Ksharagad Tablet and Ksharagad Ointment in Psoriasis Management: A Review of Their Individual and Combined Antipsoriatic Potential

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Abstract

Psoriasis is a chronic immune-mediated disease in which genetic susceptibility, environmental triggers, dendritic-cell activation and the interleukin-23/interleukin-17 axis sustain keratinocyte hyperproliferation. Ksharagada is a classical Ayurvedic polyherbal or polyherbo-mineral formulation described within Agada Tantra and has been discussed for inflammatory and skin-related indications. This narrative review evaluates the rationale for oral Ksharagada tablets, a topical Ksharagada ointment and their combined use. Modern psoriasis biology, classical concepts, pharmaceutical standardisation, ingredient-level pharmacology, experimental models and safety requirements are integrated. Direct controlled evidence for the named combination in psoriasis is presently insufficient; therefore, mechanistic relevance should not be treated as proof of efficacy. A proposed innovation—a standardised, batch-linked dual-route co-pack with an analytically fingerprinted tablet and a non-irritant ointment or hydrogel-ointment—is described as a testable development concept. Sequential quality, dermal safety, dose-ranging and controlled efficacy studies are required before clinical use or therapeutic claims.

Keywords: Ksharagada; psoriasis; Kitibha; Agada Tantra; tablet; ointment; IL-23/IL-17 axis; polyherbal formulation; standardisation; combination therapy.

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1. Introduction

Psoriasis is now understood as a systemic immune-mediated disorder rather than a purely cosmetic scaling disease. Well-demarcated erythematous plaques, silvery scale, pruritus and relapsing activity are accompanied in some patients by arthritis, cardiometabolic disease, depression and substantial impairment of quality of life. Global estimates vary because case definitions and sampling differ, but the burden is sufficiently large to justify safe, affordable and scientifically evaluated treatment options. [1]

Current care includes emollients, keratolytics, topical corticosteroids, vitamin-D analogues, phototherapy, conventional systemic agents, small molecules and biologics. These therapies have transformed outcomes, yet adherence, cost, monitoring, loss of response, contraindications and fear of adverse effects may leave important unmet needs. Traditional formulations are therefore frequently used, but their evaluation must distinguish historical use, preclinical plausibility and demonstrated clinical benefit. [2]

Ksharagada is described in Ayurvedic toxicology and has been discussed in relation to latent toxic states, inflammation and skin disorders. Published pharmaceutical work indicates that the preparation may exist as gutika or churna and requires careful control of raw-material identity, processing and finished-product properties. The formulation name alone does not guarantee compositional equivalence, because textual source, interpretation, regional substitutions and manufacturing practice may differ. [13]

The purpose of this review is to examine whether an oral tablet, a topical ointment and their combination can be developed into a coherent research programme for psoriasis. Particular attention is given to evidence gaps, because ingredient-level anti-inflammatory activity cannot automatically be extrapolated to a multi-ingredient product, topical delivery, oral bioavailability or clinical antipsoriatic efficacy. [5]

2. Review Method

A structured narrative approach was used to integrate peer-reviewed literature on psoriasis epidemiology, immunopathogenesis, severity assessment, preclinical models, topical delivery and botanical standardisation with classical and contemporary publications on Ksharagada. Preference was given to systematic reviews, consensus documents, indexed biomedical articles, recognised compendial

principles and papers reporting formulation-specific pharmaceutical observations. [31]

Evidence was interpreted by hierarchy. Direct studies of the complete Ksharagada product were considered more relevant than reports concerning isolated ingredients; controlled psoriasis studies were considered more informative than studies in unrelated inflammatory conditions; and clinical findings were not inferred from antioxidant assays alone. Recently published or non-indexed reports were used cautiously and identified as supportive rather than definitive evidence. [23]

3. Epidemiology and Clinical Burden of Psoriasis

A systematic analysis of 168 studies reported marked geographical variation, with modelled prevalence ranging from 0.14% in East Asia to 1.99% in Australasia. Western Europe, Central Europe and North America also showed comparatively high estimates, while 81% of countries lacked adequate epidemiological information. These differences caution against transferring prevalence estimates from one region to another. [24]

