Psoriasis: A Narrative Review of Pathogenesis and Herbal versus Conventional Therapy

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***Abstract*— Psoriasis is a chronic immune-mediated inflammatory skin disorder characterized by keratinocyte hyperproliferation and immune dysregulation. The condition significantly affects quality of life and is associated with systemic comorbidities such as metabolic syndrome and cardiovascular disease. Conventional therapies including corticosteroids, retinoids, coal tar, dithranol, and vitamin D analogues remain widely used but are often limited by adverse effects and recurrence. Increasing attention has been directed toward herbal therapies due to their multi-targeted pharmacological actions and improved safety profiles. Medicinal plants such as Aloe vera, Curcuma longa, Wrightia tinctoria, Nigella sativa, Mahonia Aqui folium, and Indigo naturalis demonstrate anti-inflammatory, antioxidant, and immunomodulatory activities relevant to psoriasis management. This review highlights the pathogenesis of psoriasis and compares herbal and conventional therapeutic approaches.**

***Index Terms*—Curcumin, Dermatology, Herbal therapy, Psoriasis, Wrightia tinctoria**

I. INTRODUCTION

Psoriasis is a chronic inflammatory dermatosis characterized by erythematous plaques covered with silvery-white scales and affects approximately 2–3% of the global population [18] The disease arises from genetic predisposition, environmental triggers, and immune dysregulation.

Immune activation involving dendritic cells and T helper (Th1 and Th17) cells leads to overproduction of pro-inflammatory cytokines such as TNF-α, IL-17, and IL-23, which promote keratinocyte hyperproliferation and abnormal differentiation [13,18].

Psoriasis is now considered a systemic inflammatory disorder associated with comorbidities including psoriatic arthritis, metabolic syndrome, and cardiovascular disease [18] While conventional treatments remain effective, their limitations have encouraged exploration of alternative therapies including herbal medicine.

II. PATHOGENESIS OF PSORIASIS

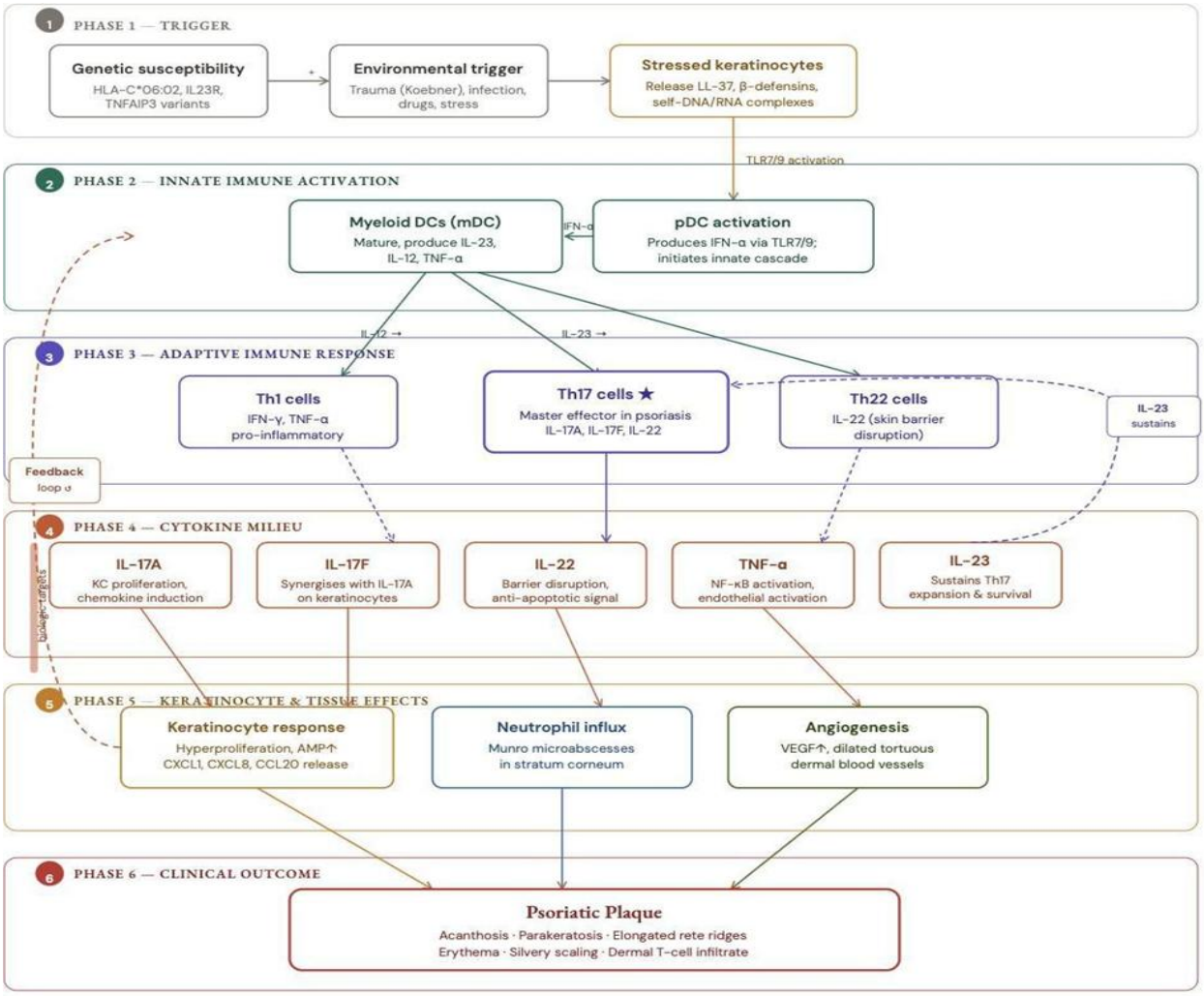

Fig.1. Pathogenesis of Psoriasis [54,55,56,57,58]

Psoriasis is a chronic immune-mediated disorder characterized by dysregulated interactions between the innate and adaptive immune systems and epidermal keratinocytes. The disease is now widely understood as an immune-driven inflammatory condition rather than a purely keratinocyte proliferation disorder [13,18].

