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Biotex Biosolace Pain Relief Cream: Pharmacological Basis, Topical Analgesic Mechanisms, and Evidence Appraisal of a Multi-Component Ayurvedic Formulation

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Abstract: Localised muscle soreness, joint discomfort and stiffness are among the most frequent reasons for using topical analgesic and counterirritant preparations. Biosolace Pain Relief Cream is publicly described as an Ayurvedic external-use formulation containing menthol, camphor (*Karpura*), ginger (*Shunthi*; *Zingiber officinale*), black pepper (*Marica*; *Piper nigrum*), turmeric (*Haridra*; *Curcuma longa*), *Capsicum* and a cream base. This narrative review critically appraises the pharmacological and clinical evidence relevant to that declared composition and to product-positioning statements concerning fast-acting cooling, soothing muscle and joint comfort, post-activity soreness and rapid absorption. The strongest direct human topical evidence among the declared actives concerns capsaicin, supported by a 2024 meta-analysis of eight randomised trials in osteoarthritis. Menthol has a well-characterised TRPM8-mediated cooling mechanism and controlled human evidence for acute pain-threshold modulation, delayed-onset muscle soreness and post-race soreness, although results are formulation- and condition-dependent. Topical curcumin has an emerging human evidence base in knee osteoarthritis, including randomised placebo-controlled trials and a 2026 double-blind trial of the VAS-101 gel. Ginger has limited and heterogeneous topical clinical evidence, while camphor and piperine are supported mainly by mechanistic or formulation studies. Experimental studies showing menthol- and piperine-associated enhancement of curcumin skin permeation provide a plausible delivery rationale but do not verify the absorption of the finished product. Overall, the literature supports a biologically coherent multi-target rationale for temporary musculoskeletal comfort. It does not establish the onset, magnitude, duration, absorption or clinical efficacy of the Biosolace finished formulation, because quantitative ingredient standardisation and product-specific controlled trials were not identified.

Keywords: topical analgesic; musculoskeletal pain; menthol; camphor; capsaicin; curcumin; ginger; piperine; TRPM8; TRPV1; osteoarthritis; delayed-onset muscle soreness.

Abbreviations: CI, confidence interval; COX-2, cyclooxygenase-2; DOMS, delayed-onset muscle soreness; DRG, dorsal root ganglion; EC_{50}, half-maximal effective concentration; HEK293, human embryonic kidney 293 cells; IL, interleukin (IL-1β, interleukin-1β; IL-6, interleukin-6; IL-8, interleukin-8);

iNOS, inducible nitric oxide synthase; IκBα, inhibitor of nuclear factor kappa B alpha; KOOS, Knee injury and Osteoarthritis Outcome Score; MMP, matrix metalloproteinase; NF-κB, nuclear factor kappa B; NNH, number needed to harm; NNT, number needed to treat; NO, nitric oxide; OA, osteoarthritis; PGE_2, prostaglandin E_2; PPT, pressure-pain threshold; q.s., quantum satis (sufficient quantity); RCT, randomized controlled trial; ROS, reactive oxygen species; SMD, standardized mean difference; TNF-α, tumour necrosis factor α; TRP, transient receptor potential; TRPA1, transient receptor potential ankyrin 1; TRPM8, transient receptor potential melastatin 8; TRPV1, transient receptor potential vanilloid 1; TRPV3, transient receptor potential vanilloid 3; VAS, visual analogue scale; WOMAC, Western Ontario and McMaster Universities Osteoarthritis Index.

1. Introduction

Musculoskeletal pain is not a single biological entity. Localised discomfort may arise after unaccustomed exercise, minor soft-tissue strain, mechanical overuse, degenerative joint disease or inflammatory sensitisation. The initiating pathology differs, yet many symptomatic states share activation or sensitisation of peripheral nociceptors by mechanical stress, protons, prostaglandins, cytokines, neuropeptides and oxidative mediators. Osteoarthritis, for example, is now understood as a whole-joint disorder in which inflammatory mediators generated by synovium, cartilage, subchondral bone and periarticular tissues influence pain processing as well as tissue catabolism [1]. Ion channels expressed by sensory neurons, including members of the TRP family, translate chemical and thermal stimuli into nociceptive signals and are therefore relevant targets for topical counterirritants [2,3].

Topical pain management is attractive when symptoms are anatomically localised because it can deliver sensory or pharmacological effects at the site of application while reducing systemic exposure compared with many oral therapies. Contemporary analyses distinguish topical non-steroidal anti-inflammatory drugs, local anaesthetics, capsaicin and counterirritants such as menthol and camphor because their mechanisms, evidence bases and optimal clinical contexts differ [4,5]. Cochrane-level evidence shows that some topical analgesic classes are effective for selected acute and chronic pain conditions, but efficacy cannot be generalised to all preparations merely because they are applied to the skin [5]. Vehicle composition, concentration, dose per unit area, duration of exposure and the clinical pain model all influence outcome.

Herbal and counterirritant topical products occupy an additional evidence problem: a finished cream may contain several bioactives, while published studies typically evaluate a purified constituent, a standardised extract or a different combination. Systematic reviews of topical herbal therapies for osteoarthritis have repeatedly emphasised heterogeneity in plant material, formulation and study quality [6,7]. A scientifically responsible product-oriented review therefore has to distinguish four levels of inference: molecular mechanism, preclinical activity, human evidence for individual actives or related formulations, and direct evidence for the marketed product itself.

Biosolace Pain Relief Cream is publicly described as containing menthol, camphor, ginger, black pepper, turmeric and *Capsicum* in a cream base. Public materials position the product for soothing muscle and joint comfort, cooling sensation, use after physical activity and easy or rapid topical application. This review examines how far those positioning statements can be supported by the literature without converting ingredient-level evidence into claims of finished-product efficacy. Particular attention is given to menthol/TRPM8, capsaicin/TRPV1, camphor-sensitive TRP channels, topical curcumin, ginger, piperine, cutaneous delivery and evidence relevant to muscle soreness and joint discomfort.

2. Narrative review methodology and evidence verification

This article was developed as a critical narrative review rather than a systematic review or meta-analysis. Searches were performed through August 2026 with PubMed as the principal biomedical index, supplemented by publisher records, DOI cross-checking, publicly accessible full-text sources and backward reference checking from systematic reviews and pivotal trials. Search concepts included topical analgesic, musculoskeletal pain, osteoarthritis, muscle strain, delayed-onset muscle soreness, menthol, TRPM8, camphor, TRPV1, TRPV3, TRPA1, capsaicin, *Capsicum*, topical curcumin, turmeric, ginger, *Zingiber officinale*, piperine, *Piper nigrum*, skin permeation and penetration enhancer. Recent systematic

reviews, meta-analyses and controlled human studies were prioritised; older mechanistic or clinical studies were retained when they established foundational receptor pharmacology or remained directly relevant [5,6].

References were cross-checked against PubMed and/or publisher metadata for authorship, year, journal and DOI where available, including the 2026 randomised trial of the VAS-101 topical curcumin formulation and a 2024 controlled study of post-race topical menthol [8,9]. Product composition and current positioning statements were derived from current product packaging and manufacturer-provided/public product information; these commercial sources are treated as product documentation and are not included in the numbered scientific reference list. No unpublished manufacturing specification, certificate of analysis or proprietary formulation dossier was available to this review. Consequently, concentrations and standardisation data that could not be verified publicly are reported as unavailable rather than inferred.

The evidence appraisal is intentionally asymmetric. Greater weight is given to systematic reviews, meta-analyses and randomised controlled trials; small uncontrolled topical studies are treated as exploratory; mechanistic receptor studies are used to explain plausibility rather than clinical efficacy; and formulation or permeation experiments are not interpreted as patient outcomes. This approach is particularly important for a multi-component cream because a plausible mechanism for each ingredient does not demonstrate that the combination is additive or synergistic.

