Tea Tree and Neem in Modern Topical Therapy: A Review

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Abstract*—Infectious skin and soft tissue disorders represent a major global health burden, increasingly complicated by methicillin-resistant Staphylococcus aureus (MRSA) and terbinafine-resistant Trichophyton indotineae. This review synthesizes the phytochemistry, antimicrobial spectrum, topical delivery innovations, clinical evidence, and translational hurdles of Melaleuca alternifolia (tea tree oil, TTO) and Azadirachta indica (neem) in dermatology. In contrast, A. indica deploys non-volatile tetranortriterpenoids (such as azadirachtin, nimbin, and nimbidin), flavonoids, and phenolics that disrupt microbial adhesion and biofilm persistence while modulating host inflammatory cascades. In vitro, both botanicals exhibit broad-spectrum activity against MRSA, dermatophytes, and Candida albicans, with TTO additionally inhibiting Cutibacterium acnes and Malassezia spp., and neem demonstrating activity against ectoparasites including Sarcoptes scabiei and Pediculus humanus capitis. Combining TTO with neem extract further enhances C. albicans growth suppression. To overcome volatility, poor aqueous solubility, and concentration-dependent contact dermatitis (particularly at TTO concentrations of 25% or higher), advanced delivery platforms—such as ethosome-based creams, nanostructured lipid carriers, nanocapsule hydrogels, and biopolymer dressings—improve dermal permeation and local tolerability. Clinically, topical TTO performs comparably to standard regimens for MRSA body decolonization and achieves a 64% mycological cure rate in interdigital tinea pedis at 50% concentration (versus 31% for placebo, with occasional moderate-to-severe dermatitis), whereas 10% neem leaf extract lotion matches 5% permethrin in scabies efficacy and safety. Broader clinical adoption depends on establishing chemical standardization for neem extracts, evaluating activity against T. indotineae, and conducting large-scale, methodologically rigorous randomized controlled trials.

***Index Terms*—Melaleuca alternifolia, Azadirachta indica, skin infections, antimicrobial resistance, topical nanocarriers**

I. Introduction

Infections of the skin and subcutaneous tissues place a heavy and growing burden on healthcare systems worldwide. Global Burden of Disease (GBD) analyses repeatedly place fungal skin diseases among the most common human infections. By 2017, dermatomycoses accounted for 10.09% of all skin disorders, building on 2016 estimates of 2.1 billion cases, which made them the fourth most frequent of the 328 diseases and injuries tracked. Superficial fungal infections (SFIs) and dermatophytoses together affect an estimated 20%–25% of the world's population [1].

In the United States, surveillance by the Centers for Disease Control and Prevention (CDC) linked dermatophyte infections to 4,981,444 outpatient visits between 2005 and 2014, and to roughly USD 845 million in direct medical costs in 2019 [2].

On the bacterial side, skin and soft tissue infections (SSTIs) are driven mainly by *Staphylococcus aureus* and beta-hemolytic streptococci, and they are often made worse by polymicrobial interaction. Pooled data

suggest that in chronic conditions such as diabetic foot ulcers, pressure ulcers, and burn wounds, up to 70% of *S. aureus* SSTIs involve co-pathogens, including *Pseudomonas aeruginosa*, *Escherichia coli*, *Enterococcus* spp., *Acinetobacter baumannii*, and *Candida albicans*. These partnerships raise antibiotic tolerance and worsen clinical outcomes. In tropical regions where scabies is endemic, the infestation damages the skin barrier and makes secondary impetigo and severe invasive SSTIs more likely [3].

At the same time, first-line antimicrobials are becoming less dependable. More than 95% of *S. aureus* isolates are now resistant to penicillin, and multidrug-resistant MRSA is classed as a serious threat and a priority pathogen by both the CDC and the World Health Organization (WHO). In medical mycology, the worldwide spread of *Trichophyton indotineae* (formerly *T. mentagrophytes* internal transcribed spacer [ITS] genotype VIII) has changed how dermatophytosis is managed. The species emerged in South Asia, where it made up 78%–94% of dermatophyte isolates in Indian multicenter surveillance (2017–2019) and as much as 96.2% in Bangladesh. It has since reached Asia, the Middle East, Europe, Africa, and the Americas [4].