Genetic predisposition plays a crucial role in psoriasis susceptibility, with multiple loci associated with immune regulation and epidermal function. Environmental triggers such as infections, trauma (Koebner phenomenon), stress, and certain medications can initiate or exacerbate disease activity in genetically susceptible individuals [18].

The early phase of psoriasis involves activation of dendritic cells, which release pro-inflammatory cytokines that stimulate T-cell differentiation. Among these, Th1 and Th17 cells play central roles in sustaining inflammation and amplifying immune responses within the skin [13].

Activated T cells migrate into the dermis and epidermis, where they release cytokines such as TNF-α, IL-17, and IL-23, which are key mediators of psoriatic inflammation [13,18]. The IL-23/IL-17 axis is considered a central pathogenic pathway in psoriasis. IL-23 promotes the expansion and survival of Th17 cells, which in turn produce IL-17 and IL-22, inducing keratinocyte hyperproliferation and abnormal differentiation [13].

This creates a self-amplifying inflammatory loop in

which keratinocytes release chemokines and cytokines that recruit additional immune cells, perpetuating chronic inflammation [13,15]. Keratinocytes in psoriatic lesions exhibit accelerated turnover, resulting in parakeratosis, scaling, and plaque formation [13].

Several intracellular signaling pathways are implicated in psoriasis pathogenesis, including NF-κB and STAT-mediated inflammatory signaling, which regulate genes involved in proliferation and immune activation [13,15]. Oxidative stress and altered apoptotic signalling further contribute to keratinocyte dysfunction and persistence of inflammatory lesions [3,6].

Although primarily cutaneous, psoriasis is increasingly recognized as a systemic inflammatory disease associated with psoriatic arthritis, metabolic syndrome, and cardiovascular disorders [18].

2.1 Genetic Factors

Psoriasis exhibits a strong genetic predisposition, supported by familial aggregation and genome-wide association studies. Several susceptibility loci, collectively termed psoriasis susceptibility regions (PSORS), have been identified, with PSORS1 on chromosome 6p21 being the most significant [32]. The HLA-Cw6 allele is strongly associated with early-onset psoriasis and increased disease severity. Other genetic variants involve genes regulating skin barrier integrity and immune signalling, including IL23R, TNFAIP3, and CARD14, which contribute to aberrant inflammatory responses [33].

2.2 Immunological Mechanisms

Psoriasis is primarily driven by dysregulated immune responses involving innate and adaptive immunity. Activated dendritic cells initiate T-cell differentiation into Th1 and Th17 subsets, leading to excessive production of cytokines such as IL-17, IL-22, and TNF-α [34]. These mediators stimulate keratinocyte proliferation and promote recruitment of neutrophils and macrophages, resulting in sustained inflammatory cascades. The IL-23/Th17 axis is considered central to disease maintenance and is a key target for modern biologic therapies [35].

2.3 Environmental Triggers

Environmental factors play a critical role in triggering psoriasis in genetically predisposed individuals. Common triggers include infections (especially streptococcal pharyngitis), psychological stress, trauma (Koebner phenomenon), and certain medications such as beta-blockers and lithium [36]. Lifestyle factors including smoking, alcohol consumption, and obesity further exacerbate disease severity by promoting systemic inflammation [37].

2.4 Molecular Pathways

At the molecular level, psoriasis involves activation of multiple signalling pathways regulating inflammation and cellular proliferation. NF-κB signalling promotes transcription of pro-inflammatory cytokines, while the JAK/STAT pathway contributes to immune cell activation and keratinocyte hyperplasia [38]. Additionally, oxidative stress and mitochondrial dysfunction have been implicated in sustaining chronic inflammation and epidermal dysregulation [39].

III. TYPES OF PSORIASIS

Psoriasis presents in several clinical forms:

A. Plaque Psoriasis

Plaque psoriasis is the most common form, accounting for nearly 80–90% of cases. It presents as raised, erythematous plaques covered with silvery scales, typically distributed over the scalp, elbows, knees, and lower back [18]. The lesions result from accelerated keratinocyte proliferation and inflammatory infiltration of T cells and neutrophils [13]

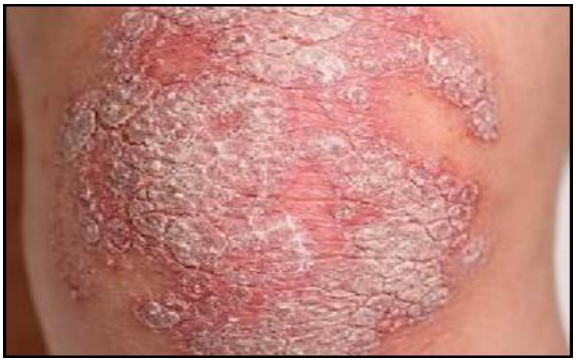

Fig.2. Plaque Psoriasis

2. Guttate Psoriasis

Guttate psoriasis is characterized by multiple small, drop-like lesions, often triggered by streptococcal infection. It is more common in children and young adults and may resolve spontaneously or progress to chronic plaque psoriasis [18].

