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Anti-inflammatory effects of herbal medicines in psoriasis: A comprehensive review of mechanisms and therapeutic potential

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Abstract

Psoriasis is a chronic, immune-mediated inflammatory skin disorder marked by erythema, scaling, and keratinocyte hyperproliferation. While conventional therapies such as corticosteroids and biologics provide symptomatic relief, their long-term use is limited by side effects and poor compliance, prompting interest in safer, plant-based alternatives. This review critically examines the anti-inflammatory effects of herbal medicines in psoriasis management, highlighting their mechanisms of action, preclinical and clinical evidence, and regulatory considerations. A comprehensive literature search of studies up to May 2025 revealed that several herbs—including Curcuma longa (curcumin), Camellia sinensis (EGCG), With Ania somniferous (withaferin A), Berberis vulgaris (berberine), and Scutellarin baicalinase (baicalin)—exert therapeutic effects by targeting key inflammatory pathways such as NF-dB, JAK/STAT, MAPKs, and the IL-17/IL-23 axis. Clinical trials have reported improvements in Psoriasis Area and Severity Index (PASI) scores and lesion clearance with minimal adverse effects. Nonetheless, challenges such as lack of standardization, potential herb–drug interactions, and inconsistent regulatory frameworks limit their widespread adoption. Overall, herbal medicines show significant promise as complementary or alternative options in psoriasis treatment, warranting further high-quality clinical trials and global efforts toward regulatory harmonization.

Keywords: Psoriasis; Herbal Medicine; Anti-Inflammatory; Phytotherapy; Immunomodulation; Curcumin; EGCG; Clinical Trials; NF-KB; IL-17; Traditional Medicine

1. Introduction

1.1. Overview of Psoriasis

Psoriasis is a chronic, immune-mediated inflammatory skin disorder characterized by erythematous, scaly plaques, primarily affecting the scalp, elbows, knees, and lower back. It is a multifactorial disease influenced by genetic, immunological, and environmental factors. The most common form is plaque psoriasis (psoriasis vulgaris), but other variants include guttate, pustular, erythrodermic, and inverse psoriasis. Psoriasis is not merely a skin disorder; it is associated with several systemic comorbidities, including psoriatic arthritis, metabolic syndrome, cardiovascular disease, and depression.[1]

1.2. Pathophysiology

Psoriasis is a chronic, immune-mediated inflammatory skin disease that predominantly affects the skin and sometimes the joints. It is characterized by epidermal hyperproliferation, abnormal keratinocyte differentiation, and a prominent inflammatory infiltrate, primarily involving T lymphocytes and dendritic cells [1,2,3].

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At the core of its pathogenesis is immune system dysregulation, where dendritic cells activate naïve T cells into Th1, Th17, and Th22 subsets, promoting the release of pro-inflammatory cytokines such as TNF-α, IL-17, IL-23, and IL-22. These cytokines stimulate keratinocytes, perpetuating a cycle of inflammation and epidermal remodeling [1,3].

The IL-23/IL-17 axis is now recognized as central to the disease mechanism, leading to recruitment of neutrophils and formation of Munro microabscesses, a histopathological hallmark of psoriasis [4]. Additionally, signaling pathways like NF-κB, JAK/STAT, and MAPK play a vital role in the upregulation of cytokines and chemokines [3,5].

Psoriasis is also influenced by genetic predisposition, with HLA-C*06:02 being the most strongly associated allele in early-onset psoriasis [2]. Environmental triggers such as infections, stress, certain medications, and trauma can initiate or exacerbate the condition in genetically susceptible individuals.

Psoriasis affects approximately 2–3% of the global population, making it one of the most prevalent chronic inflammatory skin disorders [6]. The prevalence varies by geographic region, ethnicity, and age group. For instance, the disease is more common in Northern Europe (up to 4.8%) and less prevalent in Asia and Sub-Saharan Africa (<0.5%) [7].

In the United States, around 7.5 million individuals are affected, accounting for approximately 2.2% of the population [6]. The disease exhibits a bimodal age of onset, with peaks at 15–25 years and 50–60 years [1]. Although both sexes are equally affected, women often experience earlier onset compared to men.

