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Curcuma longa Standardised for Nanocurcumin® and *Boswellia serrata* in Osteoarthritis-Related Joint Symptoms: A Critical Narrative Review and Evidence Appraisal of Biotex Curcuma + Boswellia Capsules

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Abstract: Osteoarthritis (OA) is a disorder of the entire synovial joint in which pain, stiffness, and impaired function arise from interacting cartilage, synovial, subchondral-bone, inflammatory, and nociceptive processes. This critical narrative review evaluates *Curcuma longa* and *Boswellia serrata* evidence relevant to the Biotex Curcuma + Boswellia capsule, whose current label declares 150 mg *C. longa* rhizome (Rz) standardised for Nanocurcumin® and 150 mg *B. serrata* bark (Bk; Salai Guggul) per capsule. Nanocurcumin® is curcumin presented through a nano-enabled delivery approach rather than a different pharmacologically active molecule; accordingly, the established biochemical targets and ingredient-level evidence for curcumin are directly relevant to its curcumin payload, while formulation technology determines exposure and dose efficiency. Human pharmacokinetic studies and systematic reviews demonstrate that advanced curcumin delivery systems can raise systemic exposure many-fold relative to native curcumin, providing a strong scientific basis for lower-dose delivery-enhanced curcumin strategies. Randomised trials and meta-analyses report symptom benefits with several Curcuma/curcumin preparations and standardised Boswellia extracts in OA, and clinical studies of other Curcuma–Boswellia combinations provide additional support for the combination concept. The evidence therefore gives the Biotex formulation a favourable, evidence-aligned joint-support rationale, particularly through the Nanocurcumin® active moiety and complementary botanical pharmacology. Product-specific randomised and pharmacokinetic studies would define the precise magnitude of benefit, bioavailability enhancement, and dose equivalence for the marketed capsule.

Keywords: osteoarthritis; Nanocurcumin®; nanocurcumin; curcumin; *Curcuma longa*; *Boswellia serrata*; boswellic acids; AKBA; joint pain; inflammation; nutraceutical.

Abbreviations

5-LOX, 5-lipoxygenase; AKBA, 3-O-acetyl-11-keto-β-boswellic acid; BA, boswellic acid; BSE, Boswellia serrata extract; COX-2, cyclooxygenase-2; CRP, C-reactive protein; DMC, demethoxycurcumin; BDMC, bisdemethoxycurcumin; ECM, extracellular matrix; hs-CRP, high-sensitivity C-reactive protein; IL, interleukin; KBA, 11-keto-β-boswellic acid; KOOS, Knee Injury and Osteoarthritis Outcome Score;

MAPK, mitogen-activated protein kinase; MMP, matrix metalloproteinase; NF-κB, nuclear factor kappa B; NLRP3, NOD-, LRR- and pyrin domain-containing protein 3; Nrf2, nuclear factor erythroid 2-related factor 2; NSAID, non-steroidal anti-inflammatory drug; OA, osteoarthritis; PGE2, prostaglandin E2; RCT, randomized controlled trial; ROS, reactive oxygen species; TNF-α, tumour necrosis factor-alpha; VAS, visual analogue scale; WOMAC, Western Ontario and McMaster Universities Osteoarthritis Index.

1. Introduction

Osteoarthritis is the most prevalent form of arthritis and a major contributor to chronic pain and disability with ageing[1]. Current disease models consider the entire synovial joint rather than cartilage in isolation: deterioration of articular cartilage occurs together with subchondral-bone remodelling, osteophyte development, synovial inflammation, and changes in meniscal, ligamentous, and capsular tissues[2,3,4]. Synovitis is common across OA stages and can amplify nociceptive, inflammatory, and matrix-catabolic signalling[2,3,4]. Because these processes interact across several molecular networks, multi-target interventions are biologically reasonable to investigate rather than assuming that one enzyme or cytokine accounts for the full symptom burden.

Among botanical options, turmeric (*Curcuma longa* L.) and Indian frankincense (*Boswellia serrata* Roxb. ex Colebr.) have longstanding use in traditional medicine and a growing modern evidence base in osteoarthritis[5]. Curcumin/curcuminoid preparations and boswellic-acid-rich Boswellia extracts affect overlapping but non-identical inflammatory and redox pathways, including NF-κB-associated signalling, cyclooxygenase-related mediators, 5-lipoxygenase (5-LOX), matrix metalloproteinases, and oxidative stress[5,6]. In the Biotex product, the Curcuma component is labelled as *C. longa* rhizome standardised for Nanocurcumin®. Nanocurcumin® is not a different active chemical entity from curcumin; it is a delivery-enhanced form in which curcumin remains the pharmacologically active molecule. Therefore, curcumin mechanisms and ingredient-level biological claims are relevant to Nanocurcumin®, while the magnitude of absorption, dose equivalence, and clinical response remains dependent on the specific nanoformulation. Studies are nevertheless described by the intervention actually tested so that readers can distinguish the active-molecule evidence from formulation-specific pharmacokinetics and clinical performance.

This review critically evaluates the scientific rationale and clinical evidence for *C. longa–B. serrata* combinations in OA-related pain, stiffness, mobility, inflammatory balance, oxidative stress, synovial biology, and chondrocyte/cartilage homeostasis, with specific reference to the finished product Biotex Curcuma + Boswellia Capsules. A central objective is disciplined separation of evidence tiers: (i) evidence generated with the marketed Biotex finished product; (ii) evidence from other Curcuma + Boswellia combinations; (iii) evidence from individual Curcuma/curcumin or Boswellia preparations; and (iv) preclinical mechanistic evidence. For Nanocurcumin®, this hierarchy should not be interpreted as meaning that conventional curcumin pharmacology is irrelevant: because Nanocurcumin® retains curcumin as the active molecule, established curcumin targets, mechanisms, and ingredient-level clinical observations are directly informative for the Nanocurcumin® payload. What remains formulation-dependent is the quantitative translation of dose, exposure, onset, and magnitude of benefit. For clinicians, formulators, and readers, the practical goal is therefore to distinguish active-moiety evidence from delivery-system performance and from finished-product proof. The review avoids claims of disease reversal, cartilage regeneration, pharmacological synergy, or superiority over established OA treatment unless directly supported by human evidence.