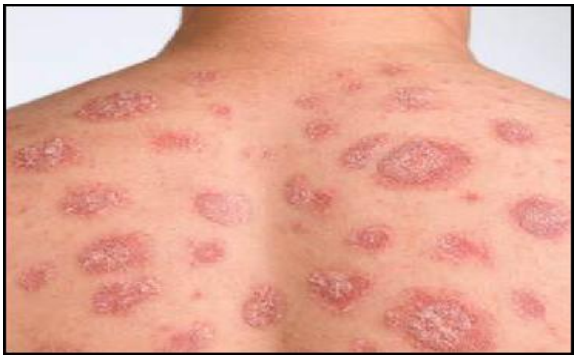

Fig.3. Guttate Psoriasis

3. Pustular Psoriasis

This variant presents with sterile pustules on an erythematous base and may be localized or generalized. It involves intense neutrophilic infiltration and cytokine activation [13].

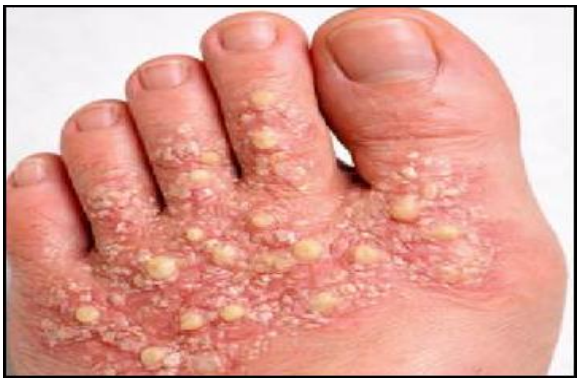

Fig.4. Pustular Psoriasis

4. Erythrodermic Psoriasis

Erythrodermic psoriasis is a rare but severe form characterized by widespread erythema and scaling involving most of the body surface area. It may be life-threatening due to impaired thermoregulation and fluid imbalance [18].

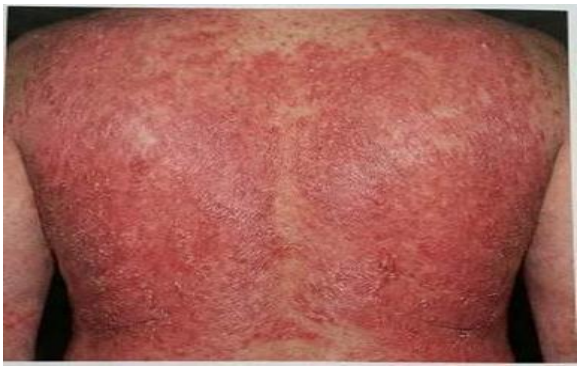

Fig.5. Erythrodermic Psoriasis

5. Inverse Psoriasis

Inverse psoriasis affects intertriginous areas such as the axillae, groin, inframammary regions, and skin folds. Lesions appear as smooth, erythematous patches with minimal scaling due to the moist environment of these areas [18].

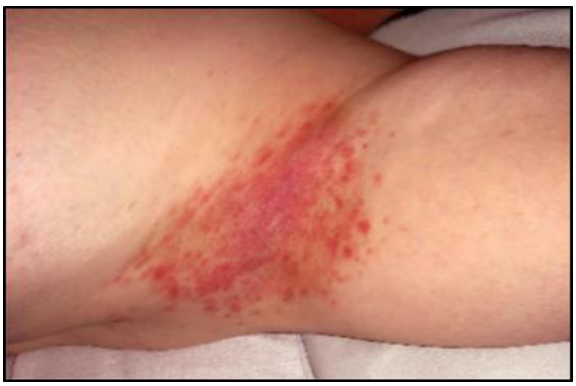

Fig.6. Inverse psoriasis.

6. Nail Psoriasis

Nail psoriasis involves the fingernails and toenails and is characterized by pitting, onycholysis, subungual hyperkeratosis, and nail discoloration. Nail involvement is commonly associated with psoriatic arthritis and may serve as an early clinical indicator of joint disease [18].

Fig.7. Nail psoriasis.

7. Scalp Psoriasis

Scalp psoriasis is a frequent manifestation that may occur independently or alongside plaque psoriasis. It presents as erythematous plaques with thick scaling on the scalp and may extend beyond the hairline. Severe cases can lead to temporary hair shedding due to inflammation [18].

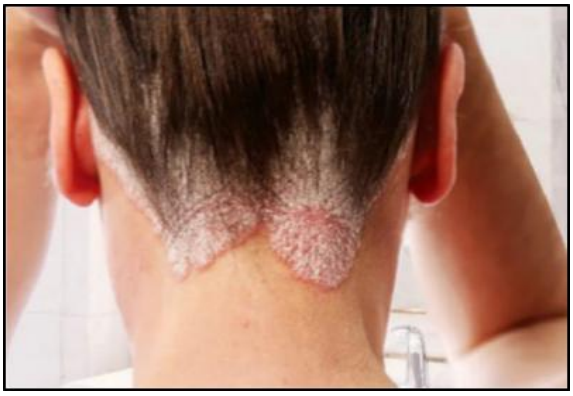

Fig. 8. Scalp psoriasis.

IV. CONVENTIONAL THERAPY

Conventional therapies remain the cornerstone of psoriasis management and are primarily aimed at reducing inflammation, normalizing keratinocyte proliferation, and modulating immune dysregulation [12,18].

4.1 Topical Treatments

Topical therapies remain first-line treatment for mild-to-moderate psoriasis. Common agents include corticosteroids, vitamin D analogues, retinoids, and calcineurin inhibitors, which reduce inflammation and normalize keratinocyte proliferation [41].

4.2 Systemic Therapy

Systemic agents such as methotrexate, cyclosporine, and acitretin are used in moderate-to-severe psoriasis. These drugs suppress immune activation but are associated with potential toxicities including hepatotoxicity, nephrotoxicity, and teratogenicity [42].

4.3 Biologics

Biologic therapies targeting specific cytokines have revolutionized psoriasis management. Agents targeting TNF-α, IL-17, and IL-23 demonstrate high efficacy and improved safety profiles compared to traditional systemic drugs [43]. Examples include adalimumab, secukinumab, and ustekinumab.