3. Biosolace formulation and ingredient profile

The publicly declared formulation contains six named active botanical or counterirritant ingredients plus a cream base. Public sources do not identify the concentration or stereo chemical form of menthol, the concentration or grade of camphor, the ginger plant part or gingerol/shogaol standardisation, the *Piper nigrum* extract specification or piperine content, the *Curcuma longa* extract specification or curcuminoid content, the *Capsicum* species or capsaicinoid concentration, or the detailed vehicle composition. These omissions matter because counterirritant effects can be sharply concentration-dependent and because plant-extract chemistry varies with species, plant part, solvent and standardisation [10]. Table 1 maps each declared ingredient to its principal pharmacology and the evidential weight it can currently carry.

Product image. Biosolace Pain Relief Cream and current packaging supplied for manuscript illustration.

Table 1. Declared Biosolace ingredients and the principal evidence domains relevant to each. Constituents listed for botanicals are literature-associated compounds and do not represent measured Biosolace standardisation; no ingredient concentration, extract ratio, excipient, pH or emulsion type is publicly disclosed.

Ingredient	Relevant constituents	Principal target/process	Most defensible role	Evidence emphasis

Menthol	Menthol; stereochemistry undisclosed	TRPM8; additional ion-channel effects at higher concentrations	Cooling and acute sensory analgesia	Moderate-to-strong human topical + mechanistic[11,12]
Camphor (*Karpura*)	Camphor	TRPV1, TRPV3 and TRPA1 modulation; additional experimental TRPM8 activation/sensitization	Counterirritant sensory modulation	Strong mechanistic; limited standalone clinical[13,14]
Ginger (*Shunthi*)	Gingerols, shogaols	TRPV1-related and inflammatory signalling	Supportive sensory/inflammatory rationale	Limited, heterogeneous topical human evidence[15,16]
Black pepper (*Marica*)	Piperine	TRPV1; NF-κB/COX-2/PGE_2/iNOS/MMP modulation; permeation effects	Mechanistic inflammatory modulation and possible delivery support	Cellular + formulation evidence[17–19]
Turmeric (*Haridra*)	Curcumin/curcuminoids	NF-κB, COX-2/PGE_2, cytokines, redox pathways	Local inflammatory-balance rationale	Emerging randomised human topical evidence[8,20]
Capsicum	Capsaicin/capsaicinoids; concentration undisclosed	TRPV1 activation and repeated-exposure defunctionalization	Localised nociceptor modulation	Strongest direct human topical evidence[10]
Cream base q.s.	Vehicle not publicly characterised	Release, partition, skin residence, sensory properties	Delivery of actives	Product-specific data required

4. Biological basis: peripheral nociception, counterirritation and TRP channels

Peripheral nociceptors terminate in epidermal and superficial dermal tissues, placing them within reach of compounds released from topical preparations. Inflammatory sensitisation lowers activation thresholds through mediators such as PGE_2, bradykinin, cytokines, protons and ROS, and ion-channel activity interfaces with these mediators in osteoarthritis and related musculoskeletal disorders rather than operating as an isolated receptor system[1,2]. TRPV1, TRPA1, and related channels detect heat, irritants, and inflammatory signals, whereas TRPM8 detects cool temperatures and cooling compounds [21,22].

Counterirritation describes the deliberate production of a mild sensory stimulus that changes the perception or transmission of underlying discomfort [4]. Menthol and capsaicin illustrate two different versions of this concept. Menthol predominantly activates TRPM8 to produce cooling; capsaicin strongly activates TRPV1 and, with sufficient repeated or sustained exposure, renders nociceptive terminals less responsive [3,11]. Camphor interacts with several thermoTRP channels: it activates and strongly desensitises TRPV1, inhibits TRPA1 and can engage TRPV3; separate experimental work also demonstrates activation and sensitisation of TRPM8 to cooling [13,14]. These mechanisms can alter perceived pain even when the underlying tissue pathology is unchanged, which is why symptomatic language such as temporary comfort is more defensible than claims of tissue healing.

The biological rationale for Biosolace is therefore best described as a layered sensory-inflammatory model: rapid sensory input from menthol and camphor; TRPV1-directed counter irritation from *Capsicum*; and additional botanical constituents with experimental inflammatory or redox effects. Figure 1 summarises the principal cutaneous pathways without implying that receptor occupancy or tissue concentrations have been measured for the product.

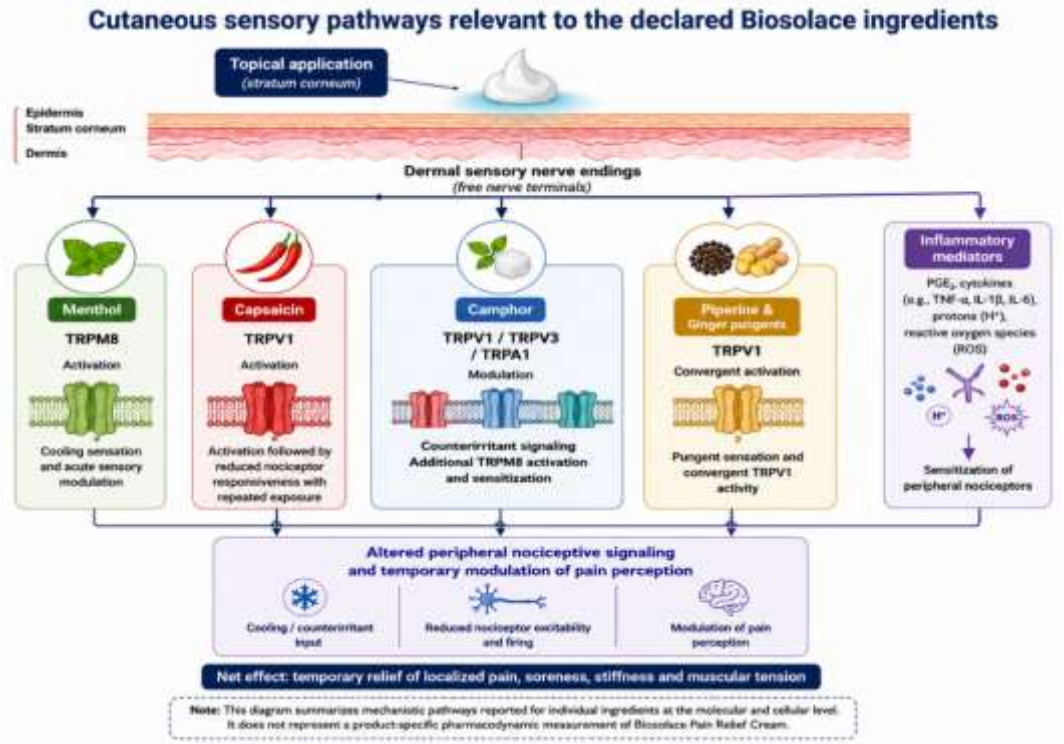

Fig 1: Cutaneous sensory pathways relevant to the declared Biosolace ingredients. Menthol is linked principally to TRPM8; capsaicin to TRPV1 activation followed by reduced nociceptor responsiveness with repeated exposure; and camphor to TRPV1/TRPV3/TRPA1 modulation, with additional experimental evidence for TRPM8 activation and sensitisation. Piperine and ginger pungents contribute convergent TRPV1-related signalling. Inflammatory mediators provide a parallel route to nociceptor sensitisation. The scheme summarises ingredient-level mechanisms rather than a product-specific pharmacodynamic measurement [11,13,14,17].

5. Menthol: TRPM8-mediated cooling and acute sensory analgesia

Menthol provides the clearest mechanistic basis for a cooling claim. Reviews of topical menthol identify TRPM8 as its principal cold-sensing target and describe concentration-dependent counterirritant and vasoactive effects [12,23]. In a foundational mechanistic study, genetic deletion or pharmacological inhibition of TRPM8 abolished L-menthol analgesia in models of acute and inflammatory pain, supporting a causal role for the channel [11]. These experiments were preclinical and therefore do not establish a clinical effect size, but they provide unusually strong target-level plausibility. TRPM8 now has a well-defined role as the primary mammalian cool sensor, which underpins the translation of menthol-evoked cooling to analgesic perception [22,24].

