectiveness, improved adherence, and reduced incidence of side effects compared to conventional synthetic therapies. Furthermore, natural products possess substantial molecular diversity and exhibit multi-targeted mechanisms of action, making them attractive candidates for psoriasis treatment. As a

result, research efforts have increasingly focused on the development of novel herbal-based therapeutic alternatives. In recent years, the use of herbal medicine has experienced a resurgence owing to its comparatively favorable safety profile. In India, the Ministry of AYUSH serves as the regulatory authority responsible for quality control, preclinical safety assessment, and clinical evaluation of herbal medicines, supported by official guidelines such as the General Guidelines for Drug Development of Ayurvedic Formulations, which ensure the safe use of herbal therapies.[15]

4. Herbal Nano-formulations for the treatment of psoriasis disease

Conventional topical formulations—including gels, ointments, creams, tinctures, and lotions—frequently exhibit inconsistent or suboptimal therapeutic performance in the delivery of natural drugs for psoriasis management. This limitation is largely associated with disruption of the cutaneous barrier in psoriatic skin, particularly the depletion of ceramides, which compromises hydration levels and reduces the skin's capacity for water retention and absorption. Therefore, to maximize the therapeutic potential of natural medicines, topical formulations must be designed to provide enhanced skin permeability alongside effective moisture-retention properties.

A substantial proportion of herbal drugs possess poor aqueous solubility due to the hydrophobic characteristics of their bioactive constituents, leading to reduced bioavailability and rapid systemic elimination. Consequently, higher or more frequent dosing regimens are often required, thereby limiting their clinical applicability. To overcome these challenges, innovative formulation approaches are essential to improve the solubility of herbal compounds while facilitating localized and sustained drug delivery at



the target site, ultimately enhancing therapeutic efficacy and patient compliance.

In recent years, nano- and micro-particulate delivery systems have attracted significant research interest for the administration of herbal therapeutics. These advanced carriers have demonstrated considerable potential in the delivery of bioactive compounds, peptides, proteins, and drugs for various dermatological conditions. Their advantages include improved targeting efficiency, enhanced therapeutic outcomes, reduced dosing requirements, minimized toxicity, and better patient adherence. Numerous herbal active constituents have been successfully encapsulated within diverse particulate systems and subsequently evaluated through biological studies to determine their safety and effectiveness in immune-mediated skin disorders.

Moreover, emerging drug delivery technologies—such as liposomes, lipospheres, nanostructured lipid carriers, nanoemulsions, crystalline systems, nanospheres, niosomes, ethosomes, microneedles, and foam-based formulations—have shown substantial promise in enhancing dermal penetration, improving skin hydration, and enabling targeted delivery of natural therapeutics to specific inflammatory mediators or cytokine-producing cells.[16]

5. Therapeutic Medicinal Plants for Psoriasis Treatment:

Numerous herbal formulations are currently available worldwide for the management of psoriasis. Medicinal plants constitute an important foundation for pharmacological research and drug discovery, as they offer several advantages over conventional synthetic therapies, including a lower incidence of adverse effects, wide accessibility, cost-effectiveness, and improved patient

acceptability. Consequently, there is growing research interest in identifying and developing herbal-based therapeutics as viable alternatives to synthetic drugs for psoriasis treatment. The subsequent section discusses selected medicinal plants employed in psoriasis therapy, along with their principal bioactive constituents and corresponding mechanisms of action.

1. Neem:

Common Name: Neem

Botanical Name: *Azadiracta indica*

Family Name: Meliaceae

Active Constituent: Azadirachtin

Mechanism of action: Azadirachtin penetrates into the deeper layers of the skin, where it contributes to disease resolution, while vitamin E and omega-6 and omega-9 fatty acids present in neem oil provide a moisturizing effect that aids in reducing cutaneous scaling and dryness.[17]

2. Basil:

Common Name: Basil

Botanical Name: *Ocimum basilicum L*

Family Name: Lamiaceae

Active Constituent: linalool, eugenol, and rosmarinic acid

Mechanism of action: Activation of nuclear factor- κ B (NF- κ B) has been implicated in the pathogenesis of several inflammatory and immune-related disorders, including osteoporosis, psoriasis, septic shock, and acquired immunodeficiency syndrome (AIDS). Previous studies have reported that *Ocimum sanctum* (holy basil) exhibits notable chemopreventive activity.