4.4 Phototherapy

Phototherapy using narrowband UVB and PUVA remains an effective non-pharmacological option. It works by inducing T-cell apoptosis and reducing epidermal proliferation, though long-term use may increase skin cancer risk [44].

V. CONVENTIONAL TREATMENT APPROACHES

1. Dithranol (Anthralin)

Dithranol exerts antiproliferative and anti-inflammatory effects by inhibiting keratinocyte hyperproliferation and modulating mitochondrial activity [1–4]. Despite efficacy, its use is limited by irritation and staining.

2. Tazarotene

Tazarotene is a topical retinoid that regulates gene expression through retinoic acid receptors, promoting normalization of keratinocyte differentiation and reducing inflammation [7–10].

3. Corticosteroids

Topical corticosteroids are widely used first-line agents due to potent anti-inflammatory and immunosuppressive effects. Long-term use may lead to adverse effects such as skin atrophy and tachyphylaxis [12–15].

4. Coal Tar

Coal tar has antiproliferative and antipruritic properties, though its use has declined due to cosmetic concerns and potential irritation [11].

5. Vitamin D Analogues

Vitamin D analogues regulate keratinocyte proliferation and differentiation through vitamin D receptor activation and are often used in combination therapies [18].

4.5 Limitations

Despite effectiveness, conventional therapies are limited by adverse effects, relapse upon discontinuation, and high cost of biologics. These limitations have prompted interest in safer and cost-effective alternative therapies [45].

V. HERBAL THERAPY IN PSORIASIS

5.1. Rationale for Herbal Medicine

Herbal therapies offer multi-targeted pharmacological effects including anti-inflammatory, antioxidant, and immunomodulatory actions. Their natural origin and historical usage contribute to increased patient acceptance and safety perception [46].

5.2 Common Medicinal Plants

Several botanicals including Aloe vera, Curcuma longa, Wrightia tinctoria, and Indigo naturalis have demonstrated therapeutic potential. These plants exhibit anti-psoriatic activity by modulating cytokine expression and reducing oxidative stress [47].

5.3 Phytochemicals of Interest

Bioactive compounds such as curcumin, indirubin, berberine, and thymoquinone play a key role in herbal efficacy. These phytochemicals regulate inflammatory signaling pathways and inhibit keratinocyte proliferation [48].

5.4 Mechanisms of Action

Herbal compounds exert effects through suppression of NF-κB activation, inhibition of pro-inflammatory cytokines, and restoration of antioxidant defences. Some phytochemicals also modulate T-cell differentiation, thereby interrupting the inflammatory cycle in psoriasis [49].

5.5 Types of Herbal Approaches.

1. Capsicum annuum

Capsicum annuum contains capsaicin, which modulates neurogenic inflammation by desensitizing sensory neurons and reducing substance P release. Topical capsaicin has demonstrated reductions in itching and erythema, though local irritation may occur.

Fig. 9. Capsicum annuum.

2. Aloe vera

Aloe vera contains polysaccharides and bioactive compounds that promote wound healing and reduce inflammation. Clinical studies have shown reductions in erythema, scaling, and plaque thickness with topical use [25].

Fig. 10. Aloe vera.

3. Silybum marianum

Silybum marianum (milk thistle) contains silymarin, a flavonolignan complex known for its antioxidant and anti-inflammatory properties. Although primarily studied in hepatic disorders, its ability to modulate oxidative stress and inflammatory mediators suggests potential supportive benefits in inflammatory skin conditions, including psoriasis [25].

Fig. 11. Silybum marianum (milk thistle).

4. Matricaria recutita

Matricaria recutita (German chamomile) contains flavonoids and terpenoids that contribute to its soothing and anti-inflammatory effects. Traditional topical use includes management of erythema and irritation, and its mild anti-inflammatory action may provide adjunctive benefits in
psoriasis [25].

Fig. 12. Matricaria recutita (chamomile).

5. Ulmus rubra

Ulmus rubra (slippery elm) is traditionally used for its mucilaginous and soothing properties. Topical preparations are believed to reduce irritation and inflammation in dermatological conditions. While direct clinical evidence in psoriasis is limited, traditional use supports its role among complementary herbal therapies [25].

Fig. 13. Ulmus rubra (slippery elm).

6. Curcuma longa (Turmeric)

Curcumin exhibits potent anti-inflammatory and antioxidant effects by modulating NF-κB and cytokine signalling. Emerging studies suggest improvements in psoriasis severity, particularly when used alongside conventional therapy [25].

Fig. 14. Curcuma longa (turmeric).

7. Wrightia tinctoria

Wrightia tinctoria is widely used in traditional Indian medicine. Extracts demonstrate anti-inflammatory and antiproliferative effects, with herbal oils showing improvements in scaling and pruritus [19,22].

Fig. 15. Wrightia tinctoria.

8. Nigella sativa

Nigella sativa contains thymoquinone, which exhibits antioxidant and immunomodulatory properties.

Preliminary studies suggest potential benefits in reducing inflammatory mediators and oxidative stress [23].

Fig. 16. Nigella sativa (black cumin).

9. Mahonia aquifolium

Mahonia aquifolium contains isoquinoline alkaloids such as berberine and has demonstrated clinical benefits in mild-to-moderate psoriasis, reducing erythema and scaling [24].

Fig. 17. Mahonia aquifolium.

10. Indigo naturalis

Indigo naturalis contains indirubin and tryptanthrin, which exert anti-inflammatory and antiproliferative effects. Clinical studies have shown improvements in plaque psoriasis with topical preparations [27–29].

Fig. 18. Indigo naturalis.