Global Burden of Disease data estimated approximately 64.6 million affected individuals in 2017, corresponding to an age-standardised prevalence near 0.84%. Incidence rose from 92 per 100,000 in 1990 to 99 per 100,000 in 2017, and peak incidence was observed around 55–60 years. The figures describe population burden and do not establish the suitability of any particular treatment. [1]

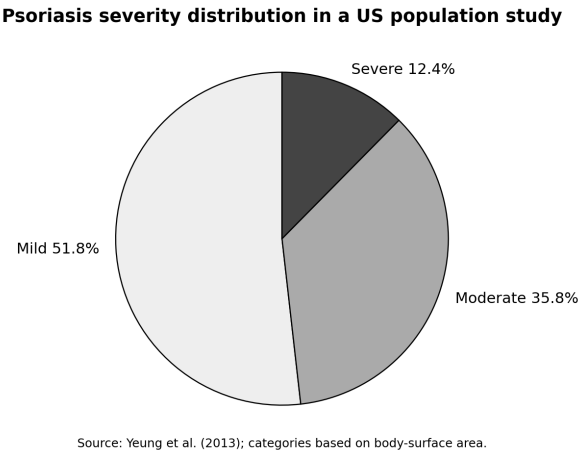


Figure 1. Severity distribution reported in a US population study. The proportions are study-specific and should not be interpreted as global estimates. [37]

Severity is multidimensional. Body-surface area, the Psoriasis Area and Severity Index, lesion location, symptoms and the Dermatology Life Quality Index may lead to different classifications in the same patient. In one population analysis using body-surface-area criteria, 51.8% of cases were mild, 35.8% moderate and 12.4% severe, which provides the dataset used in Figure 1. [37]

4. Immunopathogenesis Relevant to Treatment

Susceptibility loci, mechanical injury, infection, medications, smoking, obesity and stress can contribute to initiation or exacerbation. Activated plasmacytoid and myeloid dendritic cells release mediators that promote T-cell differentiation and trafficking. The resulting inflammatory loop links innate sensing to adaptive immune activation and explains why durable control often requires more than removal of surface scale. [20]

The IL-23/Th17 pathway is central to plaque psoriasis. IL-23 supports pathogenic T cells that produce IL-17A and related cytokines; IL-17 then drives keratinocytes to release chemokines, antimicrobial peptides and additional inflammatory mediators. Tumour necrosis factor amplifies this circuit, while IL-22 contributes to altered differentiation and epidermal thickening. [6]

Keratinocytes are active immune participants rather than passive targets. Their accelerated turnover produces scaling and impaired barrier function, whereas cytokine-stimulated expression of antimicrobial peptides and chemokines recruits neutrophils and additional lymphocytes. This biology provides measurable translational endpoints such as epidermal thickness, Ki-67, IL-17A, IL-23, TNF-α and histopathological scores. [12]

Oxidative stress is frequently reported in psoriasis and may interact with inflammation, but antioxidant activity in a chemical assay is not an accepted surrogate for lesion clearance. A botanical product proposed for psoriasis should therefore be evaluated against disease-relevant cellular, histological and clinical endpoints rather than relying only on free-radical scavenging tests. [26]

5. Ayurvedic Perspective: Kitibha, Kushtha and Agada Tantra

Ayurvedic discussions commonly relate plaque-like psoriasis to Kitibha within the broader Kushtha group, while recognising that one-to-one equivalence between classical entities and modern diagnoses is imperfect. Dosha, dushya, agni, srotas and individual constitution inform traditional assessment; modern enrolment, however, should require dermatologist-confirmed psoriasis and explicit severity criteria. [30]

Agada Tantra addresses poisons, toxic exposures and their consequences. Ksharagada is cited in Visha Chikitsa and contemporary reviews connect its properties with Vishaghna, Kushthaghna, Kandughna, Krimighna and Shothahara actions. Such terminology supplies a traditional rationale, but it should be translated into predefined biological and clinical outcomes before scientific testing. [5]

Published descriptions report predominance of pungent, bitter and astringent tastes and attributes such as lightness, dryness and sharpness, together with heating or cooling actions across different ingredients. These composite characteristics are proposed to influence Kapha, Pitta, Rakta, itching, swelling and moisture. Because the final formulation contains diverse materials, conclusions based solely on aggregate Ayurvedic properties remain hypotheses. [5]

