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Therapeutic potential of curcumin in dermatological conditions: mechanisms, clinical evidence and safety considerations

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Abstract

Curcumin, a pleiotropic polyphenol derived from *Curcuma longa*, has attracted sustained interest as a potential adjuvant therapy in dermatology because of its anti inflammatory, antioxidant, antimicrobial and immunomodulatory properties. A rapidly expanding body of preclinical research indicates that curcumin modulates multiple molecular pathways implicated in skin disease, including nuclear factor kappa B (NF kappa B), Janus kinase and signal transducer and activator of transcription (JAK–STAT) signalling, mitogen-activated protein kinases and the Nrf2 antioxidant response, with demonstrable effects on keratinocytes, fibroblasts, melanocytes, sebocytes and cutaneous immune cells [1-4]. These mechanistic data have prompted a growing number of clinical studies evaluating oral and topical curcumin, as well as advanced formulations and photodynamic applications, across a spectrum of dermatological conditions such as psoriasis, atopic dermatitis, acne vulgaris, disorders of pigmentation, chronic wounds and photoaging [5-8]. Overall, the clinical evidence suggests that curcumin may confer clinically meaningful benefit as an adjunct to standard therapy in psoriasis, pruritic and inflammatory dermatoses, limited forms of vitiligo and selected wound-healing scenarios, whereas evidence for cosmetic indications and infectious dermatoses remains preliminary [6-8]. Safety data from dermatological and non dermatological trials indicate a generally favourable profile, with mainly mild gastrointestinal and local irritant adverse effects, although emerging reports of hepatotoxicity and potential drug interactions necessitate caution with high-dose systemic or long-term use [3, 4, 16, 17]. Translation of curcumin into routine dermatological practice is hindered by poor and variable bioavailability, heterogeneity of formulations, small and methodologically limited trials and inadequate standardisation of outcomes. This narrative review synthesises mechanistic and clinical data on curcumin in skin disease, appraises the strength and limitations of current evidence and outlines priorities for future research required to define the realistic therapeutic niche of curcumin in modern dermatology.

Keywords: curcumin; turmeric; dermatology; psoriasis; atopic dermatitis; acne vulgaris; vitiligo; photodamage; chronic wounds; phytotherapy; nanotechnology.

Introduction

Curcumin is a diarylheptanoid polyphenol that constitutes the major bioactive component of the rhizome of *Curcuma longa*, a spice widely used in South and East Asian cuisine and traditional medicine. Over the past three decades it has been investigated in a broad range of chronic inflammatory and degenerative disorders, driven by extensive mechanistic and clinical data indicating potent anti-inflammatory, antioxidant and immunomodulatory actions at micromolar concentrations in vitro and at tolerated doses in vivo [1-4]. At the molecular level, curcumin can inhibit NF-kappa B activation, reduce expression of pro-inflammatory cytokines such as tumour necrosis factor alpha, interleukin 1 beta, interleukin 6 and interleukin 17, modulate JAK–STAT pathways and activate the Nrf2 antioxidant response element, among other effects [1- 4]. These pleiotropic actions underpin its designation as a "golden nutraceutical" and explain its putative relevance to skin disease, where chronic inflammation, oxidative stress and immune dysregulation are central to pathogenesis [2, 3].

In dermatology, interest in curcumin has been catalysed by several converging factors. Many chronic dermatoses such as psoriasis, atopic dermatitis and acne vulgaris require long-term therapy, and conventional systemic agents, including methotrexate, ciclosporin and oral retinoids, carry cumulative toxicity or monitoring burdens. There is, at the same time, a growing demand from patients and within integrative and lifestyle medicine for evidence-based botanical or nutraceutical options that can complement standard treatments and potentially reduce their required doses. In parallel, curcumin exhibits biological activities that map onto key pathogenic pathways in common skin diseases, including Th1, Th17 and Th22 polarisation, epidermal hyperproliferation, matrix metalloproteinase-driven dermal remodelling, dysregulated melanogenesis and microbial colonisation [5-7]. Finally, the last decade has seen substantial advances in formulation science, including nanoencapsulation, liposomal systems and phytosomal complexes that can partially overcome curcumin's poor aqueous solubility and low oral bioavailability, enabling more reliable systemic and topical exposure [9-12].

The dermatological literature on curcumin has expanded considerably since the first small trials in the early 2000s. Several systematic reviews and narrative overviews have synthesised data across conditions, generally concluding that turmeric and curcumin appear promising but that the overall quality of evidence is low to moderate, with notable heterogeneity in study design and formulations [6-8]. More recent work has shifted from crude turmeric preparations towards standardised curcumin extracts, phytosomal complexes and nanoformulations, and has begun to integrate curcumin into combination strategies, for example with acitretin in psoriasis or as a photosensitiser in photodynamic therapy for acne [8-10, 14, 15]. At the same time, large umbrella reviews of curcumin supplementation in non-dermatologic conditions have

highlighted both its acceptable safety and the risk of publication bias and small-study effects [3, 18]. Against
this background, a critical, disease-oriented appraisal focused specifically on dermatological indications is timely.