VI. HERBAL VS CONVENTIONAL THERAPY: COMPARATIVE ANALYSIS

Table 1: Comparative analysis of Conventional and Herbal Therapies in Psoriasis

Parameter	Conventional Therapy	Herbal Therapy
Mechanism of action	Target-specific cytokine inhibition	Multi-targeted action including anti-inflammatory, antioxidant, and immunomodulatory effects
Onset of action	Rapid symptomatic relief	Gradual but sustained therapeutic benefits
Safety profile	Associated with systemic adverse effects on prolonged use	Favourable safety profile with minimal side effects
Long-term use	Limited due to toxicity and relapse risk	More suitable for long-term management
Cost	Expensive, especially biologics	Cost-effective and widely accessible
Standardization	Highly standardized formulations	Variability exists but improving with modern phytopharmaceutical research
Evidence base	Strong clinical trial support	Growing scientific evidence and increasing clinical validation
Patient compliance	Reduced due to adverse effects	Higher acceptance due to natural origin and tolerability
Relapse rate	Frequent after discontinuation	May help reduce recurrence when used as adjunct therapy
Overall role	Primary treatment for acute control	Promising complementary and supportive therapy

Adapted from published studies on conventional and herbal psoriasis therapies [34,41–47,49–53].

6.1 Clinical efficacy of Herbal agents in management of Psoriasis

1. Capsicum annuum (Capsaicin)

Capsaicin exerts its anti-psoriatic effects by depleting Substance P, a neuropeptide that drives neurogenic inflammation, promotes keratinocyte hyperproliferation, and induces angiogenesis and vasodilation in psoriatic lesions. Topical application causes initial sensory nerve stimulation followed by sustained desensitization, resulting in prolonged reduction of neurogenic inflammatory activity.

In a double-blind interpatient comparison trial (n=44, 6 weeks), 0.025% capsaicin cream applied to one side of symmetrically distributed psoriatic lesions produced statistically significant reductions in scaling, erythema, and overall global evaluation scores compared to vehicle control.[59] A larger double-blind, placebo-controlled trial (n=197, 6 weeks) using the same concentration applied four times daily confirmed significantly greater global evaluation improvement at 4 weeks ($p=0.024$) and 6 weeks ($p=0.030$), along with significant pruritus relief ($p=0.002$) and reductions in combined psoriasis severity scores.[60] Adverse effects primarily burning and stinging at the application site were self-limiting and diminished with continued use.

2. Aloe vera

The anti-inflammatory and immunomodulatory properties of Aloe vera are attributed to ace Mannan, anthraquinones, and polyphenols, which modulate macrophage activity, cytokine release, and arachidonic acid metabolism mediators implicated in psoriatic plaque formation.

A double-blind, placebo-controlled trial (n=60, 4 weeks) using 0.5% Aloe vera extract cream applied three times daily achieved a cure rate of 83.3% (25/30 patients) versus 6.6% in the placebo group ($p<0.001$), with mean PASI score declining to 2.2 from baseline and no adverse events reported.[61] A head-to-head randomized trial comparing Aloe vera against 0.1% triamcinolone acetonide (TA) over 8 weeks demonstrated PASI reduction from 11.6 to 3.9 in the Aloe vera group versus 10.9 to 4.3 in the TA group, with comparable Dermatology Life Quality Index (DLQI) improvement in both arms.[62] Conversely, one double-blind trial (n=40) found no significant benefit of a commercial Aloe vera gel over placebo in stable plaque psoriasis, a finding attributed to a high placebo response rate and lack of formulation standardization.[63]

3. Curcuma longa (Turmeric / Curcumin)

Curcumin inhibits NF-κB signalling, suppresses pro-inflammatory cytokines including IL-17, IL-22, and TNF-α, and inhibits phosphorylase kinase — an enzyme markedly elevated in psoriatic skin. Its combined anti-inflammatory and anti-proliferative profile directly target core pathogenic mechanisms of psoriasis.

A 2022 meta-analysis of seven clinical RCTs and 19 preclinical studies found that curcumin significantly

improved PASI scores in both monotherapy and combination regimens compared to controls (standardized mean difference: −0.83%; 95% CI: −1.53 to −0.14; p=0.02).[64] A topical turmeric microemulsion trial (n=34) produced statistically significant reductions in erythema, desquamation, and plaque thickness versus placebo.[64] A randomized scalp psoriasis trial (n=40, 9 weeks) using twice-daily turmeric tonic demonstrated significant PASI reductions ($p<0.05$) and improved DLQI with no adverse effects.[65] An adjunctive oral curcumin trial (n=60, 12 weeks; 3 g/day combined with topical corticosteroids) confirmed safe and effective benefit for mild-to-moderate psoriasis.[66] Poor oral bioavailability of standard curcumin formulations remains a key pharmacokinetic limitation; enhanced delivery systems including Meriva, nanocurcumin, and piperine co-administration have demonstrated improved systemic absorption.

4. Nigella sativa (Black Seed)

Thymoquinone (TQ), the principal bioactive constituent of Nigella sativa, suppresses NF-κB signaling, inhibits pro-inflammatory cytokines (TNF-α, IL-6), and exerts antioxidant and anti-proliferative effects on keratinocytes mechanisms directly relevant to psoriatic pathophysiology.

A 2022 systematic review and meta-analysis of 14 RCTs evaluating Nigella sativa across skin diseases found a pooled odds ratio of 4.59 (95% CI: 2.02–10.39) in favor of treatment, including psoriasis, though significant heterogeneity across studies warrants cautious interpretation.[67] A case series (n=12) using a black seed extract preparation achieved PASI-75 response in 10 of 12 patients — a threshold equivalent to the primary endpoint of most biologic psoriasis trials.[68] A study evaluating combined topical and oral Nigella sativa found the dual-route group achieved the highest proportion of complete and good responses with lower relapse rates compared to monotherapy arms.

