



ISSN: 2231-3656

International Journal of Pharmacy and Industrial Research (IJPIR)

IJPIR | Vol. 16 | Issue 3 | Jul – Sep 2026

www.ijpir.com

DOI: <https://doi.org/10.61096/ijpir.v16.iss3.2026.822.836>

White Curcumin (Tetrahydrocurcumin) and Manjistha (*Rubia cordifolia*) in Topical Skin Care: Phytochemistry, Dermatological Mechanisms, and Evidence Supporting Biotex White Curcumin Cream

A Critical Narrative Review

Dr Leroy Rebello¹, Dr Sreesudha Chepyala², Sahithi Polineni^{3*}

^{1,2,3}Department of Research and Development, Biotex Life Solutions Pvt. Ltd., Secunderabad, Telangana, India

*Corresponding author: Sahithi Polineni

Email: sahithi.biotexlife@gmail.com



Published on:

08.09.2026

Published by:

Futuristic Publications

2026| All rights reserved.



Creative Commons Attribution 4.0 International License.

Abstract: Botanical cosmeceuticals are increasingly used to support visible tone, radiance, comfort, hydration and resilience against everyday oxidative stress. This critical narrative review examines Biotex White Curcumin Cream as a two-active topical system built on White Curcumin (Hydro Curcumin 95%), a hydrogenated curcuminoid material in which tetrahydrocurcumin predominates, derived from *Curcuma longa*, together with Manjistha (*Rubia cordifolia*). Tetrahydrocurcumin (THC) is the colourless hydrogenated form of curcumin, and its antioxidant activity is well established. Still, comparatively superior: independent assays rank its radical-scavenging potency above both curcumin and the reference antioxidant Trolox. In skin cells, THC lowers hydrogen peroxide-induced reactive oxygen species in keratinocytes, suppresses melanin and the melanogenic enzymes tyrosinase, TRP-1 and TRP-2 at sub-microgram concentrations, and improves collagen, elastin, hyaluronic acid and UVB-survival endpoints in fibroblasts; engineered vehicles raise its skin permeation seven- to seventeen-fold. *Rubia cordifolia* contributes a complementary quinone- and phenolic-rich profile: a stem-derived antityrosinase fraction has identified purpurin and ellagic acid as the active constituents, and recent topical quinone research demonstrates inflammatory and barrier-related activity on skin. The two actives therefore reach overlapping visible endpoints—even-looking tone, radiance, antioxidant defence, comfort, hydration, and healthy-looking texture—through partly independent mechanisms. Human topical evidence is thinner and is honestly reported here: it comprises patch-test and moisturisation work, one split-face firmness trial of a multi-active gel, and a four-arm facial-cream comparison that separated no THC-specific benefit from vehicle. The evidence supporting this formulation is therefore predominantly ingredient-level, and does not constitute clinical efficacy evidence for the finished product.

Keywords: tetrahydrocurcumin; white curcumin; *Curcuma longa*; *Rubia cordifolia*; Manjistha; melanogenesis; antioxidant; botanical cosmeceuticals.

1. Introduction

Visible skin quality is governed by interconnected processes rather than a single pathway. Ultraviolet radiation, pollution-derived oxidants, intrinsic metabolic stress, and low-grade inflammation can increase reactive oxygen species (ROS), amplify cytokine signalling, disturb epidermal homeostasis, alter melanogenesis, and accelerate extracellular matrix degradation. Contemporary botanical skin care

therefore increasingly favours multi-target actives that can address oxidative stress, pigmentation signalling, inflammatory balance, hydration, and matrix integrity simultaneously. Curcumin from *Curcuma longa* has been widely studied in dermatology, but its intense yellow colour, limited aqueous solubility, and chemical instability constrain its convenience in leave-on cosmetic products. Hydrogenation of curcumin yields tetrahydrocurcumin (THC), a colourless derivative with a distinct molecular and pharmacological profile^[1-5].

THC is commonly referred to in cosmetic and formulation literature as “white curcumin.” The descriptor does not indicate the botanical species *Curcuma zedoaria*; rather, it refers to the hydrogenated curcuminoid derivative originating from *Curcuma longa* chemistry. THC lacks the α,β -unsaturated double-bond system of curcumin and is white/colorless, a property that removes the characteristic yellow staining associated with conventional curcumin^[1,7].

The second botanical considered here, Manjistha (*Rubia cordifolia* L.), is a Rubiaceae plant used in South Asian traditional medicine and is chemically rich in anthraquinones, naphthoquinones, phenolics, peptides, and related secondary metabolites. Modern phytochemical reviews describe more than 100 compounds, while dermatology-relevant work has identified purpurin and ellagic acid in a stem-derived antityrosinase fraction and has demonstrated topical anti-inflammatory activity of selected Rubia quinones^[15-18].

This critical narrative review evaluates the existing evidence for White Curcumin/Hydro Curcumin 95% and Manjistha as complementary topical actives. It is organised around the claim domains that matter for a cosmetic cream: visible evenness of tone, radiance, defence against oxidative stress, comfort, hydration, and the smoothness and texture of the skin surface. It does not infer treatment of pigmentary disease, inflammatory dermatoses, wounds, or photoaging disorders from ingredient-level evidence.

2. Review approach and evidence hierarchy

Peer-reviewed literature was prioritised from PubMed-indexed journals and major full-text scientific databases, with emphasis on studies directly evaluating tetrahydrocurcumin/tetrahydrocurcuminoids, topical delivery, skin cells, skin tissue, or human topical use. For Manjistha, preference was given to studies using *Rubia cordifolia* itself, particularly stem-derived material where available, followed by purified quinones with a clear botanical relationship. Reviews were used for chemical context and evidence mapping, whereas mechanistic claims were preferentially anchored to original experimental studies. Evidence was interpreted in the following order: human topical evidence; ex vivo/human skin or primary human skin-cell evidence; in vivo skin models; cell-based mechanistic studies; biochemical and phytochemical studies. Disease-specific experiments were used only to support biological pathways and not to imply disease-treatment efficacy of the finished cosmetic. The literature search was updated through 2026. Targeted supplementary searches covered tetrahydrocurcumin/white curcumin, melanogenesis, photoaging, topical delivery, human skin, *Curcuma longa* dermatology, *Rubia cordifolia* keratinocyte biology, skin barrier, purpurin, and topical formulation evidence. Before finalisation, reference titles, journal details, years, page/article numbers, and DOIs were cross-checked against PubMed, publisher records, or the original journal record where available; duplicate title/DOI records were also screened.

3. Formulation concept: two complementary botanical actives

3.1 White Curcumin: Hydro Curcumin 95% as a tetrahydrocurcumin-rich material

For scientific clarity, the term “White Curcumin” in this manuscript refers primarily to tetrahydrocurcumin (THC), the hydrogenated curcumin derivative commonly investigated as a colourless curcuminoid. Commercial wording such as “Hydro Curcumin 95%” should be treated as a raw-material specification rather than as a finished-cream concentration. In one analytical study of a commercially obtained high-purity tetrahydrocurcuminoid sample, LC-MS resolved THC as the predominant component together with smaller amounts of related hydrogenated curcuminoids. That study illustrates why an industrial material described as “95%” should be interpreted from its batch certificate of analysis and chromatographic specification rather than from the trade descriptor alone^[5].