Unlike classic, localized *T. rubrum* infection, *T. indotineae* often causes extensive, inflamed, multifocal disease (36.9% of 974 reported cases), with prolonged and treatment-resistant courses. In vitro terbinafine resistance has been recorded in up to 76% of Indian *T. indotineae* isolates and 57.1% of tested *T. rubrum* isolates. Resistance was also found in 39% of Iraqi *T. indotineae* isolates (2019 Baghdad survey), and Iranian cohorts showed elevated minimum inhibitory concentrations (MICs) of 32 mg/L or higher. These pressures have renewed scientific interest in botanical anti-infectives that act on several targets at once. *Melaleuca alternifolia* Cheel (Myrtaceae), traditionally used by the Bundjalung First Nations people of Australia for wounds, burns, and insect bites, and *Azadirachta indica* A. Juss (Meliaceae), a cornerstone remedy across the Indian subcontinent and Southeast Asia, offer complementary chemistry. The first is rich in hydrophobic terpenes and the second in triterpenoids, and both can act on microbial membranes, biofilms, and skin inflammation.

II. Phytochemistry and Mechanisms of Action

The pharmacological difference between *M. alternifolia* and *A. indica* comes from the very different classes of secondary metabolites they contain.

Tea tree oil (TTO) is a steam-distilled essential oil made up of volatile monoterpenes, sesquiterpenes, and terpene alcohols. The international standard ISO 4730 sets compositional limits for 14 components to safeguard antimicrobial quality. Terpinen-4-ol is the main active constituent (ISO 4730 minimum ≥30%; typical 40.1%). It is accompanied by γ-terpinene (10%–28%; typical 23.0%), α-terpinene (5%–13%; typical 10.4%), 1,8-cineole (≤15%; typical 5.1%), terpinolene (1.5%–5%; typical 3.1%), ρ-cymene (0.5%–12%; typical 2.9%), α-pinene (1%–6%; typical 2.6%), and α-terpineol (1.5%–8%; typical 2.4%), along with minor sesquiterpenes such as aromadendrene and δ-cadinene [5,6].

Because these lipophilic hydrocarbons move easily into microbial membranes, TTO damages membrane structure and upsets cellular homeostasis. In *E. coli*, it disturbs potassium balance, blocks glucose-dependent

respiration, alters cell shape, allows propidium iodide to enter, and causes modest leakage of material absorbing at 280 nm. *S. aureus*, by contrast, loses viability without the whole cell lysing. In *C. albicans*, 0.25% (v/v) TTO raises membrane fluidity and permeability within 30 minutes, as shown by uptake of propidium iodide and methylene blue and by leakage of 260-nm-absorbing material at 6 hours. It also reduces respiration by 40% at 0.25% and by 95% at 1.0%, and it prevents glucose-induced acidification of the medium, which points to impaired plasma membrane ATPase. At 0.125%–0.25%, it reversibly suppresses germ-tube formation. TTO vapors also inhibit fungal growth and sporulation, and germinated conidia are considerably more susceptible than ungerminated ones. In human clinical testing, TTO has additionally been shown to reduce histamine-induced skin inflammation [7,8].

The activity of *A. indica*, in contrast, centers on non-volatile tetranortriterpenoids (limonoids), including azadirachtin, nimbin, nimbidin, nimbidol, nimbolide, and gedunin. These are joined by flavonoids (such as quercetin), phenolics, tannins, saponins, and alkaloids found in the seeds, leaves, and bark. The polarity of the extraction solvent strongly influences antimicrobial potency: ethanolic, methanolic, and ethyl acetate extracts consistently outperform purely aqueous ones in antibacterial and antidermatophytic assays. Neem constituents interfere with microbial colonization and with host inflammatory signaling through several pathways, summarized below.