2. Literature-Search and Evidence-Appraisal Approach

PubMed/MEDLINE, Scopus, Web of Science, and the Cochrane Library were searched for relevant literature, with targeted checking of journal and publisher records; searches were updated through 2026. Search concepts combined *Curcuma longa*, turmeric, curcumin, nanocurcumin, curcuminoid, NanoCurcumin, *Boswellia serrata*, frankincense, boswellic acid, AKBA, KBA, osteoarthritis, knee pain, hand osteoarthritis, stiffness, WOMAC, VAS, randomised controlled trial, bioavailability, pharmacokinetics, safety, liver injury, and herb–drug interaction. Priority was given to randomised controlled trials, systematic reviews and meta-analyses, controlled human studies, pharmacokinetic

investigations, and original mechanistic studies. Recent high-level syntheses were used to contextualise older individual trials, while original articles were preferred for specific pathway, dose, pharmacokinetic, and numerical claims. Preprints were not retained when peer-reviewed alternatives were available. Studies were interpreted according to the actual intervention tested: conventional curcumin, standardised curcuminoids, a named enhanced-delivery or nanocurcumin formulation, standardised Boswellia resin extract, isolated boswellic acid, or a defined combination. Evidence from non-OA musculoskeletal studies was treated as secondary and not used to establish OA efficacy. As a critical narrative review, the search was not presented as a PRISMA systematic review and no formal pooled re-analysis was performed. Evidence was interpreted in four levels: product-specific evidence; evidence from other Curcuma–Boswellia combinations; single-ingredient clinical evidence; and preclinical/mechanistic evidence. Particular attention was paid to whether plant part and active standardisation matched the Biotex label, because the marketed product specifies *C. longa* rhizome standardised for Nanocurcumin® and *B. serrata* bark. No independently retrievable published randomised trial, pharmacokinetic study, or analytical certificate for the marketed Biotex finished product was identified. This approach was designed to help readers distinguish biological plausibility from direct product validation and to prevent formulation-class evidence from being overstated as evidence for a specific capsule.

3. Biotex Curcuma + Boswellia Formulation

The product under appraisal is Biotex Curcuma + Boswellia Capsules, manufactured by Biotex Life Solutions Pvt. Ltd. The current product label states that each 300 mg capsule contains 150 mg *Curcuma longa* rhizome (Rz), standardised for Nanocurcumin®, and 150 mg *Boswellia serrata* bark (Bk; Salai Guggul) (Table 1; Figure 1). The capsule shell is declared as hydroxypropyl methylcellulose. The label therefore resolves the plant-part question for both botanicals: rhizome for *C. longa* and bark for *B. serrata*. This is more than a nomenclature detail. Plant part can determine which phytochemical classes are present and whether evidence from a standardised extract can reasonably be extrapolated to the marketed material. The Curcuma declaration links the product to a rhizome-derived ingredient standardised for Nanocurcumin®, but the label does not disclose total curcuminoids or the amount of curcumin within the 150 mg botanical mass. Similarly, the *B. serrata* declaration provides bark mass but not total boswellic acids, KBA, or AKBA. Consequently, the label is sufficient to define nominal formulation architecture but not sufficient to establish active-dose equivalence with clinical-trial materials.

Nanocurcumin® is a registered Biotex ingredient designation for the Curcuma component of the formulation. Its active molecular entity is curcumin; nanoengineering modifies how curcumin is dispersed, absorbed, and delivered rather than creating a different pharmacophore. This distinction supports a favourable interpretation of the evidence: biochemical targets and pharmacodynamic actions established for curcumin remain relevant to the Nanocurcumin® curcumin payload, while improved delivery can increase the amount of active compound reaching the systemic circulation[7,8,9]. Human studies illustrate the magnitude that delivery technology can achieve. In the Theracurmin® study, systemic exposure measured by AUC was about 27 times that obtained with curcumin powder[10]; in another crossover study, the liquid micellar formulation produced an approximately 185-fold AUC advantage over native curcumin when all participants were analysed together[11]. A systematic review likewise found that most evaluated advanced oral curcumin delivery systems increased bioavailability relative to free curcumin[9]. These data strongly support nanocurcumin as a more bioavailable and potentially more dose-efficient form of curcumin than unformulated curcumin. The exact quantitative enhancement for Biotex Nanocurcumin® itself should, however, be assigned only after direct product-specific pharmacokinetic characterisation.

Table 1: Biotex formulation and botanical identity.

Component	Declared content	Verification status
Curcuma longa L. rhizome (Rz), standardised for Nanocurcumin®	150 mg/capsule	Plant part and dose visible on the current label. Nanocurcumin® is a registered Biotex ingredient designation in which curcumin remains the active molecular entity. The curcumin mechanism/evidence base is therefore applicable to the payload, while the exact

		product-specific curcuminoid dose, particle characteristics, and fold bioavailability enhancement were not independently available.
Boswellia serrata bark (Bk; Salai Guggul)	150 mg/capsule	Plant part and dose visible on the current label. Total boswellic acids and KBA/AKBA are not disclosed. Most osteoarthritis trials use resin-derived standardised extracts, so chemical and clinical equivalence to the labelled bark component is not established.
Total identified botanical actives	300 mg/capsule	Label-confirmed botanical total; no independently retrievable product-specific RCT, pharmacokinetic study, or certificate of analysis identified.

Fig 1: Biotex Curcuma + Boswellia capsules and declared label composition. The available product label states that each 300 mg capsule contains 150 mg *Curcuma longa* rhizome (Rz), standardised for Nanocurcumin®, and 150 mg *Boswellia serrata* bark (Bk; Salai Guggul). The photograph documents the marketed formulation and plant-part declarations; it does not establish curcuminoid content, boswellic-acid content, particle-size characteristics, or pharmacokinetic performance.