The cosmetic significance of hydrogenation is twofold. First, loss of the conjugated double-bond system removes the intense yellow chromophore, producing a white/colorless material that is more compatible with non-staining leave-on skin care. Second, hydrogenated curcuminoids often show strong radical-scavenging and lipid-peroxidation-inhibitory activity in comparative assays. THC should

nevertheless not be presented as pharmacologically identical to curcumin; the two molecules share several biological effects but differ in molecular targets and relative potency across pathways^[1-4].

3.2 Manjistha: *Rubia cordifolia*

Manjistha (*Rubia cordifolia* L.) contains quinones and other secondary metabolites, including anthraquinones such as purpurin, alizarin, and rubiadin, along with phenolic and additional phytochemical classes. Composition varies with plant part and extraction conditions, which is important when extrapolating published data to a cosmetic raw material^[15,17]. Of particular relevance to the present formulation, a study using *R. cordifolia* stem material generated an anthraquinone-enriched fraction with antityrosinase activity and used TLC-MS bioautography to associate purpurin and ellagic acid with that activity^[16].

3.3 Declared composition of the finished cream

Biotex White Curcumin Cream is formulated with two declared botanical actives: *Curcuma longa* L., supplied as the tetrahydrocurcuminoid raw material marketed as Hydro Curcumin 95% and referred to throughout this review as White Curcumin; and an extract of *Rubia cordifolia* L. (Manjistha). Finished-cream concentrations of both actives are held in the product technical file and are not reported here.

Table 1. Principal ingredient-level chemistry and dermatological relevance.

Active	Key chemical features	Skin-relevant evidence	Most defensible cosmetic role
White Curcumin / THC	Hydrogenated curcumin derivative; colourless; phenolic and β -diketone motifs; analytical composition depends on the raw-material specification and batch.	Antioxidant; anti-melanogenic; keratinocyte oxidative protection; anti-inflammatory; topical delivery; fibroblast/ECM; photoaging models ^[2-4,6-10,23] .	Even-looking tone, radiance, antioxidant defence, comfort, texture support.
Manjistha / <i>R. cordifolia</i>	Anthraquinones (purpurin, alizarin, rubiadin), naphthoquinones, phenolics and other secondary metabolites.	Stem antityrosinase fraction; purpurin/ellagic acid; antioxidant activity; keratinocyte differentiation; <i>Rubia</i> quinone anti-inflammatory and barrier-relevant topical data ^[15-20,25,30,31] .	Tone support, antioxidant contribution, calming/comfort, complementary botanical activity.



Fig 1: Biotex White Curcumin Cream (20 g). Manufacturer product photograph, provided for product identification; the image is not presented as efficacy evidence.

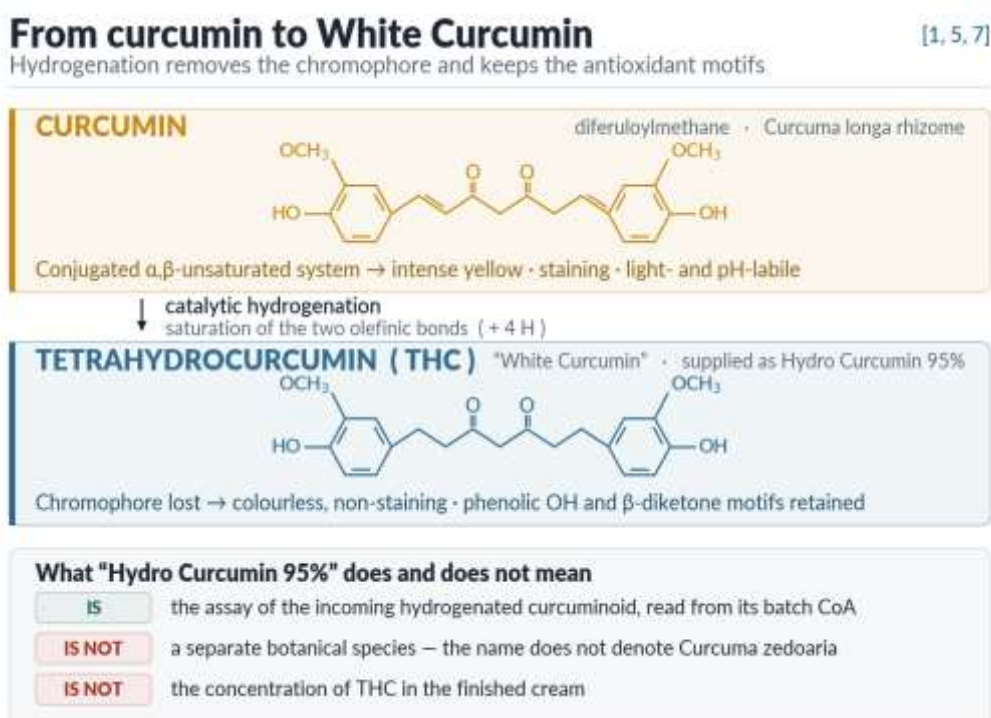


Fig 2: Chemical and formulation distinction between conventional yellow curcumin and White Curcumin (tetrahydrocurcumin). Catalytic hydrogenation of curcumin from *Curcuma longa* saturates the two olefinic bonds to give tetrahydrocurcumin, which loses the conjugated chromophore and is colourless and non-staining while retaining the phenolic and β -diketone motifs associated with antioxidant activity. The lower panel sets out what the trade descriptor "Hydro Curcumin 95%" does and does not denote.

4. Pigmentation biology and support for an even-looking skin tone

Melanogenesis is coordinated by signalling networks that converge on microphthalmia-associated transcription factor (MITF), which in turn governs the three melanogenic enzymes — tyrosinase (TYR) and its related proteins TRP-1 and TRP-2. Oxidative and inflammatory stress can augment melanogenic signalling, making redox regulation and inflammatory balance relevant to visible tone in addition to direct enzyme modulation.

4.1 Tetrahydrocurcumin

Two independent cellular studies provide direct THC evidence for pigmentation-related biology. In α -melanocyte-stimulating-hormone-stimulated B16F10 cells, THC lowered melanin accumulation and suppressed expression of the three melanogenic enzymes at both protein and mRNA levels; the same investigation found that THC protected HaCaT keratinocytes against hydrogen-peroxide-associated oxidative injury and evaluated a lecithin nanoemulsion as a delivery system^[6]. A later study distributed its endpoints across three cell lines: melanin was quantified in α -MSH-stimulated B16F0 murine melanoma cells, direct enzyme inhibition was measured against mushroom tyrosinase in a cell-free assay, and dose-dependent reductions in tyrosinase protein and in intracellular ROS were demonstrated in the A375 human line. THC was non-cytotoxic up to 100 μ M in melanoma and in normal fibroblast cells, and in the α -MSH-stimulated model it suppressed melanin synthesis at concentrations below those required for the established depigmenting agents arbutin and vitamin C^[23]. Together, these results establish an anti-melanogenic mechanism at the cellular level; they do not quantify a whitening effect in human facial skin.

A separate in vitro study assessed THC across mouse melanoma cells and human foreskin fibroblasts, and reported effect sizes worth stating. Melanin synthesis in B16-F10 cells fell by 78.5% at a THC concentration as low as 0.1 μ g/mL, while in fibroblasts at 1 μ g/mL collagen rose by 37.9%, elastin by 90.1% and hyaluronic acid by 74.2%; UVB-protection improved by 61.2% and scratch closure accelerated, with no toxicity up to 10 μ g/mL^[8]. Because these figures come from a single cell-culture study rather than from human skin, they are treated here as supporting mechanistic evidence rather than as proof of clinical change in firmness, wrinkles, hydration or repair.

