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.iss3.2026.852.870

NanoCurcumin, *Leucas aspera,* and *Boswellia serrata* in Leucas NC Cream: A Critical Review of Convergent Wound-Repair Pharmacology and Translational Potential

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Published on:

20.09.2026

Published by:

Futuristic Publications

Abstract: Cutaneous wound repair depends on coordinated control of inflammation, redox signalling, microbial pressure, fibroblast function, extracellular matrix reconstruction, vascular recovery, and epithelial closure. In diabetic and other difficult-to-heal wounds, several of these processes can fail simultaneously, creating a rationale for interventions that address more than one biological barrier. Leucas NC Cream combines NanoCurcumin Oil, NanoCurcumin Extract, *Leucas aspera* extract, and an Olibanum component derived from *Boswellia serrata*, three botanical systems with complementary wound-relevant pharmacology. This critical narrative review evaluates the formulation through a claim-rationale-evidence-inference framework, separating direct wound evidence from mechanistic or formulation-level extrapolation. NanoCurcumin has the broadest wound-specific evidence, including studies of fibroblast mobilisation, macrophage-state regulation, protease-responsive delivery, collagen deposition, vascular responses, and re-epithelialisation in acute and diabetic models, with heterogeneous but emerging human evidence. *Leucas aspera* contributes a distinct antimicrobial-redox evidence stream supported by phytochemical studies, inflammatory and antioxidant assays, membrane-disruptive antibacterial experiments, and recent fibroblast-migration data from a *Leucas*-mediated composite. *Boswellia serrata* contributes complementary evidence linking inflammatory regulation with collagen-associated and angiogenic endpoints, including both standardised-extract and topical diabetic-wound studies. Clinical curcumin-boswellic-acid research further shows that the two can be co-administered with good tolerability, supporting pharmacological compatibility. The principal synthesis of this review is that the three ingredients converge on sequential barriers to repair - microbial pressure, redox and inflammatory dysregulation, fibroblast and matrix failure, and inadequate vascular support - giving the formulation a coherent multi-target scientific rationale. Within the literature retrieved, no indexed study has yet evaluated the complete cream; product-level studies of efficacy, antimicrobial performance, safety on wounded tissue, and synergy are therefore the logical next step.

Keywords: nanocurcumin; *Leucas aspera*; *Boswellia serrata*; wound healing; diabetic foot ulcer; topical delivery.

Abbreviations: 5-LOX, 5-lipoxygenase; 12-LOX, 12-lipoxygenase; Ang-1, angiopoietin-1; Arg-1, arginase-1; COX, cyclooxygenase; DFU, diabetic foot ulcer; Dkk-1, Dickkopf-1; DNA, deoxyribonucleic acid; ECM, extracellular matrix; FBN1, fibrillin-1; GC-MS, gas chromatography–mass spectrometry;

GPX4, glutathione peroxidase 4; GRADE, Grading of Recommendations Assessment, Development and Evaluation; HAT, histone acetyltransferase; HDAC2, histone deacetylase 2; HPLC, high-performance liquid chromatography; IL-1β, interleukin-1β; IL-6, interleukin-6; iNOS, inducible nitric oxide synthase; LPS, lipopolysaccharide; MBC, minimum bactericidal concentration; MCP-1, monocyte chemoattractant protein-1; MIC, minimum inhibitory concentration; MMP-9, matrix metalloproteinase-9; NF-κB, nuclear factor kappa B; NLC, nanostructured lipid carrier; NRF2, nuclear factor erythroid 2-related factor 2; PRISMA, Preferred Reporting Items for Systematic Reviews and Meta-Analyses; RCT, randomised controlled trial; ROS, reactive oxygen species; STAT3, signal transducer and activator of transcription 3; TGF-β1, transforming growth factor-β1; Tie2, TEK receptor tyrosine kinase; TNF-α, tumour necrosis factor-α; TREM-1, triggering receptor expressed on myeloid cells 1; VEGF, vascular endothelial growth factor; ZnO, zinc oxide.

1. Introduction

Skin repair is a coordinated multicellular programme in which inflammatory cells, keratinocytes, fibroblasts, endothelial cells and extracellular-matrix components act in overlapping phases rather than as isolated sequential events[1]. In diabetes, neuropathy, vascular disease and metabolic dysfunction increase the likelihood that this programme will stall, contributing to recurrent and clinically consequential foot ulceration[2,3].

A multi-target topical strategy is biologically reasonable because chronic wounds rarely reflect a single abnormality. Microbial communities can alter host responses, organise into biofilms, and complicate antimicrobial control[4]. Reactive oxygen species (ROS) are also context-dependent: physiological redox signals participate in host defence and repair, whereas persistent excess promotes tissue injury and chronic-wound stalling[5]. In diabetic tissue, nuclear factor erythroid 2-related factor 2 (NRF2)-dependent antioxidant signalling is linked to keratinocyte behaviour, apoptosis, transforming growth factor-β1 (TGF-β1), and matrix metalloproteinase-9 (MMP-9) regulation[6], and higher wound-fluid MMP-9 has been associated with poorer healing in patients with diabetic ulcers[7].

Macrophage state adds a further regulatory layer. Effective repair requires immune cells to move from early host-defence functions toward phenotypes that support tissue restoration; diabetes can delay this transition and prolong inflammatory signalling[3,8]. The rationale for Leucas NC Cream therefore addresses this multi-barrier biology - redox imbalance, inflammatory persistence, microbial pressure, protease dysregulation, fibroblast impairment, and insufficient vascular support - rather than any single molecular target.

The formulation contains four named actives: NanoCurcumin Oil, NanoCurcumin Extract, *Leucas aspera* extract, and Olibanum oil [*Boswellia serrata* extract]. The central question of this review is whether the ingredient-level literature supports a coherent wound-repair hypothesis for their co-inclusion, and where the evidence stops short of demonstrating finished-product efficacy. Recent reviews have addressed curcumin nanoformulations, *Leucas* pharmacology, and *Boswellia* therapeutics separately, but the targeted search did not identify a recent review integrating these three evidence streams across wound-specific barriers, formulation standardisation, and translational boundaries. This cross-ingredient critical synthesis constitutes the principal novelty of the present review.

2. Literature Identification, Critical Appraisal and Review Novelty

This manuscript was developed as a targeted critical narrative review using literature available through 12 September 2026. PubMed-indexed primary studies and reviews were prioritised, with supplementary verification against publisher records from Nature Portfolio, Springer Nature, Elsevier, Wiley, ACS, Frontiers and MDPI. Search concepts combined ingredient names and formulation terms with wound healing, diabetic wound, diabetic foot ulcer, fibroblast, macrophage, collagen, angiogenesis, oxidative stress, inflammation, antimicrobial activity, biofilm and topical delivery. A supplementary formulation-mechanism search examined cutaneous application, stratum corneum, percutaneous absorption, skin retention, controlled or sustained release, vehicle or excipient effects, keratinocyte migration, macrophage polarisation and epigenetic or chromatin regulation. Combination-oriented searches included curcumin plus *Boswellia*, curcumin plus *Leucas*, *Leucas* plus *Boswellia* and the complete three-botanical system. The search was designed for critical synthesis rather than exhaustive systematic retrieval. Accordingly, a study described as not identified should be understood as absent from the literature retrieved by this targeted search rather than as proof that no such study exists. A final targeted update search was performed in September 2026 to identify newly indexed wound-relevant studies involving nanocurcumin, *Leucas aspera,* and *Boswellia serrata*.

