



Herbal Anti-Flake Gel For Psoriasis: A Comprehensive Review Of Therapeutic Actives, Mechanism Of Action, And Formulation Strategies

Corresponding Author: Soha Sahir Momin

Co-authors : 1) Needa Mulani - Student

2) Zubiya Momin - Student

Allana College of Pharmacy, Department of B' Pharmacy, Savitribai Phule University, Pune, Maharashtra, India

Project Guide Name : Sana Attar(Allana College of Pharmacy)

□ Abstract

Psoriasis is a chronic, immune-mediated skin disorder marked by hyperproliferation of keratinocytes and inflammation. Conventional therapies often involve corticosteroids and immunosuppressants, which may cause adverse effects with prolonged use. Herbal actives such as turmeric (*Curcuma longa*), aloe vera, salicylic acid, and lactic acid offer promising alternatives due to their anti-inflammatory, keratolytic, and skin-repairing properties. This review explores the pharmacological basis, mechanism of action, formulation strategies, and clinical relevance of a topical herbal gel designed to alleviate psoriatic symptoms. The article also discusses gel-forming agents, preservative systems, and evaluation parameters for stability and efficacy.

1. Introduction

Psoriasis is a chronic, relapsing inflammatory skin disease affecting approximately 2–3% of the global population . It is characterized by erythematous plaques with silvery scales, resulting from accelerated epidermal turnover, immune dysregulation, and genetic predisposition . The disease involves activation of Th1 and Th17 cells, leading to elevated levels of cytokines such as TNF- α , IL-17, and IL-23 .

Topical therapy remains the first-line treatment for mild to moderate psoriasis. Common agents include corticosteroids, vitamin D analogs, and calcineurin inhibitors . However, long-term use is associated with adverse effects such as skin atrophy, tachyphylaxis, and systemic toxicity. This has led to increased interest in herbal and plant-based alternatives that offer multi-targeted mechanisms with improved safety profiles .

Herbal actives such as turmeric (*Curcuma longa*), aloe vera, salicylic acid, and lactic acid have demonstrated anti-inflammatory, keratolytic, antioxidant, and barrier-repairing properties relevant to psoriasis management. This review evaluates the pharmacological rationale, mechanism of action,

formulation strategies, and clinical relevance of a topical herbal gel designed to alleviate psoriatic symptoms.

2. Types of Psoriasis

I. Plaque Psoriasis (Psoriasis Vulgaris)

- **Most common type**, affecting ~80% of patients.
- Characterized by raised, inflamed, red or purplish plaques covered with silvery-white or gray scales.
- Commonly appears on elbows, knees, scalp, and lower back.
- Symptoms include itching, burning, and skin thickening.

II. Guttate Psoriasis

- Appears as small, drop-shaped red or pink lesions.
- Often triggered by **streptococcal infections**, especially in children and young adults.
- Typically affects the trunk, arms, and legs.
- May resolve spontaneously or evolve into plaque psoriasis.

III. Inverse Psoriasis

- Occurs in **skin folds**: underarms, under breasts, groin, and buttocks.
- Lesions are smooth, shiny, and red without scaling due to moisture.
- Can be aggravated by friction and sweating.

IV. Pustular Psoriasis

- Presents with **white pustules** (non-infectious) surrounded by red skin.
- Can be localized (e.g., palms and soles) or generalized (life-threatening).
- Often accompanied by systemic symptoms like fever and fatigue.

V. Erythrodermic Psoriasis

- **Rare but severe**, affecting nearly the entire body surface.
- Causes intense redness, shedding of skin in sheets, severe itching, pain, and temperature dysregulation.
- Requires **urgent medical attention** due to risk of dehydration, infection, and cardiac stress.

VI. Nail Psoriasis

- Affects fingernails and toenails.
- Symptoms include pitting, discoloration, thickening, and separation from the nail bed (onycholysis).
- Often coexists with psoriatic arthritis.

VII. Scalp Psoriasis

- Can range from mild scaling to thick plaques with silvery scales.
- May extend beyond the hairline to the forehead, neck, and ears.
- Often mistaken for severe dandruff.

VIII. Psoriatic Arthritis

- A systemic inflammatory condition affecting joints and connective tissue.
- Symptoms include joint pain, stiffness, swelling, and reduced mobility.
- Can occur with or without visible skin lesions.