Conventional curcumin has also been shown to reduce melanin content and cellular tyrosinase activity in cultured human melanocytes while decreasing MITF, tyrosinase, TRP-1, and TRP-2 expression. Because THC is not pharmacologically identical to curcumin, these human-melanocyte data are best used as pathway context rather than direct proof for THC^[21].

4.2 *Rubia cordifolia* and purpurin-centred tyrosinase evidence

The Manjistha evidence adds a mechanistically distinct pigmentation pathway. Using authenticated *R. cordifolia* stem material, Insaf and colleagues prepared an alcoholic extract and an anthraquinone-enriched fraction, demonstrated tyrosinase-inhibitory activity, and identified purpurin and ellagic acid in the bioactive fraction by TLC-MS bioautography. Molecular docking placed purpurin at a stronger predicted binding affinity for tyrosinase (−7.4 kcal/mol) than the reference inhibitor kojic acid (−5.3 kcal/mol). The same work incorporated the enriched fraction into a gel and assessed release and permeation across laboratory and ex vivo skin models^[16]. The study therefore links a stem-derived Manjistha preparation both to tyrosinase inhibition and to topical-formulation feasibility.

This evidence is valuable because it is at once plant-part specific and formulation-oriented: it ties a defined plant organ to a defined enzymatic mechanism and to a workable topical vehicle, which is more than most botanical tone claims can show. It stops short of demonstrating clinical lightening by Manjistha or by the finished cream, but it establishes a biologically coherent route from a declared Manjistha ingredient to the cosmetic concept of a more even-looking tone.

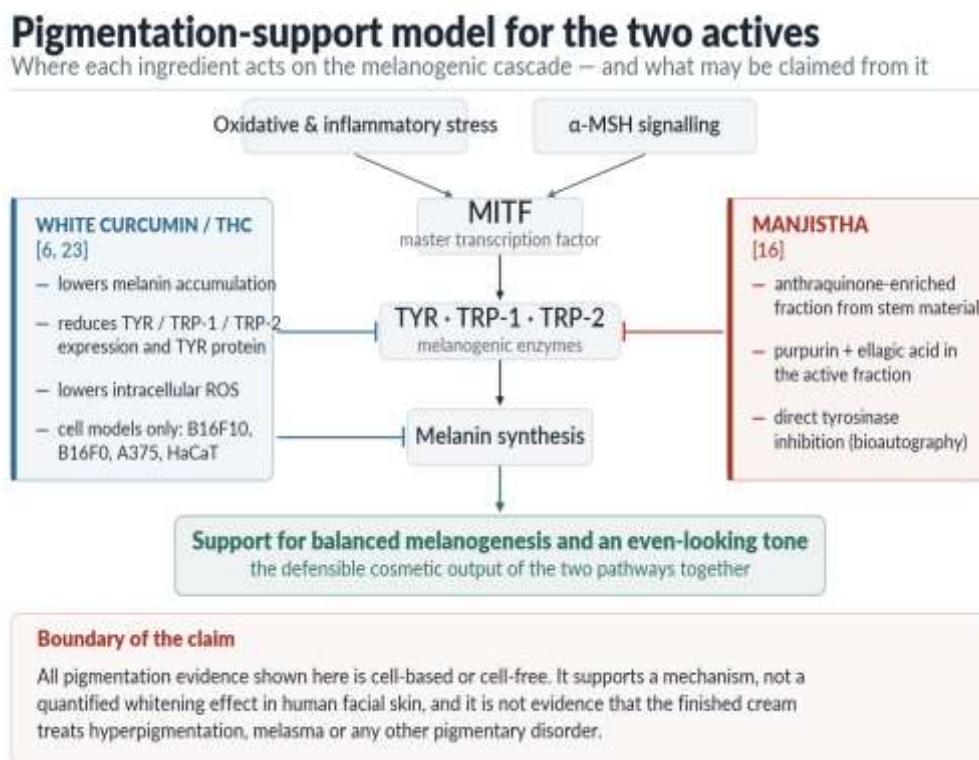


Fig 3: Integrated pigmentation-support model for White Curcumin and Manjistha. Oxidative and inflammatory stress and α -MSH signalling converge on MITF and on the melanogenic enzymes TYR, TRP-1 and TRP-2. Tetrahydrocurcumin acts on melanin formation and on melanogenic enzyme expression; the Manjistha fraction studied by Insaf and colleagues — an anthraquinone-enriched preparation from stem material, in which purpurin and ellagic acid were identified — acts by direct tyrosinase inhibition. Blunt-ended connectors denote inhibition. The convergent output is labelled as support for balanced melanogenesis and an even-looking tone, not treatment of hyperpigmentation.

5. Antioxidant defence and environmental oxidative stress

ROS participate in lipid oxidation, protein damage, inflammatory signalling, matrix metalloproteinase activation, pigment dysregulation, and loss of barrier resilience. Antioxidant activity is therefore the most convergent mechanistic domain in the White Curcumin-Manjistha combination.

5.1 Antioxidant properties of tetrahydrocurcumin

The antioxidant case for THC is unusually consistent across independent comparative studies. In the original tetrahydrocurcuminoid series, the fully hydrogenated compound showed the strongest antioxidative activity of all curcuminoids tested, in every assay system used; a follow-up study attributed this to the β -diketone moiety and again found THC more protective than parent curcumin against erythrocyte-membrane lipid peroxidation^[2,3]. A later head-to-head comparison ranked radical-scavenging potency as THC > hexahydrocurcumin = octahydrocurcumin > Trolox > curcumin, placing THC above both the reference antioxidant and its own parent compound, with the same ordering broadly reproduced in linoleic-oxidation and erythrocyte-haemolysis assays^[4]. These experiments establish antioxidant potency under controlled conditions rather than quantifying protection in human facial skin, but the consistency and direction of the finding across three decades of independent work are notable.

A physicochemical study of a high-purity THC sample adds the analytical dimension, documenting chromatographic composition, thermal behaviour, free-radical and lipid-peroxidation endpoints, and antioxidant-enzyme responses^[5]. Its value to this review is that it shows a high-purity tetrahydrocurcuminoid material can be chemically resolved and its antioxidant behaviour characterised alongside its impurity profile — the analytical basis on which a raw-material specification should rest. Its biological assays used non-cutaneous cell systems, so they are read as supporting rather than as direct skin evidence.

The same effect is measurable in skin cells. In HaCaT keratinocytes, THC pre-treatment lowered hydrogen-peroxide-induced intracellular ROS in a concentration-dependent manner — from roughly three times the unstressed level down to below baseline at the highest concentration tested — and also reduced α -MSH release from the stressed keratinocytes, a second route by which antioxidant activity may bear on pigmentation^[6]. In human foreskin fibroblasts, survival under UVB challenge improved by 61.2%^[8]. Together these observations support the statement that White Curcumin contributes to topical free-radical defence and helps skin manage everyday environmental oxidative stress.