Reported material	Botanical/material identity	Suggested quality role
Palasha kshara	Butea monosperma-derived alkaline fraction	Core processed material; identity and alkalinity control
Haridra	Curcuma longa	Curcuminoid marker candidate
Daruharidra	Berberis aristata	Berberine marker candidate
Yashtimadhu	Glycyrrhiza glabra	Glycyrrhizin marker candidate
Maricha	Piper nigrum	Piperine marker candidate
Pippali	Piper longum	Piperine-related marker candidate
Shunthi	Zingiber officinale	Gingerol/shogaol marker candidates
Saindhava	Rock salt	Inorganic identity and elemental profile

Table 1. Selected reported materials and candidate analytical roles; the final formula must follow one authenticated source. [13]

Raw materials should be authenticated by macroscopic, microscopic and chromatographic methods, with voucher specimens retained for botanicals. Tests should include foreign matter, moisture, ash values, extractives, microbial limits, aflatoxins, pesticide residues, elemental impurities and relevant contaminants. Mineral or earth-derived materials require especially careful elemental and toxicological assessment. [34]

The Kshara process can alter pH, ionic composition and extractability, so in-process controls are essential. Yield, pH, total alkalinity, carbonate content, loss on drying and elemental fingerprint should be recorded. Any use of animal-derived processing media must be declared, ethically sourced, microbiologically controlled and justified; an alternative process cannot be described as classically equivalent without comparative evidence. [13]

Finished tablets require uniformity of mass, hardness, friability, disintegration, moisture, assay or marker consistency and dissolution or dispersion profiling. A multi-marker HPTLC or HPLC fingerprint is preferable to a single marker because no one constituent can represent the entire formula. Stability-indicating methods should separate degradants and track changes under long-term and accelerated storage. [17]

7. Individual Rationale for the Oral Tablet

Oral administration is intended to expose the systemic immune and metabolic milieu, but the extent of absorption is unknown for a complex processed formulation. Putative contributions from curcuminoids, berberine, glycyrrhizin, piperine and ginger-related compounds are supported by ingredient-level pharmacology, yet their concentrations, interactions and bioavailability in the complete tablet require measurement. [11]

Curcumin can modulate NF-κB, cyclooxygenase-2 and multiple cytokine pathways in experimental systems, but poor aqueous solubility and extensive metabolism limit simple translation from in-vitro concentrations to oral dosing. Piperine can alter drug-metabolising enzymes and transporters, which may increase exposure to some compounds while also creating interaction risk with prescribed medicines. [7]

Berberine shows anti-inflammatory and metabolic effects across preclinical models, whereas glycyrrhizin-related constituents may influence inflammatory signalling. Both also illustrate why “natural” cannot be equated with harmless: berberine has interaction and reproductive cautions, and excessive liquorice exposure can cause hypertension, hypokalaemia and fluid retention through mineralocorticoid effects. [18]

An oral Ksharagada tablet should consequently be treated as a pharmacologically active multi-constituent medicine. Exclusion criteria, concomitant-medication review, liver and kidney assessment, electrolyte monitoring where justified, and prospective adverse-event collection are necessary in early human studies.

6. Ksharagada Composition and Pharmaceutical Identity

A formulation-specific monograph should begin by declaring the textual source and exact recipe. A reported Charaka-based composition includes Palasha-derived kshara along with botanicals or natural materials such as Haridra, Daruharidra, Tulasi inflorescence, Yashtimadhu, Laksha, Saindhava, Jatamansi, Harenu, Hingu, Sariva, Kushtha and Trikatu-related ingredients; other textual traditions may not be identical. [19]

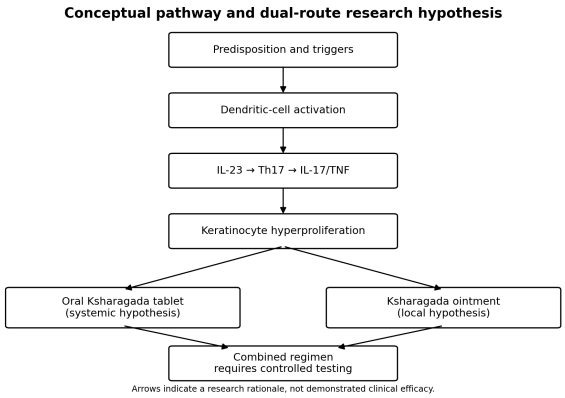
No dose should be recommended from this review alone. [33]