Several product-defining parameters remain analytically unresolved. The label identifies *B. serrata* bark, whereas the majority of pharmacological and clinical Boswellia literature concerns oleo-gum-resin or standardised resin extracts containing measurable boswellic acids[12,13,14,15,16]. Bark and resin should not be treated as chemically interchangeable, and the boswellic-acid content of the marketed bark material cannot be inferred without analytical testing. For the Curcuma arm, the scientific situation is different: Nanocurcumin® retains curcumin as the active molecule, so the established curcumin pharmacology summarised in this review remains relevant to the Nanocurcumin® payload. The unresolved issue is not whether curcumin biology applies, but how efficiently this specific formulation delivers curcumin. An independently retrievable specification for curcuminoid payload, particle-size distribution, carrier composition, dissolution/release behaviour, or product-specific bioavailability was not identified. These measurements would quantify the delivery advantage and enable dose bridging to published nanocurcumin systems. No published randomised clinical trial or human pharmacokinetic study of the finished Biotex product was located. These are verification gaps rather than evidence of product deficiency, and they should be interpreted as limits on quantitative product-specific claims rather than as limits on the applicability of established curcumin mechanisms.

4. *Curcuma longa*: Botany and Traditional Use

Curcuma longa L. is a perennial rhizomatous herb of the Zingiberaceae family cultivated widely across tropical and subtropical Asia[17]. The rhizome is the principal culinary and medicinal material and contains starch, non-volatile curcuminoids, volatile oil, proteins, sugars, and other minor constituents[17,18,19]. In traditional Indian systems, turmeric/Haridra has been used in contexts involving inflammation, wounds, digestive complaints, and musculoskeletal discomfort, but historical use should be regarded as an ethnomedical context rather than evidence of modern clinical efficacy. Modern preparations range from powdered rhizome to solvent extracts, highly standardised curcuminoid concentrates, phospholipid complexes, micelles, solid-lipid systems, and nanoparticles. These dosage forms are not pharmacokinetically interchangeable, although curcumin remains the same active molecular entity when it is incorporated into a delivery system rather than chemically derivatised. For the Biotex formulation, the current label states *C. longa* rhizome (Rz) standardised for Nanocurcumin®, which is the appropriate product-specific terminology. Published curcumin studies therefore remain relevant to the active payload, while formulation-specific studies determine how much curcumin reaches systemic or target tissues at a given dose.

5. Curcuma Phytochemistry: Curcumin, Demethoxycurcumin, Bisdemethoxycurcumin, and Turmerones

The principal diarylheptanoid curcuminoids of *C. longa* rhizome are curcumin (diferuloylmethane), demethoxycurcumin (DMC), and bisdemethoxycurcumin (BDMC)[17,20]. Curcumin usually predominates within standardised curcuminoid mixtures, while DMC and BDMC contribute smaller fractions; the exact proportions vary with genotype, geographical source, processing, solvent system, and analytical method[20,21,22]. The shared conjugated β-diketone system contributes to chromophore properties, redox chemistry, metal binding, and tautomerism, while methoxy substitution differentiates the three major curcuminoids. Turmeric rhizome also yields a volatile oil rich in α-turmerone, β-turmerone, and related sesquiterpenes[19]. These compounds are chemically distinct from curcuminoids and may contribute additional biological activity in extracts that retain the volatile fraction. From a formulation perspective, this chemistry has two practical consequences. First, a declaration of 150 mg of *C. longa* rhizome does not reveal the milligrams of curcumin or total curcuminoids delivered. Second, Nanocurcumin® should be understood as a formulation/ingredient designation rather than a new chemical entity: curcumin remains the active molecular scaffold, while nanoengineering is intended to alter its delivery characteristics. Product-specific curcuminoid assay and particle characterisation are therefore necessary before a precise dose comparison can be made.

6. *Boswellia serrata*: Botany, Resin, and Traditional Use

Boswellia serrata Roxb. ex Colebr. is a Burseraceae tree native to the Indian subcontinent and best known pharmacologically for its aromatic oleo-gum-resin[12,13,14]. Resin is obtained by tapping the trunk and contains a resinous triterpenoid fraction, volatile constituents, and polysaccharide-rich gum[12,13]. Traditional preparations of Salai/Salai Guggul have been used for inflammatory and rheumatic complaints, and this historical use helped motivate modern evaluation in osteoarthritis. Contemporary clinical research, however, is dominated by standardised oleo-gum-resin extracts rather than undifferentiated botanical material. This distinction is particularly consequential for the Biotex capsule because the marketed label identifies the *B. serrata* plant part as bark (Bk), not oleo-gum-resin. Bark and exuded resin are anatomically and chemically different materials; evidence derived from one cannot be assumed to establish the composition of the other. Thus, when this review discusses boswellic acids, AKBA, KBA, or standardised Boswellia clinical extracts, it is describing the evidence base generated predominantly from resin-derived preparations. For formulators and clinicians, direct translation to the marketed bark component requires analytical confirmation that the relevant triterpenoids are present at defined and reproducible concentrations.

7. Boswellia Phytochemistry: β-Boswellic Acid, KBA, AKBA, and Related Triterpenes

Boswellic acids are pentacyclic triterpenoids most extensively characterised in Boswellia oleo-gum-resin and standardised resin extracts[12,15]. Major analytes include β-boswellic acid, acetyl-β-boswellic acid, 11-keto-β-boswellic acid (KBA), and 3-O-acetyl-11-keto-β-boswellic acid (AKBA). Classical structure–

activity studies identified the 11-keto functionality as important to 5-LOX-related activity[16]. A 2020 structural study proposed a specific allosteric AKBA–5-LOX interaction[23]; however, Nature Chemical Biology added an Editor's Note on 31 July 2026 stating that concerns had been raised about the validity of the reported AKBA-bound crystal structure. The present review therefore treats that structural interpretation as provisional and relies principally on the older biochemical evidence and newer functional lipid-mediator data when discussing 5-LOX-related effects[16,24,25]. More broadly, boswellic acids differ in abundance, absorption, metabolism, and molecular targets[12,15,26]. β-Boswellic acid, for example, can achieve greater systemic exposure than AKBA even though AKBA is commonly used as a standardisation marker. Resin extracts also contain acetylated and non-acetylated triterpenes, volatile constituents, and other minor components that may contribute to whole-extract activity. Because the Biotex label identifies bark, resin-derived concentration profiles cannot be assigned to the marketed product without chromatographic characterisation of the actual bark ingredient and finished capsule.

Table 2: Major *Curcuma* and *Boswellia* phytochemicals.