The aim of this review is to summarise mechanistic insights relevant to skin biology, catalogue and critique the clinical data for curcumin across major dermatological disease categories, evaluate safety and methodological limitations and delineate realistic therapeutic opportunities and research priorities, rather than to promote curcumin as a panacea.

Pharmacology and mechanisms of action relevant to the skin

Curcumin exerts a wide spectrum of molecular effects that are highly relevant to dermatological pathophysiology. At the core of its anti-inflammatory action is inhibition of NF-kappa B signalling. Curcumin can prevent phosphorylation and degradation of inhibitor of kappa B, thereby reducing nuclear translocation of NF-kappa B and downstream transcription of multiple pro-inflammatory genes, including tumour necrosis factor alpha, interleukin 1 beta, interleukin 6, interleukin 8 and cyclo-oxygenase 2 [1-3]. In various cellular systems, these actions translate into decreased production of inflammatory cytokines, chemokines and adhesion molecules, reduced expression of inducible nitric oxide synthase and dampened recruitment of inflammatory cells. Curcumin also modulates JAK–STAT pathways, particularly STAT3, which is implicated in epidermal hyperproliferation and Th17 responses in psoriasis, and it can inhibit mitogen activated protein kinase pathways, including p38 and extracellular signal-regulated kinase, further contributing to anti-inflammatory and anti-proliferative effects [3, 9, 10].

Beyond these canonical signalling cascades, curcumin exerts robust antioxidant and cytoprotective effects through activation of the Nrf2 pathway. Nrf2, when stabilised and translocated to the nucleus, upregulates multiple antioxidant and phase II detoxification enzymes such as heme oxygenase 1, glutathione S-transferases and NAD(P)H quinone dehydrogenase 1. In keratinocytes and dermal fibroblasts exposed to ultraviolet radiation or pro-oxidant stimuli, curcumin reduces reactive oxygen species generation, lipid peroxidation and DNA damage, while preserving mitochondrial function and cellular viability [5, 6, 9, 10]. These effects have obvious implications for photodamage, photoaging and wound healing, where sustained oxidative stress drives tissue injury and impaired repair.

Curcumin also modulates key cellular actors in the skin. In keratinocytes, it can inhibit proliferation and promote a more differentiated phenotype, while reducing expression of keratin 16 and other markers associated with psoriatic hyperplasia [5, 9]. In dermal fibroblasts, curcumin downregulates matrix metalloproteinases such as matrix metalloproteinase 1 and matrix metalloproteinase 9, reduces collagen degradation and can stimulate collagen synthesis under some conditions, suggesting anti-wrinkle and dermal-protective properties [5, 6, 9]. In

melanocytes, curcumin has a complex profile:
at lower concentrations it can inhibit tyrosinase activity and melanin synthesis, largely through modulation of microphthalmia-associated transcription factor and related signalling, whereas at higher concentrations it may induce melanocyte apoptosis, raising concerns about cytotoxicity if not carefully dosed or formulated; its hydrogenated metabolite tetrahydrocurcumin appears to retain depigmenting effects with a more favourable safety profile in vitro and in vivo [6, 7].

In sebocytes and within the pilosebaceous unit, curcumin demonstrates antimicrobial activity against *Cutibacterium acnes* and *Staphylococcus aureus* and attenuates lipopolysaccharide-induced inflammatory responses, including reductions in interleukin 8 and interleukin 1 beta [6, 9]. Curcumin can also modulate peroxisome proliferator-activated receptor gamma and sterol regulatory element-binding protein 1 signalling, potentially influencing sebum production. These properties, combined with its photosensitising capacity under blue or visible light, underpin its exploration in photodynamic therapy for acne vulgaris [14, 15].

Curcumin affects both innate and adaptive cutaneous immunity. In macrophages and dendritic cells, it reduces expression of major histocompatibility complex and costimulatory molecules, inhibits Toll-like receptor signalling and skews cytokine production toward a less inflammatory profile [1-3, 9]. In T lymphocytes, curcumin can suppress Th1 and Th17 polarisation while enhancing regulatory T-cell function in several models. In mast cells and basophils, it inhibits degranulation and histamine release. Collectively, these effects are relevant to diseases such as psoriasis, atopic dermatitis, chronic urticaria and other immune-mediated dermatoses [5-7].

A central challenge in translating these mechanistic insights into clinical efficacy is curcumin's unfavourable pharmacokinetic profile. Orally administered curcumin is poorly absorbed from the gastrointestinal tract, undergoes extensive first-pass metabolism to glucuronide and sulfate conjugates and is rapidly eliminated. Plasma concentrations after conventional oral doses are often in the nanomolar range, well below those used in most in vitro experiments [3, 9, 10]. Multiple formulation strategies have been developed to enhance systemic bioavailability, including complexation with phospholipids (phytosomal formulations), inclusion complexes with cyclodextrins, co administration with piperine to inhibit glucuronidation and various nanoparticle-based systems such as solid lipid nanoparticles, polymeric micelles and nanocrystals [9-12]. Clinical pharmacokinetic studies indicate that some of these approaches can increase area under the curve of curcumin and its metabolites by an order of magnitude or more, although this does not necessarily guarantee proportional improvements in clinical effect and may alter safety and interaction profiles.