Ursolic acid, a triterpenoid compound derived from basil and rosemary, has been shown to suppress NF- κ B signaling by inhibiting I κ B kinase (IKK), thereby downregulating the expression of cyclin D1, cyclooxygenase-2 (COX-2), and matrix metalloproteinase-9 (MMP-9).[18]

3. Garlic:

Common Name: Garlic

Botanical Name: *Allium sativum*

Family Name: Amaryllidaceae

Active Constituents: Allin, Allicin

Mechanism of action: Antioxidant activity: Onion-derived antioxidants play a crucial role in mitigating oxidative stress, a pathological factor associated with several dermatological disorders, including psoriasis. This activity supports the maintenance of overall skin integrity and health. Skin repair and regeneration: The topical use of onion extracts or juice has been reported to facilitate cutaneous healing processes and enhance the clinical appearance of psoriatic plaques.[19]

4. Apigenin:

Common Name: Apigenin

Botanical Name: *Matricaria chamomilla* L

Family Name: Apiaceae.

Active Constituents: Flavonoid apigenin

Mechanism of action: This flavonoid demonstrates multiple pharmacological activities, including antioxidant and anti-inflammatory effects, modulation of keratinocyte proliferation and differentiation, inhibition of angiogenesis, and antibacterial action. In vivo studies employing 5 μ mol of apigenin—a non-mutagenic plant-derived

flavone and a potent inhibitor of nuclear factor kappa-light-chain-enhancer of activated B cells (NF- κ B)—have shown significant reductions in interleukin-6 (IL-6) and interleukin-12 (IL-12) levels in murine models following apigenin administration. These pro-inflammatory cytokines are known to be markedly elevated in psoriatic conditions.[20]

5. Clove:

Common Name: Clove

Botanical Name: *Syzygium aromaticum*

Family Name: Myrtaceae

Active Constituents: eugenol and isoeugenol

Mechanism of action: By stabilizing I κ B, these compounds effectively block nuclear factor- κ B (NF- κ B) activation. Murakami et al. reported that bis-eugenol, in contrast to eugenol, effectively blocks I κ B degradation and consequently reduces the production of inflammatory cytokines at both transcriptional and protein expression levels.[21]

6. Fennel:

Common Name: Fennel

Botanical Name: *Foeniculum Vulgare*.

Family: Apiaceae

Active constituents: phenyl propanoids anethole and estragole

Mechanism of action: Anethole has been shown to possess significant anti-inflammatory and anticarcinogenic activities, along with pronounced antioxidant properties. Evidence from prior research demonstrates that anethole modulates NF- κ B signaling through stabilization of I κ B, resulting in reduced NF- κ B activation..[22]



7. Turmeric:

Common Name: Turmeric

Botanical Name: *Curcuma longa*

Family: Zingiberaceae

Active constituents: Curcuminoids, curcumin

Mechanism of action: Curcumin suppresses the activity of the nuclear factor kappa B (NF- κ B) protein complex, a key regulator of inflammatory processes involved in the pathophysiology of psoriasis. In addition to its anti-inflammatory effects, curcumin promotes cutaneous wound healing and enhances the regenerative capacity of the skin.[23]

8. Ginseng:

Common name: Asian ginseng, Chinese ginseng.

Botanical Name: *Panax ginseng* C

Family: Araliaceae

Active constituents: ginsenosides, polysaccharides, polyacetylenes, proteins, lipids

Mechanism of action: These compounds exhibit a broad spectrum of pharmacological activities, including antitumor, anti-inflammatory, and immunomodulatory effects against various immune-mediated disorders. Multiple studies have reported that ginsenosides, also referred to as ginseng polysaccharides (GP), modulate several intracellular signaling pathways, notably those mediated by mitogen-activated protein kinases (MAPKs) and nuclear factor- κ B (NF- κ B). The MAPK signaling cascade involves sequential activation of key kinases, including p38, extracellular signal-regulated kinase (ERK), and c-Jun N-terminal kinase (JNK). Activation of serine/threonine kinases initiates MAPK

signaling, leading to phosphorylation of p38 MAPK, which plays a critical role in both transcriptional and post-transcriptional regulatory processes responsible for the production of inflammatory mediators.[24]