Plaque psoriasis (psoriasis vulgaris) is the most common clinical subtype, comprising more than 80% of all cases. Other forms include guttate, pustular, inverse, and erythrodermic psoriasis [1].

2. Herbal Anti-Inflammatory Mechanisms in Psoriasis

Given the central role of inflammation and immune dysregulation in the pathogenesis of psoriasis, targeting pro-inflammatory cytokines and immune pathways presents a promising therapeutic strategy. Several herbal medicines and phytochemicals exhibit potent anti-inflammatory, immunomodulatory, and antioxidant effects, making them potential adjuncts or alternatives to conventional treatments.

2.1. Modulation of Pro-inflammatory Cytokines

Many medicinal herbs downregulate cytokines central to psoriatic inflammation, particularly the IL-23/IL-17 axis and TNF-α:

- **Curcumin**, the active compound in *Curcuma longa*, inhibits the production of **IL-17**, **IL-22**, **TNF-α**, and **IL-1β** by suppressing **NF-κB** activation [8,9].
- **Resveratrol**, from *Polygonum cuspidatum* and grapes, reduces **Th17 cell differentiation** and **IL-6/IL-23 signaling**, key elements in psoriasis pathology [10].
- **Berberine**, found in *Berberis aristata*, inhibits **IL-6** and **TNF-α**, modulates **Treg/Th17 balance**, and suppresses **MAPK and STAT3 pathways** [11].

By dampening these cytokine networks, such herbs interrupt the self-perpetuating inflammatory cycle in psoriasis.

2.2. Inhibition of Key Signaling Pathways

Herbal bio actives exert their effects by interfering with intracellular signaling cascades crucial for inflammatory gene expression

- **NF-κB Pathway**: Central to psoriasis, this pathway is targeted by numerous herbs. Curcumin, **Boswellic acids** (from *Boswellia serrata*), and **Withaferin A** (from *Withania somnifera*) all inhibit NF-κB translocation to the nucleus, thereby reducing inflammatory gene transcription [8,12].
- **JAK/STAT Pathway**: Resveratrol and **Epigallocatechin gallate (EGCG)** from green tea inhibit **STAT3 phosphorylation**, a key event in Th17 cell activation and keratinocyte hyperproliferation [10,13].
- **MAPK Pathway**: Compounds such as **Apigenin** (found in *Matricaria chamomilla*) downregulate MAPK signaling, reducing cytokine production and oxidative stress [14].

2.3. Antioxidant and Anti-proliferative Effects

Oxidative stress is a contributor to psoriatic inflammation and keratinocyte damage. Several herbs exert ROS-scavenging activity and restore redox balance

- Aloe vera, rich in polyphenols and vitamins, reduces lipid peroxidation and enhances superoxide dismutase (SOD) and glutathione peroxidase (GPx) activity [15].
- Emblica officinalis (Amla) exhibits strong antioxidant effects that complement its anti-inflammatory properties, potentially reducing oxidative DNA damage in psoriatic lesions [16].
- Moreover, many herbs also possess anti-proliferative activity, reducing epidermal hyperplasia, a hallmark of psoriasis.

2.4. Immunomodulatory Effects

Rather than simple immunosuppression, herbs often modulate the immune system, restoring immune balance

- Glycyrrhiza glabra (Licorice) promotes Treg activity while reducing Th17 polarization [17].
- Nigella sativa (Black seed) exhibits both anti-inflammatory and immunoregulatory properties by altering cytokine profiles and suppressing histamine release [18].

These immunoregulatory effects help in long-term control without the adverse effects commonly associated with synthetic immunosuppressants.

2.5. Synergistic Potential with Conventional Drugs

Some herbal compounds enhance the efficacy or reduce the toxicity of conventional anti-psoriatic drugs

- **Curcumin** and **methotrexate** show **synergistic anti-inflammatory activity** in experimental models, allowing for lower doses of methotrexate with reduced toxicity [19].
- **Boswellia extracts** have been used alongside corticosteroids and demonstrated improved outcomes in psoriatic arthritis [12].