The review is not presented as a systematic review, and no Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) flow diagram is claimed. A 2025 scoping review was used to contextualise the human curcumin literature because that study searched PubMed, Scopus, Web of Science and the Cochrane Library using predefined eligibility criteria[9]. Bibliographic details in the final reference set were cross-checked against PubMed and/or publisher DOI records where available. Evidence was weighted by translational proximity: human wound studies were considered more direct than animal or cellular models; wound-specific evidence was given greater weight than non-wound pharmacology; and related species, different extracts, combination biomaterials or alternative delivery routes were treated as supportive rather than equivalent. Major interpretive statements were then assessed as four linked elements - claim, wound-biological rationale, factual evidence and bounded review-level inference.

3. Phytopharmacological Architecture and Evidence Hierarchy

The four named ingredients can be interpreted as three interacting evidence domains. NanoCurcumin Oil and NanoCurcumin Extract form a curcumin-centred delivery and regenerative platform; *Leucas aspera* contributes a botanical antimicrobial-redox domain; and *Boswellia serrata*-derived Olibanum contributes an inflammation-matrix-vascular domain. Together, these domains give the formulation broad coverage of wound-repair biology; how fully that coverage is realised in the cream depends on the identity and standardisation of its raw materials, excipients, concentrations, release characteristics, and local tissue exposure. The formulation architecture and evidence hierarchy are summarised in Table 1 and Figure 1.

The four actives are identified here by their declared ingredient names. Quantitative composition (% w/w), extract ratios, plant parts, extraction solvents and marker-compound specifications of the corresponding raw materials are not reported in this review. Because these parameters determine whether the cited experimental preparations are comparable with the formulation's raw materials, each ingredient-to-product inference in the following sections is conditional on the characterisation requirements set out in Section 10.

Table 1. Active components of Leucas NC Cream and evidence-based scientific positioning.

Component	Evidence-linked role	Strongest evidence domain	Key limitation
NanoCurcumin Oil	Lipophilic nanocurcumin delivery component; rationale includes local cutaneous retention and formulation-dependent release from topical lipid-carrier literature	Topical lipid nanocarriers and wound-specific curcumin NLC studies	Actual carrier architecture, local bioavailability, percutaneous behaviour and release from the cream are unreported
NanoCurcumin Extract	Principal curcumin bioactive platform	Fibroblast mobilisation, diabetic-wound models, macrophage regulation, ECM and re-epithelialisation	Different nanoformulations are not interchangeable.
Leucas aspera extract	Antimicrobial, antioxidant, anti-inflammatory and emerging fibroblast-supportive botanical component	Mechanistic antibacterial studies; phytochemical pharmacology; 2026 *Leucas*-mediated scratch model	Direct wound evidence for isolated extract remains limited; standardisation is critical.
Olibanum [*Boswellia serrata* extract]	Inflammatory regulation, ECM/collagen support, angiogenesis-associated and anti-apoptotic activity	2019 standardised DFU study; 2025 β-boswellic-acid mechanistic study; 2026 topical *Boswellia* gel study	Purified constituent, resin extract, and essential oil are not interchangeable; the exact raw material must be defined

Abbreviations: DFU, diabetic foot ulcer; ECM, extracellular matrix; NLC, nanostructured lipid carrier.

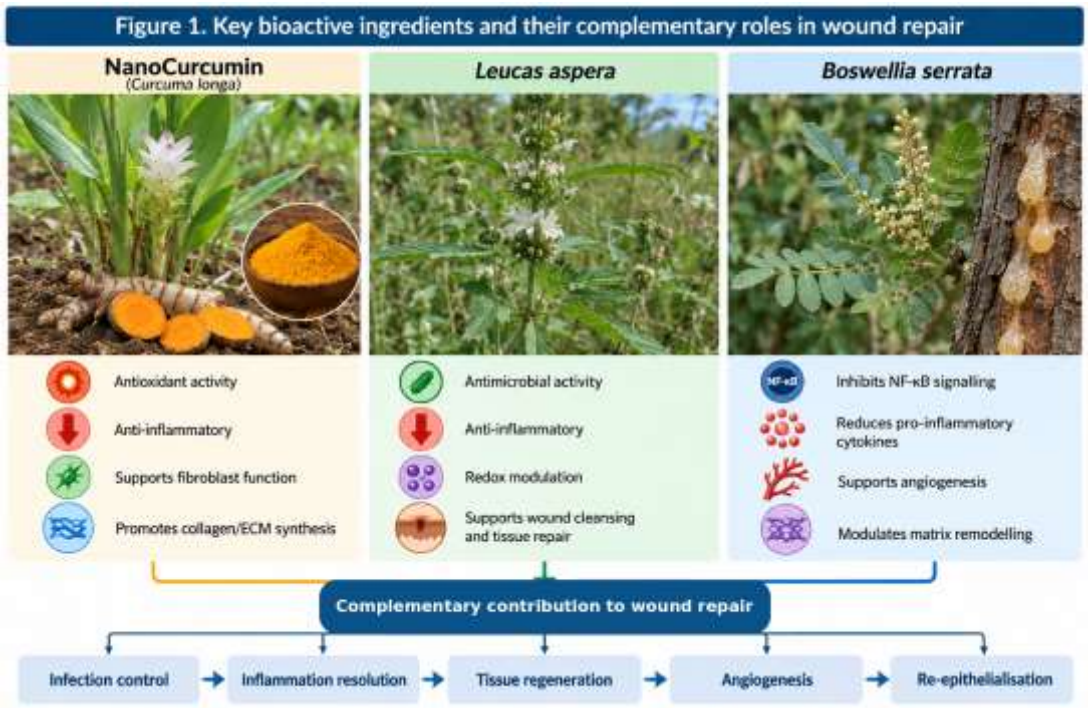

Fig 1: Key bioactive ingredients and their complementary evidence domains in wound repair. NanoCurcumin, *L. aspera,* and *B. serrata* are mapped to the areas in which the literature provides their strongest support. Graphic labels are schematic summaries of ingredient-level findings, not claims of clinical activity or pharmacological synergy for the finished formulation.

The evidence is intentionally treated as asymmetric. NanoCurcumin has the widest span of wound-specific studies, *Boswellia* has particularly strong preclinical diabetic-wound evidence, and *Leucas* has a smaller but distinctive antimicrobial and emerging wound-related literature. This asymmetry is central to the critical interpretation: the ingredients are not assumed to contribute equally, and the review-level conclusion for each component is limited to what its strongest evidence can support.

4. NanoCurcumin as the Principal Regenerative Platform

4.1 Pharmaceutical rationale for nanoformulated curcumin

Curcumin, a major polyphenol of *Curcuma longa*, has substantial wound-relevant pharmacology but presents formulation challenges because of poor aqueous solubility and limited conventional systemic exposure[10]. Reviews of wound-focused delivery systems show why nanoengineering is attractive: carrier design can alter local stability, release, and tissue exposure, but not every nanocurcumin preparation behaves identically[11-13]. Table 2 summarises representative high-value curcumin and nanocurcumin wound studies.

Table 2. Selected high-value evidence for curcumin and nanocurcumin in wound repair.