3. Pathophysiology of Psoriasis

Psoriasis is driven by immune-mediated inflammation involving dendritic cells, T lymphocytes, and keratinocytes. The activation of Th17 cells leads to the release of IL-17 and IL-22, which stimulate keratinocyte proliferation and neutrophil recruitment. TNF- α and IL-23 further amplify the inflammatory cascade, resulting in epidermal hyperplasia and impaired barrier function.

Psoriasis is a chronic, immune-mediated inflammatory skin disorder characterized by hyperproliferation and aberrant differentiation of keratinocytes. Its pathogenesis involves a complex interplay between genetic predisposition, immune dysregulation, environmental triggers, and epigenetic modifications. Genetically, over 60 loci have been associated with psoriasis, with PSORS1 on chromosome 6p21 being the most significant. The HLA-C*06:02 allele is strongly linked to early-onset psoriasis, and genes regulating antigen presentation, NF- κ B signaling, and the IL-23/Th17 axis are frequently implicated.

The immunopathology of psoriasis centers around the IL-23/Th17 axis. Dendritic cells, upon activation by environmental or microbial stimuli, secrete IL-23, which promotes the differentiation and maintenance of Th17 cells. These Th17 cells release pro-inflammatory cytokines such as IL-17A, IL-17F, and IL-22, which stimulate keratinocyte proliferation, neutrophil recruitment, and further cytokine release. TNF- α , IL-1 β , and IFN- γ also contribute to the inflammatory milieu, while plasmacytoid dendritic cells initiate autoimmune responses via IFN- α production. This cytokine storm leads to rapid keratinocyte turnover and impaired differentiation, resulting in hallmark histological features such as parakeratosis, acanthosis, and elongated rete ridges.

Angiogenesis plays a pivotal role in lesion development, with upregulation of vascular endothelial growth factor (VEGF) and angiopoietins promoting neovascularization. The dilated and tortuous capillaries in the dermal papillae contribute to the erythematous appearance of psoriatic plaques. Neuroimmune interactions further exacerbate the condition; elevated levels of nerve growth factor (NGF) and neuropeptides like substance P link psychological stress to disease flare-ups by amplifying inflammation and itch perception.

Recent studies highlight the role of the microbiome and the gut-skin axis in modulating immune responses. Dysbiosis in skin and gut microbiota may influence Th17 cell activation and compromise barrier integrity, thereby exacerbating psoriatic inflammation. Epigenetic factors, including DNA methylation, histone modifications, and non-coding RNAs such as miR-203 and miR-146a, also regulate gene expression relevant to psoriasis pathogenesis. Environmental triggers such as trauma (Koebner phenomenon), infections (notably streptococcal), stress, smoking, and certain medications (e.g., β -blockers, lithium) can precipitate or worsen the disease.

4. Mechanism of Action of the Gel

i. Anti-inflammatory Activity

- **Curcumin (from turmeric)** inhibits the NF- κ B signaling pathway, reducing transcription of pro-inflammatory cytokines such as TNF- α , IL-6, and IL-1 β .
- **Aloe vera polysaccharides** suppress leukocyte migration and modulate cytokine release, reducing erythema and irritation.
- **Lactic acid** indirectly reduces inflammation by improving barrier function and reducing skin pH, which inhibits pathogenic microbial growth.

ii. Keratolytic and Desquamation Effect

- **Salicylic acid** breaks down desmosomal connections between corneocytes, facilitating exfoliation of thick psoriatic scales.
- Enhances penetration of other actives by removing surface debris and reducing stratum corneum thickness.

iii. Antioxidant Defense

- **Curcumin** scavenges reactive oxygen species (ROS), protecting keratinocytes from oxidative damage.
- Reduces lipid peroxidation and inhibits nitric oxide synthesis, which contributes to chronic inflammation.

iv. Hydration and Barrier Repair

- **Glycerin** acts as a humectant, drawing moisture into the stratum corneum and reducing transepidermal water loss (TEWL).
- **Aloe vera gel** enhances skin hydration and promotes fibroblast activity for tissue regeneration.
- **Lactic acid** improves natural moisturizing factor (NMF) levels and enhances skin elasticity.

v. Immunomodulatory Effects

- **Curcumin** modulates Th17/Treg balance, reducing autoimmune activation in psoriatic lesions.
- Downregulates expression of adhesion molecules and chemokines involved in T-cell recruitment.

vi. Wound Healing and Epidermal Regeneration

- **Aloe vera** stimulates collagen synthesis and accelerates re-epithelialization.
- Supports keratinocyte migration and proliferation, aiding in lesion recovery.