Table 1. Comparison of the two botanicals [9,10]

Feature	***Melaleuca alternifolia* (tea tree oil)**	***Azadirachta indica* (neem)**
Plant source and extraction	Steam-distilled essential oil from leaves	Seed oil; aqueous, ethanolic, methanolic, or ethyl acetate extracts of leaves, seeds, and bark
Principal bioactives	Terpinen-4-ol (≥30%, typical 40.1%), γ-terpinene (23.0%), α-terpinene (10.4%), 1,8-cineole (5.1%)	Limonoids/triterpenoids (azadirachtin, nimbin, nimbidin, nimbidol, nimbolide, gedunin), quercetin, tannins, saponins
Direct antimicrobial mechanisms	Partitioning into the lipid bilayer, K^+ leakage, respiratory arrest, plasma membrane ATPase inhibition, germ-tube blockade	Bactericidal action (MBC/MIC <4 for leaf extracts); reduced cell-surface hydrophobicity and adhesion in *C. albicans*
Antibiofilm activity and host modulation	Reduces histamine-induced skin inflammation; antibiofilm activity	Suppresses *S. aureus*/MRSA biofilm adherence and eradicates mature biofilms; purified nimbidin inhibits macrophage migration, iNOS induction and NO release, prostaglandins, and neutrophil degranulation
Chemical standardization	Internationally standardized under ISO 4730	No harmonized topical pharmacopeial standard; large variation between batches, solvents, and geographic sources

One mechanistic caveat from recent molecular studies deserves emphasis: most of the antimicrobial and wound-healing effects credited to neem come from crude extract studies, not from assays of purified constituents. Where purified compounds have been tested, nimbidin directly inhibits macrophage chemotaxis. It lowers nitric oxide (NO) and prostaglandin release by suppressing the induction of inducible nitric oxide synthase (iNOS), without inhibiting the enzyme's catalytic activity directly. It also dampens degranulation of stimulated human neutrophils, reducing the release of β-glucuronidase, myeloperoxidase, and lysozyme. Separately, an aqueous extract of *A. indica* stem bark raised tissue hydroxyproline, DNA, total protein, and local nitrite levels in experimental excision wounds, which supports maturation of granulation tissue[11] .

Notably, combining TTO with neem extract suppressed *C. albicans* growth significantly more than either agent alone in intraoral testing. This suggests that terpene-driven membrane permeabilization and triterpenoid-driven anti-adhesion effects may complement each other. However, formal studies of cutaneous synergy against MRSA and dermatophytes remain limited.

III. Antimicrobial Spectrum Against Skin Pathogens

Both botanicals show broad in vitro activity against Gram-positive and Gram-negative skin bacteria, dermatophytes, yeasts, and ectoparasites. Comparing the numbers directly requires care, because TTO potency is usually expressed as % (v/v), whereas *A. indica* extracts and isolated limonoids are reported as mass per volume (µg/mL or mg/mL) or as mass per well or disc (µg), depending on the assay design [12].

Table 2. In vitro susceptibility of skin pathogens

Pathogen group / species	***M. alternifolia* (TTO)**	***A. indica* (neem)**
MRSA / *S. aureus*	MIC 0.04%–0.35% (v/v); MBC 0.5% (v/v); slower time-kill than most bacteria	Leaf ethyl acetate extract: 16 µg (*S. aureus*), 16–32 µg (MRSA); ethanolic: 32 µg (MBC/MIC <4). Leaf ethanolic extract inhibits biofilm adherence at 62.5 µg/mL (*S. aureus*) and 125 µg/mL (MRSA); ripe seed extract: *S. aureus* MBIC 100 µg/mL, MBEC 300 µg/mL. Bark ethanol MIC/MBC 12.5 mg/mL [13]
S. epidermidis	No specific % (v/v) MIC reported in the reviewed studies	Isolated seed limonoids: MIC 32–128 µg/mL
Trichophyton rubrum	MIC 0.03%–0.6% (v/v); MBC/MFC 0.25%–1% (v/v); active against nail isolates	Active in leaf and seed extracts; seed extracts show lower MICs than leaf extracts [14]

T. mentagrophytes	MIC 0.11%–0.44% (v/v); MBC/MFC 0.25%–0.5% (v/v)	Active in ethanolic and oil/extract assays
T. tonsurans	MIC 0.004%–0.016% (v/v); MBC/MFC 0.12%–0.5% (v/v)	Active across *Trichophyton* isolates (ethanolic > aqueous across 88 clinical isolates) [15]
Candida albicans	Quantitative MIC/MFC values not available in the reviewed studies; 0.25% permeabilizes membranes and blocks 40% of respiration (95% at 1.0%); 0.125%–0.25% blocks germ tubes	Leaf extracts: 64 µg; bark ethanol MIC/MFC 12.5 mg/mL; inhibits adhesion and biofilm; suppression enhanced when combined with TTO [16]
Malassezia spp.	*M. furfur*: MIC 0.03%–0.12%, MBC/MFC 0.5%–1.0%; *M. sympodialis*: MIC 0.016%–0.12%, MBC/MFC 0.06%–0.12%	No quantitative MIC values reported in the reviewed studies
Ectoparasites (*Sarcoptes*, *Pediculus*, *Demodex*)	Evaluated in *Demodex* eyelid wipes (clinical evidence inconclusive); no quantitative *Sarcoptes* MICs available	Active against *S. scabiei* var. *cuniculi* larvae and *P. humanus capitis*; 10% leaf lotion clinically effective in human scabies [17]