Study	Model/formulation	Evidence signal	Interpretive value	Ref.
Dai et al., 2017	Curcumin/gelatin nanofibres; fibroblasts and rat acute wounds	Greater fibroblast motility and repair-associated tissue endpoints, with Dkk-1/Wnt and MCP-1 implicated mechanistically	Direct nanocurcumin wound mechanism	[14]
Karri et al., 2016	Curcumin-loaded chitosan nanoparticles in collagen-alginate scaffold; diabetic wounds	Improved diabetic wound repair while addressing curcumin formulation limitations	Strong preclinical DFU relevance	[15]

Liu et al., 2018	MMP-9-responsive curcumin nanoparticle hydrogel; diabetic mice	Pathology-responsive release with faster healing	Links delivery design to chronic-wound protease biology	[16]
Cao et al., 2024	High-glucose fibroblasts and DFU rat model	Improved fibroblast survival/migration and vascular responses via miR-152-3p/FBN1/TGF-β	Direct diabetic-wound mechanism	[17]
Kant et al., 2014	Topical curcumin gel; diabetic rat wounds	Lower inflammatory/protease markers with stronger antioxidant defence and tissue repair	Direct topical diabetic-wound evidence	[18]
Astaneh & Fereydouni, 2025 (online 2024)	Nanocurcumin zein nanofibres; macrophages and rat wounds	Lower iNOS and higher Arg-1 accompanied by improved closure, epithelial restoration, and collagen deposition	Direct immunomodulatory wound evidence	[19]
Li et al., 2022	Systematic review/meta-analysis of 9 diabetic animal studies	Overall benefit for healing rate and vessel density; inflammatory findings heterogeneous	Higher-level preclinical synthesis	[20]
Agharazi et al., 2025	Randomised topical turmeric study; 76 DFU patients	Greater ulcer-area reduction vs placebo at five weeks	Human topical support; not nanocurcumin	[21]
Mokhtari et al., 2021	Oral nanocurcumin RCT; 60 grade-3 DFU patients	Metabolic/antioxidant improvement without significant wound-size benefit	Important negative human evidence; route matters	[22]
Esposito et al., 2014	Lipid vehicles; stratum corneum-epidermis membranes and in vivo skin assessments	Distinct vehicle-dependent disposition profiles, ranging from early penetration to greater stratum-corneum retention and slower release	Strong formulation-science evidence for percutaneous delivery and release; intact-skin model, not open-wound exposure	[23]
Calderon-Jacinto et al., 2022	Curcumin NLCs embedded in Carbopol hydrogel; Franz diffusion membrane model.	Hydrogel incorporation altered membrane accumulation and release while maintaining cytocompatibility.	Shows that vehicle/excipient architecture can change local delivery behaviour	[24]

Abbreviations: Arg-1, arginase-1; DFU, diabetic foot ulcer; Dkk-1, Dickkopf-1; FBN1, fibrillin-1; iNOS, inducible nitric oxide synthase; MCP-1, monocyte chemoattractant protein-1; MMP-9, matrix metalloproteinase-9; NLC, nanostructured lipid carrier; RCT, randomised controlled trial; TGF-β, transforming growth factor-β.

At the level of formulation rationale, lipid nanocarriers provide a useful comparator for the NanoCurcumin Oil component. In the study by Rapalli et al., a curcumin nanostructured lipid carrier (NLC) of approximately 96 nm showed greater uptake by keratinocytes and greater ex vivo cutaneous deposition than non-nanostructured comparators[25]. This supports lipid-mediated local delivery as a formulation principle, while leaving the actual carrier architecture, tissue distribution, and wound-bed exposure of the product-specific NanoCurcumin Oil unresolved.

For cutaneous application, topical delivery should not be equated with transdermal systemic delivery. In wound-directed use, the more relevant pharmaceutical objective is adequate local exposure in the wound bed and adjacent viable epidermal and dermal tissue. In the Rapalli study, the NLC gel prolonged curcumin liberation in vitro and increased ex vivo skin permeation and retention relative to free-curcumin gel[25]. These findings identify local retention and controlled release as realistic, measurable attributes for a nanocurcumin cream (Section 10.1).

In a rabbit full-thickness excision model, NLC-encapsulated curcumin produced greater early wound closure than free curcumin and showed stronger antioxidant and antimicrobial activity under the study conditions[26]. The result shows that nanocarrier formulation can enhance the wound-repair performance of curcumin over the free compound, supporting the rationale for a nanoformulated curcumin component.

4.2 Fibroblast mobilisation and inflammatory resolution

Dai et al. showed in a gelatin-nanofibre system that curcumin enhanced fibroblast movement and growth and improved several in vivo indices of tissue repair[14]. Their mechanistic experiments linked these effects to reduced Dickkopf-1 (Dkk-1) restraint on Wnt signalling together with lower monocyte chemoattractant protein-1 (MCP-1)-associated chemotactic activity. The combined pattern suggests facilitation of the shift from inflammatory recruitment toward proliferative repair rather than a simple, non-specific suppression of inflammation.

The critical inference is that NanoCurcumin is relevant to phase transition as well as to redox control: it has direct evidence connecting immune recruitment and fibroblast behaviour within a wound model. Because the Dkk-1/Wnt-MCP-1 mechanism was shown in a gelatin nanofibre platform, its expression in other NanoCurcumin preparations is a question for platform-specific testing.

Fibroblast biology also requires phase-sensitive wording. Migration, proliferation and transient myofibroblast differentiation can support granulation tissue, matrix deposition and wound contraction, whereas persistent or excessive activation can contribute to pathological fibrosis. Accordingly, the more defensible formulation-level concept is regulated dermal fibroblast activation rather than fibroblast activation as an invariably beneficial endpoint[1,14].

4.3 Diabetic wounds, MMP-9-responsive delivery and matrix repair

Diabetic-wound studies extend the evidence beyond acute repair models. In one approach, curcumin-loaded chitosan nanoparticles were incorporated into a collagen-alginate scaffold, combining a local delivery strategy with a provisional matrix for diabetic wounds[15]. A separate chitosan-nanoparticle study reported improved repair together with attenuation of macrophage-associated inflammatory activity in diabetic rats[27].

The pathological wound environment has also been exploited as a release trigger. Liu et al. packaged curcumin nanoparticles in gelatin microspheres within a thermosensitive system responsive to matrix metalloproteinases and reported improved healing in diabetic mice[16]. This strategy is biologically pertinent because MMP-9 is frequently excessive in non-healing diabetic wounds, and higher wound-fluid MMP-9 has been associated with poorer clinical healing[7]. Thus, the study links delivery design to a disease-relevant protease abnormality rather than treating prolonged release as an isolated formulation advantage.

Cao et al. combined high-glucose fibroblast experiments with a rat diabetic foot ulcer (DFU) model and reported improved fibroblast survival and migratory behaviour alongside better vascular and wound-repair readouts after curcumin treatment[17]. Manipulation of miR-152-3p and fibrillin-1 (FBN1) placed this response within a miR-152-3p/FBN1/TGF-β regulatory axis. Separately, topical curcumin gel in diabetic rats altered inflammatory, proteolytic, and antioxidant markers in parallel with improved histological repair[18]. These studies therefore support effects at both cellular and wound-microenvironment levels, without indicating that a single pathway explains the total response.

Re-epithelialisation should likewise be interpreted at the cellular level. Epidermal keratinocyte migration and proliferation at the wound margin are required to restore epithelial continuity. Curcumin biomaterial literature links curcumin-containing systems with improved epithelial restoration and keratinocyte-associated migration, while nanoformulated curcumin wound models report enhanced re-epithelialisation[13,14]. This supports keratinocyte migration as a wound-relevant curcumin process, but not as a demonstrated effect of the finished Leucas NC formulation.