Phytochemical	Class	Source in literature	Key pharmacological note
Curcumin (diferuloylmethane)	Diarylheptanoid curcuminoid	*C. longa* rhizome; curcumin is the chemical entity underlying curcumin/nanocurcumin evidence[17]	Curcumin generally predominates in standardised curcuminoid mixtures; NF-κB-, COX-2-, and Nrf2-related activity is supported in preclinical literature[20,27,28,29].
Demethoxycurcumin	Diarylheptanoid curcuminoid	C. longa rhizome[17,19]	Minor curcuminoid contributing to total curcuminoid composition[17,19].
Bisdemethoxycurcumin	Diarylheptanoid curcuminoid	C. longa rhizome[17,19]	Minor curcuminoid contributing to total curcuminoid composition[17,19].
Turmerones	Bisabolane sesquiterpenes (volatile oil)	*C. longa* rhizome oil[17,18,19]	Representative volatile-oil constituents with experimentally reported biological activity; their specific contribution to clinical OA outcomes remains uncertain[17,18].
β-Boswellic acid	Pentacyclic triterpene	*B. serrata* oleo-gum-resin [12,13,14,15]	Resin-derived boswellic acid; 5-LOX-related pharmacology is formulation- and constituent-dependent [12,16].
Acetyl-β-boswellic acid	Pentacyclic triterpene	*B. serrata* oleo-gum-resin [13,14,15]	Major resin triterpene documented in Boswellia phytochemical profiles [14,15].
11-Keto-β-boswellic acid	11-keto pentacyclic triterpene	*B. serrata* oleo-gum-resin [12,13,14,15]	11-keto boswellic acid used as a pharmacokinetic analyte in human Boswellia studies [30].

3-O-Acetyl-11-keto-β-boswellic acid	11-keto pentacyclic triterpene	*B. serrata* oleo-gum-resin [12,13,14,15]	Resin-derived AKBA is a major mechanistic and standardisation marker in several studied extracts [12,16,31,33].

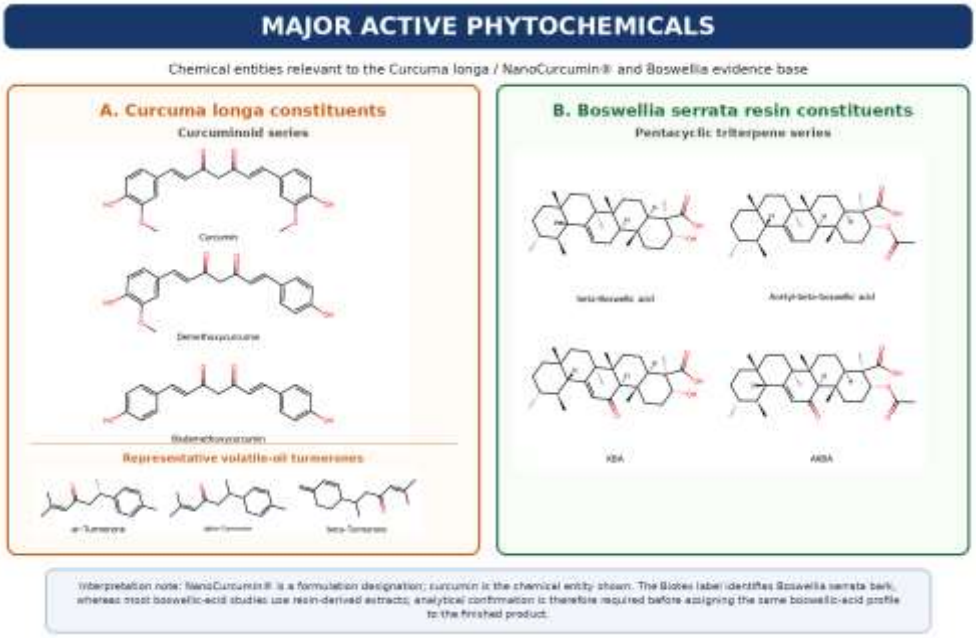

Fig 2: Major active phytochemicals relevant to the evidence base. The left panel shows curcumin, DMC, BDMC, and representative turmerones from *C. longa*; the right panel shows β-boswellic acid, acetyl-β-boswellic acid, KBA, and AKBA, which are principally characterised in Boswellia resin-derived preparations. Nanocurcumin® is a formulation designation rather than a distinct chemical molecule. Because the Biotex label identifies *B. serrata* bark, the boswellic acid structures summarise resin-based literature and should not be assumed to define the marketed bark component without analytical confirmation.

8. Standardisation and Quality Control

Standardisation is essential because turmeric powder, purified/standardized curcumin, and nanocurcumin are different dosage forms even when the active curcumin molecule is the same. For a Curcuma-derived ingredient, quality assessment should document botanical identity, plant part, extraction process, total curcuminoids, individual curcumin/DMC/BDMC concentrations, relevant impurities, and stability[32]. If a nanoformulation is used, additional parameters become important because they determine how effectively the same curcumin payload is delivered: particle-size distribution, polydispersity, carrier composition, payload or encapsulation characteristics where applicable, dissolution/release behaviour, and stability over shelf life. These parameters quantify the delivery advantage; they do not redefine curcumin's pharmacology. For Boswellia, the analytical profile should specify plant part and, when resin-derived pharmacology is invoked, total boswellic acids and individual markers such as KBA and AKBA[12,31,33]. Identity testing is especially important here because the Biotex label specifies bark whereas most OA trials used resin extracts. For readers evaluating the product scientifically, the principal Curcuma question is therefore how much bioavailable curcumin is delivered, not whether curcumin's established mechanisms apply to Nanocurcumin®.

9. Pharmacokinetics and Bioavailability of Conventional Curcumin, NanoCurcumin, and Boswellic Acids

Conventional curcumin. Oral curcumin is constrained by poor water solubility, incomplete gastrointestinal uptake, extensive intestinal and hepatic conjugation, and relatively rapid clearance[7]. As a result, large oral doses may still yield limited circulating concentrations of unconjugated parent curcumin. This

pharmacokinetic limitation does not negate curcumin's pharmacology; rather, it explains why formulation technology has become central to translating the molecule's biological activity into practical oral dosing.