Topical delivery faces distinct challenges and opportunities. Curcumin is lipophilic and poorly water soluble but has an affinity for lipid-rich environments including the stratum corneum. Simple ointments and creams containing curcumin or turmeric extracts can
deposit pigment in the stratum corneum and superficial epidermis, leading to visible yellow

staining but not necessarily therapeutically relevant concentrations in deeper viable layers. Advanced topical systems, including microemulgels, liposomes, solid lipid nanoparticles, nanostructured lipid carriers, polymeric nanofibres, hyaluronic acid based hydrogels and microneedle arrays, have demonstrated enhanced penetration, prolonged residence time and more controlled release of curcumin in ex vivo human skin and animal models [9-12]. Some of these formulations also exploit co-delivery of other actives such as acitretin or chlorin photosensitisers to achieve synergistic effects in psoriasis or photoaging models.

Formulations and routes of administration in dermatology

Curcumin has been administered in dermatology research through oral, topical and, less commonly, combined or device-assisted routes, each with distinct pharmacokinetic and practical implications. Oral administration typically uses standardised curcumin extracts in capsule or tablet form, often in the range of 500 to 2000 milligrams per day, with or without bioavailability enhancers such as piperine or phospholipid complexes [3, 9, 10]. Phytosomal curcumin formulations in which curcuminoids are complexed with phosphatidylcholine have been widely studied and demonstrate substantially higher plasma concentrations than unformulated curcumin at equivalent doses. Oral curcumin is attractive in chronic inflammatory dermatoses where systemic immune modulation is desirable, including psoriasis, atopic dermatitis and pruritic disorders, and it may also contribute to systemic antioxidant and anti-inflammatory effects in patients with comorbidities [6-8].

Topical administration aims to deliver curcumin directly to lesional skin while limiting systemic exposure. Vehicles include ointments, creams, gels, lotions, foams, pastes and masks. Simple oil-based ointments or creams containing turmeric powder or curcumin extract are frequently used in traditional practice and have been evaluated in some early clinical trials, but they are limited by variable active content, unstable curcumin, relatively poor penetration and pronounced yellow staining of skin and clothing. More sophisticated vehicles such as microemulgels can solubilise curcumin in mixed surfactant systems, enhancing its dispersion and penetration; several small trials in psoriasis and vitiligo have used such formulations [6-8]. Hydrogels based on hyaluronic acid, chitosan or other polymers can provide moist wound environments and sustained local release, which is attractive for chronic wounds and ulcers [11-13].

Nanotechnology-based topical systems represent a major recent advance. Solid lipid nanoparticles and nanostructured lipid carriers encapsulate curcumin in lipid matrices that can merge with the stratum corneum lipid lamellae, enhancing penetration and providing a depot effect [11]. Polymeric nanoparticles and micelles can increase aqueous dispersion, protect

curcumin from degradation and enable controlled release.

Nanofibrous scaffolds, prepared by electrospinning curcumin-loaded polymers, can serve as wound dressings that combine physical coverage with release of curcumin and other actives [12, 13]. Microneedle arrays coated with or containing curcumin have been explored in preclinical studies to bypass the stratum corneum barrier and deliver the drug into the viable epidermis and superficial dermis. While many of these systems are still at the preclinical stage, some have progressed to early-phase clinical trials in psoriasis and wound healing [9-13].

A third mode of delivery is photodynamically assisted application. Curcumin has intrinsic photosensitising properties in the blue and visible light spectrum; upon light activation, it can generate reactive oxygen species that exert antimicrobial and cytotoxic effects. In acne vulgaris, topical curcumin solutions or gels have been applied and then irradiated with blue or light-emitting diode-based sources to achieve photodynamic therapy, targeting *C. acnes* colonisation and sebaceous gland hyperactivity [14, 15]. Similar approaches are being explored experimentally for photoaging and superficial infections. In these settings, curcumin is effectively a topical photosensitising agent rather than a conventional anti-inflammatory drug, and its safety and efficacy profile depends on light parameters as much as on drug formulation.

Across indications, several patterns emerge. Most of the clinical evidence in psoriasis and pruritic dermatoses involves oral curcumin, often in phytosomal form, used as an adjunct to topical corticosteroids or systemic agents. Topical microemulgels and creams dominate studies in psoriasis, vitiligo, hyperpigmentation and cosmetic photoaging, reflecting the localised nature of these conditions. In chronic wounds and burns, hydrogels, nanofibres and nanomicellar systems predominate, aligning with modern wound-care principles [6-8, 11-13]. In acne, both oral supplements and topical photodynamic applications have been evaluated. The relative strength of evidence tends to be greatest for oral phytosomal formulations in psoriasis and for topical formulations in limited vitiligo and hyperpigmentation, whereas nanotechnology-based systems, microneedles and photodynamic combinations are supported primarily by preclinical or early pilot data [7-10, 14, 15].