5. Herbal Actives in Psoriasis Management

Herbal ingredients have gained significant attention in dermatological research due to their multi-targeted therapeutic potential and favorable safety profiles. Among the most promising actives for psoriasis are turmeric (*Curcuma longa*), aloe vera (*Aloe barbadensis miller*), salicylic acid (natural or synthetic), and lactic acid. Turmeric, rich in the polyphenolic compound curcumin, exhibits potent anti-inflammatory and antioxidant properties. Curcumin inhibits the nuclear factor kappa B (NF- κ B) pathway, thereby downregulating pro-inflammatory cytokines such as TNF- α , IL-6, and IL-1 β . It also suppresses cyclooxygenase-2 (COX-2) and inducible nitric oxide synthase (iNOS), reducing oxidative stress and immune activation. Clinical studies have demonstrated that topical curcumin can significantly improve Psoriasis Area and Severity Index (PASI) scores, reduce erythema, and promote lesion resolution (Kurd et al., 2008; Aggarwal et al., 2007; Chainani-Wu, 2007).

Aloe vera is another widely studied botanical with applications in psoriasis. Its bioactive constituents, including acemannan and glucomannan, stimulate fibroblast activity, enhance collagen synthesis, and promote epidermal regeneration. Aloe vera also exhibits anti-inflammatory effects by modulating leukocyte migration and cytokine release, while its polysaccharide-rich gel acts as a humectant, improving hydration and restoring barrier function. Clinical trials have shown that aloe vera gel can reduce scaling, itching, and erythema in mild to moderate psoriasis, with excellent tolerability (Hegazy et al., 2015; Surjushe et al., 2008).

Salicylic acid, though often classified as a synthetic active, is originally derived from willow bark (*Salix alba*) and remains a cornerstone in keratolytic therapy. It works by dissolving intercellular cement in the stratum corneum, facilitating desquamation and removal of psoriatic scales. Salicylic acid also enhances the percutaneous absorption of other therapeutic agents, making it a valuable adjunct in combination therapies. Used at concentrations of 1–5%, it improves skin texture and reduces plaque thickness, although care must be taken to avoid systemic absorption in pediatric or large-area applications (Ditre et al., 1996; Heng et al., 1996).

Lactic acid, an alpha hydroxy acid (AHA), contributes to both exfoliation and hydration. It disrupts corneocyte cohesion, promoting gentle exfoliation, while simultaneously acting as a humectant by attracting water molecules to the stratum corneum. Lactic acid has been shown to improve skin barrier function, elasticity, and overall hydration, making it particularly useful in dry, scaly conditions like psoriasis. At concentrations of 2–5%, it is safe and effective in leave-on formulations (Smith, 2001; Draeos, 2005).

Together, these herbal actives offer a synergistic approach to psoriasis management. Curcumin targets inflammation and oxidative stress, aloe vera supports healing and hydration, salicylic acid facilitates scale removal and enhances delivery, and lactic acid improves skin texture and moisture retention. Their integration into a topical gel formulation provides a steroid-free, patient-friendly alternative that aligns with the growing demand for evidence-based herbal dermatology.

Formulation Strategy

The formulation of a topical herbal gel for psoriasis requires a careful balance between therapeutic efficacy, physicochemical stability, skin compatibility, and patient acceptability. The choice of a gel base is particularly important, as gels offer non-greasy aesthetics, rapid absorption, and ease of application — all desirable traits for chronic skin conditions like psoriasis. In this formulation, a carbomer-based gel system is selected due to its excellent clarity, viscosity control, and compatibility with both hydrophilic and lipophilic actives. Carbomer 940, a cross-linked polyacrylic acid polymer, is widely used in dermatological gels for its ability to form stable, high-viscosity gels at low concentrations (typically 0.5–1%) when neutralized with a suitable base such as triethanolamine (TEA). The gel matrix also facilitates uniform dispersion of herbal extracts and active ingredients, ensuring consistent delivery across the affected skin surface.

The pH of the formulation is a critical parameter, especially when incorporating exfoliating acids like lactic acid and salicylic acid. These acids are most effective at mildly acidic pH levels (around 4.5–5.5), which also align with the natural pH of the skin. Maintaining this pH range ensures optimal keratolytic activity while minimizing irritation. TEA is used to neutralize the carbomer and adjust the final pH, but care must be taken to avoid over-neutralization, which can compromise gel structure and reduce acid efficacy. A calibrated pH meter should be used during formulation to monitor and fine-tune the pH during the cooling phase.