9. Indigo naturalis:

Common Name: Indigo naturalis

Botanical Name: *Indigofera tinctoria*

Family: Fabaceae

Active constituents: Indirubin

Mechanism of action: Furthermore, it downregulates pro-inflammatory cytokine production, particularly TNF- α and IL-6, while simultaneously decreasing serum monocyte chemoattractant protein-1 (MCP-1) levels. In vitro studies further demonstrate that IN suppresses lipopolysaccharide (LPS)-induced production of TNF- α and IL-6. Indigo and indirubin have been identified as the principal bioactive constituents responsible for these effects.[25]

10. Calendula :

Common Name: Calendula

Botanical Name: *calendula officinalis*

Family: Asteraceae

Active constituents: quercetin

Mechanism of action: Studies have shown that extracts from *Calendula officinalis* flowers are effective in both acute and chronic inflammation. Anti-inflammatory activity has been demonstrated in carrageenan- and dextran-induced acute inflammation models, as well as in formalin-induced chronic inflammation models in mice. The effects are chiefly mediated by



downregulating pro-inflammatory cytokines, including IL-1 β , IL-6, TNF- α , and IFN- γ , as well as inhibiting COX-2 activity and reducing prostaglandin output.[26]

11. Mahonia aquifolium:

Common Name: Mahonia aquifolium

Botanical Name: Oregon grape

Family: Berberidaceae

Active constituents: berberine

Mecahnism of action: Berberine has been demonstrated to exert anti-inflammatory activity through multiple mechanisms, including inhibition of lipoxygenase activity and suppression of lipid peroxidation, thereby contributing to its immunomodulatory effects.[27]

12. Aloe vera :

Common Name: Aloe vera

Botanical Name: *aloe barbadensis*

Family: asodelaceae

Active constituents: aloin, emodin

Mechanism of action: Various parts of the plant, particularly the gel, exhibit beneficial therapeutic effects. Aloe vera gel has been reported to alleviate erythema and scaling associated with psoriasis. The phytochemical composition of Aloe vera includes anthraquinones, mucopolysaccharides, steroids, saponins, and salicylic acid. Among these constituents, anthraquinones and acemannan are considered the primary active compounds, demonstrating antibacterial properties and therapeutic relevance in the management of psoriasis. Salicylic acid also contributes to the plant's overall pharmacological activity.[28]

13. Sandalwood :

Common name : Sandalwood

Botanical name : indian sandalwood,, Santalum album

Family: santalaceae

Active constituent: α -santalo,, α -, β -, and epi- β -santalene

Mechanism of action: α -Santalol is the principal compound responsible for the characteristic aroma of sandalwood and has demonstrated the ability to inhibit specific inflammatory pathways, suggesting its potential to alleviate psoriasis-associated complications. The therapeutic profile of sandalwood oil is further supported by the presence of santalenes, including α -, β -, and epi- β -santalene. Beyond its topical effects, the aromatic properties of sandalwood are associated with calming and relaxing responses, which may provide additional benefits in the management of psoriasis.[29]

14. Tea tree oil:

Common Name: Tea tree oil

Botanical Name: *Melaleuca alternifolia*

Family: Myrtaceae

Active constituents: Terpinen-4-ol, alphaterpineol, alpha-pinene

Mechanism of action: Tea tree oil, which is rich in terpenoid compounds such as terpinen-4-ol, exhibits pronounced anti-inflammatory activity with potential relevance in psoriasis management. In addition, its broad-spectrum antimicrobial properties further enhance its therapeutic value. Tea tree oil also demonstrates immunomodulatory effects, which may contribute to regulating



immune responses, thereby limiting excessive keratinocyte proliferation and inflammatory processes characteristic of psoriatic plaques.[30]

15. Liquorice:

Common name: Liquorice

Botanical name: *Glycyrrhiza glabra*,

Family: leguminasae

Active constituent: glycyrrhizin, glycyrrhizic acid, liquiritin

Mechanism of action: Licorice contains a diverse array of bioactive constituents, including glycyrrhizin, glycyrrhizic acid, and liquiritin, all of which exhibit notable anti-inflammatory activity. These compounds collectively suppress the production of key pro-inflammatory cytokines, such as interleukin-6 (IL-6) and tumor necrosis factor- α (TNF- α), which play central roles in the pathogenesis of psoriasis. Additionally, licorice-derived constituents modulate the activation of nuclear factor kappa B (NF- κ B), a critical regulator of inflammatory signaling, thereby contributing to the protection of skin cells and supporting the healing of psoriatic lesions.[31]

16. Mango:

Common name: Mango

Botanical name: *Mangifera indica* L.