2.6. Anti-Inflammatory Herbal Medicines in Psoriasis: Active Compounds and Molecular Targets

Table 1 Herbal Medicines with Anti-psoriatic Potential and Their Molecular Mechanisms

Herbal Source	Active Compound(s)	Molecular Target(s)	Key Effects in Psoriasis	References
Curcuma longa (Turmeric)	Curcumin	NF-κB, IL-17, IL-23, TNF-α, MAPK	↓ Cytokines, ↓ keratinocyte proliferation	[20,21]
Boswellia serrata	Boswellic acids	5-LOX, TNF-α, NF-κB	Anti-inflammatory, used in psoriatic arthritis	[22,23]
Glycyrrhiza glabra	Glycyrrhizin, liquiritigenin	Th17/Treg balance, IL-6, COX-2	Immunomodulation, ↓ scaling & erythema	[23]
Nigella sativa	Thymoquinone	IL-1β, IL-6, TNF-α, ROS	Antioxidant, ↓ cytokines	[24]
Aloe vera	Polysaccharides, aloin	ROS, SOD, GPx, IL-8	Wound healing, anti-oxidative, ↓ skin inflammation	[25]
Withania somnifera	Withaferin A	NF-κB, TNF-α, IL-1β	↓ keratinocyte proliferation, anti-inflammatory	[26]
Emblica officinalis	Ascorbic acid, tannins	ROS, TNF-α, IL-6	Antioxidant, anti-inflammatory	[27]
Camellia sinensis	EGCG (Epigallocatechin gallate)	STAT3, IL-17, IL-22	↓ epidermal thickness, Th17 inhibition	[28]

Polygonum cuspidatum	Resveratrol	IL-23, IL-6, STAT3	↓ dendritic cell activation, anti-proliferative	[29]
Berberis aristata	Berberine	MAPK, STAT3, IL-6, TNF-α	Immunoregulation, ↓ epidermal hyperplasia	[31]

2.7. Mechanisms of Anti-Inflammatory Effects of Herbal Medicines in Psoriasis

Herbal medicines exert their anti-psoriatic effects through diverse mechanisms that modulate immune signaling, oxidative stress, keratinocyte activity, and cytokine production. This section outlines the major cellular and molecular pathways targeted by bioactive herbal compounds.

2.8. Suppression of Pro-Inflammatory Cytokines

Psoriasis is characterized by elevated levels of pro-inflammatory cytokines such as IL-17, IL-23, TNF-α, and IL-6, which drive the Th17 and Th1 immune responses. Several phytochemicals have demonstrated the ability to downregulate these cytokines:

- **Resveratrol** (from Polygonum cuspidatum) inhibits IL-23-mediated dendritic cell activation and reduces IL-17 secretion by T cells [32-34].
- **Baicalin** (from Scutellaria baicalensis) inhibits IL-17/IL-23 axis and TNF-α expression, thereby alleviating psoriasis-like inflammation [33].

2.9. Inhibition of NF-κB, JAK/STAT, and MAPK Signaling Pathways

Key intracellular pathways such as NF-κB, MAPK, and JAK/STAT mediate inflammatory gene transcription in psoriatic skin.

- **Withaferin A** (from *Withania somnifera*) inhibits STAT3 phosphorylation and NF-κB nuclear translocation, reducing psoriatic plaque formation [35].
- **Apigenin** (from *Matricaria chamomilla*) inhibits p38 MAPK and NF-κB signaling, leading to decreased keratinocyte proliferation and inflammatory mediator release [36].

2.10. Antioxidant and Free Radical Scavenging Effects

Oxidative stress plays a key role in keratinocyte hyperproliferation and inflammatory cytokine expression in psoriasis.

- **Quercetin** (from *Allium cepa*, *Ginkgo biloba*) neutralizes reactive oxygen species (ROS) and downregulates NF-κB signaling in skin inflammation models [38].
- **Epigallocatechin gallate (EGCG)** (from green tea) reduces oxidative damage in psoriatic lesions and improves skin barrier function [39].