5.2 Antioxidant contribution of Manjistha

Independent *R. cordifolia* studies support an antioxidant contribution from Manjistha chemistry. An alcoholic extract influenced iron-dependent redox reactions and lipid peroxidation, while isolated rubiadin inhibited experimentally induced lipid oxidation^[19,20]. A more recent side-by-side comparison of root, stem and leaf material found antioxidant activity in all three organs but the highest antioxidant content in the root, and it showed that yields shift further with the extraction solvent^[17]. In root material, hydroxyanthraquinones were identified as major radical-scavenging phenolics, and their activity depended on substitution pattern^[31]. Taken together, these studies support an anthraquinone/phenolic antioxidant rationale, while also showing that antioxidant strength varies by organ, so figures obtained from one plant part should not be transferred to a preparation made from another.

The dual-active formulation therefore combines a chemically defined hydrogenated curcuminoid antioxidant with a polyphenol/anthraquinone-rich botanical. This convergence is more defensible than attributing the total antioxidant claim to any single pathway.

6. Inflammatory balance and skin comfort

Inflammatory signalling can amplify erythema, discomfort, oxidative stress, melanogenic signalling, and barrier dysfunction. A cosmetic “skin comfort” claim is therefore best supported by evidence showing modulation of inflammatory mediators rather than by disease-treatment terminology.

6.1 White Curcumin

THC has been evaluated in both general inflammatory models and skin-focused delivery studies. In non-cutaneous acute-inflammation experiments, THC and octahydrocurcumin reduced oedema and vascular-permeability responses and were more effective than curcumin itself at suppressing COX-2 expression and TAK1–NF- κ B signalling^[10]. More directly relevant to topical use, a THC solid-lipid-nanoparticle hydrogel achieved approximately 17-fold greater ex vivo skin permeation than a free-THC gel, together with correspondingly stronger anti-inflammatory activity in vivo and a non-irritant, stable, occlusive formulation profile^[7]. That result supports local-delivery plausibility, with the caveat that the nanoparticle vehicle differs from a conventional cosmetic cream.

A separate high-purity THC study measured a concentration-dependent fall in three inflammatory mediators — tumour necrosis factor alpha, interleukin-1 beta and macrophage inflammatory protein-1 alpha — in cultured mouse splenocytes, with THC suppressing each more effectively than curcumin^[5]. As this cytokine work sits outside a cutaneous model it serves as secondary mechanistic support, and the cosmetic inference drawn from it is correspondingly bounded: support for inflammatory balance and comfort, not treatment of dermatitis or other inflammatory disease.

6.2 Manjistha

Recent work on *R. cordifolia* quinones provides skin-specific mechanistic evidence. In activated keratinocyte and macrophage systems, alizarin, purpurin and mollugin reduced inflammatory cytokine and chemokine responses through JAK1/STAT3-associated signalling, and the study compared topical permeation of the three quinones directly^[18]. In an imiquimod-driven mouse model, topical alizarin attenuated psoriasiform inflammatory changes, reduced average epidermal thickness from 116 to 78 μ m and restored barrier function in lesional skin. For the present cosmetic review these results are used to establish that *Rubia* quinones interact with cutaneous inflammatory and barrier biology — not to suggest psoriasis treatment. A separate purpurin review adds broader antioxidant-linked anti-inflammatory context at the constituent level^[30].

7. Extracellular matrix, photoaging biology, and visible texture

Visible skin texture depends on epidermal organisation, dermal extracellular matrix, hydration, and repeated exposure to oxidative and ultraviolet stress. THC has several lines of evidence relevant to these processes.

One in vitro study using human foreskin fibroblasts recorded THC-associated gains in collagen, elastin and hyaluronic-acid measurements, improved viability under UVB challenge and greater migration in a scratch assay — the three endpoints a texture claim would want to rest on^[8]. They are matrix and stress-response endpoints in culture, so they cannot be read as increased dermal collagen or wrinkle reduction in people; what they support is the conservative texture-oriented language used here, namely maintenance of smooth, resilient, healthy-looking skin.

In a UV-photoaging mouse model, THC treatment was associated with improvements in several macroscopic and histologic measures, including wrinkle-related features, epidermal thickness, and collagen/elastic-fiber organization. Transcriptomic analysis in the same study implicated inflammatory, extracellular-matrix, and pigmentation-related networks^[9]. This adds in vivo biological plausibility for photoaging-related pathways while remaining preclinical evidence.

A human split-face trial provides limited formulation-level support. Sommerfeld randomised 28 women to a double-blind, placebo-controlled comparison of a multi-active gel (Tricutan) whose declared actives are tetrahydrocurcumin, asiatic acid and ursolic acid at 0.1% each, together with dimethylaminoethanol; asiatic acid and ursolic acid are the isolated triterpenes associated with *Centella asiatica* and rosemary, respectively. Twenty-five women completed the four-week protocol, and the treated side registered a significant gain in firmness on ultrasound shear-wave propagation; three participants left the study early with mild contact irritation^[12]. Because the vehicle delivered four actives at once, neither the benefit nor the tolerability signal can be attributed to THC on its own^[12,14].

The broader *Curcuma longa* lineage contributes the one piece of human topical matrix evidence in this field. Muta and colleagues isolated gelatinase-inhibitory constituents from a turmeric-rhizome extract and then tested a cream containing that extract on human facial skin, where treatment was associated with higher elasticity measurements and fewer stratum-corneum sites showing gelatinase activity^[24]. The tested material was turmeric extract rather than isolated THC, so this stands as pathway and botanical-lineage support rather than as direct White Curcumin evidence — but it demonstrates that the matrix mechanism invoked here is measurable on human skin.

Parent-curcumin wound-healing literature was reviewed during evidence screening but was not retained as a core support domain because it is both molecule-distant from THC and indication-distant from the intended cosmetic claims. Accordingly, the texture/photoaging interpretation in this article is anchored to THC-specific fibroblast and UV-photoaging studies and to human topical *Curcuma* skin evidence rather than to wound-healing outcomes^[8,9,24].

8. Hydration, barrier support, and softness

Hydration requires careful attribution because an emulsion vehicle can itself alter corneometric measurements. In a 36-woman study, tetrahydrocurcuminoid-containing test materials produced substantially less irritation than the SDS positive control, while the finished cream (marketed as GPO “curmin” cream, the spelling used by that study) increased measured hydration after twice-daily use^[11]. Because the hydration comparison evaluated the complete cream rather than isolated THC, it supports formulation-level moisturization and topical tolerability, not a THC-specific moisturising mechanism.

A larger four-arm facial study provides an important counterbalance. Eighty women were assigned to curcuminoid-cream variants containing THC and/or liposome or to vehicle and used the assigned cream twice daily for four weeks. At the end of treatment, the groups did not differ significantly in hydration, elasticity, or colour; within-group changes occurred in several arms, including the vehicle arm^[32]. This result argues against attributing moisturization or colour change to THC alone and supports conservative wording that hydration and softness are properties of the finished cream system.

The broader *Curcuma longa* record adds independent, if indirect, hydration context. In an eight-week randomised, double-blind, placebo-controlled study, an orally administered hot-water extract significantly increased facial water content at both four and eight weeks, while companion keratinocyte experiments showed increased hyaluronan production and suppression of UVB-induced TNF- α and IL-1 β ^[22]. The oral route and the chemically distinct extract place this as supportive background for the hyaluronan mechanism rather than as direct evidence for topical THC.

For Manjistha, the relevant epidermal evidence is species-level. An ethyl acetate fraction prepared from *R. cordifolia* root modulated HaCaT keratinocytes in vitro and promoted keratinocyte differentiation in a topical mouse-tail model, increasing both the number and thickness of the granular

layer^[25]. A dermocosmetic review subsequently listed *R. cordifolia* root extract among a small set of botanicals with demonstrated barrier-reinforcing or differentiation-related activity after topical application^[26]. Both studies used root rather than stem material, so they establish epidermal plausibility for the species rather than hydration performance for any particular Manjistha preparation.