4.4 Macrophage phenotype and immune-phase transition

Macrophage phenotype is one determinant of whether a diabetic wound remains dominated by inflammatory signalling or progresses toward tissue restoration[8]. In zein nanofibres containing nanocurcumin, macrophages displayed a marker pattern shifted away from the inflammatory state, with lower inducible nitric oxide synthase (iNOS) and higher arginase-1 (Arg-1), together with lower interleukin-1β (IL-1β), interleukin-6 (IL-6) and tumour necrosis factor-α (TNF-α) expression[19]. In the corresponding rat wounds, the same construct improved closure, epithelial restoration, and collagen deposition. These observations support immune-state modulation as one plausible contribution of nanocurcumin while remaining specific to the tested zein-nanofibre platform.

These findings strengthen the claim that curcumin can participate in immune-phase regulation, but they should not be generalised beyond the tested nanofibre construct. A meta-analysis of nine diabetic animal studies likewise found an overall benefit for wound-healing rate and vessel density while reporting less consistent anti-inflammatory findings across models[20]. The balanced review-level conclusion is that NanoCurcumin has multi-domain wound activity, with macrophage regulation representing one supported mechanism rather than the sole explanation for repair.

4.5 Human wound evidence and translational limits

Human evidence is heterogeneous but now extends beyond isolated reports. A 2025 scoping review included 19 clinical wound studies of curcumin-containing interventions, 14 of which were randomised trials; favourable findings were common, but heterogeneity in wound type, formulation, route, dose and duration prevented a uniform clinical recommendation[9].

Clinical findings also underline the importance of route and formulation. A placebo-controlled DFU study involving 76 participants reported a larger reduction in ulcer area after five weeks of topical turmeric ointment[21]. By contrast, 12 weeks of oral nanocurcumin in 60 patients with grade-3 DFU improved several metabolic and antioxidant biomarkers without a corresponding significant advantage in wound-size outcomes[22]. The trials are not directly comparable, but together they caution against assuming that systemic biochemical improvement necessarily produces local wound closure. Product-specific exposure and wound endpoints are therefore more informative for a topical formulation than extrapolation from oral nanocurcumin.

5. *Leucas aspera*: Distinct Botanical Contribution to Wound Biology

5.1 Phytochemical basis and standardisation relevance

Leucas aspera is a Lamiaceae species reported to contain triterpenoids, sterol-related compounds, lignans, flavonoids, diterpenes and other phenolic constituents[28-31]. Reversed-phase high-performance liquid chromatography (HPLC) has been used to quantify oleanolic and ursolic acids across *Leucas* species, demonstrating that chemical standardisation of this botanical is technically feasible[31]. Selected *L. aspera* evidence relevant to wound biology is summarised in Table 3.

Table 3. Selected *Leucas aspera* evidence relevant to wound biology.

Study	Model	Main relevance	Boundary of inference	Ref.
Sadhu et al., 2003	Activity-guided phytochemistry	Radical-scavenging and prostaglandin-response inhibition; active lignans/flavonoids isolated	Mechanistic, not direct wound evidence	[29]
Sadhu et al., 2006	Isolated diterpenes/glycosides	Several compounds inhibited prostaglandin-induced contractions	Constituent-level anti-inflammatory support	[30]
Shukla et al., 2016	HPLC comparison across *Leucas* species	Quantification of oleanolic and ursolic acids	Supports standardisation, not efficacy	[31]

Kripa et al., 2011	Adjuvant-arthritis model	Inflammatory-marker and antioxidant-enzyme modulation	Supportive systemic pharmacology	[32]
Chew et al., 2012	Different plant parts; in-vitro assays	Antioxidant and antibacterial activity	Extract- and part-dependent	[33]
Saritha et al., 2015	Mechanistic bacterial study	Loss of bacterial membrane integrity with increased permeability, ultrastructural damage, and release of intracellular contents	Direct antimicrobial mechanism; not wound treatment	[34]
Poorani et al., 2026	*Leucas*-mediated chitosan-ZnO composite; L929 scratch assay	Antioxidant, antibacterial, and anti-inflammatory activity with approximately 96.6% scratch-gap closure under the best composite condition	Composite system; effect cannot be assigned to *Leucas* alone	[35]

Abbreviations: HPLC, high-performance liquid chromatography; L929, mouse fibroblast cell line; ZnO, zinc oxide.

The reason standardisation matters is that a chemically diverse extract is not a single reproducible intervention. Plant part, solvent, and extraction conditions can alter the constituents that reach the final formulation. Documenting botanical identity, plant part, extraction process, and a chromatographic fingerprint therefore allows the *Leucas* evidence to be linked with confidence to the extract used in Leucas NC Cream.

5.2 Antioxidant and anti-inflammatory evidence

Bioactivity-guided work on *L. aspera* identified fractions with free-radical-scavenging activity and reduced responsiveness in a prostaglandin-driven smooth-muscle assay, followed by isolation of several lignan and flavonoid constituents[29]. Subsequent phytochemical work isolated diterpenes and glycosides that were active in the same functional assay[30]. These findings support redox and inflammation-related pharmacology at the constituent level, but they are not direct wound-healing experiments.

In an adjuvant-arthritis model, ethanolic *L. aspera* extract changed inflammatory and endogenous antioxidant endpoints, including TNF-α-related and redox-enzyme measures[32]. Independent experiments using different plant parts also demonstrated antioxidant and antibacterial activity[33], while more recent extract comparisons showed that antimicrobial and anti-inflammatory readouts varied with extraction solvent[36]. The defensible synthesis is therefore extract-dependent redox and inflammatory pharmacology, not a generic wound-healing effect.

5.3 Antimicrobial relevance to the wound microenvironment

Within the reviewed evidence, the most distinctive contribution of *Leucas* is its antimicrobial pharmacology. This is relevant because wound-associated microorganisms and biofilms can perpetuate inflammatory signalling and complicate treatment[4]. In a mechanistic *Escherichia coli* study, ethanolic *L. aspera* extract progressively disrupted membrane integrity, producing loss of electrochemical membrane function, greater permeability, visible surface deformation, and release of intracellular contents[34].

This evidence identifies membrane injury as a plausible antibacterial mode of action for *Leucas* constituents. Translating this mechanism into product-level antimicrobial data calls for standardised minimum inhibitory concentration (MIC)/minimum bactericidal concentration (MBC) testing, time-kill and biofilm assays and, ideally, testing against clinically relevant wound isolates. At review level, *Leucas* is best positioned as a supportive microbial-pressure component that complements, rather than replaces, antimicrobial therapy.

5.4 Emerging wound-related evidence

The most direct recent wound-related study incorporated *L. aspera* leaf extract into a chitosan–zinc oxide (ZnO) nanocomposite rather than evaluating the extract alone[35]. The resulting material showed antioxidant, antibacterial, and anti-inflammatory activity, acceptable cytocompatibility, and marked L929

fibroblast gap closure under the best test condition (approximately 96.6%). Because every component of the composite can influence the assay, this percentage cannot be assigned specifically to *L. aspera*.

That distinction is crucial because both chitosan and ZnO can contribute biological activity. The study cannot establish that *L. aspera* by itself produces near-complete wound closure. Its legitimate contribution to this review is different: *Leucas*-derived chemistry can coexist within a wound-directed material that supports antimicrobial control and fibroblast migration at the same time. This supports a novel formulation-level interpretation of *Leucas* as a possible bridge between microbial-pressure control and a repair-permissive environment, while leaving direct isolated-extract wound efficacy unresolved.