Clinical evidence by disease entity

Psoriasis

Psoriasis is a chronic, immune-mediated inflammatory dermatosis characterised by epidermal hyperproliferation, aberrant keratinocyte differentiation and a dominant Th17 and Th1 cytokine milieu centred on the interleukin 23 and interleukin 17 axis. NF-kappa B activation, oxidative stress and elevated tumour necrosis factor alpha are prominent features in lesional skin and systemic circulation. Given curcumin's ability to inhibit NF-kappa B and STAT3, reduce key cytokines and modulate oxidative pathways, its use in psoriasis is mechanistically plausible [1-3, 5-10].

Preclinical studies in imiquimod-induced and other murine models of psoriasis show that oral or topical curcumin reduces epidermal thickness, scaling and erythema, downregulates interleukin 17, interleukin 22 and interleukin 23 and normalises keratinocyte differentiation markers [5, 9, 10]. Nanostructured lipid carriers and other nanoformulations further enhance these effects by improving cutaneous penetration and local retention [9-12]. These data provided the foundation for clinical exploration.

Several small clinical trials and observational studies have evaluated curcumin in plaque psoriasis. Randomised controlled trials of oral phytosomal curcumin given in combination with topical corticosteroids versus topical corticosteroids alone in patients with mild to moderate psoriasis have reported higher proportions of patients achieving at least 50 percent reduction in Psoriasis Area and Severity Index and larger decreases in serum interleukin 22 in the curcumin arm, although absolute effect sizes were modest and sample sizes small [7, 9]. Another randomised trial examined curcumin solid lipid nanoparticles as an adjunct to acitretin in moderate to severe psoriasis, finding greater reductions in Psoriasis Area and Severity Index and Dermatology Life Quality Index than with acitretin alone, without additional laboratory toxicity, again in a small cohort [9, 11].

Topical curcumin formulations have also been investigated. In a double-blind study of patients with mild to moderate plaque psoriasis, a curcumin-containing microemulgel applied to lesional skin twice daily was compared with vehicle over several weeks; lesions treated with curcumin showed greater improvement in erythema, scaling and induration scores than vehicle, although the study did not include an active comparator such as topical vitamin D analogues or corticosteroids [6, 7]. Cosmetic acceptability was limited by yellow staining, although this was less pronounced than with crude turmeric pastes. Other small case series and uncontrolled studies using turmeric ointments or combined oral and topical regimens generally report improvement in lesion appearance and symptoms but are difficult to interpret due to lack of control groups and variable concomitant therapies [5-7].

Systematic reviews focusing on curcumin in psoriasis, which include both oral and topical interventions, conclude that curcumin appears to improve Psoriasis Area and Severity Index and patient-reported outcomes compared with baseline or placebo but emphasise that the overall certainty of evidence is low because of risk of bias, heterogeneity of formulations and small numbers of participants [7-10]. In clinical practice, these data support considering curcumin as a potential adjunct to standard topical or systemic therapy in motivated, well-counselled patients with mild to moderate plaque psoriasis who seek complementary approaches and have no contraindications to curcumin. The evidence does not support its use as monotherapy in moderate or severe disease, nor as a substitute for biologic agents or conventional systemic drugs,
and long-term efficacy, impact on comorbid psoriatic arthritis and optimal dosing remain insufficiently characterised.

Atopic dermatitis and other eczematous or pruritic dermatoses

Atopic dermatitis is characterised by epidermal barrier dysfunction, type 2 skewed immunity with interleukin 4, interleukin 13 and interleukin 31 dominance, chronic pruritus and a propensity to colonisation with *Staphylococcus aureus*. Oxidative stress and activation of NF-kappa B and JAK–STAT pathways also contribute to disease pathophysiology. Curcumin could, in principle, ameliorate atopic dermatitis by reducing type 2 and Th17 cytokines, stabilising mast cells, enhancing barrier function and modulating the skin microbiome [5-7, 9, 10].

In murine models of atopic dermatitis induced by ovalbumin or house dust mite extract, topical or oral curcumin reduces epidermal thickness, inflammatory infiltrates and serum immunoglobulin E levels, lowers interleukin 4 and interleukin 13 and increases filaggrin expression in the epidermis [6, 9, 10]. Essential oil fractions of *Curcuma longa* have similarly attenuated atopic dermatitis-like lesions and pruritus in animal models. These preclinical results provide a rationale for translation but do not directly inform human efficacy.

Human data are more limited than in psoriasis. Early small open-label studies suggested that oral turmeric or curcumin preparations could improve pruritus and lesion severity in atopic dermatitis, but these trials lacked control groups and used non-standardised outcome measures. More recently, randomised pilot trials of phytosomal curcumin formulations in adults with mild to moderate atopic dermatitis have reported reductions in Scoring Atopic Dermatitis index and pruritus scores compared with baseline, with trends toward greater improvement than placebo, although they were underpowered for definitive conclusions [7, 8]. Curcumin has also been studied in pruritic dermatoses that share features with eczematous disorders. Randomised trials in patients with chronic cutaneous complications from sulfur mustard exposure and in those with uraemic pruritus showed that oral curcumin-based supplements reduced pruritus intensity and improved sleep quality compared with placebo, again in small cohorts [7, 8].