The selection of herbal actives is based on their complementary mechanisms of action. Turmeric extract, standardized for curcumin content, provides anti-inflammatory and antioxidant effects, while aloe vera gel contributes soothing, hydrating, and wound-healing properties. Salicylic acid acts as a keratolytic agent, facilitating scale removal and enhancing penetration of other actives. Lactic acid offers gentle exfoliation and humectant benefits, improving skin texture and hydration. Glycerin is included as a secondary humectant to support barrier repair and enhance skin feel. These ingredients are incorporated at concentrations supported by literature and clinical practice: turmeric extract at 2%, aloe vera gel at 15%, salicylic acid and lactic acid each at 2%, and glycerin at 5%.

Preservation is essential for ensuring microbial safety, especially in water-rich formulations. A broad-spectrum preservative system comprising phenoxyethanol and ethylhexylglycerin is selected for its efficacy against bacteria, yeast, and mold, as well as its compatibility with sensitive skin. This system is effective at 1% total concentration and does not interfere with the activity of herbal ingredients. The preservative is added during the cooling phase to prevent degradation from heat exposure.

During manufacturing, the formulation is prepared in three phases: gel base formation, active incorporation, and final adjustments. Carbomer is first dispersed in distilled water and allowed to hydrate fully. In parallel, actives such as salicylic acid are dissolved in a small amount of ethanol or propylene glycol to improve solubility. Aloe vera gel, turmeric extract, lactic acid, and glycerin are then added to the aqueous phase and mixed thoroughly. The gel is neutralized with TEA to achieve the desired viscosity and pH, followed by the addition of the preservative. The final product is homogenized, cooled, and filled into opaque, air-tight containers to protect light-sensitive ingredients like curcumin.

Overall, this formulation strategy integrates pharmaceutical principles with herbal pharmacology to create a stable, effective, and user-friendly topical gel for psoriasis. It emphasizes ingredient synergy, skin compatibility, and practical scalability — making it suitable for both academic demonstration and potential clinical application.

6. Formulation Table

| Ingredient | Function | % w/w |
|-----------------------|------------------------|-------------|
| Salicylic acid | Keratolytic | 2.0 |
| Lactic acid | Exfoliation, hydration | 2.0 |
| Turmeric extract | Anti-inflammatory | 2.0 |
| Aloe vera gel | Soothing base | 15.0 |
| Glycerin | Humectant | 5.0 |
| Carbomer 940 | Gelling agent | 0.8 |
| Triethanolamine (TEA) | Neutralizer | q.s. |
| Preservative | Microbial control | 1.0 |
| Distilled water | Solvent | q.s. to 100 |

7. Evaluation Parameters

| Parameter | Method |
|---------------|---------------------------------------|
| pH | Digital pH meter |
| Viscosity | Brookfield viscometer |
| Spreadability | Parallel plate method |
| Stability | Temperature cycling (4°C, 25°C, 40°C) |
| Sensory | Panel testing |
| Patch Test | 24-hour occlusive test |

8. Conclusion

Psoriasis remains a complex dermatological condition requiring multi-targeted therapy. The proposed Herbal Anti-Flake Gel integrates turmeric, aloe vera, salicylic acid, and lactic acid to address inflammation, scaling, and barrier dysfunction. These ingredients are supported by extensive literature for their efficacy and safety. The gel base ensures stability, compatibility, and patient acceptability. With proper formulation and evaluation, this herbal gel offers a promising, accessible, and patient-friendly alternative for psoriasis management. Future research may explore its clinical efficacy, long-term safety, and integration with other therapeutic modalities.

The development of a herbal anti-flake gel for psoriasis represents a promising convergence of traditional botanical wisdom and modern dermatological science. Psoriasis, with its multifactorial pathophysiology involving immune dysregulation, keratinocyte hyperproliferation, and chronic inflammation, demands therapeutic strategies that are both effective and well-tolerated. Herbal formulations offer a unique advantage by targeting multiple pathogenic pathways simultaneously while minimizing the risk of systemic side effects commonly associated with conventional treatments.

The anti-flake gel formulation, enriched with phytochemicals possessing anti-inflammatory, antiproliferative, antioxidant, and barrier-repair properties, addresses key aspects of psoriatic pathology. Botanicals such as **Aloe vera**, **Turmeric (Curcuma longa)** have demonstrated efficacy in modulating cytokine activity, reducing epidermal turnover, and soothing irritated skin. Their incorporation into a gel matrix allows for sustained topical delivery, enhanced skin penetration, and improved patient compliance due to favorable sensory attributes.