6. *Boswellia serrata*-Derived Olibanum: Inflammation, Matrix and Angiogenesis

6.1 Raw-material distinction

Boswellia serrata produces an oleo-gum resin containing non-volatile pentacyclic triterpenoids, including boswellic acids, together with a smaller volatile-oil fraction[37]. Steam-distilled frankincense essential oil is chemically different from a boswellic-acid-rich resin extract and is dominated by volatile terpenes, as illustrated by a recent wound study of an oil characterised by gas chromatography–mass spectrometry (GC-MS)[38]. Because the confirmed formulation ingredient is described as Olibanum oil [*Boswellia serrata* extract], resin-extract and essential-oil evidence should not be treated as interchangeable; the strongest literature link depends on the analytical definition of the actual raw material. Selected *Boswellia*/Olibanum evidence relevant to wound repair is summarised in Table 4.

Table 4. *Boswellia*/Olibanum evidence relevant to wound repair.

Study	Material/model	Main findings	Translational interpretation	Ref.
Zhang et al., 2019	Standardised *B. serrata* extract; experimental DFU; oral dosing	Greater contraction and hydroxyproline with coordinated shifts in inflammatory/apoptotic signalling, matrix markers and the VEGF-Ang-1/Tie2 axis	Strong mechanistic diabetic-wound evidence; route differs from cream	[39]
Han et al., 2025	β-Boswellic acid; high-glucose fibroblasts and diabetic rat wounds	Improved fibroblast migration; reduced oxidative/ferroptotic stress; faster closure with greater granulation and collagen deposition; STAT3-GPX4 implicated	Constituent-level evidence only; purified β-boswellic acid is not equivalent to the formulated Olibanum raw material	[40]
Mukadam et al., 2026	*B. serrata* extract alone and *B. serrata*-zinc topical gel; diabetic rats	Improved wound area together with lower cytokine/MMP-9 activity, stronger antioxidant defences and better collagen-I/VEGF and histological endpoints	Strongest direct topical *B. serrata* evidence identified; best gel also contained zinc	[41]
Venkatesan et al., 2025	Chemically characterised frankincense essential oil; rat excision wounds	Less inflammatory/oxidative injury with improved collagen and epithelial restoration	Supportive Olibanum oil evidence; not equivalent to resin extract	[38]
Badr et al., 2023	*B. carteri* topical formulation vs silver sulfadiazine; randomised burn trial	Progressive healing in both arms without significant difference in time to healing among completers	Human genus-level topical feasibility only	[42]

Abbreviations: Ang-1, angiopoietin-1; DFU, diabetic foot ulcer; GPX4, glutathione peroxidase 4; MMP-9, matrix metalloproteinase-9; STAT3, signal transducer and activator of transcription 3; Tie2, TEK receptor tyrosine kinase; VEGF, vascular endothelial growth factor.

6.2 Standardised *Boswellia serrata* in experimental diabetic wounds

In the 2019 diabetic-wound model, orally administered standardised *B. serrata* extract increased wound contraction and hydroxyproline while reducing the oxido-inflammatory burden[39]. Molecular readouts showed partial normalisation of inflammatory/apoptotic signalling together with preservation of TGF-β1, collagen-I, vascular endothelial growth factor (VEGF), angiopoietin-1 (Ang-1) and TEK receptor tyrosine kinase (Tie2) repair signals. This study is particularly informative because it links inflammatory control to structural and vascular repair within the same diabetic-wound model. The review-level inference is that *Boswellia* may act as an inflammation-to-matrix-to-vascular bridge rather than as an anti-inflammatory ingredient alone. The main limitation is route: the standardised extract was administered orally, so equivalence to the topical Olibanum component cannot be assumed.

A complementary constituent-level study evaluated β-boswellic acid in high-glucose human skin fibroblasts and diabetic rat wounds[40]. The compound improved impaired fibroblast migration, reduced biochemical and molecular features of ferroptotic/oxidative injury, and accelerated wound closure with greater granulation and collagen deposition. Pharmacological interference with signal transducer and activator of transcription 3 (STAT3) weakened part of the cellular response, supporting a candidate STAT3–glutathione peroxidase 4 (GPX4)/ferroptosis axis. The study strengthens the mechanistic plausibility of *Boswellia*-derived constituents in diabetic wound repair; because it used purified β-boswellic acid, the corresponding response to the formulation's Olibanum material remains to be characterised.

6.3 New topical *Boswellia* evidence

Mukadam et al. subsequently compared topical *B. serrata* preparations in diabetic rat wounds[41]. The zinc-containing gel achieved the largest reduction in wound area, while the *B. serrata*-only arm also improved closure relative to diabetic controls. Treated wounds showed a broader shift toward lower inflammatory/protease activity, stronger antioxidant defences and improved collagen-I, VEGF and histological repair endpoints. The zinc formulation therefore cannot be treated as evidence for *Boswellia* alone, although the extract-only arm strengthens the topical preclinical case.

6.4 Supportive frankincense-oil and human topical evidence

Supportive evidence is also available for frankincense essential oil. In a 2025 rat excision model, chemically characterised oil treatment was associated with less inflammatory/oxidative damage and apoptosis-related signalling together with better contraction, collagen deposition and epithelial recovery[38]. This extends the wound relevance of Olibanum-derived materials and is most directly applicable where the Olibanum component is an essential-oil fraction rather than a resin extract.

Human topical evidence is emerging at genus level. In a randomised double-blind trial of second-degree burns, a standardised *Boswellia carteri* formulation achieved healing times similar to those of the conventional comparator, 1% silver sulfadiazine, among study completers[42]. This supports the topical feasibility of *Boswellia* preparations, although a species-specific *B. serrata* wound trial has not yet been reported. Across the *Boswellia* literature, inflammatory resolution, extracellular matrix (ECM) support, and vascular signalling converge consistently in preclinical models, with raw-material identity and route as the main translational qualifiers.

7. Combination Pharmacology and Translational Application

The comparative experiments of Ammon et al. indicate that curcumin and boswellic acids reach inflammatory pathways through partly different biochemical routes[43]. Curcumin influenced 5-lipoxygenase (5-LOX) together with platelet 12-lipoxygenase (12-LOX)/cyclooxygenase (COX)-related activity and also behaved as a reducing antioxidant, whereas boswellic acids preferentially suppressed leukotriene formation through 5-LOX without reproducing the same 12-LOX/COX pattern. Their mechanistic non-equivalence is therefore a stronger basis for complementarity than the broad observation that both are anti-inflammatory.

Human osteoarthritis studies provide evidence that curcumin and boswellic-acid preparations can be co-administered. In a randomised, double-blind three-arm trial involving 201 participants, the combination was well tolerated and showed advantages over comparator arms for some outcomes[44]. A subsequent review judged the preclinical data compatible with additive activity and considered clinical synergy possible, pending definitive confirmation[45].

For wound care, these studies support pharmacological compatibility and a clear rationale for complementary pathway coverage. Targeted searching did not identify a convincing indexed study of NanoCurcumin plus *L. aspera*, *L. aspera* plus *B. serrata*, or the complete NanoCurcumin-*Leucas*-*Boswellia* system as one wound formulation, which positions the combination as a novel formulation concept. Its scientific value lies in evidence convergence: it brings non-identical evidence domains together in a single topical system, and experimental studies can now test whether this convergence yields additive benefit.