5. Mahonia Aqui folium

Mahonia Aqui folium contains the alkaloids berberine, berbamine, and oxyacanthine, which inhibit keratinocyte proliferation, suppress lipoxygenase-mediated leukotriene synthesis, and modulate T-lymphocyte activity collectively targeting the core inflammatory-proliferative axis of psoriasis.

A randomized, double-blind, placebo-controlled multicentre trial (n=200, 12 weeks) using Mahonia aquifolium cream (Reliéva) twice daily demonstrated statistically significant improvements in PASI scores and Quality of Life Index versus vehicle ($p<0.05$), with adverse effects in fewer than 1% of patients.[69] An intraindividual randomized trial (n=82) applying bark extract ointment to one side and placebo to the contralateral side found

statistically significant patient-assessed differences across all psoriasis severity grades.[70] A 2018 systematic review of seven studies found five demonstrating statistically significant benefit; one comparative study found Mahonia cream equal to or superior to both calcipotriol and tazarotene gel in all 33 enrolled patients, and 63% of patients in another study rated Mahonia cream equivalent to or better than their prior standard treatment.[71]

6. Indigo naturalis

Indirubin, the primary alkaloid of Indigo naturalis, inhibits cyclin-dependent kinases (CDKs), suppresses TNF-α-induced NF-κB activation, reduces CD3+ T-lymphocyte infiltration, downregulates Ki-67 expression, and restores filaggrin a key epidermal differentiation protein deficient in psoriatic skin.

A randomized, observer-blind, vehicle-controlled intrapatient trial (n=42, 12 weeks) in recalcitrant plaque psoriasis demonstrated highly significant reductions in scaling, erythema, and induration ($p<0.001$), with a mean clinical score of 6.3 in treated versus 12.8 in vehicle-treated lesions. [72] A controlled histological and immunohistochemical study (n=14) confirmed significant clinical score reductions alongside decreased Ki-67 and CD3 immunostaining and increased filaggrin expression, providing cellular-level mechanistic validation.[73] A dose-response randomized double-blind trial evaluating four indirubin concentrations in Lindioil formulation (200, 100, 50, and 10 μg/g, twice daily, 8 weeks) using PASI-75 and PASI-90 as primary endpoints established a clear dose-response relationship. [74] A systematic review comparing topical plant extracts identified Indigo naturalis as having the strongest and most consistent evidence base among all traditional plant extracts for psoriasis.[75] Topical formulations are generally well tolerated; oral use has been associated with gastrointestinal effects and isolated reports of pulmonary arterial

hypertension, necessitating caution.

VII. CHALLENGES

Several challenges hinder widespread adoption of herbal therapies:

- Lack of standardization in herbal formulations
- Limited high-quality clinical trials
- Variability in phytochemical content
- Regulatory and quality control concerns [51]

VIII. FUTURE PERSPECTIVES

Future research should focus on integrative therapeutic strategies combining conventional and herbal approaches. Advances in pharmacogenomics and personalized medicine may enable tailored treatment based on individual genetic and immunological profiles [52]. Development of standardized herbal extracts and large-scale clinical trials will further validate their clinical utility.

IX. CONCLUSION

Psoriasis is a multifactorial immune-mediated disorder requiring comprehensive management strategies. While conventional therapies remain the cornerstone of treatment, their limitations necessitate exploration of complementary approaches. Herbal therapies provide promising alternatives due to their multi-targeted mechanisms and favorable safety profiles. Future research should emphasize evidence-based integration of herbal and modern therapies to improve long-term patient outcomes [53].

REFERENCES

[1] L. Kemény, T. Ruzicka, and O. Braun-Falco, "Dithranol: A review of the mechanism of action in the treatment of psoriasis vulgaris," *Skin Pharmacol.*, 1990.

[2] J. Berth-Jones, A. C. Chu, W. A. Dodd, M. Ganpule, W. A. D. Griffiths, R. P. Haydey, *et al.*, "A multicentre comparison of calcipotriol ointment and short-contact dithranol therapy in chronic plaque psoriasis," 1990s.

[3] McGill, A. Frank, N. Emmett, *et al.*, "Anthralin accumulates in keratinocyte mitochondria and induces apoptosis," *FASEB J.*, 2005.

[4] T. Benezeder, C. Painsi, V. Patra, *et al.*, "Dithranol targets keratinocytes and inhibits IL-36 inflammatory loop," *eLife*, 2020.

[5] G. M. Kavanagh, *et al.*, "Effects of dithranol on neutrophil superoxide generation in psoriasis," *Br. J. Dermatol.*, 1996.

[6] K. U. Schallreuter, *et al.*, "Anthralin inhibits elevated thioredoxin reductase in psoriasis," *JAMA Dermatol.*, 1987.

[7] R. A. S. Chandraratna, "Tazarotene: The first receptor-selective topical retinoid for psoriasis," 1997.

[8] M. Duvic, "Molecular mechanisms of tazarotene action in psoriasis," 1997.

[9] G. D. Weinstein, "Tazarotene cream in psoriasis," 2003.

[10] M. Lebwohl, *et al.*, "Tazarotene 0.1% gel plus corticosteroids in psoriasis," 1998.

[11] S. Sekhon, C. Jeon, M. Nakamura, L. Afifi, D. Yan, J. J. Wu, *et al.*, "Mechanism of action of coal tar in psoriasis," 2010s.

[12] J. D. Ference and A. R. Last, "Topical corticosteroids: Choice and application," *Am. Fam. Physician*, vol. 79, no. 2, pp. 135–140, 2009.

[13] L. Uva, D. Miguel, C. Pinheiro, J. Antunes, D. Cruz, J. Ferreira, *et al.*, "Mechanisms of action of topical corticosteroids in psoriasis," *Int. J. Endocrinol.*, 2012, Art. no. 561018.