Human studies support an acute sensory effect of topical menthol, but the magnitude varies by formulation and pain model. In a randomised study, pressure-pain threshold increased within approximately 15 minutes after application of a menthol-based topical analgesic, with comparable responses across tested upper- and lower-body sites [25]. In a double-blind crossover study of knee osteoarthritis, pain declined during several functional tasks within the menthol condition, although superiority over placebo was not consistently demonstrated [26]. Together, these findings support rapid sensory modulation while also showing that a cooling response or within-group improvement does not by itself establish a treatment effect over vehicle.

Exercise-associated soreness provides a more direct bridge to post-activity positioning. In an experimental DOMS study, participants treated with 3.5% topical menthol reported less discomfort than those treated with ice, and electrically evoked tetanic force was higher under the menthol condition [23,27]. A subsequent study linked menthol treatment during DOMS with changes in pain and corticospinal inhibition, supporting a predominantly neurosensory interpretation rather than a demonstrated acceleration of tissue repair [28]. In 2024, 4% topical menthol used after distance racing attenuated the rise in muscle soreness and reduced perceived exertion during later running [9]. These studies were small and formulation-specific. A separate placebo-controlled muscle-damage study found that soreness improvement was largely explained by the placebo gel, although menthol appeared to better preserve lower-body power [29].

Not all menthol-containing combinations are superior. A comparison of isolated menthol with menthol plus capsaicin during exercise in the heat found no clear additive perceptual advantage from the combination [30]. A bibliometric review of camphor- and menthol-containing agents similarly concluded that human evidence is promising but limited and heterogeneous [31]. In a randomised double-blind comparison, two mentholated creams produced improvement over seven days in acute musculoskeletal pain, with greater improvement when oxygenated glycerol triesters were added; because both arms contained menthol, the trial cannot isolate a placebo-adjusted menthol effect [32].

A larger multicenter study randomised 208 adults with mild-to-moderate muscle strain to a patch containing 10% methyl salicylate plus 3% L-menthol or placebo; the active patch produced significantly greater movement-related pain relief over eight hours[33]. This supports the clinical utility of a menthol-containing counterirritant patch, but methyl salicylate was a co-active. Conversely, 385 participants with acute ankle sprain randomised to 1% diclofenac/3% menthol, diclofenac alone, menthol alone or placebo showed no significant advantage of the combination for the primary or secondary pain outcomes [34]. Together, these trials demonstrate a core principle for Biosolace: efficacy depends on concentration, vehicle, co-actives and pain model, and cannot be inferred simply from the presence of menthol.

Menthol is also vasoactive. Topical 5% menthol gel increased cutaneous blood flow at the treated site and modestly across the same dermatome without a systemic response [35], an observation consistent with early placebo-controlled data showing menthol-induced increases in cutaneous perfusion [36]. Such vascular effects may contribute to local sensory experience, but they should not be described as clinically meaningful "improved circulation" in the absence of product-specific evidence. For Biosolace, menthol most defensibly supports cooling sensation, acute sensory modulation and a limited ingredient-level rationale for post-exercise soreness.

6. Camphor: multi-TRP counterirritant pharmacology

Camphor is a classical component of topical balms and liniments. Experimental receptor studies show that camphor activates and rapidly desensitises TRPV1, can engage TRPV3, and inhibits TRPA1[13]. Separate work demonstrates activation and sensitisation of TRPM8 to cooling [14]. This multi-channel profile provides a plausible basis for the mixed cooling, warming and counterirritant sensations associated with camphor-containing preparations, while remaining distinct from menthol's predominantly TRPM8-centered pharmacology.

Clinical attribution to camphor alone remains limited because most human studies use multicomponent preparations. A review of camphor- and menthol-containing topical agents found potentially useful clinical signals but insufficient evidence for definitive recommendations [31]. Observational data from a patch containing methyl salicylate, menthol and camphor reported improvements in pain and function, but the contribution of any single active cannot be isolated [37]. In a small human study of nine adults, topical camphor at 5%–20% produced an early, concentration-related cooling sensation that subsequently shifted toward warmth, illustrating dose-sensitive sensory effects rather than standalone analgesic efficacy [38].

Thus camphor should be positioned as a mechanistically credible sensory co-active rather than a primary clinical-evidence anchor. It can strengthen the concept of a cooling/warming counterirritant architecture, but statements implying a quantified analgesic contribution are not supportable from the available evidence.

7. Capsicum and capsaicin: the strongest direct topical analgesic evidence

Capsaicin is the pungent vanilloid most strongly associated with *Capsicum* analgesic preparations. It binds TRPV1 on nociceptive fibres, initially causing depolarisation, burning and neuropeptide release; repeated or sufficiently intense exposure produces reduced responsiveness and functional defunctionalisation of peripheral nociceptor terminals [3,4]. This is a direct sensory-neuronal mechanism rather than a conventional cyclooxygenase-inhibition mechanism.

The clinical evidence for topical capsaicin is substantially larger than for the other declared botanicals. A 2024 systematic review and meta-analysis of eight double-blind osteoarthritis trials (498 participants) favoured topical capsaicin for pain reduction (SMD −0.84; 95% CI −1.48 to −0.19), although heterogeneity was substantial, certainty was low to very low, and local burning occurred more frequently with active treatment[10]. Evidence from neuropathic-pain studies further shows that dose and delivery strategy materially influence outcome. A later Cochrane review of repeated low-concentration (<1%) capsaicin regimens judged the available data insufficient for reliable conclusions about clinically meaningful efficacy in chronic neuropathic pain[39], whereas a separate Cochrane review found modest benefit from the 8% capsaicin patch in selected peripheral neuropathic conditions[40]. An earlier systematic review pooling chronic musculoskeletal and neuropathic pain studies also suggested a modest analgesic effect accompanied by frequent local adverse reactions [41]. In chronic nonspecific low back pain, a randomised placebo-controlled trial of a capsicum plaster found greater short-term improvement after three weeks than placebo, again underscoring formulation-specific rather than class-wide efficacy [42].

Individual trials provide additional context. In a 12-week multicenter study of 113 patients with osteoarthritis, 0.025% capsaicin cream outperformed vehicle on pain outcomes; physician global improvement was reported in 81% of capsaicin-treated participants versus 54% of controls, while early local burning was common[43]. A separate double-blind arthritis trial likewise favoured repeated topical capsaicin over vehicle [44]. Network meta-analysis found no statistically significant difference between topical NSAIDs and capsaicin when licensed doses were compared indirectly in osteoarthritis [45]. In a series of n-of-1 trials, overall mean pain reduction did not differ significantly between capsaicin and ibuprofen gel, although individual treatment preference varied substantially [46]. High-concentration capsaicin exposure has also been associated with reduced heat sensitivity and diminished epidermal nerve-fibre immunostaining in human skin, supporting peripheral nociceptor defunctionalisation as a biological mechanism [47].

For Biosolace, *Capsicum* provides the strongest ingredient-level evidence for topical analgesia. However, "Capsicum" on a label is not equivalent to a known capsaicin dose. Species, extract type and capsaicinoid standardisation determine receptor exposure and tolerability, so the product cannot be assumed to reproduce the effect sizes seen with 0.025%, 0.075% or other standardised capsaicin preparations unless its capsaicin content is analytically established.

8. Turmeric and curcumin: mechanisms and emerging topical evidence

Turmeric introduces a different pharmacological dimension. Curcumin and related curcuminoids have been studied across inflammatory and redox pathways relevant to musculoskeletal disease, including NF-κB, COX-2/PGE_2, pro-inflammatory cytokines, iNOS and matrix-degrading enzymes [48,49]. In chondrocyte models, curcumin attenuates IL-1β-driven inflammatory and catabolic signalling, including MMP expression and NF-κB pathway activation [50,51]. These findings provide mechanistic plausibility, but most experiments use purified curcumin at concentrations that may not be reproduced in skin or periarticular tissues after application of an unspecified turmeric ingredient [52]. For a topical product, the human topical evidence is therefore more directly informative than the much larger oral-curcumin literature [53].