2.11. Immunomodulation and T Cell Regulation

The dysregulation of Th17, Th1, and Treg cells contributes to the chronicity of psoriasis.

- **Berberine** (from *Berberis vulgaris*) has been shown to rebalance Th17/Treg ratios by modulating dendritic cell maturation [40].
- **Apigenin** suppresses Th17 cell differentiation and upregulates regulatory T cells, restoring immune balance [41].

2.12. Inhibition of Keratinocyte Hyperproliferation

Hyperproliferation and abnormal differentiation of keratinocytes are hallmarks of psoriatic skin.

- **Shikonin** (from *Lithospermum erythrorhizon*) induces keratinocyte apoptosis via caspase-3 activation and halts the cell cycle in G0/G1 phase [42].
- **Gami-Yukmijihwang-tang**, a multi-herb formulation, inhibits excessive keratinocyte growth and reduces psoriatic inflammation [43].

3. Synergistic Use of Herbal and Conventional Therapies in Psoriasis

The integration of herbal medicine with conventional treatment for psoriasis has gained interest due to the potential to enhance therapeutic efficacy, reduce dosage-related toxicity, and overcome drug resistance. While conventional therapies such as corticosteroids, methotrexate, and biologics target immune pathways directly, herbal medicines can modulate oxidative stress, inflammatory cytokines, and keratinocyte behavior through complementary mechanisms.

3.1. Rationale for Combination Therapy

- **Reduced Side Effects:** Combining herbs with standard drugs may allow lower dosages of pharmaceuticals, reducing risks like hepatotoxicity (e.g., with methotrexate).
- **Targeting Multiple Pathways:** Herbal compounds such as curcumin, boswellic acids, and resveratrol have **multi-target actions** that can complement single-pathway biologics.
- **Improved Patient Compliance:** Natural therapies often appeal to patients seeking holistic or "chemical-free" treatments, potentially improving long-term adherence.

3.2. Evidence from Studies

3.2.1. Curcumin + Topical Corticosteroids

- Topical application of curcumin with corticosteroids showed **enhanced reduction of erythema and scaling**, with no significant adverse events.
- Curcumin inhibits **NF-κB**, a central pathway also modulated by corticosteroids, allowing synergism. [64-65]

3.2.2. Aloe vera + Calcipotriol

- In a split-body clinical trial, Aloe vera gel combined with calcipotriol improved **moisture retention** and reduced **scaling severity** more effectively than calcipotriol alone. [66-68]

3.2.3. Boswellia + NSAIDs or DMARDs

In psoriatic arthritis, Boswellia extract used as an adjunct to conventional DMARDs reduced joint pain, morning stiffness, and inflammatory markers.[69]

3.2.4. Resveratrol + Methotrexate (Preclinical)

- In murine models, combination therapy enhanced anti-inflammatory efficacy and minimized hepatotoxicity induced by methotrexate.[70]

3.3. Challenges and Considerations

- **Herb–Drug Interactions:** Some herbal medicines can influence CYP450 enzymes, potentially altering the metabolism of biologics and immunosuppressants.
- **Standardization:** Lack of consistency in herbal formulations can lead to variable clinical outcomes.
- **Regulatory Gaps:** Few herbal drugs are approved under standardized pharmaceutical guidelines, limiting their integration into evidence-based practice.[71]

Table 2 Synergistic Use of Herbs with Conventional Therapies

Herbal Agent	Combined With	Observed Outcome	Reference
Curcumin	Topical corticosteroids	Enhanced anti-inflammatory effects	Kurd SK et al., 2008
Aloe vera	Calcipotriol	Improved hydration, reduced scaling	Paulsen E et al., 2005
Boswellia serrata	NSAIDs/DMARDs	Reduced joint pain and systemic inflammation	Sengupta K et al., 2008
Resveratrol	Methotrexate (preclinical)	Increased efficacy, reduced hepatotoxicity	Fouad AA et al., 2013

4. Limitations and Safety Concerns of Herbal Medicine in Psoriasis

While herbal medicines offer promising anti-inflammatory and immunomodulatory properties, their clinical adoption is hindered by several limitations. Addressing these challenges is essential to ensure safe and effective integration with conventional therapies.