- **RSA kills slowly.** Although 0.5% (v/v) TTO eradicates MRSA, time-kill studies show it is killed more slowly than most Gram-positive and Gram-negative clinical isolates, which are generally eliminated in under 60 minutes. Selectively bred chemotypes such as Clone 88 kill faster than standard TTO.
- **Neem potency varies widely.** It changes sharply with plant organ, extraction solvent, and geographic origin. Seed extracts show lower MICs than leaf extracts against dermatophytes. Leaf ethyl acetate and ethanol fractions inhibit MRSA at microgram levels, whereas crude bark ethanol requires milligram-scale concentrations (12.5 mg/mL). Identically extracted leaves from two different sites even produced different MRSA susceptibility profiles.
- **Neem acts against biofilms.** Its antibiofilm activity against *S. aureus* and MRSA (adherence inhibition at 62.5–125 µg/mL; MBEC of 300 µg/mL for ripe seed extract) addresses the biofilm tolerance seen in chronic SSTIs [18,19] .

IV. Topical Delivery Systems and Formulations

Conventional topical vehicles, such as 5% TTO gels, 10% TTO creams, 25%–50% TTO solutions, and 10% neem leaf lotions, face built-in physicochemical limits. Unencapsulated TTO evaporates quickly from the skin surface, dissolves poorly in water, penetrates intact stratum corneum unevenly, and can cause local irritation or allergic dermatitis at high concentrations. Crude *A. indica* oil and organic extracts have similar problems: they are hydrophobic, chemically unstable, strongly scented, and released unpredictably from simple ointment bases [20].

Nanocarrier engineering can overcome these limits by shielding volatile terpenes and hydrophobic limonoids, improving their deposition in the skin, and controlling their release.

Table 3. Delivery platforms

Delivery platform	Active tested	Formulation and physicochemical/biological metrics	Translational profile and evidence gaps
Ethosome-based cream	TTO (terpinen-4-ol)	Sonication plus phase inversion; encapsulation efficiency (EE) 76.19 ± 3.26%, particle size 333.6 nm, zeta potential −35.3 mV	Better ex vivo permeation and retention; non-cytotoxic to HaCaT keratinocytes; reduced clinical severity, white blood cells, eosinophils, and IgE in vivo [21]
Nanostructured lipid carriers (NLCs)	TTO (with hesperidin)	TTO-loaded hesperidin NLCs tested for cytotoxicity, skin irritancy, and in vivo wound healing	Lipid-core stabilization with dual anti-inflammatory and wound-healing support; quantitative EE% and human RCTs unavailable [22]
Nanocapsule and nanoemulsion hydrogels	TTO	Polymeric nanocapsules and nanoemulsions incorporated into hydrogel bases	Anti-edema effect, faster wound healing, and bacterial growth inhibition, with no irritation on hairless animal skin [23]
Polymeric hydrogels and nanofibers	Essential oils; neem biomaterials	Biopolymer hydrogels and electrospun chitosan/cellulose acetate nanofibers (documented for essential oils such as eucalyptus oil in *S. aureus* wounds); neem	Prolonged release and exudate absorption; quantitative EE%, permeation, and human data for

		biomaterial platforms are still conceptual	TTO or neem liposomes, SLNs, and nanofibers remain scarce [24]

Among the fully characterized systems, ethosome-based TTO creams use ethanol-rich phospholipid vesicles to fluidize stratum corneum lipids. They reach an encapsulation efficiency of 76.19 ± 3.26%, a particle diameter of 333.6 nm, and a zeta potential of −35.3 mV, which prevents aggregation. They also increase dermal retention ex vivo without harming HaCaT keratinocytes. Placing TTO nanocapsules or nanoemulsions inside hydrogels further limits burst evaporation, reduces edema, and supports non-irritating wound repair in animal models [25].