Jamali and colleagues randomised 72 older adults with knee osteoarthritis to 5% curcumin ointment or Vaseline twice daily for six weeks. By the final assessment, pain was lower with curcumin, and the mixed-model estimate corresponded to an approximately 11.3-mm advantage on a 100-mm VAS relative to placebo [20]. This provides direct placebo-controlled evidence for a defined 5% curcumin ointment, but not for an unspecified turmeric concentration in a multicomponent cream.

Azadbakht and colleagues studied 60 patients with knee osteoarthritis using 10% topical curcumin versus 1% diclofenac; both groups improved on VAS and WOMAC over the short treatment period [54].

Because there was no inert placebo and the study was not designed as a rigorous equivalence trial, the results should not be interpreted as proof that curcumin is equivalent to diclofenac. Nevertheless, the trial contributes human evidence that a sufficiently concentrated topical curcumin formulation can be clinically active.

Baharizade and colleagues evaluated a self-nano-emulsifying polyethene glycol organogel designed to improve topical curcumin delivery [55]. The optimised system showed sustained release and measurable skin permeation, and its clinical arm reported greater eight-week improvements in pain, stiffness and physical-function difficulty than placebo. Because the intervention used a specialised nano-emulsifying organogel, the study is particularly informative about the importance of delivery technology and should not be treated as directly interchangeable with a conventional cream.

In 2026, Lopresti, Antony and Smith randomised 60 adults aged 45–75 years with knee osteoarthritis to VAS-101 topical curcumin gel or placebo every second day for 28 days. KOOS pain favoured the active formulation by 5.12 points (95% CI 0.47–9.77; d = 0.62), and daily pain ratings also improved, while several other KOOS and performance outcomes did not differ significantly between groups; temporary skin staining was reported [8]. The result strengthens the topical-curcumin evidence base while remaining specific to the tested VAS-101 formulation.

The academically defensible role of turmeric in Biosolace is therefore an inflammatory/redox-modulating rationale supported by an increasingly credible topical-curcumin clinical literature. The key translational unknown is dose: without curcuminoid standardisation and release data, the exposure achieved by the marketed cream remains unknown.

9. Ginger: plausible biology, limited topical evidence

Ginger contains pungent constituents such as gingerols and shogaols that interact with sensory and inflammatory pathways relevant to pain. In preclinical work, 8-shogaol produced analgesic effects through TRPV1-related mechanisms in acute and inflammatory pain models [16]. Earlier electrophysiological research also showed that zingerone can activate vanilloid-sensitive sensory neurons, with response kinetics distinct from those of capsaicin and piperine [56]. These findings support mechanistic plausibility but do not establish clinical efficacy after topical ginger application.

The human topical evidence for ginger remains limited. A systematic review identified only a small number of controlled topical studies and highlighted important methodological weaknesses [15]. A later systematic review and meta-analysis found that pooled topical ginger comparisons did not produce statistically significant improvements in pain or knee function versus standard treatment, and the overall evidence was heterogeneous and of poor methodological quality[57]. Oral ginger has a separate clinical literature [58–60], but those findings are not used here to substantiate dermal delivery or topical efficacy.

Tosun and colleagues evaluated self-knee massage with ginger oil in 68 patients receiving standard osteoarthritis treatment; the intervention group had lower VAS pain and better WOMAC outcomes at follow-up [61]. However, ginger oil, massage, attention and standard therapy were bundled, so the independent pharmacological effect of ginger cannot be isolated. Therkleson reported improvements in pain, fatigue and function in 20 adults using ginger compresses or patches, but the study was small and lacked the rigour of a placebo-controlled efficacy trial [62]. Niempoog and colleagues compared a combined ginger/plai gel with diclofenac in knee osteoarthritis and found improvement in both groups; because the herbal arm contained two *Zingiber* species and no inert placebo, it does not establish standalone topical ginger efficacy [63].

Ginger should therefore be presented as a supportive botanical rather than a primary clinical-evidence driver. The formulation rationale is plausible through pungent TRP-channel and inflammatory signalling, but claims of analgesic efficacy for ginger itself should remain qualified.

10. Black pepper and piperine: TRPV1, inflammatory signalling and delivery science

Piperine, the principal pungent alkaloid associated with black pepper, is relevant to both sensory pharmacology and formulation science. In IL-1β-stimulated human osteoarthritic chondrocytes, piperine shifted the cellular response toward a less inflammatory and less catabolic profile, with lower prostaglandin and nitric oxide output, reduced expression of several matrix-degrading and inflammatory

mediators, and attenuation of NF-κB signalling [18]. A separate study in human fibroblast-like synoviocytes found concentration-dependent suppression of inflammatory outputs including IL-6, PGE_2, COX-2 and MMP-13[19]. These cell-level findings do not demonstrate that topical black pepper produces comparable tissue concentrations in vivo.

Electrophysiological studies of human TRPV1 indicate that piperine is a genuine receptor agonist, but it requires substantially higher concentrations than capsaicin to reach half-maximal activation. Under the experimental conditions used, piperine produced a larger maximal current response and receptor responsiveness declined more rapidly during sustained or repeated exposure [17]. This establishes convergent TRPV1 biology without implying that piperine is clinically more potent or more effective than capsaicin; exposure and clinical evidence remain very different[64,65].

Piperine may also modify the cutaneous delivery of co-formulated phytochemicals. In an experimental cellulose-composite membrane, curcumin transport increased as piperine loading increased; at the highest tested piperine level, the permeation rate was approximately 1.9-fold that of the piperine-free comparator[66]. Separate vesicular and nanocarrier studies likewise support the possibility that co-formulation with piperine can alter curcumin delivery through skin models [67,68]. These systems are not equivalent to the marketed cream, so the data support a formulation hypothesis rather than evidence of Biosolace absorption.

11. Integrated multi-target rationale: complementarity without assuming synergy

When the ingredient evidence is integrated, Biosolace can be conceptualised as a multi-target topical rather than as six independent analgesics. Menthol contributes rapid cooling and TRPM8 signalling; *Capsicum* provides the strongest route to repeated-exposure TRPV1 analgesia; camphor broadens counterirritant signalling across TRPV1/TRPV3/TRPA1; curcuminoids contribute an inflammatory and redox rationale with emerging topical human evidence; ginger adds pungent TRP-related and inflammatory mechanisms; and piperine adds TRPV1 activity, human-cell anti-inflammatory evidence and a possible delivery-enhancing role[10,11,13,18,20].

The term synergy should nevertheless be avoided. Pharmacological synergy requires evidence that the combined effect exceeds an expected additive effect at defined doses. No factorial or isobolographic study was identified for the finished Biosolace formulation; indeed, the available menthol/capsaicin exercise study found no advantage of the combination over isolated menthol for several perceptual outcomes[30], and the diclofenac/menthol ankle-sprain trial showed that adding menthol to another active did not necessarily improve clinical analgesia[34]. These negative or neutral findings do not argue against Biosolace; they demonstrate why product-specific testing is necessary. Table 2 summarises the mechanistic evidence for each declared ingredient.

Table 2. Mechanistic evidence relevant to the declared Biosolace actives. All entries are cell-, tissue- or animal-level unless stated otherwise; efficacy at receptor level does not quantify clinical effect.