9. Topical delivery and formulation relevance

The biological value of a topical active depends on release from the vehicle, partitioning into the stratum corneum, local retention and stability. THC has an inherent formulation advantage over conventional curcumin — it is colourless and does not stain — but, being crystalline and poorly water-soluble, its skin delivery still depends on the vehicle. Engineering that vehicle raises skin exposure substantially: a lecithin nanoemulsion increased permeation roughly sevenfold over an unformulated THC suspension, and a solid-lipid-nanoparticle gel about 17-fold over free THC in gel^[6,7].

A similar formulation dependence is evident for Manjistha-related materials. The stem antityrosinase fraction was incorporated into a gel and assessed in release/permeation experiments, while a later study compared skin permeation of individual *Rubia* quinones under normal and inflammation-simulated conditions^[16,18]. These data support the general principle that biological activity and skin exposure depend on the specific extract, constituent, and vehicle.

For Biotex White Curcumin Cream the translational reading is that an emulsion vehicle offers a workable platform for presenting both actives to the stratum corneum, and that the formulation route is therefore consistent with the published delivery literature. What that literature cannot supply is the numbers: skin concentrations, permeation rates and biological effect sizes are vehicle-specific, so values reported for nanoemulsions and lipid nanoparticles should not be transferred to a conventional cream without formulation-specific measurement.

10. Human topical evidence relevant to White Curcumin

Direct human evidence for topical THC is modest. The available record includes irritation/moisturization testing, a multi-active split-face firmness trial, a disease-specific tetrahydrocurcuminoid study, and a four-arm facial-cream study in which THC did not show a distinct between-group benefit for hydration, elasticity, or colour^[11-14,32]. Broader systematic reviews of turmeric/curcumin dermatology provide additional translational context but involve heterogeneous molecules, formulations, routes, and indications. Vaughn and colleagues retained 18 human studies in their clinical review; Barbalho and colleagues synthesised controlled and clinical Curcuma evidence across several skin outcomes; and Patel and colleagues appraised 18 randomised controlled trials of turmeric or curcumin in dermatological indications^[27-29]. A narrower review confined to topically applied curcumin reaches the same conclusion from the opposite direction, observing that most of the clinical record concerns oral dosing and that comparatively few trials test topical application at all^[14]. These reviews strengthen the human-skin context for Curcuma-derived actives without establishing THC-specific efficacy or efficacy of the finished Biotex product.

Table 2. Human topical evidence involving tetrahydrocurcumin/tetrahydrocurcuminoids relevant to cosmetic positioning.

Study	Design	THC exposure	Main finding	Interpretation for this review
Wattanakrai et al., 2007 ^[11]	36 female volunteers; closed-patch irritation testing plus cream hydration assessment	Tetrahydrocurcuminoid test materials / commercial curcumin cream	Test materials were less irritating than SDS; the complete cream increased corneometric hydration	Supports topical tolerability and formulation-level moisturization; does not isolate a THC moisturising effect.

Sommerfeld, 2007 ^[12]	Randomised, double-blind, placebo-controlled split-face study; 28 women enrolled, 25 completed	0.1% THC within a four-active gel (with asiatic acid, ursolic acid, DMAE)	Firmness improved significantly on the treated side; 3 of 28 withdrew with mild contact irritation	Human formulation-level evidence; the THC contribution cannot be separated from the three co-actives.
Asawanonda & Klahan, 2010 ^[13]	Preliminary randomised comparative study; 10 patients with vitiligo	Topical tetrahydrocurcumin cream plus targeted NB-UVB	Repigmentation was numerically greater than NB-UVB alone but not statistically significant	Disease-specific exposure/tolerability evidence only; not evidence for cosmetic brightening.
Wanitphakdeedecha et al., 2016 ^[32]	Four-arm facial study; 80 women; twice-daily use for 4 weeks	Curcuminoid cream variants with THC and/or liposome versus vehicle	No significant between-group differences at week 4 for hydration, elasticity, or colour; within-group changes also occurred with vehicle	Direct human evidence cautioning against THC-specific hydration, elasticity, or colour claims.
Muta et al., 2018 ^[24]	Human facial-skin topical study plus skin-equivalent / biochemical work	<i>Curcuma longa</i> rhizome-extract cream; not THC-specific	Higher facial elasticity and fewer gelatinase-positive stratum-corneum sites were reported after topical use	Botanical-lineage and matrix-pathway context; not direct White Curcumin evidence.
Vaughn 2016; Barbalho 2021; Di Lorenzo 2023; Patel 2024 ^[14,27-29]	Independent reviews of human Curcuma/curcumin dermatology, one of them confined to topical application	Heterogeneous turmeric/curcumin in interventions; not THC-specific	Human evidence spans inflammatory, pigmentary, erythema, moisture, scarring/wound and other dermatologic settings, but most of the record concerns oral rather than topical dosing	Strengthens Curcuma-to-human-skin context without establishing THC or finished-product efficacy.

11. Integrated claim-support framework for Biotex White Curcumin Cream

The formulation is best read as a complementary two-active system rather than as a collection of independently proven claims, and on that reading the evidence is coherent. White Curcumin/THC is strongest where the assays are most direct: it outranks both curcumin and Trolox in comparative radical-scavenging assays, lowers melanin and melanogenic-enzyme endpoints in cultured cells at sub-microgram concentrations, protects keratinocytes from oxidative injury, is measurably deliverable from engineered vehicles, modulates COX-2 and TAK1–NF-κB signalling, and improves matrix and photoaging endpoints in preclinical models. Manjistha contributes what THC does not: antityrosinase activity demonstrated in stem material with its active constituents identified, a separate anthraquinone/phenolic antioxidant chemistry, and quinone-mediated inflammatory and barrier effects demonstrated on skin. The two therefore act through partly independent routes toward the same visible endpoints. Human evidence adds limited formulation-level translation and also demonstrates vehicle effects and multi-active confounding^[11-18,23-32]. The resulting claim framework rests on this convergence, while keeping finished-product efficacy distinct from ingredient-level evidence.

Table 3. Claim-to-evidence matrix for the finished-product narrative.

Cosmetic claim domain	White Curcumin evidence	Manjistha evidence	Evidence-calibrated wording
Even-looking tone / visible radiance	THC reduces melanin and melanogenic enzyme/protein endpoints in cellular models and also lowers oxidative stress ^[6,23] .	A stem-derived fraction inhibits tyrosinase; purpurin and ellagic acid were associated with the active fraction ^[16] .	Helps support balanced melanogenesis, a more even-looking tone, and natural radiance.
Antioxidant defense	Biochemical studies show radical-scavenging and lipid-oxidation protection; keratinocyte oxidative-stress data add skin-cell relevance ^[2-4,6] .	Extract, rubiadin, and root hydroxyanthraquinone studies support antioxidant chemistry ^[17,19,20,31] .	Provides botanical antioxidant support against everyday environmental oxidative stress.
Skin comfort	Skin-focused delivery data plus general inflammatory-mechanism studies support local anti-inflammatory plausibility ^[7,10] .	Rubia quinones modulate inflammatory mediators in skin-related models; purpurin adds constituent-level context ^[18,30] .	Helps calm and comfort skin exposed to everyday stressors.
Hydration/softness	Human cream studies show formulation-level hydration signals but also strong vehicle effects; fibroblast hyaluronic-acid data are mechanistic only ^[8,11,32] .	Root-derived keratinocyte-differentiation/barrier evidence is supportive but not direct hydration proof for a Manjistha extract ^[25,26] .	Helps maintain hydration, softness, and smooth feel as part of the cream system.
Texture / age-support	Fibroblast ECM endpoints, a UV-photoaging mouse study, a multi-active split-face trial, and human topical Curcuma matrix evidence support texture-related plausibility ^[8,9,12,24] .	Antioxidant/inflammatory and epidermal-organisation evidence provides complementary support ^[18,25,31] .	Supports smooth, resilient, healthy-looking skin texture and helps defend against the visible effects of everyday environmental stress.