[14] B. Gottlieb, *et al.*, "In vivo mechanisms of action of topical corticosteroids in psoriasis," *J. Am. Acad. Dermatol.*, vol. 53, no. 1, pp. 77–85, 2005.

[15] P. J. Barnes, "Anti-inflammatory actions of glucocorticoids," *Clin. Sci.*, vol. 94, no. 6, pp. 557–572, 1998.

[16] L. Mallbris, *et al.*, "," *J. Invest. Dermatol.*, vol. 105, pp. 765–770, 1995.

[17] D. D. Bikle, "," *J. Steroid Biochem. Mol. Biol.*, vol. 89–90, pp. 375–379, 2004.

[18] Menter, *et al.*, "Guidelines of care for the management of psoriasis," *J. Am. Acad. Dermatol.*, vol. 69, pp. 60–78, 2013.

[19] "Wrightia tinctoria review," *J. Coastal Life Med.*, 2014.

[20] "Potential herbal constituents for psoriasis treatment," BITS Pilani, India.

[21] "Fight psoriasis naturally through Ayurveda,"

Indo Am. J. Pharm. Res., 2016.
[22] "Systems pharmacology approach to Wrightia tinctoria," *J. Altern. Complement. Med.*, 2017.
[23] L. P. Dwarampudi, D. Palaniswamy, M. Nithyanantham, and P. S. Raghu, "Antipsoriatic activity of Nigella sativa seeds," *Pharmacogn. Mag.*
[24] M. Wiesenauer and R. Lüdtke, "Mahonia aquifolium in psoriasis vulgaris: An intraindividual study," 1990s.
[25] "Review of natural compounds for psoriasis treatment," 2023.
[26] Izadyari Aghmiuni and A. Akbarzadeh Khiavi, "Medicinal plants for psoriasis," 2020s.
[27] H. M. Cheng, *et al.*, "Clinical efficacy of Indigo naturalis in psoriasis," *BMC Complement. Altern. Med.*, 2017.
[28] C. Wang, *et al.*, "Mechanism of action of Indigo naturalis in psoriasis," *Ann. Med.*, 2024.
[29] Y. Zhao, *et al.*, "Indigo naturalis nanofibrous patches in psoriasis," *Front. Pharmacol.*, 2025.
[30] "Polyherbal gel for scalp psoriasis," *World J. Pharm. Med. Res.*
[31] D. Sarkar, *et al.*, "Traditional Indian medicinal plants for psoriasis," 2010s.
[32] F. O. Nestle, D. H. Kaplan, and J. Barker, "Psoriasis," *N. Engl. J. Med.*, 2009.
[33] L. C. Tsoi, *et al.*, "Genome-wide association studies of psoriasis," *Nat. Commun.*, 2017.
[34] M. A. Lowes, *et al.*, "Pathogenesis and therapy of psoriasis," *Nature*, 2014.
[35] Blauvelt, "IL-23/IL-17 axis in psoriasis," *Lancet*, 2018.
[36] E. M. Griffiths, *et al.*, "Environmental triggers in psoriasis," *Lancet*, 2021.
[37] J. Takeshita, *et al.*, "Psoriasis and comorbidities," *J. Am. Acad. Dermatol.*, 2017.
[38] M. Goldminz, *et al.*, "NF-κB in psoriasis," *J. Invest. Dermatol.*, 2013.
[39] S. Briganti and M. Picardo, "Oxidative stress in psoriasis," *Free Radic. Biol. Med.*, 2003.
[40] W. H. Boehncke and M. P. Schön, "Psoriasis phenotypes," *Lancet*, 2015.
[41] Menter, *et al.*, "Guidelines of care for psoriasis," *J. Am. Acad. Dermatol.*, 2009.
[42] W. Armstrong and C. Read, "Systemic therapy in psoriasis," *JAMA*, 2020.
[43] Rendon and K. Schäkel, "Biologics in psoriasis," *J. Clin. Med.*, 2019.
[44] A. Elmets, *et al.*, "Phototherapy guidelines," *J. Am. Acad. Dermatol.*, 2019.
[45] J. E. Greb, *et al.*, "Psoriasis management challenges," *Nat. Rev. Dis. Primers*, 2016.
[46] Y. Panahi, *et al.*, "Herbal therapies in dermatology," *Phytother. Res.*, 2019.
[47] R. Sarkar, *et al.*, "Medicinal plants in psoriasis," *Indian Dermatol. Online J.*, 2017.
[48] S. J. Hewlings and D. S. Kalman, "Curcumin: A review," *Foods*, 2017.
[49] J. Kim, *et al.*, "Natural anti-inflammatory compounds," *Int. J. Mol. Sci.*, 2020.
[50] E. Ernst, "Herbal versus conventional medicine," *Br. J. Clin. Pharmacol.*, 2005.
[51] M. Ekor, "The growing use of herbal medicines: Issues relating to adverse reactions and challenges in monitoring safety," *Front. Pharmacol.*, 2014.
[52] E. A. Ashley, "Towards precision medicine," *JAMA*, 2016.
[53] R. Parisi, *et al.*, "Global burden of psoriasis," *J. Invest. Dermatol.*, 2020.
[54] M. Kamata and Y. Tada, "Dendritic cells and macrophages in the pathogenesis of psoriasis," *Front. Immunol.*, vol. 13, Art. no. 941071, 2022.
[55] M. Kamata and Y. Tada, "Crosstalk between keratinocytes and immune cells in psoriasis," *Front. Immunol.*, vol. 14, Art. no. 1286344, 2023.
[56] J. Xiao, *et al.*, "Advances in the pathogenesis of psoriasis: From keratinocyte perspective," *Cell Death Dis.*, vol. 13, p. 81, 2022.
[57] J. C. Szepietowski, *et al.*, "The immunology of psoriasis—Current concepts in pathogenesis," *Clin. Rev. Allergy Immunol.*, 2024.
[58] M. A. Lowes, M. Suárez-Fariñas, and J. G. Krueger, "Immunology of psoriasis," *Annu. Rev. Immunol.*, vol. 32, pp. 227–255, 2014.
[59] J. E. Bernstein, L. C. Parish, M. Rapaport, M. M. Rosenbaum, and H. H. Roenigk Jr., "Effects of topically applied capsaicin on moderate and severe psoriasis vulgaris," *J. Am. Acad. Dermatol.*, vol. 15, no. 3, pp. 504–507, 1986.
[60] N. Ellis, B. Berberian, V. I. Sulica, *et al.*, "A double-blind evaluation of topical capsaicin in pruritic psoriasis," *J. Am. Acad. Dermatol.*, vol. 29, no. 3, pp. 438–442, 1993.
[61] T. A. Syed, S. A. Ahmad, A. H. Holt, S. A.