8. Integrated Mechanistic Interpretation

The formulation is most coherent when mapped onto barriers to repair rather than presented as three unrelated botanical summaries. NanoCurcumin contributes the strongest direct evidence for fibroblast mobilisation, macrophage-state regulation and matrix-associated repair[14,17-20]. *Leucas* contributes a distinctive antimicrobial component, supported by bacterial membrane disruption plus redox and inflammatory pharmacology[29,32-36]. *Boswellia* contributes the clearest ingredient-level evidence linking cytokine regulation with collagen-associated and angiogenic responses in diabetic wounds[38-41]. The integrated formulation hypothesis is shown in Figure 2.

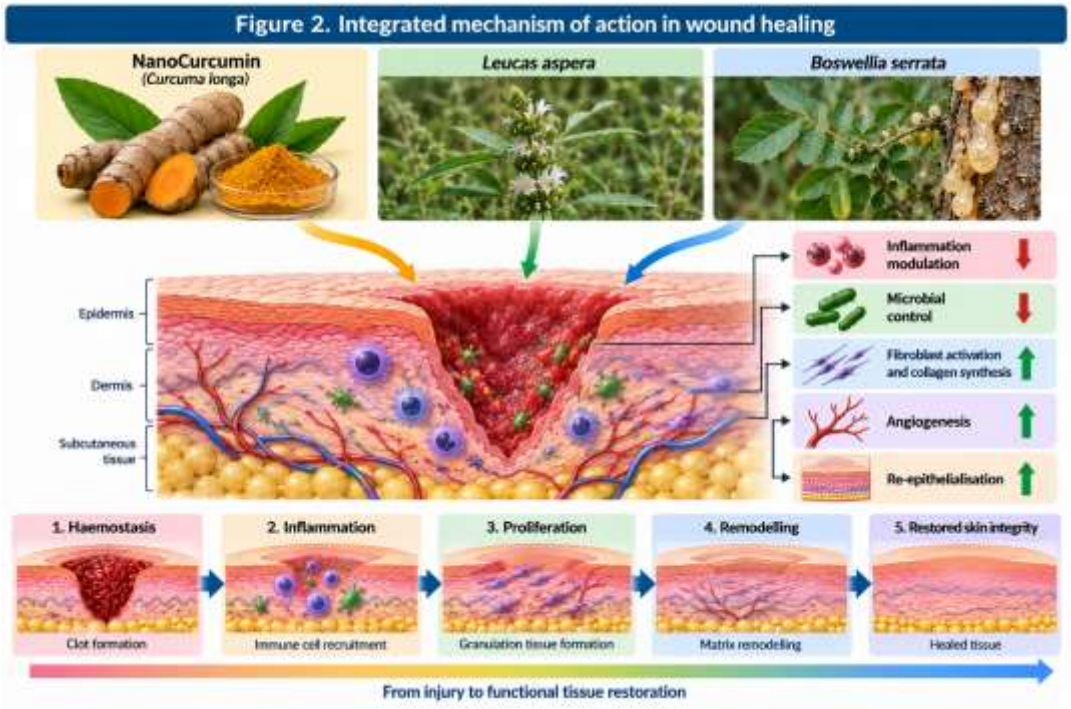

Fig 2: Proposed wound-repair pathways represented in the ingredient-level evidence. The diagram maps processes supported by studies of NanoCurcumin, *L. aspera,* and *B. serrata*, including inflammatory modulation, reduced microbial burden, regulated fibroblast/ECM activity, angiogenesis, and re-epithelialisation. The arrows indicate review-level mechanistic convergence rather than a demonstrated causal mechanism of the finished formulation. Labels such as “microbial control” and “fibroblast activation” should be interpreted as schematic shorthand for reduced microbial burden and regulated fibroblast/ECM activity in the cited models.

Within this model, redox balance and inflammatory resolution are upstream permissive events rather than therapeutic endpoints in isolation. Lower microbial pressure may remove an additional source of inflammatory stimulation; viable, migrating fibroblasts can then support granulation and ECM formation; and VEGF/Ang-1-Tie2-associated signalling can support vascular reconstruction before epithelial closure. The novel synthesis of this review is that the scientific strength of Leucas NC Cream lies in combining evidence streams that address sequential obstacles to healing, rather than in any single new pathway. This sequence provides a testable mechanistic framework for the finished cream.

9. Relevance to Diabetic and Difficult-to-Heal Wounds

The diabetic-wound context is particularly relevant because two formulation components have direct diabetic-wound evidence. Curcumin has been studied in chitosan nanoparticles, collagen-alginate

scaffolds, protease-responsive hydrogels, high-glucose fibroblast systems and topical-gel models[15-18,27]. *Boswellia serrata* has both standardised oral-extract evidence[39] and newer direct topical evidence[41]. Contemporary DFU reviews further emphasise that persistent inflammation, impaired angiogenesis and dysfunctional fibroblast and immune-cell responses interact rather than occur independently[3].

These ingredient studies intersect with recognised DFU biology. NRF2-dependent antioxidant signalling influences keratinocyte behaviour, apoptosis, TGF-β1 and MMP-9 under diabetic conditions[6]; high wound-fluid MMP-9 predicts poorer healing in human diabetic ulcers[7]; and delayed macrophage transition can prolong the inflammatory phase[8]. Curcumin and *Boswellia* address several of these domains experimentally, while *Leucas* adds antimicrobial support relevant to a wound environment in which microbial communities can amplify inflammation[4].

The evidence remains asymmetric and should be reported as such. NanoCurcumin has the broadest wound-specific literature, *Boswellia* has strong mechanistic diabetic-wound evidence, and *Leucas* has emerging wound relevance with a comparatively distinctive antimicrobial contribution. The review-level conclusion is not that all ingredients are equally effective, but that their strongest evidence domains are potentially complementary.

10. Phytochemical Standardisation and Pharmaceutical Characterisation

Translation from literature to the actual product depends on demonstrating that the materials used in Leucas NC Cream are sufficiently comparable with those studied experimentally. For NanoCurcumin Oil and NanoCurcumin Extract, relevant characterisation includes curcuminoid composition, particle-size distribution where applicable, polydispersity, surface charge where applicable, loading or dispersion state, chemical stability and release from the cream matrix. If "nano" is retained as a defining product descriptor, at least one directly measured nanoscale attribute should support that designation.

10.1 Cutaneous delivery, stratum-corneum interactions and formulation-dependent release

Skin-delivery terminology requires particular care. In intact skin, the stratum corneum is the dominant barrier to percutaneous transport, and the vehicle can determine where and how rapidly curcumin is deposited. Esposito et al. showed that two lipid systems produced different disposition profiles: one favoured earlier penetration, whereas the other produced greater stratum-corneum retention with a slower release pattern[23]. Since an open wound disrupts this barrier, intact-skin data demonstrate the importance of excipient architecture but cannot directly predict wound-bed exposure.

A second delivery platform supports the same formulation-science principle. Incorporation of curcumin NLCs into a Carbopol hydrogel changed membrane accumulation and release behaviour[24], and Rapalli et al. likewise reported prolonged release with greater skin retention from an NLC gel[25]. These studies make release, retention, diffusion, and excipient compatibility measurable targets for the finished cream, and their direct measurement would establish its cutaneous delivery profile.