Neem oil nanoemulsions have been developed for larvicidal vector control, and biomaterial dressings (hydrogels, films, and nanofibers) are recognized as promising platforms for neem wound therapy. Even so, the dermatological studies reviewed here contain no quantitative data on encapsulation efficiency, skin permeation, or clinical efficacy for neem nanoformulations, nor for TTO liposomes, solid lipid nanoparticles (SLNs), or electrospun nanofibers.

V. Clinical Efficacy and Therapeutic Evidence

Human clinical evidence is unevenly distributed. TTO has been tested in 46 RCTs across dentistry (n = 18), dermatology (n = 9), infectious disease (n = 9), ophthalmology (n = 6), podiatry (n = 3), and other fields (n = 1), although overall trial quality was rated poor to modest. *A. indica*, on the other hand, has strong controlled clinical data mainly in scabies, while its evidence in wound healing is largely preclinical [26].

Table 4. Clinical and preclinical evidence

Indication	Botanical intervention	Comparator / study design	Key efficacy and safety findings
Acne vulgaris	5% TTO (8 weeks); TTO RCTs	Placebo / standard comparators (RCTs)	5% TTO reduced inflammatory acne at 8 weeks; a 2023 systematic review concluded that more high-quality trials are needed to confirm benefits for lesions and skin inflammation. No neem acne RCTs were identified [27]
Interdigital tinea pedis	10% w/w TTO cream	1% tolnaftate cream vs. placebo (double-blind RCT)	Relieved symptoms about as well as 1% tolnaftate, but was no better than placebo for mycological cure [25]

Interdigital tinea pedis	25% and 50% TTO solution	Placebo (randomized, blinded RCT)	64% mycological cure with 50% TTO vs. 31% with placebo; 3.8% (4 patients) developed moderate-to-severe dermatitis, which resolved after treatment stopped [24]
MRSA decolonization	Topical TTO regimens	Standard decolonization regimens (RCT review)	Body decolonization efficacy similar to standard care; intranasal TTO caused mucosal irritation [23]
Scabies	Neem + turmeric paste (pilot); 10% neem leaf extract lotion	5% permethrin and 10% brotowali extract (2025 RCT)	97% cure in 3–15 days in a 1992 pilot; in the 2025 comparative trial, 10% neem leaf lotion was as effective and safe as 5% permethrin and improved quality of life [25]
Wounds (preclinical models)	TTO nanocapsule/nano emulsion hydrogels; *A. indica* aqueous bark extract	Vehicle and reference drug in animal excision/incision models	TTO hydrogels improved healing and reduced edema; aqueous neem bark extract increased wound contraction from day 10 onward, hydroxyproline (13.31 vs. 7.76 mg/g), DNA, and breaking strength (289.40 vs. 122.20 g; reference 258.60 g) [28]

Acne vulgaris. An 8-week trial summarized in herbal monograph databases reported reduced inflammatory acne with 5% TTO, which fits with its in vitro activity against *C. acnes* and its antibiofilm effects. However, the systematic review by Kairey et al. (2023) concluded that existing trials are not methodologically rigorous enough to confirm benefits for acne lesions or local inflammation. The reviewed literature identified no controlled clinical trials of topical neem for acne.

Superficial fungal infections. TTO has a narrow therapeutic window here. A 1992 double-blind trial found that a 10% (w/w) TTO cream matched 1% tolnaftate in relieving the symptoms of tinea pedis but did not beat placebo in achieving mycological cure. Raising the strength to 25% and 50% TTO solutions in a 2002 randomized, placebo-controlled trial lifted the mycological cure rate to 64% (with 50% TTO) against 31% for placebo, but 3.8% of TTO-treated participants developed moderate-to-severe dermatitis. For comparison, standard 1% topical terbinafine clears interdigital tinea pedis within 1 week. The retrieved evidence contains no controlled human RCTs of TTO or neem for onychomycosis or *T. indotineae* dermatophytosis [29].