Family: mangifera

Active constituent: Mangiferin

Mechanism of action: Previous studies have demonstrated that mangiferin can suppress macrophage-mediated inflammatory responses, thereby modulating skin inflammation. This effect involves the downregulation of pro-inflammatory

mediators, including tumor necrosis factor- α (TNF- α), along with major inflammatory biomarkers, including inducible nitric oxide synthase (iNOS), interleukin-1 β , and interleukin-6, which are implicated in the pathogenesis of psoriatic lesions.[32]

17. Capsicum annuum:

Common name: Capsicum annuum

Botanical name: *Capsicum annuum* L

Family: Solanaceae

Chemical constituent: capsaicinoids, capsaicin,

Mechanism of action: Neuropeptides and neural markers, including neuropeptide Y, protein gene product 9.5 (PGP-9.5), nerve growth factor (NGF), calcitonin gene-related peptide (CGRP), and substance P (SP), have been associated with pruritus in psoriasis. Elevated levels of NGF, increased density of PGP-9.5-positive nerve fibers, and a higher number of NGF-immunoreactive keratinocytes have been observed in pruritic skin and are correlated with the severity of itch.[33]

18. Ginger:

Common Name: Ginger

Botanical Name: *Zingiber officinale*

Family: Zingiberaceae

Chemical constituents: shogaols, gingerols

Mechanism of action: Gingerol has been shown to inhibit the activity of nitric oxide synthase and cyclooxygenase-2 (COX-2), both of which are regulated by nuclear factor- κ B (NF- κ B). Additionally, gingerol may reduce platelet aggregation. Owing to its anti-inflammatory



properties, gingerol represents a promising herbal therapeutic candidate for the management of psoriasis.[34]

19. St.john's wort:

Common Name: St.john's wort

Botanical Name: *Hypericum perforatum*

Family: Hypericaceae

Chemical constituents: phloroglucinols

Mechanism of action: The primary bioactive constituents responsible for these pharmacological effects are hyperforin and adhyperforin. In addition, these compounds modulate serotonin receptor activity, influence ion channel function—potentially altering calcium and sodium flux into cells—and can affect the activity of cytochrome P450 enzymes.[35]

20. Naringin:

Common Name: Naringin

Botanical Name: *genus Citrus*.

Family: Polyphenols

Chemical constituents: flavanone naringenin, disaccharide neohesperidose

Mechanism of action: Naringin, a flavonoid predominantly found in citrus fruits such as grapefruit and oranges, as well as in cooked tomato paste, cherries, beans, and oregano, exhibits several therapeutic effects relevant to psoriasis. Its pharmacological actions include anti-inflammatory activity and inhibition of chemokine production. In a clinical investigation with 20 subjects, Deenonpoe found that naringin effectively decreased the production of pro-inflammatory cytokines TNF- α and IL-6, which

are central to psoriasis-related immune dysregulation.[36]

CONCLUSION

Psoriasis remains an incurable yet manageable disease that requires long-term, patient-centric therapy. Although modern synthetic and biologic treatments effectively control symptoms, their limitations such as high cost, adverse effects, and recurrence highlight the need for safer alternatives. Herbal medicines, with their multidirectional mechanisms, natural bioactive compounds, and favorable safety profiles, have emerged as valuable options in psoriasis management. The integration of nanotechnology with herbal formulations further enhances therapeutic outcomes by improving drug solubility, skin penetration, and sustained release. However, rigorous preclinical and clinical evaluations, standardization of herbal extracts, and regulatory validation are essential to translate these natural remedies into reliable, evidence-based therapies. Future research should focus on understanding molecular mechanisms, developing novel delivery systems, and ensuring quality control to establish herbal medicine as a scientifically grounded, effective treatment strategy for psoriasis.

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