4.1. Variability and Lack of Standardization

- **Inconsistent Composition:** Herbal products often vary in active ingredient content due to differences in **plant species, cultivation conditions, harvesting methods**, and extraction techniques.
- **Quality Control Issues:** Adulteration, contamination with heavy metals, pesticides, or microbial agents can compromise safety.
- **Example:** Studies on *St. John's Wort* show wide variability in hypericin concentration, affecting reproducibility of results. [72-75]

4.2. Herb–Drug Interactions

- Certain herbal compounds can **induce or inhibit cytochrome P450 enzymes**, impacting the metabolism of systemic psoriasis treatments.
- **Curcumin**, for example, may inhibit CYP3A4, potentially increasing serum levels of immunosuppressants like cyclosporine.
- **Ginkgo biloba** and **St. John's Wort** have been reported to interact with **methotrexate** and **biologics**, altering their pharmacokinetics. [76-78]

4.3. Dosage, Bioavailability, and Formulation Challenges

- Many herbal actives (e.g., curcumin, resveratrol) have **poor oral bioavailability**, requiring **nanocarriers, liposomes, or pipeline co-administration** to enhance absorption.
- Inconsistent dosing regimens across studies limit the establishment of **standard therapeutic doses**. [81-82]

4.4. Regulatory and Legal Barriers

- Herbal medicines are regulated differently across regions (e.g., **dietary supplements in the US**, **traditional medicines in India/China**), leading to
  - Inconsistent clinical use
  - Limited pharmacovigilance
  - Minimal post-market surveillance.

5. Challenges and Future Directions

Despite promising preclinical and clinical findings, the integration of herbal medicines into mainstream psoriasis treatment faces several challenges. These obstacles span from scientific validation to regulatory acceptance, but they also point toward crucial areas for future research.

5.1. Current Challenges

- Limited High-Quality Clinical Trials
- Incomplete Mechanistic Understanding

Although anti-inflammatory and immunomodulatory effects are documented, molecular pathways involved (e.g., JAK/STAT, NF-κB, IL-17 axis) require further elucidation through systems biology and omics approaches

- Regulatory Gaps

Global disparities in regulation and a lack of harmonized standards complicate clinical adoption and global marketing of herbal products.

5.2. Future Directions

5.2.1. Clinical Validation

There is an urgent need for large, multi-center randomized controlled trials (RCTs) evaluating standardized herbal formulations, with long-term follow-ups for efficacy and safety.

5.2.2. Mechanistic Studies

Advanced molecular studies—including transcriptomics, proteomics, metabolomics, and network pharmacology—are required to identify targets and optimize formulations.

6. Conclusion

Psoriasis remains a challenging chronic inflammatory skin disease that demands long-term management strategies with minimal adverse effects. The limitations of current pharmacological therapies, including toxicity and immune suppression, have driven increasing interest in herbal medicines due to their multitargeted anti-inflammatory, antioxidant, and immunomodulatory properties.

A wide range of herbal compounds—including *Curcumin*, *Resveratrol*, *Baicalin*, *Apigenin*, *Withaferin A*, *Berberine*, and *Green Tea Catechins*—have demonstrated promising preclinical efficacy by modulating crucial inflammatory pathways such as NF-dB, MAPKs, and IL-17/IL-23 axes. Some have even advanced to clinical trials, showing improvements in erythema, scaling, and lesion severity without serious side effects.

However, the full integration of herbal therapies into conventional dermatological practice is hampered by issues such as lack of standardization, limited clinical validation, safety concerns, and regulatory inconsistencies. Addressing these limitations through rigorous clinical trials, molecular mechanistic studies, and global regulatory harmonization is vital for advancing herbal medicines from alternative to evidence-based mainstream therapy.

In the future, a holistic and integrative approach, combining the strengths of herbal medicine with conventional treatment, has the potential to improve clinical outcomes, reduce side effects, and offer personalized care in psoriasis management.

Compliance with ethical standards

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