Dual-active mechanism for Biotex White Curcumin Cream

Two ingredient-level evidence streams and the cosmetic outcomes on which they converge

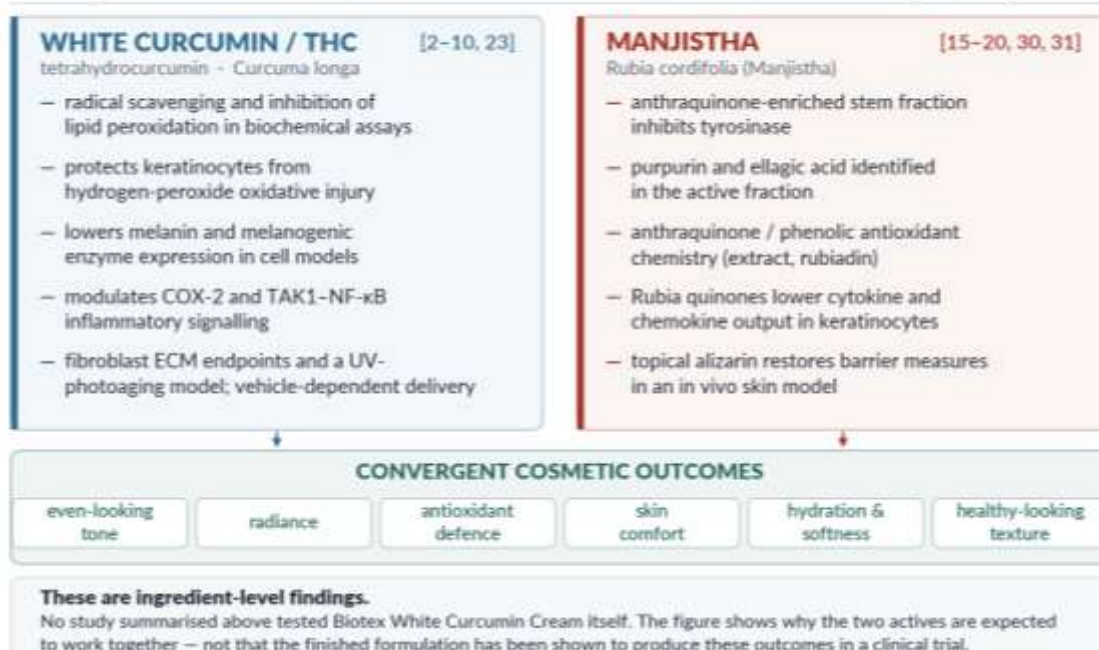


Fig 4: Integrated dual-active mechanism for Biotex White Curcumin Cream. Left column, White Curcumin/THC: radical scavenging and inhibition of lipid peroxidation, keratinocyte protection against oxidative injury, reduced melanin and melanogenic enzyme expression, COX-2 and TAK1–NF-κB modulation, and fibroblast extracellular matrix and UV-photoaging endpoints. Right column, Manjistha: tyrosinase inhibition by an anthraquinone-enriched stem fraction with purpurin and ellagic acid identified in the active fraction, anthraquinone and phenolic antioxidant chemistry, and quinone-mediated inflammatory and barrier modulation. The two streams converge on the six cosmetic outcome domains shown; all findings are ingredient-level.

Evidence hierarchy behind the formulation

What kind of study supports each layer — and where the finished product sits

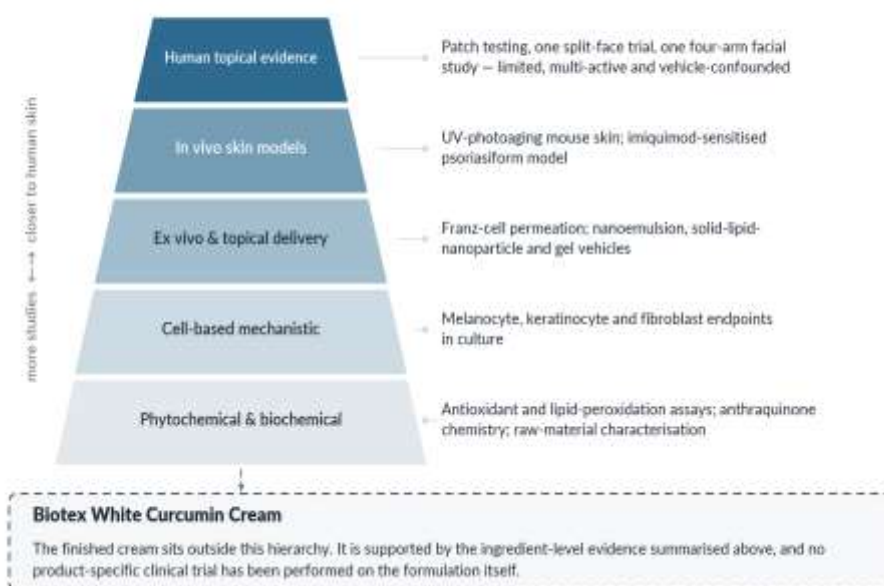


Fig 5: Evidence hierarchy for Biotex White Curcumin Cream. From the base upward: phytochemical and biochemical evidence; cell-based mechanistic studies; ex vivo and topical-delivery work; in vivo skin

models; and, at the apex, the limited human topical evidence for tetrahydrocurcumin-containing formulations. The finished product is shown separately, outside the hierarchy, as a formulation supported by ingredient-level evidence rather than by a product-specific clinical trial.

12. Safety and topical tolerability

The human tolerability record, though small, is favourable. In closed-patch testing on 36 volunteers, tetrahydrocurcuminoid-containing materials produced significantly less irritation than an SDS positive control^[11]. In a vitiligo study combining a tetrahydrocurcuminoid cream with targeted NB-UVB, reported adverse effects were slight and did not interrupt treatment^[13]. Broader randomised curcumin and turmeric dermatology trials have likewise been reported as having an excellent safety profile^[29]. The one adverse signal comes from the multi-active Tricutan split-face trial, in which three of 28 enrolled women withdrew with mild irritant contact dermatitis^[12] — a gel carrying four actives plus 3% dimethylaminoethanol, so the reaction cannot be assigned to THC. Taken together the data support acceptable topical use of the studied formulations while confirming that tolerability is formulation-specific and that safety testing of the finished Biotex cream remains necessary in its own right.

Rubia cordifolia has a long history of traditional use, but topical cosmetic safety is less comprehensively characterised than its phytochemistry and pharmacology. Because botanical extracts contain multiple constituents, prudent product labelling should retain standard external-use precautions and acknowledge the possibility of individual hypersensitivity. The evidence reviewed here does not justify describing either ingredient or the finished cream as universally non-irritating or hypoallergenic.