Nanocurcumin and enhanced-delivery systems. Nanocurcumin retains curcumin as the active molecule while using nanoscale or related formulation strategies to improve dispersion, dissolution, absorption, stability, or residence time. The mechanistic claims established for curcumin therefore remain applicable to the curcumin payload, whereas the amount of exposure obtained from a given dose is formulation dependent. Human data provide clear proof of principle: Theracurmin® generated roughly 27-fold higher AUC than curcumin powder[10], and liquid micellar curcumin generated an approximately 185-fold AUC increase over native curcumin in the overall study population[11]. A systematic review of advanced oral systems concluded that most studied platforms improved oral bioavailability compared with free curcumin[9]. For readers, the practical implication is that nanocurcumin should be regarded as a delivery-improved form of curcumin, so lower nominal doses may be pharmacologically meaningful; the exact dose equivalence of Nanocurcumin® must still be established from its own composition and pharmacokinetic profile.

Boswellic acids. Resin-derived boswellic acids are lipophilic and show constituent-specific oral exposure; KBA and AKBA often have low systemic concentrations after conventional extracts[12,26]. Food can materially change absorption[34], and modified lipid formulations can alter pharmacokinetics[30]. These findings are relevant to the broader Boswellia evidence base but do not establish pharmacokinetics for the Biotex bark component. Because the marketed product does not disclose a boswellic-acid profile, neither AKBA exposure nor equivalence to resin-based trial materials can be estimated from the 150 mg bark declaration alone.

10. Osteoarthritis Pathobiology

Osteoarthritis is a heterogeneous whole-joint disease rather than an isolated disorder of articular cartilage[1,2,3,4]. Structural changes include progressive loss and fibrillation of cartilage, altered chondrocyte phenotype, subchondral-bone remodelling and sclerosis, osteophyte formation, synovial inflammation, and changes in menisci, ligaments, capsule, and periarticular tissues[1,2]. Mechanical stress, ageing-associated cellular dysfunction, metabolic factors, damage-associated molecular patterns, and innate-immune signals can activate NF-κB and MAPK pathways in chondrocytes and synoviocytes. The resulting cytokine and eicosanoid milieu—commonly involving IL-1β, TNF-α, IL-6, PGE2, leukotrienes, reactive oxygen species, and nitric oxide-related pathways—favours expression of matrix metalloproteinases and aggrecanases that degrade type II collagen and aggrecan[3,4,35]. Oxidative stress and mitochondrial dysfunction can further impair chondrocyte survival, while NLRP3-associated inflammatory signalling has been implicated in experimental OA[35,36]. Importantly, pain does not correlate perfectly with cartilage loss because articular cartilage is aneural; nociception arises from innervated synovium, subchondral bone, capsule, periarticular structures, and sensitised peripheral/central pathways. This complexity explains why a multi-target botanical strategy is mechanistically plausible, but it also explains why changes in inflammatory biomarkers cannot automatically be interpreted as structural disease modification or clinically meaningful pain relief.

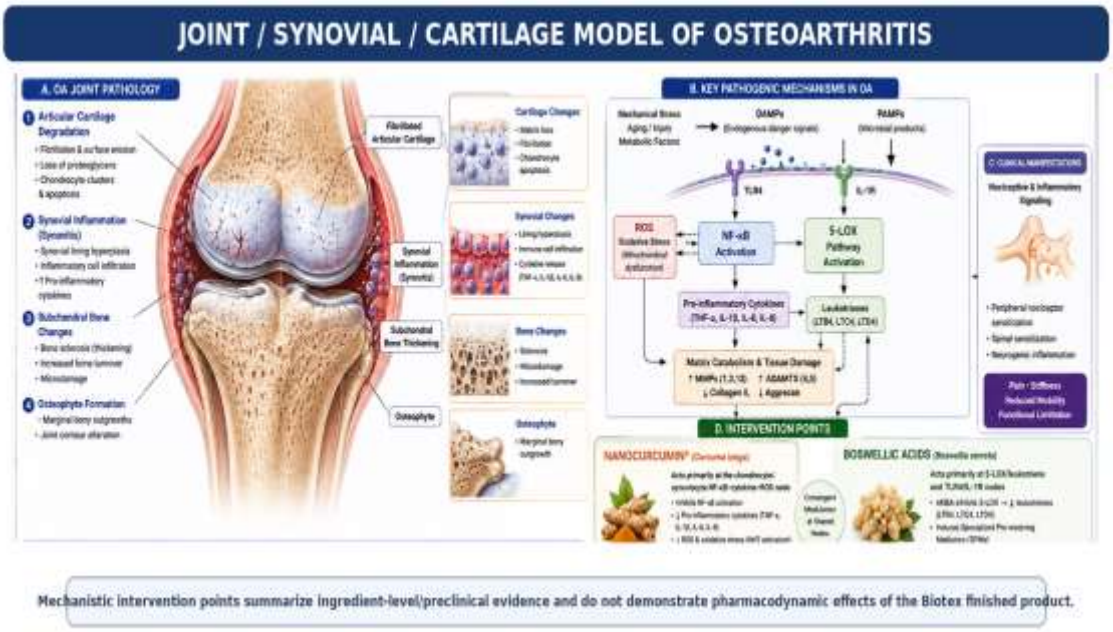

Fig 3: Joint/synovial/cartilage biological model of osteoarthritis and intervention points. The schematic summarises cartilage fibrillation, chondrocyte stress, synovitis, subchondral-bone change, osteophytes, inflammatory signalling, and pain-related outputs. The Nanocurcumin® intervention arm represents established curcumin active-moiety mechanisms, which remain pharmacologically applicable when curcumin is delivered in nanoformulated form; nanoengineering is expected to improve exposure rather than create different molecular targets. The boswellic-acid arm is derived predominantly from resin-based studies, whereas the marketed product label specifies *B. serrata* bark.