Bacterial decolonization and wound care. Topical TTO regimens for the body achieve MRSA decolonization rates comparable to standard hospital protocols, whereas intranasal application irritates the mucosa. In rat wound models, an aqueous *A. indica* stem bark extract significantly accelerated contraction from day 10 onward. It also raised tissue hydroxyproline (13.31 ± 6.65 vs. 7.76 ± 3.88 mg/g in vehicle controls, $p < 0.05$; reference 13.81 ± 6.90 mg/g), DNA content (20.99 ± 0.68 µg/100 mg, $p < 0.01$), total protein (100.53 ± 7.88 vs. 68.59 ± 6.19 mg/g, $p < 0.05$), and nitrite levels (3.05 ± 0.03 vs. 1.41 ± 0.08 mMol/g, $p < 0.01$). In incised wounds, breaking strength rose to 289.40 ± 29.45 g versus 122.20 ± 16.51 g for vehicle ($p < 0.05$; reference 258.60 ± 20.96 g). By contrast, clinical studies that used uncharacterized cold-pressed neem oil in chronic wounds cannot establish which compound is responsible, nor definitive clinical efficacy [30].

Scabies. This is where *A. indica* has its strongest clinical evidence. A 1992 pilot trial of a neem and turmeric combination achieved a 97% cure rate within 3–15 days without adverse reactions.

VI. Safety, Standardization, and Regulatory Aspects

Adverse events occurred in 60% of the TTO RCTs reviewed by Kairey et al. (2023). They were mostly minor, except at topical concentrations of 25% or higher, where irritant and allergic contact dermatitis become clinically important. The earlier belief that 1,8-cineole causes TTO's skin irritation has been refuted by controlled human data showing that 1,8-cineole is not an irritant. Application to mucosal surfaces consistently carries a risk of irritation.

For *A. indica*, a topical 10% leaf extract lotion and a neem-turmeric paste showed favorable skin safety in scabies studies. Solvent choice remains critical, however: in experimental wounds, ethanol and ethanol-water bark extracts produced poorer collagen deposition and epidermal structure than aqueous bark extract [31].

Standardization marks the sharpest regulatory difference between the two botanicals. *M. alternifolia* benefits from the compositional tolerances of ISO 4730 and from clonal selection (for example, Clone 88) to optimize terpinen-4-ol activity while limiting irritation. *A. indica*, by contrast, has no equivalent harmonized topical pharmacopeial standard in the reviewed literature, and its antimicrobial potency and limonoid profile vary considerably with plant tissue, solvent, and harvest location [32].

To bring these botanicals safely into clinical care alongside WHO AWaRe antibiotic stewardship and susceptibility-guided management of *T. indotineae*, validated marker quantification and clear regulatory boundaries are needed. Without them, unstandardized botanicals could delay definitive antimicrobial therapy in severe or necrotizing SSTIs.

VII. Future Perspectives and Conclusion

Moving *M. alternifolia* and *A. indica* from empirical adjuncts to standardized dermatological therapies will require four research priorities.

- **Susceptibility profiling in the era of resistance.** Standardized TTO chemotypes, purified neem limonoids, and TTO–neem combinations should be tested quantitatively against molecularly confirmed *T. indotineae* isolates and polymicrobial MRSA biofilms.
- **Chemical standardization of neem.** Validated chromatographic specifications that quantify key triterpenoids (such as nimbidin and azadirachtin), together with limits for residual solvents, are essential to overcome the heterogeneity of current extracts.
- **Dose-sparing nanocarriers.** Co-encapsulating TTO and neem bioactives in ethosomes, NLCs, or hydrogel/nanofiber dressings offers a practical way to improve follicular delivery while keeping TTO concentrations well below the ≥25% threshold for dermatitis.
- **Methodologically rigorous RCTs.** Future human trials should use mycological or molecular cure endpoints (culture or PCR), active standard-of-care comparators (for example, 1% terbinafine or mupirocin), and standardized reporting of adverse events [33].

In conclusion, *M. alternifolia* and *A. indica* offer complementary membrane-disrupting, antibiofilm, and anti-inflammatory actions against the major skin pathogens. Current clinical evidence supports 10% neem leaf extract lotion as a safe and effective alternative to 5% permethrin for scabies. It also supports topical TTO as an adjunct for MRSA body decolonization and for mild inflammatory acne, whereas conventional TTO preparations perform worse than standard allylamines in dermatophytosis. Pairing standardized phytochemicals with lipid-based and polymeric nanocarriers is the most credible route to clinically reliable, resistance-resilient topical botanicals .

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