Ingredient	Key mechanistic finding	Model/system	Reference
Menthol	TRPM8 deletion/blockade abolishes L-menthol analgesia in acute and inflammatory pain	Mouse, genetic/pharmacological	[11]
Menthol	Topical application increases cutaneous blood flow across the dermatome	Human crossover (5% gel)	[35]
Camphor	Activates/desensitizes TRPV1, inhibits TRPA1 and engages TRPV3	Heterologous expression/DRG neurons	[13]
Camphor	Activates and sensitises TRPM8 to cooling	HEK293/rDRG neurons	[14]
Capsaicin	TRPV1 activation followed by desensitization/defunctionalization; reduced epidermal nerve-fibre immunostaining after high-concentration patch	Human skin/histology; receptor studies	[3,47]

Piperine	TRPV1 agonism with a larger maximal current under the experimental conditions and rapid desensitisation during sustained/repeated exposure	HEK293 whole-cell patch clamp	[17]
Piperine	Inhibits IL-1β-induced PGE_2, IL-6, COX-2, MMP-13 in human synoviocytes	Human cells	[19]
Piperine	Inhibits IL-1β-induced PGE_2/NO, MMP-3/13, iNOS/COX-2, NF-κB in human OA chondrocytes	Human chondrocytes	[18]
Ginger (8-shogaol)	Targets TRPV1; analgesic in acute and inflammatory pain models	Rodent models	[16]
Ginger (zingerone)	Vanilloid-receptor agonist with distinct kinetics	Rat trigeminal ganglion cells	[56]
Curcumin	Suppresses NF-κB activation, COX-2, PGE_2, IL-1β/TNF-α/IL-6, MMPs in chondrocytes	Human/rodent chondrocytes	[48–50]

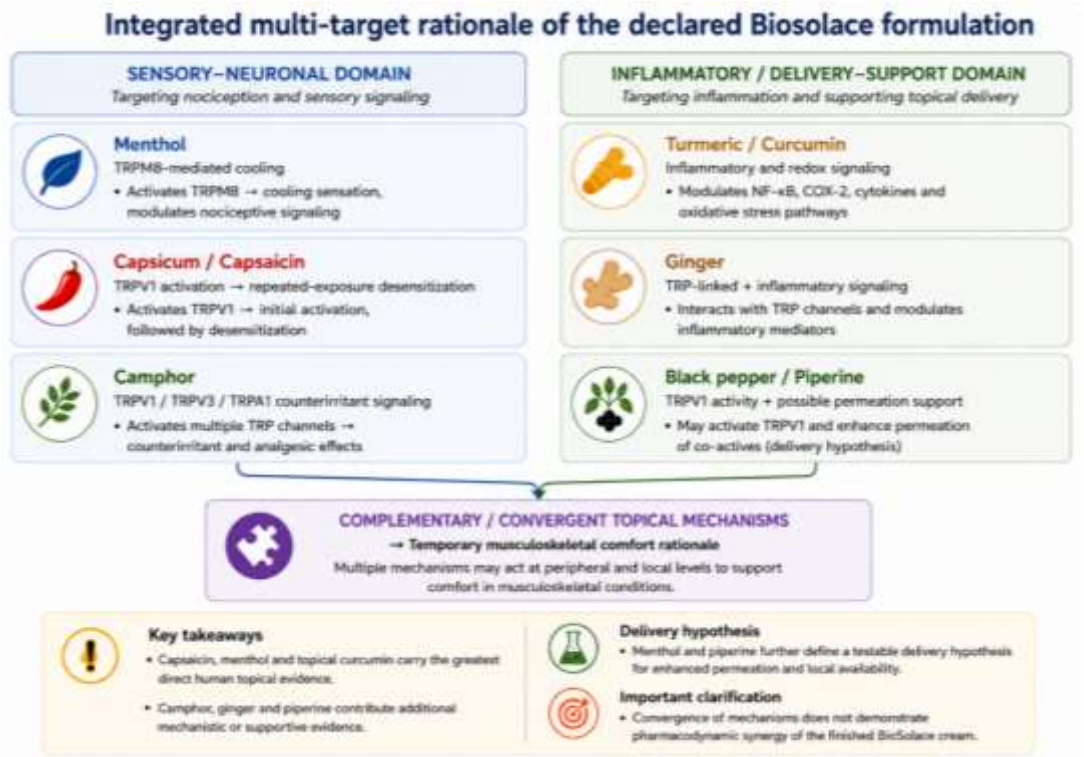

Fig 2: Integrated multi-target rationale for the declared Biosolace formulation. Capsaicin, menthol and topical curcumin carry the greatest direct human topical evidence; camphor, ginger and piperine contribute additional mechanistic or supportive evidence; menthol and piperine further define a testable delivery hypothesis. Convergence does not demonstrate pharmacodynamic synergy of the finished cream [8,10,11,20].

12. Formulation science: skin barrier, vehicle and permeation interactions

Topical pharmacology is inseparable from formulation science. An active compound must be released from the vehicle, partition into the stratum corneum, diffuse through appropriate skin layers and remain sufficiently available near sensory endings or superficial tissues. Lipophilic phytochemicals such as curcumin and piperine can be limited by poor aqueous solubility, crystallisation, inadequate thermodynamic activity or strong retention in the vehicle, and the choice of carrier system materially changes dermal penetration[69]. This is one reason clinical data from a patch, ointment, nano-emulgel or organogel cannot be transferred quantitatively to a conventional cream [5,55].

Menthol is widely investigated as a terpene-type penetration enhancer. In topical curcumin gels containing 0–12.5% menthol, addition of menthol increased curcumin flux across excised rat epidermis; the same formulations were also evaluated in a carrageenan paw-oedema model [70]. Piperine enhanced curcumin transport in a different experimental membrane system [66], while newer nanocarrier studies show that curcumin-piperine co-delivery can modify cutaneous penetration [67,68]. These observations make the co-presence of menthol, black pepper and turmeric a reasonable formulation-science hypothesis, but they do not establish the magnitude of delivery from Biosolace.

The translational boundary is important. No peer-reviewed Biosolace-specific in vitro release study, Franz-cell permeation experiment, tape-stripping analysis, dermal-retention assessment, rheological characterisation, or human pharmacokinetic study was identified in the sources reviewed. Experimental permeation enhancement by menthol or piperine therefore cannot validate the commercial descriptor "quick absorption" or establish dermal bioavailability. Perceived dry-down and molecular delivery are different properties: a cream may disappear quickly from the skin surface yet deliver little active, or remain emollient while sustaining local release. Product substantiation would require direct release, permeation/retention, rheology, spreadability and dry-down measurements, with blinded onset testing if a time-to-effect claim is intended.

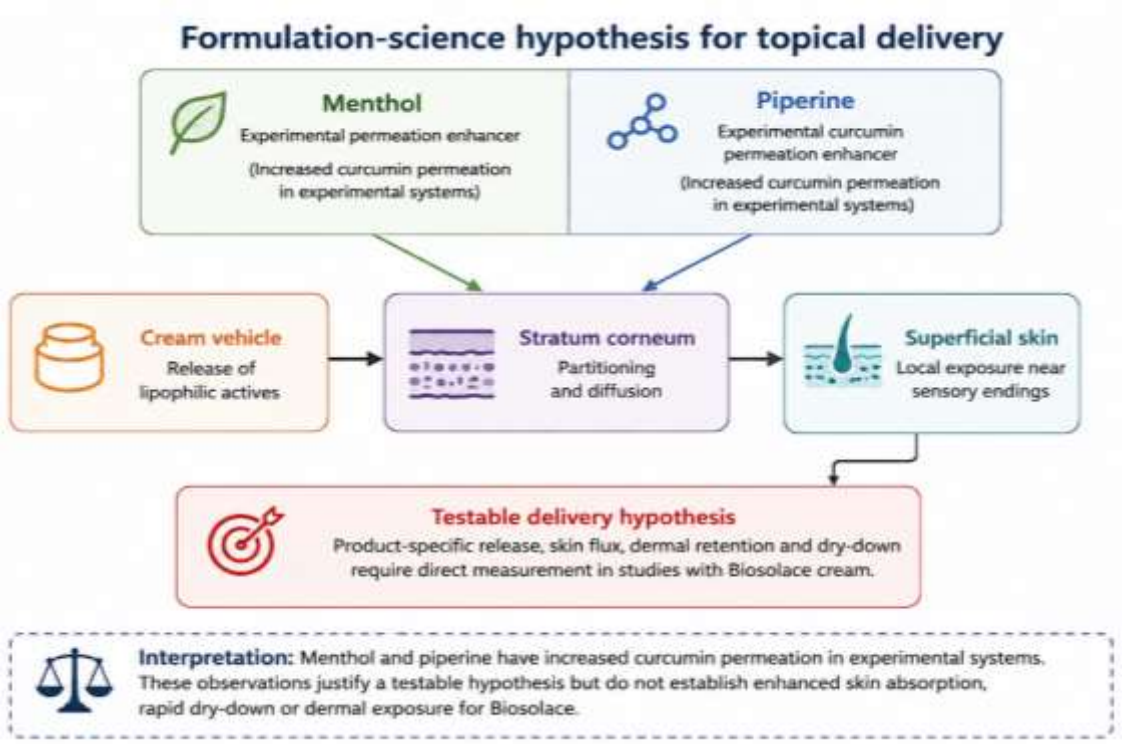

Fig 3: Formulation-science hypothesis for topical delivery. Menthol and piperine have increased curcumin permeation in experimental systems [66,70]. These observations justify a testable hypothesis but do not establish enhanced skin absorption, rapid dry-down or dermal exposure for Biosolace [67,68].