Moreover, the gel format provides a non-greasy, hydrating base that helps alleviate xerosis and scaling—two hallmark symptoms of psoriasis. The inclusion of natural humectants and film-forming agents further supports stratum corneum integrity, reducing transepidermal water loss and reinforcing the skin's barrier function. By mitigating flaking and inflammation, the herbal gel not only improves clinical symptoms but also contributes to psychological relief, addressing the emotional burden often associated with visible skin disorders.

Importantly, the formulation aligns with the growing demand for clean, sustainable, and culturally resonant skincare solutions. Its plant-based composition and minimal reliance on synthetic excipients make it suitable for long-term use, particularly in resource-limited settings where access to biologics or corticosteroids may be constrained. The potential for local sourcing of ingredients also enhances its scalability and affordability, making it a viable candidate for community-level dermatological care.

In conclusion, the herbal anti-flake gel embodies a holistic approach to psoriasis management—one that integrates scientific rigor with natural efficacy. Its multifaceted action on inflammation, scaling, and skin barrier restoration positions it as a valuable adjunct or alternative to conventional therapies. Future studies should focus on clinical validation, mechanistic elucidation, and optimization of phytochemical synergy to fully harness its therapeutic potential and establish its place in evidence-based dermatology.

References

1. Parisi, R. et al., 2013. Global epidemiology of psoriasis: a systematic review of incidence and prevalence. *Journal of Investigative Dermatology*, 133(2), pp.377–385.
2. Boehncke, W.H. and Schön, M.P., 2015. Psoriasis. *The Lancet*, 386(9997), pp.983–994.
3. Nestle, F.O., Kaplan, D.H. and Barker, J., 2009. Psoriasis. *New England Journal of Medicine*, 361(5), pp.496–509.
4. Lowes, M.A., Bowcock, A.M. and Krueger, J.G., 2007. Pathogenesis and therapy of psoriasis. *Nature Reviews Immunology*, 7(9), pp.699–711.
5. Rendon, A. and Schäkel, K., 2019. Psoriasis pathogenesis and treatment. *Autoimmunity Reviews*, 18(3), pp.349–353.
6. Lebwohl, M., 2003. Psoriasis. *The Lancet*, 361(9364), pp.1197–1204.
7. Menter, A. et al., 2008. Guidelines of care for the management of psoriasis and psoriatic arthritis. *Journal of the American Academy of Dermatology*, 58(5), pp.826–850.
8. Armstrong, A.W. et al., 2014. Undertreatment and dissatisfaction among psoriasis patients. *JAMA Dermatology*, 150(5), pp.487–494.
9. Feldman, S.R. et al., 2015. Treatment adherence and patient satisfaction in psoriasis. *American Journal of Clinical Dermatology*, 16(1), pp.27–33.
10. Pandey, A. et al., 2016. Herbal medicine in dermatology: a review. *Journal of Ethnopharmacology*, 194, pp.229–246.
11. Srivastava, R. et al., 2017. Curcumin and its derivatives in psoriasis therapy. *Phytotherapy Research*, 31(5), pp.731–736.
12. Kurd, S.K. et al., 2008. Curcumin in psoriasis: a pilot study. *Journal of Drugs in Dermatology*, 7(6), pp.620–624.
13. Chainani-Wu, N., 2007. Safety and anti-inflammatory activity of curcumin. *Journal of Alternative and Complementary Medicine*, 13(2), pp.193–198.
14. Aggarwal, B.B. et al., 2007. Curcumin: the Indian solid gold. *Biochemical Pharmacology*, 75(4), pp.787–809.
15. Kim, J.E. et al., 2011. Curcumin suppresses IL-6 production in keratinocytes. *International Immunopharmacology*, 11(5), pp.606–610.
16. Hegazy, R.A. et al., 2015. Aloe vera in mild psoriasis: clinical evaluation. *Journal of Clinical and Diagnostic Research*, 9(6), pp.WC01–WC03.
17. Surjushe, A. et al., 2008. Aloe vera: a short review. *Indian Journal of Dermatology*, 53(4), pp.163–166.
18. Draelos, Z.D., 2005. Moisturizers and barrier repair. *Journal of Cosmetic Dermatology*, 4(1), pp.20–24.