8. Individual Rationale for Ksharagada Ointment

Topical treatment can concentrate exposure at plaques and reduce systemic exposure, but formulation composition controls penetration, occlusion, hydration and irritation. A conventional hydrophobic ointment may soften scale and improve barrier function, whereas an emulsion or hydrogel-ointment can release hydrophilic and lipophilic markers differently. Vehicle-only controls are therefore essential. [3]

Kshara-containing material may be alkaline or irritant if improperly standardised. For application to inflamed skin, pH, buffering capacity, spreadability, rheology, content uniformity, preservative effectiveness, microbial quality and in-vitro release must be established. Skin-corrosion and irritation screening should precede repeated-dose testing, and compromised skin requires additional caution. [22]

A rational ointment development sequence would screen compatible bases, quantify marker release through a synthetic membrane, evaluate permeation and retention in excised skin, and compare intact with barrier-impaired conditions. Franz diffusion studies can inform formulation selection, but flux alone does not establish antipsoriatic activity or clinical safety. [21]



Topical endpoints should include erythema, scaling, thickness, transepidermal water loss, local tolerability and histology. When an imiquimod-induced mouse model is used, the psoriasis-like phenotype is transient and dominated by innate and IL-23/IL-17 responses; it does not reproduce the full chronic human disease. [32]

9. Combined Oral and Topical Strategy

Combination therapy is conceptually attractive because oral and topical products may address different compartments: systemic inflammatory drivers and local plaque/barrier changes. However, complementary routes do not prove synergy. The combined regimen must outperform each monotherapy by a prespecified margin and should not merely appear better because total exposure or supportive vehicle effects are greater. [29]

A four-group comparison—vehicle or control, tablet alone, ointment alone and combination—provides the minimum design needed to separate effects. Random allocation, blinded outcome assessment, equal handling, predefined exclusions and transparent reporting reduce bias. If a reference treatment is included, it should be analysed separately and used with an ethically and scientifically justified dose. [25]

Figure 2. Flow diagram linking psoriasis biology to a dual-route research hypothesis. It does not demonstrate efficacy. [6]

Potential disadvantages of combination use include cumulative irritation, reduced adherence, difficulty attributing adverse events, interactions among constituents and an inflated expectation of benefit. A factorial design can estimate oral, topical and interaction effects more efficiently than disconnected experiments, provided assumptions and sample-size calculations are specified in advance. [15]

10. Proposed New Innovation: Standardised Dual-Route Ksharagada Co-Pack

The proposed innovation is a batch-linked co-pack containing an analytically fingerprinted Ksharagada tablet and a dermatologically optimised Ksharagada ointment or hydrogel-ointment. The invention concept lies not in asserting a new cure, but in controlling two routes as one investigational system with shared raw-material traceability, route-specific specifications, dose accountability and a staged evidence programme. [17]

The oral unit would use authenticated ingredients, a declared classical source, controlled Kshara preparation, multi-marker chromatography and dissolution or dispersion specifications. The topical unit would use a low-irritancy vehicle selected through pH, rheology, release, skin-retention and safety testing. Marker ranges would be linked across both units to the same qualified botanical lots where feasible. [8]

Module	Core specification or study	Purpose
Identity module	DNA/microscopy where appropriate; HPTLC/HPLC fingerprint	Prevents substitution and documents batch similarity
Tablet module	Mass, hardness, friability, disintegration, marker assay, dissolution	Defines oral product performance
Topical module	pH, viscosity, spreadability, content	Defines local delivery and

	uniformity, release, microbial quality	usability
Safety module	Elements, pesticides, aflatoxins, microbes, dermal irritation, systemic toxicity	Controls foreseeable product risks
Evidence module	Factorial efficacy design, blinded scoring, cytokines, histology, PK markers	Separates route and interaction effects
Packaging module	Linked batch codes, dosing diary, lesion map, adverse-event card	Improves accountability and adherence

Table 2. Development architecture for the proposed dual-route co-pack. Values must be set from development data rather than invented targets. [8]

A useful technical extension is a marker-balanced topical microdomain system in which hydrophilic material is dispersed within an emollient continuous phase, or a hydrogel-ointment provides controlled release without strong alkalinity at the skin surface. The design would be selected by performance data, not by novelty language, and would require compatibility and stability testing. [3]