Overall, the evidence base for curcumin in atopic dermatitis and related pruritic dermatoses consists of supportive preclinical data, small pilot trials and extrapolation from related conditions. Curcumin cannot be recommended as stand-alone treatment for atopic dermatitis, but may be cautiously considered as an adjunct to emollients and anti-inflammatory therapy in selected adults, particularly where chronic pruritus is a dominant complaint and conventional options are insufficient or poorly tolerated. More robust trials are required to define its place, if any, alongside targeted therapies such as biologics and JAK inhibitors.

Acne vulgaris and other follicular disorders

Acne vulgaris is a multifactorial disorder involving follicular hyperkeratinisation, sebum overproduction, colonisation with *Cutibacterium acnes* and complex innate and adaptive immune responses. Curcumin's antimicrobial activity against Gram-positive bacteria, its ability to reduce interleukin 8 and other inflammatory mediators in sebocytes and keratinocytes

and its photosensitising properties have prompted evaluation in acne management [5-7, 9, 14, 15].

In vitro, curcumin inhibits growth of *C. acnes* at micromolar concentrations and reduces biofilm formation, while diminishing inflammatory cytokine release from keratinocytes exposed to bacterial products. These findings led to the development of topical curcumin formulations, both as conventional creams and gels and as photosensitisers for photodynamic therapy. Several small clinical studies have explored oral curcumin containing supplements in acne as adjuncts to standard topical regimens, with some suggesting faster reduction in inflammatory lesion counts and erythema, but with heterogeneous formulations and limited methodological rigour [6-8].

Curcumin-mediated photodynamic therapy has been more systematically investigated. In a split-face randomised study of patients with mild to moderate acne, Zhang and colleagues applied a 1 percent curcumin mask to one side of the face followed by 445 nm blue light, while the contralateral side received light alone; at two weeks after the final session, the curcumin-treated side showed significantly greater reductions in total and inflammatory lesion counts than the light-only side, with good tolerability [14]. A larger randomised, double-blind study evaluated a gel containing curcumin-rich *Curcuma longa* actives used with blue light-emitting diode therapy compared with vehicle plus light or light alone; the combination produced the largest reduction in inflamed lesions and the greatest improvement in acne-specific quality of life indices, again without serious adverse effects [15]. Adverse events across studies have generally been mild and transient, including erythema, burning and occasional post-inflammatory hyperpigmentation in darker phototypes.

Evidence for curcumin in other follicular disorders such as hidradenitis suppurativa remains anecdotal, consisting of case reports and patient surveys suggesting that oral turmeric supplements may reduce pain and drainage in some individuals; these observations have not been tested in controlled trials and should be interpreted cautiously. Overall, current data support curcumin as a potentially useful adjunct in acne vulgaris, particularly in the context of photodynamic protocols, but there is insufficient evidence to recommend it as monotherapy for moderate or severe acne or to define its role relative to established treatments such as topical retinoids, benzoyl peroxide, oral antibiotics and isotretinoin.

Disorders of pigmentation

Disorders of pigmentation in which curcumin has been explored include hyperpigmentary conditions such as melasma and post-inflammatory hyperpigmentation, as well as hypopigmentary disorders such as vitiligo. Curcumin’s effects on melanogenesis are concentration-dependent and context-specific, which complicates its therapeutic deployment. In hyperpigmentation, curcumin and tetrahydrocurcumin can inhibit tyrosinase activity and melanin synthesis in melanocyte cultures and skin models [5, 6]. Small randomised trials of tetrahydrocurcumin creams in facial hyperpigmentation have reported reductions in pigment

intensity comparable to those achieved with hydroquinone, with fewer reports of irritation, but sample sizes are small and follow-up short [6, 7].

Vitiligo presents a distinct challenge, with autoimmune destruction of melanocytes leading to depigmented macules and patches. Curcumin has been proposed as an adjunctive agent that might protect melanocytes from oxidative stress and modulate immune responses by activating Nrf2 and inhibiting interferon gamma-driven pathways [5-7, 9, 10]. A limited number of small randomised or quasi-randomised trials of turmeric-based creams, sometimes combined with narrowband ultraviolet B phototherapy, suggest modest improvements in repigmentation compared with vehicle, but the data are sparse and heterogeneous [7, 8]. Overall, curcumin-containing preparations may have a modest adjuvant effect in pigmentary disorders, but they are far from established therapies and should not replace phototherapy or topical calcineurin inhibitors.

Chronic wounds, ulcers and impaired wound healing

Curcumin's wound-healing potential is supported by extensive preclinical evidence. In rodent models of acute incisional wounds, excisional wounds and burns, topical or locally delivered curcumin accelerates wound closure, enhances re-epithelialisation, increases angiogenesis and granulation tissue formation and improves collagen deposition and tensile strength [5, 9, 10]. These effects are attributed to modulation of inflammatory cytokines, reduction of oxidative stress, normalisation of matrix metalloproteinase activity and promotion of transforming growth factor beta-mediated repair pathways. Nanoencapsulated curcumin, nanofibrous scaffolds and curcumin loaded hydrogels frequently outperform simple ointments in these models, reflecting improved solubility and controlled release [11-13].