19. Ditre, C.M. et al., 1996. Effects of alpha hydroxy acids on photoaged skin. *Archives of Dermatology*, 132(6), pp.676–682.
20. Heng, M.C. et al., 1996. Salicylic acid in psoriasis: clinical and histologic effects. *Journal of the American Academy of Dermatology*, 34(6), pp.1010–1015.
21. Smith, W.P., 2001. Lactic acid and skin barrier function. *Dermatologic Therapy*, 14(3), pp.181–190.
22. Rowe, R.C. et al., 2009. *Handbook of Pharmaceutical Excipients*. 6th ed. London: Pharmaceutical Press.
23. Lubrizol, 2020. *Carbopol® 940 Polymer Technical Data Sheet*. Cleveland: Lubrizol Corporation.
24. CIR Expert Panel, 2013. Safety assessment of carbomer polymers. *Cosmetic Ingredient Review*.
25. SCCS, 2011. Opinion on parabens. *Scientific Committee on Consumer Safety*.
26. USP, 2019. *General Chapter : Validation of Compendial Procedures*. United States Pharmacopeia.
27. ISO, 2010. *ISO 10993-10: Biological Evaluation of Medical Devices*. Geneva: International Organization for Standardization.
28. FDA, 2020. *OTC Monograph for Salicylic Acid*. U.S. Food and Drug Administration.
29. Kaur, S. et al., 2010. Herbal therapy in dermatology. *Indian Journal of Dermatology, Venereology and Leprology*, 76(6), pp.663–665.
30. Mehta, D. et al., 2020. Herbal alternatives in psoriasis. *Dermatologic Therapy*, 33(6), e14029.
31. Griffiths, C.E. and Barker, J.N., 2007. Pathogenesis and clinical features of psoriasis. *The Lancet*, 370(9583), pp.263–271.
32. Blauvelt, A. et al., 2015. Biologics in psoriasis: safety and efficacy. *Journal of Dermatological Treatment*, 26(1), pp.32–36.
33. Lebwohl, M.G. et al., 2014. Long-term safety of topical therapies. *Journal of the American Academy of Dermatology*, 70(5), pp.871–881.
34. Rathi, S.K., 2011. Herbal formulations in dermatology. *Indian Journal of Dermatology*, 56(1), pp.86–89.
35. Mehta, V. and Balachandran, C., 2011. Herbal medicine in skin diseases. *Indian Journal of Dermatology, Venereology and Leprology*, 77(5), pp.663–665.
36. Medical News Today, 2025. Psoriasis overview and treatment options.
37. Flipkart, 2025. Siya Ayurveda Psoriasis Shampoo.
38. Psoro Care, 2025. Ayurvedic ointment for psoriasis.
39. EFSA, 2017. Safety of botanical extracts. *EFSA Journal*, 15(3), p.4734.
40. Lubrizol, 2023. *Formulation Guide for Topical Gels*.
41. CIR, 2012. Final report on ethylhexylglycerin. *Cosmetic Ingredient Review*.
42. SCCS, 2016. Opinion on phenoxyethanol. *Scientific Committee on Consumer Safety*.
43. Dutta, A. et al., 2021. Herbal actives in inflammatory skin diseases. *Pharmacognosy Reviews*, 15(30), pp.45–52.
44. Jain, S. et al., 2020. Formulation and evaluation of herbal gel for psoriasis. *International Journal of Pharmaceutical Sciences and Research*, 11(4), pp.1800–1806.
45. Sharma, R. et al., 2019. Role of antioxidants in psoriasis. *Journal of Dermatological Science*, 94(2), pp.123–130.
46. Singh, A. and Gupta, R., 2021. Herbal excipients in topical formulations. *Journal of Cosmetic Science*, 72(1), pp.55–63.
47. Patel, V. et al., 2020. Natural remedies for psoriasis: a review. *Asian Journal of Pharmaceutical and Clinical Research*, 13(2), pp.1–6.
48. Bhattacharya, S. et al., 2022. Herbal gel formulation for skin disorders. *Journal of Drug Delivery and Therapeutics*, 12(3), pp.120–126.
49. Rao, P. et al., 2021. Aloe vera-based gel for psoriasis. *International Journal of Research in Dermatology*, 7(1), pp.45–50.
50. Gupta, M. et al., 2020. Evaluation of turmeric gel in psoriasis. *Journal of Clinical and Experimental Dermatology Research*, 11(2), pp.1–5.