Digital adherence support could be incorporated through a QR-coded diary that records tablet intake, topical application, lesion photographs under standard lighting and symptom scores. This is a research-support feature rather than a diagnostic device; privacy, consent, data retention and image standardisation must be addressed before implementation. [35]

Patentability cannot be concluded from a literature review. Before describing the system as an invention in a filing, a professional prior-art search should examine Ksharagada formulations, psoriasis indications, dual-route packs, topical vehicles, marker combinations and dosing systems. Novelty, inventive step, enablement and jurisdiction-specific exclusions must be assessed by qualified counsel. [36]

11. Preclinical Evaluation Plan

Development should begin with chemical and pharmaceutical characterisation before efficacy screening. The complete tablet and topical product should be profiled alongside raw materials, placebo vehicle and selected marker standards. Acceptance criteria must be derived from qualified batches and validated analytical procedures rather than copied from unrelated herbal products. [10]

Stage	Work package	Representative outputs
Stage 1	Authentication and contaminant control	Identity, microscopy, fingerprint, elements, pesticides, aflatoxins, microbes
Stage 2	Dosage-form development	Tablet performance; topical pH, rheology, release and stability
Stage 3	Safety screening	Cytotoxicity, irritation/corrosion, sensitisation rationale, dose range
Stage 4	Mechanistic assays	Keratinocyte viability, proliferation and cytokine-responsive markers
Stage 5	Mouse proof-of-concept	Control, tablet, ointment, combination; blinded scoring and histology
Stage 6	Translation decision	Integrated benefit–risk review and human-study readiness

Table 3. Sequential evidence plan designed to prevent premature efficacy claims. [25]

In-vitro work may use cytokine-stimulated human keratinocytes to examine viability, proliferation and expression of inflammatory mediators. Concentration ranges should be limited by solubility, cytotoxicity and achievable exposure. An apparent fall in cytokine release caused by cell death must not be misinterpreted as anti-inflammatory activity. [22]

For topical selection, release testing should be discriminatory between batches and followed by ex-vivo skin retention or permeation. Mass balance is important because low receptor-phase recovery can represent retention, degradation, adsorption or analytical loss. Histology of treated skin should be read by a blinded pathologist using a predefined scoring system. [21]

The imiquimod mouse model can provide a rapid psoriasis-like inflammatory phenotype, with daily clinical scoring of erythema, scale and thickness. A modified PASI-like score should be described as an animal score rather than the human PASI. Ear or skin thickness, spleen weight, epidermal thickness and selected cytokines can support interpretation. [32]

Albino mice should be used only with institutional animal ethics approval, humane endpoints, refined procedures and a justified sample size. Investigators should follow applicable national rules, the 3Rs and ARRIVE reporting. Randomisation and blinding should be documented, and sex as a biological variable should be considered. [25]

12. Suggested Experimental Groups and Outcomes

Group	Intervention	Primary interpretive role

G1	Normal/untreated where scientifically necessary	Baseline skin and systemic measures
G2	Disease model + vehicle/control	Model severity and vehicle effect
G3	Disease model + tablet	Oral-route effect
G4	Disease model + ointment	Topical-route effect
G5	Disease model + tablet + ointment	Combined effect and interaction
G6	Disease model + reference therapy, optional	Assay sensitivity

Table 4. Illustrative group structure; doses, duration and animal numbers require protocol-specific justification. [15]

The primary endpoint should be selected before data collection and could be a blinded composite skin score or epidermal thickness at a fixed time. Secondary endpoints may include individual erythema, scale and thickness scores, histology, spleen weight, cytokines, oxidative markers, tolerability, body weight and clinical observations. Multiplicity and missing data should be handled prospectively. [25]

Demonstrating combination benefit requires more than statistical significance versus vehicle. The analysis should test the oral-by-topical interaction in a factorial model and report effect sizes with confidence intervals. If the combination is similar to the better monotherapy, the additional complexity and exposure may not be justified. [15]

13. Clinical Translation

Human translation should begin only after reproducible product specifications, adequate toxicology and regulatory consultation. An initial study would primarily assess tolerability, local reactions, adherence, pharmacokinetic marker exposure where feasible and laboratory safety. Patients should continue evidence-based rescue care according to protocol, and severe or unstable disease should not be enrolled without specialist oversight. [9]