11. Curcumin and Nanocurcumin Mechanisms Relevant to Nanocurcumin®

Because Nanocurcumin® retains curcumin as the active molecular entity, molecular mechanisms established for curcumin are applicable to the curcumin payload of Nanocurcumin®; nanoengineering changes delivery and exposure rather than the core target profile of curcumin. In IL-1β-stimulated human chondrocytes, curcumin has been shown to inhibit NF-κB activation and nuclear translocation, suppress MMP-3, and counter cytokine-associated loss of type II collagen[28]. Related rat-chondrocyte work links NF-κB inhibition with regulation of MMP-13, collagen II, and cell proliferation[37]. Three-dimensional OA models also show suppression of inflammatory, apoptotic, and catabolic proteins alongside preservation of cartilage-associated transcriptional markers[38]. Curcumin can reduce COX-2/PGE2 signalling, pro-inflammatory cytokines, reactive oxygen/nitrogen species, and AP-1/NF-κB-associated MMP transcription[27,39]. The redox arm is equally relevant: curcumin-based nanoparticle systems have activated Nrf2-associated antioxidant responses and reduced NLRP3-related inflammatory signalling in experimental OA[40], while curcumin combined with catalase enhanced NRF2/HO-1 signalling and protected chondrocytes from oxidative injury[29]. Thus, the qualitative mechanism-of-action evidence for curcumin carries forward to nanocurcumin formulations, including Nanocurcumin®. What remains formulation-specific is the magnitude of tissue exposure and therefore the dose required to reproduce those pharmacodynamic effects in vivo.

12. Boswellia Mechanisms: 5-LOX/Leukotrienes, NF-κB, MAPK, Cytokines, NLRP3, and Matrix-Related Pathways

Boswellia pharmacology should be interpreted at the constituent and extract level. Classical biochemical studies support modulation of 5-lipoxygenase (5-LOX) and leukotriene biosynthesis by AKBA and related boswellic-acid chemistry[16,25]. A 2020 structural study proposed a specific allosteric AKBA–5-LOX interaction[23], but Nature Chemical Biology added an Editor's Note on 31 July 2026 raising concerns about the validity of the reported AKBA-bound crystal structure; accordingly, the present review does not rely on that structural model as proof of mechanism. Functional studies provide additional support: frankincense

preparations have modified leukotriene output and increased pro-resolving lipid mediators in experimental human innate-immune-cell systems[24]. Beyond lipid mediators, β-boswellic acid has been reported to attenuate TLR4/IL-1R-associated signalling in OA-relevant chondrocytes, osteoblasts, and synoviocytes, with downstream effects involving ROS, p38 MAPK, NF-κB, NLRP3, and matrix-related pathways[41]. An enriched *B. serrata* gum-resin extract reduced multiple IL-1β-induced inflammatory and matrix-catabolic mediators while preserving aggrecan and type II collagen in a chondrocyte model[42]. Peer-reviewed work published in 2024 further linked AKBA with reduced synovial inflammation and matrix-degrading responses through Nrf2/HO-1-associated mechanisms in cellular and rat OA models[43]. Together, these findings support a broad resin-derived anti-inflammatory and matrix-preserving mechanism base, but they should not be assumed to define the Biotex bark component until its constituent profile is analytically established.

13. Complementary Mechanisms of Nanocurcumin® (Curcumin Payload) and Boswellia-Derived Actives

The combination rationale rests on complementary and partially overlapping pharmacology rather than proven synergy. Nanocurcumin® contributes the established curcumin target profile while nanoformulation is intended to improve exposure and delivery efficiency. Curcumin and nanoformulated curcumin systems influence NF-κB-associated cytokine signalling, COX-2/PGE2, oxidative stress, Nrf2, NLRP3, and matrix-catabolic enzymes[10,44,45,46,47,48,49]. Resin-derived Boswellia constituents add a distinct 5-LOX/leukotriene and lipid-mediator dimension and also affect innate-immune receptors, NF-κB/MAPK, oxidative, inflammasome, and MMP-related pathways[26,30,50,51,52,53]. The two arms therefore approach OA biology from more than one upstream direction while converging on common downstream nodes such as inflammatory cytokines, ROS, NF-κB, NLRP3, and matrix-degrading enzymes. Such convergence is compatible with complementary or potentially additive activity, but it is not by itself evidence of pharmacological synergy. Formal synergy requires quantitative dose–response combination analysis[54]. For the Biotex product, this framework provides a coherent mechanistic rationale, with the strongest direct transfer of evidence occurring on the Nanocurcumin®/curcumin side and a need for constituent confirmation on the labelled Boswellia bark side.

Table 3: Molecular targets and pathways of NanoCurcumin (curcuminoid payload) and boswellic acids.

Target/pathway	Curcumin active-moiety evidence (applicable to Nanocurcumin® payload)	Boswellia resin-derived actives	Convergence
NF-κB	Inhibits NF-κB signalling and p65 nuclear translocation in IL-1β-stimulated chondrocyte models [28,37,38].	β-Boswellic acid and enriched resin extracts suppress TLR4/IL-1R-linked NF-κB signalling and p65 activation in articular-cell/chondrocyte models [41,42].	Shared reduction of pro-inflammatory transcription and cytokine signalling [5].
COX-2 / PGE2	Curcumin suppresses COX-2/PGE_2-related inflammatory signalling in experimental OA systems [27,39].	Enriched *B. serrata* gum-resin extract reduced IL-1β-induced COX-2/PGE_2 responses in SW1353 chondrocytes [42].	Complementary modulation of inflammatory eicosanoid signalling.
5-LOX / leukotrienes	—	AKBA and related resin constituents modulate 5-LOX/leukotriene biosynthesis [16,25]; standardised frankincense preparations can also shift lipid-mediator profiles	Complementary eicosanoid/lipid-mediator arm.

		toward pro-resolving mediators [24].	
NLRP3 inflammasome	Curcumin-based nanoparticles activated Nrf2 and inhibited NLRP3 in an experimental OA model [40].	β-Boswellic acid reduced TLR4-associated NLRP3 signalling in articular-cell models [41].	Shared inflammasome node.
MAPK (ERK, p38, JNK)	Curcumin modulates MAPK-linked inflammatory and catabolic signalling in OA models [38,39].	Enriched gum-resin extract suppressed ERK, p38, and JNK phosphorylation in chondrocytes [42].	Shared kinase cascade.
MMPs / ECM	Curcumin reduced MMP expression while supporting type II collagen and matrix-associated markers in chondrocyte models [28,37,38].	Enriched *B. serrata* resin extract reduced MMP-1/-3/-13 and preserved aggrecan/type II collagen in vitro [42].	Convergence on matrix-catabolic pathways.
Nrf2 / antioxidant	Nrf2/HO-1-related antioxidant effects are supported by OA chondrocyte and animal studies [29,40].	AKBA reduced experimental synovitis through Nrf2/HO-1 signalling [43]; β-boswellic acid also reduced ROS-linked signalling in articular-cell models [41].	Redox-homeostasis convergence.
Cytokines (IL-1β, TNF-α, IL-6)	Curcumin reduced OA-related pro-inflammatory cytokine signalling in preclinical models [27,39].	Boswellia-derived actives reduced IL-1β/TNF-α/IL-6-related responses in chondrocyte and synovitis models [41,42,43].	Shared cytokine axis.