Clinical data, although more limited, generally align with these findings. In small trials of patients with partial-thickness burns, curcumin-containing dressings or nanomicellar formulations have been associated with faster wound epithelialisation, reduced pain and lower infection rates compared with standard dressings [6, 13]. Other studies have examined curcumin-based topical formulations in diabetic foot ulcers or venous leg ulcers, often in combination with other herbal agents or honey. Some report improved wound area reduction and granulation tissue quality compared with baseline or control dressings, but methodological shortcomings, including lack of blinding, small sample sizes and concomitant interventions, preclude firm conclusions [6-8, 11-13].

For dermatologists, the most relevant potential applications are in partial-thickness burns, donor-site wounds and chronic leg ulcers where topical curcumin hydrogels or nanofibrous dressings might complement standard of care, particularly in resource limited settings. At present, however, there are no widely approved curcumin-based wound dressings with robust phase 3 data, and curcumin should be considered experimental in this domain.

Photodamage, photoaging and ultraviolet-induced skin injury

Ultraviolet radiation induces DNA damage, oxidative stress, inflammation and matrix degradation in the skin, culminating in photoaging and carcinogenesis. Curcumin's antioxidant, anti-inflammatory and matrix metalloproteinase-modulating properties, together with its capacity to activate Nrf2, have stimulated interest in its photoprotective and anti-photoaging potential [5, 6, 9-11]. In multiple in vitro studies, curcumin protects keratinocytes and fibroblasts from ultraviolet B-induced apoptosis, reduces reactive oxygen species levels, decreases lipid peroxidation and attenuates expression of matrix metalloproteinase 1 and matrix metalloproteinase 3, while preserving procollagen synthesis. In hairless mouse models, topical or oral curcumin reduces ultraviolet induced erythema, epidermal hyperplasia and wrinkle formation, with histological preservation of collagen and elastic fibres [5-7, 9-11].

Human data for curcumin in photodamage are relatively scarce and mainly involve cosmetic outcomes. Small studies of curcumin-containing creams or lotions applied to photoaged facial skin report modest improvements in wrinkle scores, skin elasticity and pigmentation over several months, but these trials are often open label, involve multi ingredient formulations and use subjective outcome measures [6, 7]. Curcumin is also being incorporated into cosmetic products marketed for "brightening", "glow" or "anti aging" purposes, frequently in combination with other botanicals and vitamins. While such products may confer some benefits due to the combined antioxidant load, robust evidence specifically attributing clinical benefit to curcumin is lacking, and regulatory oversight for cosmeceuticals is less stringent than for medicinal products. Curcumin cannot be recommended as a substitute for established photoprotective measures such as broad-spectrum sunscreens, photoprotective clothing and behavioural sun avoidance, although it may ultimately find a role as an adjunctive ingredient in evidence based cosmeceutical regimens [6-8, 11, 18].

Infectious dermatoses

Curcumin exhibits broad-spectrum antimicrobial activity in vitro, including antibacterial, antifungal and antiviral effects. It disrupts bacterial membranes, interferes with quorum sensing and inhibits virulence factors in several pathogens and can potentiate the activity of conventional antibiotics [1-3, 5-7]. In fungi, curcumin has shown inhibitory effects on *Candida* species and dermatophytes, often through induction of oxidative stress and disruption of cell-wall integrity. Antiviral activity has been demonstrated against enveloped viruses in cell culture at relatively high concentrations.

Despite this impressive preclinical profile, clinical data on curcumin in infectious dermatoses are sparse. Some early studies and case reports describe use of turmeric pastes or curcumin-containing creams in superficial fungal infections such as tinea corporis or pityriasis versicolor with anecdotal improvement, but these reports lack controls and microbiological confirmation

[6, 7]. Curcumin's antimicrobial properties may nevertheless contribute indirectly to observed benefits in conditions such as acne vulgaris and infected chronic wounds, where reduction of bacterial load is relevant. In the absence of robust randomised controlled trials, curcumin should not be relied upon as sole therapy for infectious dermatoses, but it may be a useful adjunct to conventional antimicrobials within carefully monitored research settings.

Other and emerging indications

A range of other dermatological conditions have been explored for curcumin therapy, often at an early or exploratory stage. In chronic urticaria, case series and small uncontrolled studies have reported reductions in wheal frequency and antihistamine requirements with oral turmeric supplements, hypothesised to reflect mast cell stabilising and anti-inflammatory effects, but randomised controlled trials are lacking [6, 7]. In alopecia areata, topical formulations containing curcumin in combination with capsaicin and other actives have been evaluated, with some suggestion of improved hair regrowth, though attribution of benefit to curcumin is impossible. Curcumin has also been incorporated into multi-ingredient oral supplements marketed for "hair, skin and nail" health, but these combinations have not been rigorously evaluated in controlled dermatologic trials [6, 7, 18].

Patients with hidradenitis suppurativa frequently self-experiment with turmeric or curcumin supplements based on anecdotal reports of reduced pain and drainage. Survey-based studies indicate that a subset of patients perceive benefit, but these observations are highly subject to bias and placebo effects, and no randomised controlled trials of curcumin in hidradenitis suppurativa have been published to date [7, 8]. Other emerging areas include pruritic dermatoses such as lichen simplex chronicus and neuropathic itch, where anti-pruritic effects observed in sulfur mustard-induced
pruritus and uraemic pruritus suggest potential applicability. At present, however, data are too limited to support specific recommendations.