For *Leucas aspera*, the minimum scientific description should include authenticated species, plant part, extraction process, extract ratio, and a chromatographic fingerprint. Oleanolic acid, ursolic acid, or other validated markers may be useful depending on the actual material[31]. For the *Boswellia*/Olibanum component, the raw-material type should be stated explicitly; boswellic-acid markers are appropriate for resin-rich extracts, whereas GC-MS profiling is more informative for volatile-oil fractions[37,38].

Finished-product characterisation should include appearance, pH, rheology or viscosity, relevant marker/assay content, microbiological quality, preservative effectiveness where applicable, stability and compatibility with wounded tissue. For a wound-directed cream, release testing and cytocompatibility provide a more direct bridge to the biological claims than cosmetic characterisation alone.

11. Safety and Translational Considerations

Human curcumin wound studies summarised in the 2025 scoping review reported few treatment-related safety signals overall, although tolerability varied by formulation and route[9]. Traditional sources describe topical use of *L. aspera*, but standardised long-term exposure on damaged skin remains poorly characterised[28]. *Boswellia*-derived topical preparations have entered human testing, and the burn trial reported hypersensitivity withdrawals and specifically cautioned about contact dermatitis[42].

Safety of the finished cream cannot therefore be inferred from historical use of its separate ingredients. Application to an open or chronic wound requires control of microbial quality, contaminants, pH and excipient compatibility, together with product-specific cytocompatibility and irritation/sensitisation assessment. Clinically infected DFUs should continue to receive evidence-based wound care and antimicrobial treatment when indicated[2].

12. Strengths, Evidence Gaps and Limitations

The principal strength of the Leucas NC concept is convergence across several recognised barriers to healing rather than dependence on one pathway. High-quality wound and DFU literature establishes the relevance of redox balance, microbial ecology, protease control, macrophage-state transition, fibroblast function, and angiogenesis[1-8]. The ingredient literature then provides complementary evidence across these domains, with the highest translational weight carried by nanocurcumin and *Boswellia* studies and a distinctive antimicrobial contribution from *Leucas*. The claim-rationale-evidence synthesis is consolidated in Table 5, and the overall evidence hierarchy and translational gap are illustrated in Figure 3.

Table 5. Major review claims structured as rationale, factual evidence, and bounded novel inference.

Major review claim	Why the claim is reasonable	Factual evidence	Novel review-level conclusion and boundary
Chronic and diabetic wounds justify a multi-target framework	Redox imbalance, inflammatory persistence, microbial ecology, protease excess, macrophage dysfunction, and impaired vascular/cellular repair can coexist.	High-level wound biology, DFU, microbiome, NRF2, MMP-9, and macrophage literature[1-8].	A multi-axis formulation is biologically rational; this does not show that Leucas NC Cream itself modifies all axes.
NanoCurcumin is the principal regenerative evidence platform	It has the widest span of direct wound-specific delivery, fibroblast, immune, ECM, and vascular studies among the ingredients.	NLC, nanofibre, chitosan, MMP-responsive, fibroblast, macrophage, and diabetic-wound studies[14-20,25-27].	NanoCurcumin carries the strongest direct wound rationale in the formulation; different nano-systems are not interchangeable with the product.
NanoCurcumin may help the wound transition from inflammation toward proliferation.	Successful repair requires inflammatory recruitment to resolve while fibroblast and reparative immune functions emerge.	Dkk-1/Wnt-MCP-1 evidence[14] plus macrophage-marker and wound-repair data[19].	Phase-transition regulation is a plausible curcumin mechanism, not a demonstrated universal property of NanoCurcumin Oil/Extract.
Leucas aspera provides a distinctive microbial-redox contribution	Microbial pressure can amplify inflammation; *Leucas* has membrane-level antibacterial evidence plus antioxidant and anti-inflammatory pharmacology.	Activity-guided phytochemistry, inflammatory models, extraction studies, membrane-disruption experiments, and the 2026 composite scratch model[29,32-36].	*Leucas* may bridge microbial-pressure control and repair-permissive biology; isolated-extract wound efficacy remains limited.
Boswellia may bridge inflammatory resolution with matrix and vascular repair.	Diabetic repair requires control of cytokine excess together with collagen/ECM reconstruction and angiogenesis.	Standardised *B. serrata* DFU study, β-boswellic-acid fibroblast/DFU study, topical *B. serrata* study and supportive frankincense-oil evidence[38-41].	*Boswellia* shows a coherent inflammation-matrix-vascular profile, with an emerging ferroptosis/STAT3 dimension; route, constituent form, and raw-material identity limit direct transfer to the cream.

Curcumin and *Boswellia* are complementary rather than redundant	Their anti-inflammatory actions overlap but are not biochemically identical.	5-LOX/12-LOX/COX-related mechanistic data and human co-administration evidence[43-45].	Co-inclusion has a rational pharmacological basis; wound-specific additivity or synergy is not established.
The complete formulation is novel primarily through evidence convergence	The strongest evidence domains of the ingredients map to different sequential barriers to repair.	Integrated evidence across NanoCurcumin, *Leucas* and *Boswellia*[14-20,27,29,32-36,38-41,43-45].	The novelty is the combination of evidence streams, not the discovery of a new pathway; the complete product remains untested.
Ingredient-level convergence is not equivalent to clinical efficacy	The cited studies use different materials, routes, models, and wound types, and no direct finished-product trial was identified.	Translational limitations are summarised throughout and human evidence in[9,21,22,42].	Leucas NC Cream should be framed as a testable translational hypothesis until product-specific efficacy, safety, antimicrobial performance and synergy are demonstrated.
Topical performance is vehicle- and barrier-dependent	Cutaneous exposure depends on the stratum-corneum barrier, carrier architecture, vehicle/excipient interactions and release behaviour.	Curcumin lipid vehicles and NLC/hydrogel systems show formulation-dependent penetration, retention and release[23-25].	Local cutaneous exposure and sustained release are credible formulation objectives, but product-specific percutaneous absorption, transdermal flux and local bioavailability remain unmeasured.
Epigenetic mechanisms are an emerging research dimension rather than an established product mechanism.	Macrophage phenotype and inflammatory memory can be shaped by chromatin and DNA-methylation programmes during wound repair, and curcumin has documented immune-cell epigenetic effects.	Wound-macrophage epigenetic review evidence plus curcumin studies involving high-glucose monocytes and TREM-1/p300-associated chromatin regulation[46-48].	Epigenetic regulation is biologically plausible but hypothesis-generating. Targeted DNA methylation, locus-specific demethylation, promoter hyperacetylation, or phenotype rescue should not be attributed to the finished formulation without direct data.

Abbreviations: COX, cyclooxygenase; DFU, diabetic foot ulcer; Dkk-1, Dickkopf-1; DNA, deoxyribonucleic acid; ECM, extracellular matrix; 5-LOX, 5-lipoxygenase; 12-LOX, 12-lipoxygenase; MCP-1, monocyte chemoattractant protein-1; MMP-9, matrix metalloproteinase-9; NLC, nanostructured lipid carrier; NRF2, nuclear factor erythroid 2-related factor 2; STAT3, signal transducer and activator of transcription 3; TREM-1, triggering receptor expressed on myeloid cells 1.

The main limitation is the translational distance between the cited interventions and the finished formulation. A gelatin nanofibre, chitosan nanoparticle, lipid carrier, and MMP-responsive hydrogel are not pharmaceutically equivalent. The strongest recent *Leucas* wound study used a chitosan-ZnO composite rather than isolated extract[35]; the 2019 *Boswellia* study used oral standardised extract[39]; and the strongest 2026 topical *Boswellia* result came from a zinc-containing formulation[41]. Human wound evidence is most

developed for turmeric/curcumin interventions, remains limited for *Boswellia,* and has not been established directly for *Leucas*.