13. Human clinical evidence most relevant to Biosolace positioning

Table 3 brings together the most decision-relevant human studies rather than attempting to list every publication. The pattern is clear: evidence is strongest for standardised capsaicin; menthol studies directly address acute sensory pain and exercise soreness but are small or combination-dependent; topical curcumin evidence has strengthened with modern placebo-controlled trials; and ginger evidence remains difficult to separate from massage, co-extracts and weak study design. No human trial in the table evaluates the exact Biosolace formulation.

Table 3. Selected human clinical evidence most relevant to Biosolace claims. Study products, doses and vehicles differ from Biosolace; the table is intended for evidence mapping rather than direct efficacy extrapolation.

Study/design	Condition	Topical intervention	Main finding	Interpretive limitation
Higashi et al., 2010; RCT, n=208[33]	Mild-moderate muscle strain	10% methyl salicylate + 3% L-menthol patch vs placebo	Movement-related SPID8 favoured the active patch (182.6 vs 130.1; P = 0.005)	Supports a menthol-containing counterirritant patch, not menthol alone
Taylor et al., 2012; double-blind, n=66[32]	Acute musculoskeletal pain	Mentholated cream vs mentholated cream + oxygenated glycerol triesters	Both improved; combination greater by day 8	No inert placebo; menthol contribution not isolated
Topp et al., 2013; crossover, n=20[26]	Knee OA	3.5% menthol gel vs placebo	Within-menthol pain reductions; limited between-treatment superiority	Small sample; important counterexample to overclaiming
Johar et al., 2012; randomized, n=16[23,27]	DOMS	3.5% menthol vs ice	Lower perceived discomfort; greater tetanic force	Small experimental study
Hill & Sumida, 2002; randomized, n=18/group[71]	DOMS	Menthol/methyl salicylate vs capsaicin vs placebo cream	Menthol/salicylate cream reduced DOMS pain (VAS 2.79 → 1.22); capsaicin group unchanged	Single application; counterirritant-class contrast
Gillis et al., 2020; randomized, N=47 (18 control; 14 placebo; 15 menthol)[29]	Exercise-induced muscle damage	4.0% menthol vs placebo vs control	Soreness improvement was largely placebo-related; menthol better preserved vertical-jump performance.	Negative for a specific soreness effect; small groups
Fujii et al., 2024; crossover, n=11[9]	Post-race muscle soreness	4% menthol vs vehicle	Attenuated post-race soreness and perceived exertion	Small trained-athlete sample
Lai et al., 2017; RCT, n=385[34]	Acute ankle sprain	1% diclofenac/3% menthol; diclofenac; menthol; placebo	No significant advantage for combination; more skin events with combination	Shows formulation/context dependence
Altman et al., 1994; RCT, n=113[43]	Osteoarthritis	Capsaicin 0.025% cream vs vehicle, 12 weeks	Pain reduction at weeks 4, 8, 12; 81% vs 54% improved by physician global	Burning/stinging in 46%; vehicle-controlled monotherapy
Tshering et al., 2024; meta-analysis, 8 RCTs, n=498[10]	Osteoarthritis	Topical capsaicin vs placebo	Pain SMD −0.84; burning risk ratio 5.56	Low/very-low certainty; heterogeneous concentrations

Persson et al., 2018; network meta-analysis[45]	Osteoarthritis	Topical capsaicin vs topical NSAIDs	May be equally effective for pain relief	Evidence-level equivalence, not Biosolace equivalence
Jamali et al., 2020; placebo RCT, n=72[20]	Knee OA	5% curcumin ointment vs Vaseline	Approx. 11.33-mm treatment difference in VAS pain	Specific ointment and dose
Azadbakht et al., 2022; clinical trial, n=60[54]	Knee OA	10% curcumin vs 1% diclofenac	Both improved VAS/WOMAC	No inert placebo; equivalence not established
Baharizade et al., 2024; double-blind clinical study[55]	Knee OA	Self-nano-emulsifying curcumin organogel vs placebo	Large improvements in pain, stiffness and function	Delivery system highly formulation-specific
Lopresti et al., 2026; double-blind RCT, n=60[8]	Knee OA	VAS-101 topical curcumin vs placebo	KOOS pain beta 5.12; d = 0.62; daily pain improved	Small trial; formulation-specific
Tosun et al., 2017; controlled, n=68[61]	Knee OA	Ginger-oil self-massage + standard care vs standard care	Lower VAS pain and WOMAC/function	Massage and care confounding
Therkleson, 2014; pilot, n=20[62]	Chronic OA	Ginger compress or patch	Pain, fatigue and function improved	Small; no inert placebo efficacy comparison
Niempoog et al., 2012; double-blind RCT, n=100[63]	Knee OA	4% ginger + plai gel vs 1% diclofenac	Both groups improved KOOS	Combined herbal product; no placebo

14. Post-workout soreness and muscular tension

Post-activity soreness is one of the more directly supported positioning domains because several menthol studies used exercise-related pain models. Across the Johar DOMS experiment, the Hill and Sumida counterirritant comparison and the Fujii post-race study, menthol-containing topical treatments were associated with lower perceived soreness or discomfort in at least some outcomes, while neurophysiological work identified accompanying changes in corticospinal inhibition [9,27,28,71]. These findings support symptomatic sensory modulation rather than accelerated structural muscle recovery. The Gillis placebo-controlled study also demonstrates that improvement in soreness can be strongly influenced by placebo and protocol effects, and capsaicin did not improve DOMS pain in the Hill and Sumida model [29,71].

"Physical tension" and "tight muscles" are common consumer descriptors but are not standardised pharmacological endpoints. Some menthol trials measured mobility, soreness or pain, and counterirritant sensory input may make a tight or fatigued area feel more comfortable [26,33]. However, the literature reviewed does not establish reduction of muscle tone, spasticity or objective contracture. Accordingly, "helps ease post-workout soreness" is supportable as an ingredient-level rationale if it is not presented as clinically proven for Biosolace or as evidence of faster tissue recovery; "localised muscular discomfort after physical activity" is more scientifically precise than broad claims of recovery.

15. Joint pain and stiffness

Joint-discomfort support draws primarily from standardised capsaicin and topical-curcumin osteoarthritis studies. The 2024 capsaicin meta-analysis provides the strongest aggregate evidence for pain reduction,

and the curcumin trials add placebo-controlled evidence, with the 2024 organogel study capturing stiffness and function outcomes [8,10,20,55]. Trials of topical botanical mixtures for knee or hip discomfort illustrate both promise and the prominence of placebo effects in joint-pain settings [72]. Ginger shows some positive individual studies, but systematic review-level evidence remains uncertain, and oral ginger outcomes cannot be transferred to topical use [57,59]. These data support a rationale for temporary symptomatic joint comfort, especially in osteoarthritis-like pain models. They do not support disease-modifying language such as cartilage regeneration, reversal of osteoarthritis or treatment of inflammatory arthritis. Because Biosolace capsaicin and curcuminoid exposures are unknown, the manuscript should not imply that trial-level outcomes are expected from the finished cream.