14. Antioxidant and Cartilage/Connective-Tissue Relevance

Oxidative stress is both a consequence and amplifier of OA-related inflammation. Excess ROS can damage mitochondrial function, promote chondrocyte senescence or death, and enhance catabolic signalling that accelerates loss of collagen and aggrecan[35]. The Nrf2/ARE pathway coordinates endogenous antioxidant defences including HO-1, NQO1, glutathione-associated systems, superoxide dismutase, and other cytoprotective responses[55]. Experimental curcumin and curcumin-nanoparticle studies support activation of Nrf2/HO-1, suppression of oxidative stress, and secondary restraint of NLRP3-associated inflammation[29,40]. Boswellia-derived constituents also reduce ROS and matrix-catabolic responses in OA-relevant cellular models[41,42,43]. These observations support the terms 'antioxidant', 'matrix-preserving', or 'chondroprotective' when clearly identified as preclinical. They do not demonstrate regeneration of articular cartilage, reversal of established structural OA, or restoration of joint space in humans. Readers should therefore separate biochemical preservation of matrix markers in cell/animal experiments from clinically proven structure modification, which would require appropriately designed human imaging or biomarker trials.

14.1 Secondary Hepatic Evidence and Relevance Limits

Evidence outside the joint includes hepatic effects, but these are secondary to the present review. Meta-analyses in NAFLD and related populations report reductions in ALT and/or AST with turmeric/curcumin supplementation, with substantial heterogeneity across formulations, doses, and study durations[45,46,47,56,57]. Mechanistic reviews and preclinical studies link curcumin to Nrf2 and NF-κB-related hepatic pathways, while experimental Boswellia/boswellic-acid studies report antioxidant, anti-inflammatory, and antifibrotic effects[48,49,50,51,52,53,58,59]. These findings concern other curcumin/nanocurcumin preparations and predominantly resin-derived Boswellia materials; they do not establish hepatoprotection by the Biotex finished product. Because rare turmeric/curcuminoid-associated liver injury is recognised in the safety

literature[60,61,62,63,64], hepatic efficacy claims should remain separate from safety monitoring and should not be a primary positioning claim for this joint-health formulation.

15. Human Clinical Evidence for Curcumin/Curcuminoids

Human evidence for Curcuma/curcuminoid interventions in OA is sizeable compared with many botanical ingredients, although dose, extraction, and delivery systems vary substantially across trials. Because nanocurcumin retains curcumin as its active molecule, these clinical findings are relevant to the therapeutic potential of the Nanocurcumin® payload; enhanced delivery chiefly changes the exposure achieved from a given nominal dose. Systematic reviews and meta-analyses generally show reductions in pain and improvements across WOMAC domains relative to placebo[65,66,67,68,69]. Zeng et al. pooled 15 RCTs involving 1,621 participants and found favourable effects on pain, function, and stiffness, with adverse-event rates similar to placebo[65]. Daily et al. likewise reported a pooled analgesic advantage for turmeric extracts/curcumin[66], and later syntheses reached broadly consistent conclusions[67,68,69]. The 2026 network meta-analysis synthesised 20 RCTs involving 1,633 participants; among its findings, modified Curcuma preparations produced lower VAS pain scores than placebo[70]. Individual studies also illustrate why formulation matters: conventional curcuminoid trials often used higher doses[71], whereas enhanced-delivery preparations such as Curcugen®, solid-lipid curcumin, Theracurmin®, and nanomicellar curcumin have been evaluated at lower nominal doses[10,44,72,73]. Henrotin et al.[74] reported a mixed efficacy pattern rather than a uniformly negative result. Overall, the literature supports curcumin as the active therapeutic principle and supports advanced delivery as a rational way to improve exposure; the remaining question for Nanocurcumin® is the quantitative performance of this specific formulation rather than whether curcumin pharmacology is relevant to it.

Representative controlled trials illustrate this formulation dependence. Curcuminoids at 1,500 mg/day improved WOMAC, VAS, and Lequesne outcomes versus placebo[71], whereas lower-dose enhanced-delivery preparations such as Curcugen® and solid-lipid curcumin also reported symptomatic or functional improvements[72,73]. A randomised trial of nanomicellar curcumin reported significant WOMAC improvement[44]. Henrotin et al.[74] produced mixed rather than uniformly negative results: bio-optimised *C. longa* doses improved patient global assessment and pain relative to placebo, whereas KOOS change and the cartilage biomarker sColl2-1 did not differ significantly. This pattern reinforces a practical point for readers: 'curcumin' is not a single clinically interchangeable intervention, and outcomes should be interpreted in relation to the actual formulation studied.

16. Human Clinical Evidence for Boswellia

Standardised Boswellia extracts have been evaluated in multiple knee OA trials, with most preparations derived from oleo-gum-resin and standardised to boswellic acids rather than bark. 5-Loxin® (30% AKBA) at 100 or 250 mg/day improved pain and physical function and reduced synovial-fluid MMP-3 over 90 days[31]. Aflapin® at 100 mg/day produced early symptomatic improvement in a randomised placebo-controlled study[75]. Boswellin® improved pain, stiffness and function, reduced hs-CRP, and in its small 48-patient study reported exploratory radiographic changes in joint gap and osteophytes[76]. BOSMAX® improved WOMAC, VAS and Lequesne scores and reduced TNF-α and hs-CRP over 90 days[77]. The pooled evidence is less uniform than individual positive trials might suggest. A 2020 meta-analysis of seven trials reported significant improvements in pain and function[78], whereas a 2024 systematic review/meta-analysis found high heterogeneity and no statistically significant overall effect across all comparisons, with clearer benefit in placebo-controlled subgroups[79]. The 2026 network meta-analysis found significant improvements in WOMAC pain, stiffness, and knee function for modified Boswellia formulations[70]. For readers considering the Biotex formulation, the key limitation is material identity: these results concern standardised resin extracts. They cannot establish efficacy of 150 mg *B. serrata* bark unless the marketed bark is analytically linked to the same active triterpenoid profile.