A subsequent randomised study could use a double-dummy or assessor-blinded factorial design. Dermatologist-confirmed plaque psoriasis, stable baseline disease and defined body-surface-area or PASI criteria would improve interpretability. Outcomes may include PASI change, physician global assessment, target-lesion severity, pruritus, Dermatology Life Quality Index and patient-reported treatment satisfaction. [16]

Standard care should not be withheld merely to establish traditional-product efficacy. Comparator selection depends on severity and local guidelines, while add-on designs may be more ethical for patients who require active therapy. Trial registration, protocol publication, adverse-event definitions and CONSORT-compliant reporting are essential. [28]

14. Safety, Interactions and Regulatory Considerations

Safety evaluation must cover both routes and the complete formulation. Botanical misidentification, microbial contamination, pesticides, aflatoxins, elemental impurities, variable alkalinity and undeclared pharmaceutical ingredients are preventable hazards. A certificate of analysis should be linked to every research batch and retained samples should permit investigation of unexpected events. [34]

Potential herb–drug interactions deserve particular attention because psoriasis patients may use methotrexate, ciclosporin, acitretin, apremilast, biologics, antihypertensives or antidiabetic medicines. Piperine-containing materials may modify enzymes or transporters, and liquorice-derived constituents may affect blood pressure and potassium. Concomitant medication must be documented rather than treated as background noise. [33]

Topical safety is not established by oral safety. Irritation, contact allergy, photoreactivity and effects on damaged skin require route-specific assessment. Patch testing, repeated application and stopping rules should be considered in clinical development, and participants must be instructed to avoid mucosa, eyes and broken or infected areas unless a protocol specifically supports such use. [22]

The regulatory category may differ by jurisdiction and by whether the product is classical, proprietary, herbal, cosmetic or intended as an investigational medicine. Claims of treating psoriasis create a medicinal purpose and require appropriate evidence. Labelling should state composition, dosage form, batch, storage, warnings and limits of evidence without implying regulatory approval. [14]

15. Critical Appraisal of the Evidence

The strongest evidence in this topic concerns psoriasis immunobiology and established outcome measures. Formulation-specific Ksharagada publications contribute classical rationale and pharmaceutical description, but direct, replicated antipsoriatic evidence for the tablet, ointment and their combination is sparse. This imbalance limits causal conclusions and dose recommendations.

Ingredient-level studies are useful for generating mechanisms but are vulnerable to overextension. Extract type, concentration, purity and exposure in cell or animal experiments may bear little resemblance to a finished multi-ingredient product. Synergy can be claimed only when interaction is formally tested; the presence of multiple anti-inflammatory constituents is not sufficient.

Narrative reviews also carry selection and interpretation bias. The present synthesis therefore uses explicit evidence boundaries and avoids invented

numerical efficacy. A future systematic review may become appropriate when multiple controlled formulation-specific studies are available, with protocol registration and risk-of-bias assessment.

16. Research Gaps and Future Directions

Priority gaps include agreement on the authenticated formula, validated multi-marker methods, comparative batch data, oral exposure, topical release, dermal safety and complete-product pharmacology. Resolving these fundamentals is more valuable than immediately expanding to large efficacy experiments with an unstable or poorly characterised product.

Mechanistic research should connect observed effects to achievable product exposure and include orthogonal endpoints. Cytokine changes should be supported by histology or cellular measures, while oxidative assays should be interpreted as supportive. Omics approaches may be exploratory, but confirmatory assays and correction for multiple testing are required.Personalised use could eventually examine phenotype, lesion site, metabolic comorbidity and traditional constitution, but subgroup claims must be prespecified and adequately powered. Biomarker-guided hypotheses should not replace clinically meaningful endpoints or dermatologist assessment.

17. Conclusion

Ksharagada tablet and Ksharagada ointment offer a plausible dual-route research concept for psoriasis because the disease combines systemic immune dysregulation with local epidermal and barrier abnormalities. Classical indications and ingredient-level pharmacology support investigation, but they do not demonstrate that either product—or their combination—is effective in psoriasis.

The most defensible innovation is a standardised co-development platform: one authenticated source formula, analytically linked oral and topical products, route-specific safety controls and a factorial evidence strategy. Progression should depend on quality and safety gates, blinded controlled outcomes and transparent reporting. Until such evidence exists, the concept should remain investigational and should not replace dermatologist-directed care.

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