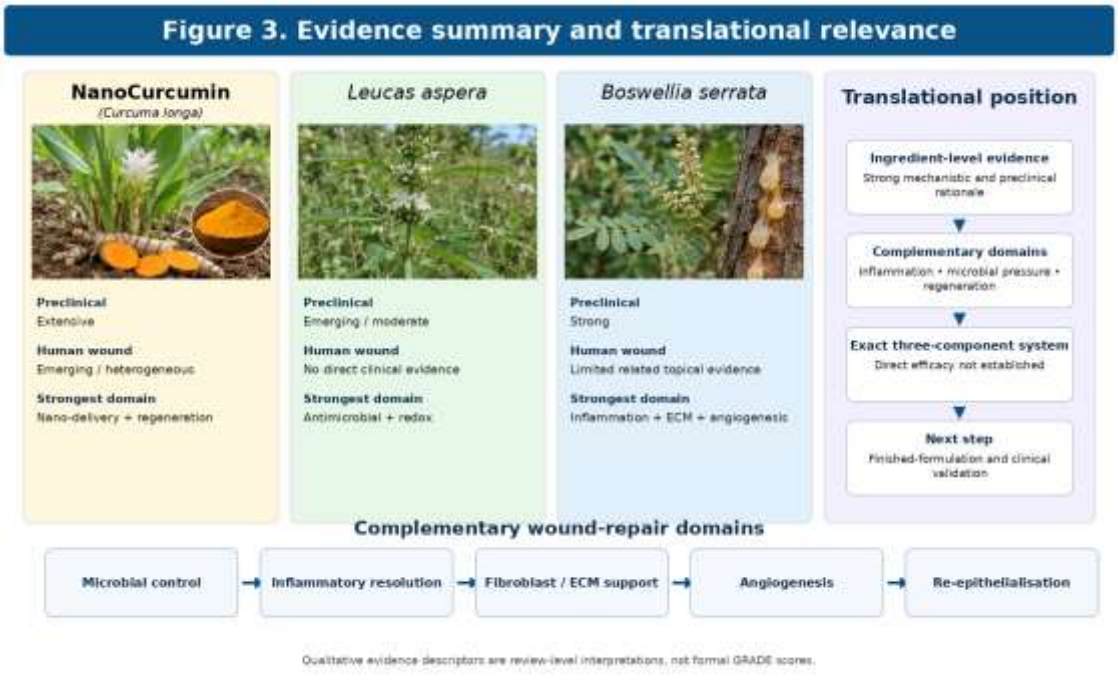

Fig 3: Evidence summary and translational relevance. The relative evidence base for NanoCurcumin, *Leucas aspera* and *Boswellia serrata* is summarised across preclinical, human and mechanistic domains. Qualitative descriptors are review-level interpretations rather than formal GRADE scores. Direct clinical efficacy of the exact three-component finished formulation remains unestablished.

The current literature also does not establish additive or synergistic wound healing for the complete formulation. Accordingly, the strongest scientifically defensible position is mechanistic coherence plus ingredient-level evidence convergence. Product-specific physicochemical characterisation, antimicrobial and biofilm testing, cell compatibility, preclinical wound studies and appropriately designed human trials are required before efficacy or synergy can be claimed.

12.1 Emerging epigenetic dimension

Epigenetic regulation is relevant to macrophage plasticity because inflammatory and reparative phenotypes are partly stabilised by chromatin-dependent transcriptional programmes[46]. Curcumin can influence this layer of regulation in immune-cell systems outside wound models. Under high-glucose conditions, it altered the balance between p300/histone acetyltransferase (HAT) and histone deacetylase 2 (HDAC2) activity and reduced nuclear factor kappa B (NF-κB)-linked cytokine transcription in monocytes[47]; in lipopolysaccharide (LPS)-stimulated macrophages, it reduced p65 occupancy at the triggering receptor expressed on myeloid cells 1 (TREM-1) promoter and local p300 activity, accompanied by lower histone H3/H4 acetylation[48]. These experiments demonstrate immune-cell epigenetic activity, not an established epigenetic mechanism for NanoCurcumin-mediated wound repair.

For that reason, stronger mechanistic terms - targeted deoxyribonucleic acid (DNA) methylation, locus-specific demethylation, reversal of aberrant methylation, promoter hyperacetylation/open chromatin, or epigenetic rescue - are best reserved for future product-specific studies. The appropriate conclusion is hypothesis-generating: future product-specific work could pair macrophage-phenotype assays with chromatin immunoprecipitation, chromatin-accessibility profiling, and locus-specific methylation analyses.

13. Conclusion

Across the three natural-product systems, the evidence supports a coherent but asymmetric wound-repair framework. NanoCurcumin provides the broadest direct wound evidence, linking formulation strategies with fibroblast mobilisation, macrophage-state modulation, matrix reconstruction, vascular responses, and epithelial repair. *Leucas aspera* contributes a distinct antimicrobial-redox evidence stream supported by

phytochemical, membrane-level antibacterial, and emerging fibroblast-migration data. *Boswellia serrata* contributes particularly strong preclinical evidence connecting inflammatory regulation with collagen-associated and angiogenic endpoints, including recent topical diabetic-wound data. Leucas NC Cream brings these complementary evidence streams together in a single topical formulation, giving it a coherent, multi-target scientific rationale for wound-repair support.

Curcumin-boswellic-acid studies further support pharmacological complementarity[43-45]. The central conclusion of this review is that the formulation is noteworthy because its ingredients converge on sequential barriers to healing - microbial pressure, redox/inflammatory dysregulation, fibroblast and matrix failure, and inadequate vascular support. Within the literature retrieved by this targeted narrative search, no indexed peer-reviewed study has yet evaluated the complete NanoCurcumin-*Leucas*-*Boswellia* combination as Leucas NC Cream; product-specific characterisation, preclinical wound studies, and controlled clinical evaluation are therefore the logical next steps to confirm its efficacy and define its place in wound care.

Declarations

Ethics approval and consent to participate: Not applicable to this narrative review.
Consent for publication: Not applicable.
Funding: No external funding was received for this work. The review was conducted within the Department of Research and Development, Biotex Life Solutions Pvt. Ltd., as part of the authors' institutional research activities.
Competing interests: All authors are employees of Biotex Life Solutions Pvt. Ltd. Leucas NC Cream, the named formulation discussed in this review, is associated with Biotex Life Solutions Pvt. Ltd. This employment and product-development relationship is disclosed as a potential competing interest. No original efficacy data for the finished formulation are presented in this review, and product-related conclusions are explicitly limited to ingredient-level evidence and translational hypotheses.
Author contributions: All authors contributed to conceptualization, literature identification and appraisal, evidence interpretation, manuscript drafting, critical revision, and approval of the final manuscript. All authors accept responsibility for the integrity and accuracy of the work.
Data availability: No new datasets were generated or analysed for this critical narrative review.
Acknowledgements: None.

Declaration of generative AI and AI-assisted technologies in the manuscript preparation process

During the preparation of this work, the authors used OpenAI ChatGPT to support manuscript structuring and language refinement. The authors reviewed and edited all AI-assisted content, independently verified the cited literature and scientific interpretations, and take full responsibility for the content of the manuscript.

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