16. Claim-by-claim evidence appraisal

The following appraisal applies the ingredient-level standard throughout. Each public positioning statement is judged by (i) the strength of relevant published evidence and (ii) whether the finished product can inherit that evidence.

Fast-acting cooling and soothing relief. Menthol provides a strong ingredient-level basis for cooling through TRPM8, and controlled human studies demonstrate sensory or pain-threshold effects on a short time scale after topical application [11,25,73]. The phrase "fast-acting," however, is a finished-product onset claim and requires a defined time-to-effect for the marketed formulation. A menthol-containing patch produced significant analgesic benefit during an eight-hour assessment, but that result cannot establish Biosolace onset [33]. The scientifically defensible interpretation is that relatively rapid sensory modulation is plausible because menthol is present, while Biosolace-specific onset remains unquantified. Camphor may add counterirritant sensory input, but current evidence does not support assigning equal analgesic contribution to menthol and camphor [13,31,38].

Sore muscles and post-workout soreness. Direct exercise-soreness studies support temporary reduction in soreness perception at ingredient level, with the caveats of small samples, placebo effects and formulation dependence noted above, and no evidence of enhanced muscle healing or recovery biology[9,27,29].

Stiff joints and joint discomfort. Capsaicin meta-analytic evidence and placebo-controlled topical curcumin trials support a rationale for temporary symptomatic joint comfort, not disease modification [8,10,20].

Localised physical or muscular tension. Reframe in scientific prose as "localised muscular discomfort or perceived tightness" unless objective muscle-tone data are generated; the reviewed literature does not establish reduction of muscle tone or spasticity.

Quick or rapid absorption. Public materials describe an easy-to-apply cream base and rapid absorption; these are formulation and sensory-use properties rather than pharmacodynamics analgesic claims, and no product-specific release, permeation or dry-down data were identified. This claim should therefore be classified as unverified within the peer-reviewed evidence base [66,70].

Table 4. Evidence-based appraisal of Biosolace positioning statements. Strength refers to the relevance of published ingredient-level evidence and should not be interpreted as a clinical efficacy grade for the finished product.

Claim domain	Best evidence	Strength/boundary	Recommended manuscript wording
Cooling sensation	Menthol/TRPM8; human sensory studies[11,25]	Strong ingredient-level rationale	"Menthol provides a mechanistic basis for cooling sensation."
Fast-acting soothing relief	Acute menthol PPT effects; menthol-containing patch evidence[33]	Moderate ingredient-level; product onset unknown	"Relatively rapid sensory modulation is plausible; Biosolace-specific onset has not been established."

Sore muscles / post-workout soreness	Menthol DOMS and post-race studies[9,27]	Moderate direct ingredient evidence	"Ingredient-level evidence supports temporary reduction in exercise-associated soreness perception."
Joint discomfort/stiffness	Capsaicin meta-analysis; topical curcumin RCTs[8,10]	Moderate-to-strong ingredient evidence	"Evidence supports a rationale for temporary symptomatic joint comfort, not disease modification."
Physical tension / tight muscles	Indirect pain/soreness/mobility outcomes[26,33]	Low-to-moderate, nonspecific endpoint	Prefer "localised muscular discomfort or perceived tightness."
Quick/rapid absorption	Public product description; no Biosolace release/permeation study located[70]	Unestablished in peer-reviewed product data	Treat as a formulation property requiring direct testing.
Multi-ingredient synergy	No Biosolace factorial trial; some combinations neutral[30,34]	Not established	Use "complementary" or "convergent" rather than "synergistic."

17. Safety and tolerability

Local sensory adverse effects are expected with counterirritants because the same receptor systems that modify pain can also generate burning, stinging or dysesthesia. In the 2024 osteoarthritis meta-analysis, application-site burning was more frequent with topical capsaicin than placebo (risk ratio 5.56; NNH approximately 3) [10]. In a 12-week capsaicin monotherapy trial, early burning or stinging was also common [43]. The low-concentration Cochrane review described frequent local reactions while concluding that efficacy data in chronic neuropathic pain were insufficient for reliable estimates[39]; high-concentration patch studies likewise report local treatment-site reactions despite modest benefit in selected neuropathic conditions[40]. Menthol can produce irritation or experimental cold hyperalgesia at higher concentrations, and combination trials show that adding sensory actives does not necessarily improve efficacy [34,36,73].

Camphor deserves separate attention because ingestion can cause serious toxicity, including seizures. An evidence-based poison-control guideline documents roughly 10,000 annual camphor ingestion exposures in US poison-centre data and recommends emergency referral for ingestion above 30 mg/kg, supportive care for convulsions, and, for asymptomatic topical exposures, simple skin washing with observation[74]. This supports strict external-use labelling and safe storage away from children; the ingestion literature should not be misread as evidence that ordinary intact-skin use at appropriate concentrations is systemically toxic — it is a route-of-exposure safety distinction.

Botanical ingredients can also cause irritant or allergic contact reactions in susceptible individuals. Because Biosolace concentrations and full excipients are not publicly characterised, a concentration-specific dermatological risk assessment cannot be performed. Practical risk minimisation is consistent with public directions: external use only, avoidance of eyes and mucous membranes, avoidance of broken or irritated skin, hand washing after use and discontinuation if significant irritation develops. Occlusion, heating pads or intensive external heat can alter exposure and should not be recommended unless specifically supported by tested product instructions.

18. Evidence hierarchy and translational boundaries

The principal scientific risk in a product-oriented narrative review is evidence slippage: a receptor study becomes a clinical claim, a purified constituent becomes a botanical extract, or a trial of one vehicle becomes proof for another. Figure 4 makes those boundaries explicit. Mechanistic evidence establishes plausibility; single-ingredient human trials establish that a pharmacological concept can work in a defined

formulation; related combination studies show that co-formulation can help, do nothing or increase adverse events; only product-specific testing can establish the magnitude and reproducibility of Biosolace outcomes.

No peer-reviewed randomised clinical trial, onset-of-action study, dose-response study or skin-permeation study of the finished Biosolace formulation was located in the sources reviewed. The available ingredient-level evidence provides a strong scientific foundation for the formulation, while direct product-specific studies would further define its clinical performance: evidence-supported rationale, not product-specific proof.

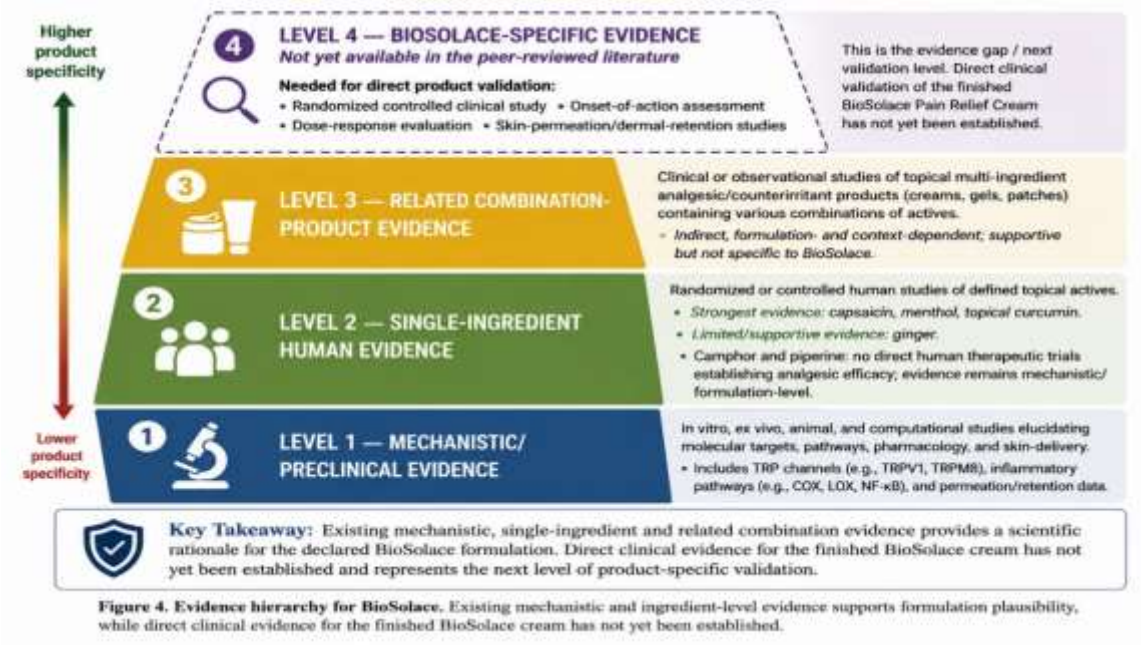

Fig 4: Evidence hierarchy for Biosolace. Existing mechanistic and ingredient-level evidence supports formulation plausibility, while direct clinical evidence for the finished BioSolace cream has not yet been established.