Safety, tolerability and interactions

Safety considerations for curcumin in dermatology encompass both topical and systemic use. In general, curcumin has been regarded as safe and well tolerated in numerous clinical trials across diverse indications, with doses up to several grams per day used for months in some studies [3, 4, 18]. Topical curcumin preparations can cause irritant or allergic contact dermatitis, contact urticaria and, more commonly, transient yellow staining of the skin and clothing. Patch-test series have identified turmeric and curcumin as occasional allergens, particularly in occupational settings such as food industry workers, though prevalence appears low. In cosmetic or therapeutic use, most reports of topical reactions involve erythema, pruritus

or burning at the application site, which generally resolve upon discontinuation [5-7].

Systemic adverse effects with oral curcumin are most frequently gastrointestinal, including nausea, dyspepsia, diarrhoea and abdominal discomfort, which are usually mild and self-limiting. Meta-analyses of curcumin supplementation in non-dermatologic conditions have generally found no significant increase in serious adverse events compared with placebo, although reporting of adverse events is variable across trials [3, 18]. Laboratory abnormalities, when reported, have included minor elevations in liver enzymes and alkaline phosphatase, which usually resolve without sequelae.

More concerning are emerging case reports and pharmacovigilance signals of hepatotoxicity associated with high-dose curcumin supplements, sometimes in combination with bioavailability enhancers such as piperine. Case reports and case series describe cholestatic or mixed-pattern liver injury temporally related to curcumin use, with resolution after discontinuation and, in some instances, recurrence upon rechallenge [16, 17]. A case series from a large liver-injury network and regulatory safety reviews suggest that hepatotoxicity is rare relative to widespread use but potentially severe, particularly with enhanced-absorption formulations. The mechanisms remain incompletely understood but may involve idiosyncratic drug-induced liver injury or interactions with concomitant medications [17].

Curcumin has multiple potential pharmacokinetic and pharmacodynamic interactions. It can modulate cytochrome P450 enzymes and transporters in vitro, raising theoretical concerns about interactions with anticoagulants, antiplatelet agents, hypoglycaemic therapies and certain chemotherapeutic or immunosuppressive drugs [3, 9, 10]. In dermatology, this is particularly relevant for patients receiving systemic methotrexate, ciclosporin, azathioprine or biologic agents, where robust clinical interaction data are lacking. Special populations warrant particular attention. In children, data on curcumin use beyond dietary turmeric intake are sparse; most paediatric experience comes from
short-term trials in non-dermatologic conditions. In pregnancy and lactation, evidence is insufficient to support high-dose curcumin supplementation, and animal studies raise concerns about uterotonic effects at very high doses, so curcumin supplements are generally not recommended beyond culinary use [3, 18]. In patients with chronic liver or kidney disease, or those on multiple medications, careful assessment of potential interactions and monitoring of liver function tests may be prudent if curcumin is used.

From a dermatologic safety standpoint, it is important to note that curcumin can alter the appearance of skin lesions through staining, which may complicate clinical assessment or dermoscopic evaluation. Additionally, photosensitising properties exploited in photodynamic protocols could theoretically increase sensitivity to light under some conditions, although in practice most studies have not reported significant phototoxic reactions outside the controlled treatment setting [14, 15].

Methodological quality and limitations of current evidence

The overall quality of evidence for curcumin in dermatology is constrained by several recurring methodological issues. Most clinical studies are small, single-centre trials or pilot studies with sample sizes typically ranging from 20 to 100 participants. Such studies are underpowered to detect modest but clinically meaningful effects, prone to type I and type II errors and vulnerable to imbalances in baseline characteristics. Many lack explicit sample-size calculations or pre-specified primary endpoints [6-8].

There is substantial heterogeneity in curcumin formulations, doses and routes of administration across studies. Trials variously use crude turmeric powder, standardised curcumin extracts, phytosomal complexes, nanoencapsulated curcumin, microemulgels and multi-ingredient herbal mixtures that include curcumin among other actives, with dosing regimens ranging from a few hundred milligrams to several grams per day orally and from once to multiple daily applications topically [7-10]. This heterogeneity makes it difficult to compare results across studies or to derive generalisable dosing recommendations and raises questions about which component is responsible for observed effects in multi-ingredient formulations.

Outcome measures and follow-up durations are often suboptimal. Many studies rely on investigator-assessed ordinal scales or non-validated composite scores rather than standardised measures such as Psoriasis Area and Severity Index, Scoring Atopic Dermatitis, Eczema Area and Severity Index or validated pruritus scales. Follow-up periods are usually short, commonly 4 to 12 weeks, which may be insufficient to capture full therapeutic effects, relapse rates or delayed adverse events, especially in chronic diseases such as psoriasis and vitiligo [7, 8]. Blinding and allocation concealment are frequently inadequate or poorly described, particularly in trials using conspicuously yellow preparations that may unblind participants and investigators.