13. Evidence boundaries and limitations

The principal limitation is translational distance. Much of the strongest mechanistic evidence for THC derives from melanoma cells, keratinocytes, fibroblasts, animal inflammation models, or UV-photoaging models. These systems are appropriate for biological plausibility but cannot establish the magnitude of cosmetic benefit in human facial skin. Human topical evidence exists but is limited and frequently involves multi-active formulations, which prevents attribution of the observed effect to THC alone^[6-14].

A second limitation is chemical equivalence. “White Curcumin,” “tetrahydrocurcumin,” “tetrahydrocurcuminoids,” and “Hydro Curcumin 95%” are often used commercially in overlapping ways, but they are not automatically interchangeable analytical terms. A raw material standardised to 95% may refer to THC itself or to total tetrahydrocurcuminoids depending on supplier specification. The scientific interpretation of the Biotex ingredient should therefore follow its certificate of analysis and chromatographic profile. This manuscript treats THC as the principal pharmacologically relevant constituent of the stated White Curcumin/Hydro Curcumin 95% material^[5].

For Manjistha, plant part and extraction method materially influence composition. The direct antityrosinase study used stem material, whereas other antioxidant and anti-inflammatory studies cited here used roots, whole extracts or isolated quinones. Readers comparing this literature should therefore attend to the plant organ and the extraction solvent reported in each study before transferring a finding from one preparation to another. Finally, none of the cited ingredient studies constitutes a clinical efficacy study of the finished Biotex White Curcumin Cream. The product should therefore be positioned through evidence convergence rather than product-specific therapeutic efficacy.

14. Conclusion

White Curcumin (Hydro Curcumin 95%) and Manjistha form a scientifically coherent two-active topical concept. THC is a colourless hydrogenated curcumin derivative whose antioxidant activity is not merely established but comparatively superior — ranking above curcumin and above the reference antioxidant Trolox in independent radical-scavenging comparisons — and it carries direct experimental evidence for melanogenesis modulation, oxidative-stress protection in skin cells, demonstrable topical delivery, inflammatory-pathway modulation, and preclinical extracellular-matrix and photoaging effects^[1-10,23]. Manjistha contributes a distinct quinone and phenolic chemistry; a stem-derived *R. cordifolia* fraction has shown tyrosinase-inhibitory activity with purpurin and ellagic acid identified in the active fraction, while newer *Rubia* quinone studies add skin-specific inflammatory, barrier and permeation evidence^[15-20,30,31]. The two actives are complementary rather than redundant: they reach the same visible endpoints by partly separate mechanisms.

Taken together, the literature supports evidence-calibrated cosmetic positioning of Biotex White Curcumin Cream around even-looking tone, radiance, antioxidant defence, skin comfort, hydration and softness as properties of the cream system, and healthy-looking texture. Human topical studies add

translational support but also expose multi-active confounding and vehicle effects, so the weight of the evidence remains mechanistic and ingredient-level^[11-14,24,27-29,32]. Accordingly, no product-specific clinical efficacy claim is made here, and the reviewed evidence does not establish that the finished formulation treats pigmentary disease, inflammatory dermatoses, wounds or established photoaging. What the review does establish is a defensible evidential basis for the cosmetic positioning of a two-active White Curcumin and Manjistha system, together with a clarified nomenclature for a raw material whose commercial description has run ahead of its scientific definition.

Abbreviations

THC, tetrahydrocurcumin; CUR, curcumin; ROS, reactive oxygen species; TYR, tyrosinase; TRP-1, tyrosinase-related protein-1; TRP-2, tyrosinase-related protein-2; MITF, microphthalmia-associated transcription factor; ECM, extracellular matrix; UV, ultraviolet; UVB, ultraviolet B; NF- κ B, nuclear factor kappa B; JAK, Janus kinase; STAT3, signal transducer and activator of transcription 3; COX-2, cyclooxygenase-2; NB-UVB, narrowband ultraviolet B; SDS, sodium dodecyl sulfate.

Declarations

Funding: No external funding was received for preparation of this review.

Conflict of interest: All three authors are employed by, or affiliated with, Biotex Life Solutions Pvt. Ltd., the developer and manufacturer of Biotex White Curcumin Cream. No other competing interests are declared.

Author contributions: All authors contributed to the conception and design of this review, to the identification, appraisal and interpretation of the published literature, and to the drafting and critical revision of the manuscript for important intellectual content. All authors read and approved the final version and agree to be accountable for all aspects of the work.

AI-assisted editing statement: Generative AI tools were used only for language refinement, structural organisation, and literature navigation. All scientific interpretation, verification of cited sources and final approval of the manuscript are the responsibility of the named authors, who accept full accountability for the content.

Data availability: No new datasets were generated or analysed. All data discussed are contained within the cited published sources listed in the References.

Ethics approval and consent to participate: Not applicable. This article is a narrative review of previously published literature and involved no human participants, human material, human data or animal experimentation.

References

- Aggarwal BB, Deb L, Prasad S. Curcumin differs from tetrahydrocurcumin for molecular targets, signaling pathways and cellular responses. *Molecules*. 2015; 20(1):185-205. doi:10.3390/molecules20010185.
- Osawa T, Sugiyama Y, Inayoshi M, Kawakishi S. Antioxidative activity of tetrahydrocurcuminoids. *Biosci Biotechnol Biochem*. 1995; 59(9):1609-1612. doi:10.1271/bbb.59.1609.
- Sugiyama Y, Kawakishi S, Osawa T. Involvement of the β -diketone moiety in the antioxidative mechanism of tetrahydrocurcumin. *Biochem Pharmacol*. 1996; 52(4):519-525. doi:10.1016/0006-2952(96)00302-4.
- Somparn P, Phisalaphong C, Nakornchai S, Unchern S, Morales NP. Comparative antioxidant activities of curcumin and its demethoxy and hydrogenated derivatives. *Biol Pharm Bull*. 2007; 30(1):74-78. doi:10.1248/bpb.30.74.
- Trivedi MK, Panda P, Sethi KK, Gangwar M, Mondal SC, Jana S. Solid and liquid state characterization of tetrahydrocurcumin using XRPD, FT-IR, DSC, TGA, LC-MS, GC-MS, and NMR and its biological activities. *J Pharm Anal*. 2020; 10(4):334-345. doi:10.1016/j.jpha.2020.02.005.
- Tang X, Dong Q, Li J, Li F, Michniak-Kohn BB, Zhao D, et al. Anti-Melanogenic Mechanism of Tetrahydrocurcumin and Enhancing Its Topical Delivery Efficacy Using a Lecithin-Based Nanoemulsion. *Pharmaceutics*. 2021; 13(8):1185. doi:10.3390/pharmaceutics13081185.