19. Limitations of the review and current evidence

- The review is narrative rather than systematic and therefore does not provide a preregistered search protocol, duplicate screening or formal risk-of-bias scoring.
- Biosolace quantitative composition, extract ratios and marker-compound standardisation were not publicly available to this review; menthol concentration and stereochemistry, camphor grade, capsaicinoid content, piperine content, curcuminoid content, pH, emulsion type and excipients are therefore reported as undisclosed rather than inferred.
- Capsicum species and capsaicinoid content are unknown; trial doses of standardised capsaicin cannot be assumed to match product exposure.
- Cream-base composition, rheology, release, skin flux, dermal retention and systemic exposure have not been publicly characterised.
- Human evidence is concentrated in knee osteoarthritis, muscle strain, experimental DOMS and a few exercise models; it should not be extrapolated to every cause of back, muscle or joint pain.
- Topical botanical studies are heterogeneous in plant part, solvent, extract concentration and vehicle, and many positive ginger or curcumin findings derive from oral administration [53,57].
- Positive mechanistic studies in cells or animals establish plausibility rather than clinical efficacy.
- Several key trials are small, short-term or non-placebo-controlled, so effect sizes should be treated as provisional.
- No product-specific controlled clinical trial of Biosolace was identified, and no evidence demonstrates pharmacodynamic synergy among all declared ingredients; biological complementarity does not prove synergy.

20. Discussion

The declared Biosolace formulation has a coherent pharmacological rationale because its constituents occupy complementary sensory, inflammatory and formulation-related domains. Menthol provides the strongest basis for cooling and acute sensory modulation [11,12]. Capsaicin is the most clinically established topical analgesic principle represented in the formula, particularly for osteoarthritis pain [10,45]. Topical curcumin now has a sufficiently developed human evidence base to extend beyond purely theoretical anti-inflammatory reasoning [8,20,55]. Ginger remains supportive because topical studies are limited and heterogeneous [15,57]; camphor has strong receptor-level plausibility but weak standalone clinical attribution[13,31,38]; and piperine contributes mechanistic TRPV1, human-cell inflammatory and experimental delivery evidence [17–19,66].

This evidence profile argues against presenting all ingredients as equally proven. A more credible narrative assigns different evidentiary roles: capsaicin, menthol and topical curcumin are the clinical anchors; camphor supports counterirritant sensory biology; ginger is a supportive botanical with uncertain topical efficacy; piperine is a mechanistic and delivery contributor. Such a hierarchy strengthens rather than weakens a product review because it prevents unsupported equivalence among ingredients.

The post-exercise positioning has a direct human evidence bridge through menthol. DOMS and post-race studies show that topical menthol can reduce perceived soreness, while acute human pain-threshold studies show changes within minutes [9,25,27,28]. The correct conclusion is symptomatic: sensory discomfort may be reduced. Neither this literature nor the formulation establishes faster recovery of damaged muscle fibres. Similarly, osteoarthritis evidence from capsaicin and curcumin supports symptomatic joint comfort but not cartilage regeneration or structural disease modification [8,10].

The formulation-science question remains unresolved. Menthol and piperine can enhance curcumin permeation in experimental systems, and modern curcumin organogels show that delivery optimisation can materially improve topical performance [55,66,70]. Biosolace could therefore possess meaningful cutaneous delivery characteristics, but this possibility must be measured rather than assumed. The public "quick absorption" descriptor should remain distinct from transdermal or dermal bioavailability: a cream can feel dry quickly yet deliver little active, or remain emollient while sustaining local release.

Combination effects are also inherently uncertain. The neutral diclofenac/menthol ankle-sprain trial and the absence of additional benefit from menthol/capsaicin co-application in exercise illustrate that mechanistic complementarity does not guarantee clinical additivity [30,34]. Conversely, the positive methyl salicylate/menthol patch and mentholated-cream studies show that some combination vehicles can be effective [32,33]. These contrasting results support the central methodological position of this review: The Biosolace combination is supported by a coherent pharmacological rationale and a substantial body of ingredient-level evidence; direct evaluation of the finished formulation would provide the next level of validation.

For scientific communication, the most defensible product-facing language is therefore "topical musculoskeletal comfort" or "temporary symptomatic support." Cooling sensation, post-activity soreness and localised joint discomfort can be discussed with ingredient-level evidence and appropriate qualifiers. "Fast-acting" should be linked to menthol's acute sensory pharmacology but not assigned a product-specific time threshold. "Quick absorption" should be clearly identified as a public product characteristic that has not been verified through peer-reviewed formulation data. "Synergy," "healing," "cartilage repair" and "anti-inflammatory treatment" should be avoided unless supported by product-specific experiments.

21. Conclusion

Biotex Biosolace Pain Relief Cream combines menthol, camphor, ginger, black pepper, turmeric and Capsicum in a topical cream base. Ingredient-level evidence provides a biologically plausible multi-target rationale centred on TRPM8-mediated cooling, TRPV1-directed counter irritation/desensitization, additional thermoTRP modulation, inflammatory/redox pathways and potential effects on cutaneous delivery. Capsaicin has the strongest direct human topical analgesic evidence; menthol has clinically relevant evidence for acute sensory modulation and exercise-associated soreness; and topical curcumin has an expanding randomised evidence base in knee osteoarthritis [8,10,11,20]. Ginger evidence remains limited

and heterogeneous, while camphor and piperine are supported mainly by mechanistic or formulation-level studies [13,15,17–19]. Overall, the available evidence supports Biosolace as a scientifically rational multi-component topical formulation for temporary musculoskeletal comfort, with particularly strong ingredient-level support for cooling, analgesic sensory modulation, exercise-associated soreness and localised joint discomfort. Product-specific clinical studies would further establish the magnitude, onset and duration of these effects.

Declarations

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Conflicts of interest. All authors are employees of Biotex Life Solutions Pvt. Ltd., the company associated with the product discussed in this review. The review was undertaken as part of the company's research and development activities. This employment relationship is disclosed as a potential competing interest. The authors report no additional competing financial or non-financial interests.

Ethics approval. Not applicable. This article is a narrative review of previously published literature; no new studies involving human participants, animals, or human tissue were conducted by the authors.

Data availability. Not applicable. No new experimental or clinical data were generated or analysed for this narrative review. The scientific evidence evaluated is contained in the published sources cited in the reference list; product composition and positioning information was derived from the current product label and publicly available product information described in the manuscript.

Author contributions. All authors contributed to the conceptualisation of the review, literature searching and verification, evidence appraisal, scientific interpretation, manuscript drafting, critical revision, development and review of tables and figures, and final approval of the manuscript. All authors have read and approved the final version and accept responsibility for the accuracy, integrity and scientific content of the work.

Use of artificial intelligence. AI-assisted tools were used as supportive tools for grammar and language correction, literature searching and discovery, gathering and organising information, and development of illustrative images and figures. AI-generated outputs were not accepted as scientific confirmation without author review. All scientific claims, references, interpretations, factual statements and figure content were independently checked and confirmed by the authors. Scientific validation, responsibility for the manuscript content and final approval of the manuscript rest solely with the authors.

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