Ahmad, S. H. Ahmad, and M. Afzal, "Management of psoriasis with Aloe vera extract in a hydrophilic cream: A placebo-controlled, double-blind study," *Trop. Med. Int. Health*, vol. 1, no. 4, pp. 505–509, 1996.

[62] Choonhakarn, P. Busaracome, B. Sripanidkulchai, and P. Sarakarn, "A prospective, randomized clinical trial comparing topical aloe vera with 0.1% triamcinolone acetonide in mild to moderate plaque psoriasis," *J. Eur. Acad. Dermatol. Venereol.*, vol. 24, no. 2, pp. 168–172, 2010.

[63] Paulsen, L. Korsholm, and F. Brandrup, "A double-blind, placebo-controlled study of a commercial Aloe vera gel in the treatment of slight to moderate psoriasis vulgaris," *J. Eur. Acad. Dermatol. Venereol.*, vol. 19, no. 3, pp. 326–331, 2005.

[64] S. Zhang, *et al.*, "Efficacy and safety of curcumin in psoriasis: Preclinical and clinical evidence and possible mechanisms," *Front. Pharmacol.*, vol. 13, Art. no. 903160, 2022.

[65] Sarafian, M. Afshar, P. Mansouri, J. Asgarpanah, K. Raoufinejad, and M. Rajabi, "Topical turmeric microemulgel in the management of plaque psoriasis: A clinical evaluation," *Iran J. Pharm. Res.*, vol. 14, no. 3, pp. 865–876, 2015.

[66] P. Bahraini, M. Rajabi, P. Mansouri, G. Sarafian, R. Chalangari, and Z. Azizian, "Turmeric tonic as a treatment in scalp psoriasis: A randomized placebo-controlled clinical trial," *J. Cosmet. Dermatol.*, vol. 17, no. 3, pp. 461–466, 2018.

[67] E. Antiga, V. Bonciolini, W. Volpi, E. Del Bianco, and M. Caproni, "Oral curcumin (Meriva) as an adjuvant treatment in psoriasis vulgaris," *Biomed Res. Int.*, 2015, Art. no. 283634.

[68] N. Nasiri, M. Ilaghi Nezhad, F. Sharififar, M. Khazaneha, M. J. Najafzadeh, and N. Mohamadi, "Therapeutic effects of Nigella sativa on skin disease: A systematic review and meta-analysis of randomized controlled trials," *Evid. Based Complement. Alternat. Med.*, 2022, Art. no. 7993579.

[69] J. Ahmed, Y. I. Azhar, and I. A.-H. Khaleel, "Evaluation of efficacy, safety and antioxidant effect of Nigella sativa in patients with psoriasis," *J. Clin. Exp. Invest.*, vol. 5, no. 2, pp. 186–193, 2014.

[70] S. Bernstein, H. Donsky, W. Gulliver, D. Hamilton, S. Nobel, and R. Norman, "Treatment of mild to moderate psoriasis with Mahonia aquifolium extract: A double-blind, placebo-controlled, multicenter study," *Am. J. Ther.*, vol. 13, no. 2, pp. 121–126, 2006.

[71] M. Augustin, U. Andrees, H. Grimme, E. Schöpf, and J. Simon, "Effects of Mahonia aquifolium ointment on skin markers in psoriasis," *Forsch. Komplementarmed.*, vol. 6, Suppl. 2, pp. 19–21, 1999.

[72] M. Janeczek, L. Moy, E. P. Lake, and J. Swan, "Review of the efficacy and safety of topical Mahonia aquifolium," *J. Clin. Aesthet. Dermatol.*, vol. 11, no. 12, pp. 42–47, 2018.

[73] Y. K. Lin, C. J. Chang, Y. C. Chang, W. R. Wong, S. C. Chang, and J. H. Pang, "Clinical assessment using indigo naturalis in psoriasis," *Arch. Dermatol.*, vol. 144, no. 11, pp. 1457–1464, 2008.

[74] Y. K. Lin, W. R. Wong, Y. C. Chang, *et al.*, "Efficacy and safety of topical indigo naturalis ointment," *Dermatology*, vol. 214, no. 2, pp. 155–161, 2007.

[75] Y. K. Lin, L. C. See, Y. C. Chang, Y. H. Huang, J. L. Chen, T. C. Tsou, *et al.*, "Comparison of indirubin concentrations in indigo naturalis ointment," *Br. J. Dermatol.*, vol. 178, no. 1, pp. 124–131, 2018.

[76] S. Deng, B. H. May, X. Liu, C. Lu, and C. C. Xue, "Topical herbal formulae in psoriasis: A systematic review and meta-analysis," *Phytomedicine*, vol. 20, no. 11, pp. 977–986, 2013.