Publication bias and selective reporting likely contribute to an overly optimistic impression of benefit. Positive small studies are more likely to be published than negative or inconclusive ones, and some trials report improvement without detailing absolute effect sizes, variability or statistical analyses [7, 8, 18]. Standardisation of curcumin content and quality control is often lacking. Herbal products may vary widely in curcumin content, purity, presence of other curcuminoids and contaminants such as heavy metals or synthetic dyes, and clinical studies rarely include independent verification of the curcumin content and composition of the interventions used [6-8]. Finally, very few studies directly compare curcumin-based interventions with established standard-of-care treatments in a head-to-head fashion, making it difficult to position curcumin relative to existing options in terms of efficacy, safety, cost effectiveness and patient preference.

Future directions and research priorities

To clarify the therapeutic potential of curcumin in dermatology and move beyond preliminary enthusiasm, several concrete research priorities can be identified. First, there is a need for well-

designed, adequately powered randomised controlled trials of standardised curcumin formulations in specific skin diseases, particularly psoriasis, atopic dermatitis, acne vulgaris, vitiligo and chronic wounds. These trials should incorporate rigorous methodology, including proper randomisation and allocation concealment, double blinding where feasible, validated disease-specific outcome measures and clinically meaningful primary endpoints such as Psoriasis Area and Severity Index 75 response, Scoring Atopic Dermatitis change, lesion-count reductions, repigmentation percentages or wound-area reduction [7-10].

Second, formulation science must be more tightly integrated into clinical research. Trials should use well-characterised, standardised curcumin products with documented curcumin content, excipients and bioavailability data. Comparative studies of different formulations, such as phytosomal versus conventional curcumin or nanoencapsulated versus simple topical preparations, would help determine whether complex delivery systems translate into clinically relevant advantages [9-12]. Pharmacokinetic and pharmacodynamic studies focused on skin, including measurement of curcumin and metabolite levels in lesional and perilesional tissue, would provide valuable mechanistic insights and inform dose selection.

Third, longer-term safety and effectiveness need to be assessed. Most existing trials are short term. Prospective cohort studies or extension arms of randomised trials should evaluate the sustainability of benefits, relapse rates after discontinuation and potential cumulative toxicities, particularly hepatic, renal and haematologic, over six to twelve months or longer [3, 4, 16-18]. Registries capturing real-world use of curcumin in
dermatology, ideally with standardised adverse-event reporting, could complement trial data.

Fourth, there is scope for exploring rational combination therapies. Curcumin could be combined with topical corticosteroids, vitamin D analogues, calcineurin inhibitors, retinoids or biologic agents in psoriasis and atopic dermatitis to assess whether it can reduce required doses or mitigate adverse effects, leveraging its anti-inflammatory and antioxidant properties [5-7, 9, 10]. In acne, integration of curcumin-based photodynamic therapy with topical retinoids and benzoyl peroxide might offer synergistic benefits [14, 15]. In vitiligo, combining turmeric-based creams with narrowband ultraviolet B or excimer-laser therapy could be further evaluated. Such combination studies must be designed to disentangle the specific contribution of curcumin and to monitor for interactions.

Finally, translational research should continue to elucidate curcumin's effects on specific cellular and molecular pathways in human skin, including its impact on resident memory T cells, innate lymphoid cells, skin microbiota and epithelial barrier function. Integration of omics technologies, such as transcriptomics and metabolomics, in clinical studies could reveal biomarkers of response and identify patient subgroups more likely to benefit, paving the way for more personalised use of curcumin-based interventions [9, 10].

Conclusion

Curcumin occupies an intriguing position at the interface of traditional medicine, nutraceuticals and modern dermatology. Extensive mechanistic and preclinical data indicate that it targets multiple pathways central to skin inflammation, oxidative stress, matrix remodelling and melanogenesis. Over the past two decades, a growing number of clinical studies have explored its oral and topical use in diverse dermatologic conditions, with the strongest, although still limited, evidence emerging for psoriasis, chronic pruritic disorders, selected aspects of vitiligo and wound healing [5-10]. For these indications, curcumin appears to offer modest adjunctive benefits when used alongside standard therapies, with a generally favourable short-term safety profile.

However, the current evidence base is constrained by small, heterogeneous and often methodologically suboptimal trials, variability in formulations and dosing and incomplete safety and interaction data. Claims regarding curcumin as a broad-spectrum or stand-alone treatment for skin disease are not supported by high-level evidence. In mainstream dermatologic practice, curcumin should therefore be viewed as a potential adjunctive or complementary agent rather than a replacement for established therapies, and its use should be individualised, carefully monitored and grounded in transparent discussion with patients about the limits of current knowledge. The realistic therapeutic potential of curcumin in dermatology will depend on the execution of rigorous, disease-
specific clinical trials using standardised, bioavailable formulations, accompanied by detailed safety monitoring and mechanistic correlative studies. If such research confirms meaningful benefits with acceptable risks, curcumin may find a defined niche as an adjuvant in the management of chronic inflammatory dermatoses, pigmentary disorders and wound healing, as well as a scientifically grounded ingredient in selected cosmeceutical products.

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