7. Kakkar V, Kaur IP, Kaur AP, Saini K, Singh KK. Topical delivery of tetrahydrocurcumin lipid nanoparticles effectively inhibits skin inflammation: in vitro and in vivo study. *Drug Dev Ind Pharm.* 2018; 44(10):1701-1712. doi:10.1080/03639045.2018.1492607.
8. Trivedi MK, Gangwar M, Mondal SC, Jana S. Protective effects of tetrahydrocurcumin (THC) on fibroblast and melanoma cell lines in vitro: its implication for wound healing. *J Food Sci Technol.* 2017; 54(5):1137-1145. doi:10.1007/s13197-017-2525-8.
9. Xu C, Xiong QW, Li Y, Zhao JN, Zhang L, Li XL. Explore the multitarget mechanism of tetrahydrocurcumin preventing on UV-induced photoaging mouse skin. *Heliyon.* 2022; 8(8):e09888. doi:10.1016/j.heliyon.2022.e09888.
10. Zhang ZB, Luo DD, Xie JH, Xian YF, Lai ZQ, Liu YH, et al. Curcumin's Metabolites, Tetrahydrocurcumin and Octahydrocurcumin, Possess Superior Anti-inflammatory Effects in vivo Through Suppression of TAK1-NF- κ B Pathway. *Front Pharmacol.* 2018; 9:1181. doi:10.3389/fphar.2018.01181.
11. Wattanakrai P, Suwanachote S, Kulkollakarn S, Rajatanavin N. The study of human skin irritation of a novel herbal skin care product and ingredients by human single closed patch testing. *J Med Assoc Thai.* 2007; 90(6):1116-1122. PMID:17624205.
12. Sommerfeld B. Randomised, placebo-controlled, double-blind, split-face study on the clinical efficacy of Tricutan on skin firmness. *Phytomedicine.* 2007; 14(11):711-715. doi:10.1016/j.phymed.2007.09.015.
13. Asawanonda P, Klahan SO. Tetrahydrocurcuminoid cream plus targeted narrowband UVB phototherapy for vitiligo: a preliminary randomized controlled study. *Photomed Laser Surg.* 2010; 28(5):679-684. doi:10.1089/pho.2009.2637.
14. Di Lorenzo R, Forgione F, Bernardi A, Sacchi A, Laneri S, Greco G. Clinical Studies on Topical Curcumin. *Skin Pharmacol Physiol.* 2023; 36(5):235-248. doi:10.1159/000535100.
15. Wen M, Chen Q, Chen W, Yang J, Zhou X, Zhang C, et al. A comprehensive review of *Rubia cordifolia* L.: Traditional uses, phytochemistry, pharmacological activities, and clinical applications. *Front Pharmacol.* 2022; 13:965390. doi:10.3389/fphar.2022.965390.
16. Insaf A, Parveen R, Srivastava V, Samal M, Khan M, Ahmad S. TLC-MS-Bioautographic Identification of Antityrosinase Compounds and Preparation of a Topical Gel Formulation from a Bioactive Fraction of an RSM-Optimized Alcoholic Extract of *Rubia cordifolia* L. stem. *J AOAC Int.* 2023; 106(6):1598-1607. doi:10.1093/jaoacint/qsad076.
17. Humbare RB, Sarkar J, Kulkarni AA, Juwale MG, Deshmukh SH, Amalnerkar D, et al. Phytochemical Characterization, Antioxidant and Anti-Proliferative Properties of *Rubia cordifolia* L. Extracts Prepared with Improved Extraction Conditions. *Antioxidants (Basel).* 2022; 11(5):1006. doi:10.3390/antiox11051006.
18. Lin CF, Chen HY, Alalaiwe A, Hsiao YT, Jhong CL, Chang SH, et al. Harnessing Quinone Derivatives from *Rubia cordifolia* for Topical Therapy: Unveiling Structure-Activity and Structure-Permeation Relationships to Suppress Psoriasiform Inflammation. *ACS Pharmacol Transl Sci.* 2025; 8(7):2270-2289. doi:10.1021/acspsci.5c00352.
19. Tripathi YB, Sharma M. The interaction of *Rubia cordifolia* with iron redox status: a mechanistic aspect in free radical reactions. *Phytomedicine.* 1999; 6(1):51-57. doi:10.1016/S0944-7113(99)80035-X.
20. Tripathi YB, Sharma M, Manickam M. Rubiadin, a new antioxidant from *Rubia cordifolia*. *Indian J Biochem Biophys.* 1997; 34(3):302-306. PMID:9425750.
21. Tu CX, Lin M, Lu SS, Qi XY, Zhang RX, Zhang YY. Curcumin inhibits melanogenesis in human melanocytes. *Phytother Res.* 2012; 26(2):174-179. doi:10.1002/ptr.3517.
22. Asada K, Ohara T, Muroyama K, Yamamoto Y, Murosaki S. Effects of hot water extract of *Curcuma longa* on human epidermal keratinocytes in vitro and skin conditions in healthy participants: a randomized, double-blind, placebo-controlled trial. *J Cosmet Dermatol.* 2019; 18(6):1866-1874. doi:10.1111/jocd.12890.

23. Su YS, Lee KT, Hsu YC. Tetrahydrocurcumin (THC) as a melanogenesis inhibitor in melanoma cell lines. *Biochem Res Int.* 2025;2025: 6256669. doi:10.1155/bri/6256669.
24. Muta K, Inomata S, Fukuhara T, Nomura J, Nishiyama T, Tagawa YI, et al. Inhibitory effect of the extract of rhizome of *Curcuma longa* L. in gelatinase activity and its effect on human skin. *J Biosci Bioeng.* 2018; 125(3):353-358. doi:10.1016/j.jbiosc.2017.10.001.
25. Lin ZX, Jiao BW, Che CT, Zuo Z, Mok CF, Zhao M, et al. Ethyl acetate fraction of the root of *Rubia cordifolia* L. inhibits keratinocyte proliferation in vitro and promotes keratinocyte differentiation in vivo: potential application for psoriasis treatment. *Phytother Res.* 2010; 24(7):1056-1064. doi:10.1002/ptr.3079.
26. Casetti F, Wölfl U, Gehring W, Schempp CM. Dermocosmetics for dry skin: a new role for botanical extracts. *Skin Pharmacol Physiol.* 2011; 24(6):289-293. doi:10.1159/000329214.
27. Vaughn AR, Branum A, Sivamani RK. Effects of turmeric (*Curcuma longa*) on skin health: a systematic review of the clinical evidence. *Phytother Res.* 2016; 30(8):1243-1264. doi:10.1002/ptr.5640.
28. Barbalho SM, de Sousa Gonzaga HF, de Souza GA, de Alvares Goulart R, de Sousa Gonzaga ML, de Alvarez Rezende B. Dermatological effects of *Curcuma* species: a systematic review. *Clin Exp Dermatol.* 2021; 46(5):825-833. doi:10.1111/ced.14584.
29. Patel P, Wang JY, Mineroff J, Jagdeo J. Evaluation of curcumin for dermatologic conditions: a systematic review. *Arch Dermatol Res.* 2024; 316(1):37. doi:10.1007/s00403-023-02754-8.
30. Singh J, Hussain Y, Luqman S, Meena A. Purpurin: a natural anthraquinone with multifaceted pharmacological activities. *Phytother Res.* 2021; 35(5):2418-2428. doi:10.1002/ptr.6965.
31. Cai Y, Sun M, Xing J, Corke H. Antioxidant phenolic constituents in roots of *Rheum officinale* and *Rubia cordifolia*: structure-radical scavenging activity relationships. *J Agric Food Chem.* 2004; 52(26):7884-7890. doi:10.1021/jf0489116.
32. Wanitphakdeedecha R, Ungakornpairote C, Manuskiatti W, Panich U, Akarasereenont P. The effects of tetrahydrocurcumin (THC) in curcuminoid cream on the hydration, elasticity and color of human skin. *Thai J Dermatol.* 2016; 32(2):106-115. Available from: <https://he02.tci-thaijo.org/index.php/TJD/issue/view/19049>