17. Human Clinical Evidence for Curcuma + Boswellia Combinations

Combination trials are important because they test the central Curcuma–Boswellia concept rather than inferring it from two separate monotherapies. Haroyan et al. randomised 201 participants with knee OA to

a curcuminoid–boswellic-acid combination, curcuminoids alone, or placebo for 12 weeks[6]. In that trial, the combination improved selected performance measures and WOMAC pain relative to placebo, whereas the curcuminoid-only arm showed benefits across fewer outcomes; the pattern is compatible with added value but does not establish pharmacological synergy. Kizhakkedath compared a *C. longa* + *B. serrata* formulation (500 mg twice daily) with celecoxib in a small trial in which 28 participants completed treatment, reporting favourable symptomatic outcomes and acceptable tolerability[80]. In hand OA, an open-label study of 232 patients reported improvement in pain-related outcomes over three months[81], followed by a multicentre double-blind randomised trial involving 162 participants, where three months of the botanical combination reduced hand-pain scores more than placebo[82]. A separate randomised trial in participants with acute musculoskeletal pain provoked by exercise reported analgesic benefit from a turmeric–Boswellia formulation[83], but this is supportive musculoskeletal evidence rather than OA-specific proof. Rhuleave-K adds another OA-specific combination dataset[84]. Importantly, the 2026 network meta-analysis found that evidence for mixed Curcuma–Boswellia formulations was still too limited for a firm estimate of their added clinical value[70]. Thus, the combination literature is encouraging and directionally positive, while remaining formulation-specific and not automatically equivalent to the marketed Biotex capsule.

Interpretation requires care because each combination trial used different extracts, standardisations, doses, and delivery systems. None tested the marketed Biotex product, and the majority of Boswellia-containing trials used resin-derived preparations rather than the bark stated on the Biotex label. The overall direction of combination evidence is encouraging for symptom-oriented research, but it does not establish dose equivalence, product-specific efficacy, or synergy.

Table 4: Major human clinical trials relevant to *C. longa–B. serrata* combinations and their ingredients.

Study (year)	Design/sample	Intervention	Key outcomes
Haroyan et al., 2018[6]	RCT, 3-arm, 12 weeks, n = 201, knee OA	Curamin® (350 mg curcuminoids + 150 mg boswellic acid, TID) vs CuraMed® (333 mg curcuminoids, TID) vs placebo	The combination showed broader statistically significant benefit than curcuminoids alone, including performance-based outcomes and WOMAC pain; this supports possible incremental benefit but not formal synergy.
Kizhakkedath, 2013[80]	RCT, 4 weeks, 30 enrolled / 28 completed, knee OA	*C. longa* + *B. serrata* extract (500 mg BID) vs celecoxib 100 mg BID	Favourable symptom scores with combination
Henrotin et al., 2025[82]	Multicentre double-blind RCT, 3 months, n = 162, hand OA	Patented *C. longa* + *B. serrata* extracts vs placebo	Pain reduction greater than placebo (between-group difference −8.5 mm; p = 0.03); several patient-reported secondary outcomes improved; adverse events similar
Gupta et al., 2026[84]	RCT, 12 weeks, n = 100, mild-to-moderate OA	Rhuleave-K® (turmeric + Boswellia extracts in sesame oil, 500 mg/day)	Large within-group reductions were reported in total WOMAC and its pain/stiffness/function domains; CRP declined, and no adverse events were reported during the 12-week study.
Panahi et al., 2014[71]	RCT, 6 weeks, knee OA	Curcuminoids 1,500 mg/day vs placebo	WOMAC (p = 0.001), VAS (p < 0.001), Lequesne (p = 0.013) improved
Lopresti et al., 2021[72]	RCT, 8 weeks, n = 101, knee OA	Curcugen® 500 mg BID vs placebo	KOOS pain p = 0.009; pain-medication reduction 37% vs 13%
Hashemzadeh	RCT, 6 weeks, knee	Nanomicellar curcumin	WOMAC pain, stiffness, and function

et al., 2020[44]	OA	vs placebo	improved; supports clinical activity of enhanced-delivery curcumin
Sengupta et al., 2008[31]	RCT, 90 days, knee OA	5-Loxin® (30% AKBA) 100 or 250 mg/day vs placebo	Pain and physical function improved; synovial MMP-3 reduced
Majeed et al., 2019[76]	RCT, 120 days, n = 48, knee OA	Boswellin® (AKBA + β-boswellic acid) vs placebo	Pain, stiffness, function and hs-CRP improved; exploratory radiographic findings in this small trial also favoured the extract.
Jayaram et al., 2026[77]	RCT, 90 days, n = 150, knee OA	BOSMAX® vs placebo	WOMAC −24.55 points ($p < 0.05$); TNF-α −104.75 ng/L; hs-CRP −5.67 ng/mL
Inprasit et al., 2026[70]	Systematic review and network meta-analysis; 20 RCTs; n = 1,633	Curcuma, Boswellia, and mixed formulations in knee OA	Modified Curcuma and modified Boswellia formulations showed domain-specific advantages; evidence for the mixed formulations remained too limited for firm conclusions.

18. Pain, Stiffness, Mobility, and Physical-Function Outcomes

Pain is the most consistently improved outcome across Curcuma and Boswellia clinical literature, whereas stiffness, objective function, and structural measures show greater variability. Curcuma meta-analyses report improvements in WOMAC pain and VAS, with smaller or less consistent effects on stiffness in some individual trials[65,66,67,68,69,71]. Boswellia studies report improvements in pain and physical function, but pooled estimates depend strongly on comparator and extract type[31,75,78,79]. Combination trials extend these findings to performance measures, WOMAC domains, patient global assessment, and, in the 2025 hand-OA RCT, a statistically greater pain reduction versus placebo[6,82]. These outcomes are clinically more informative than mechanistic biomarkers because they reflect what patients experience. Even so, a statistically significant mean change is not automatically a large individual benefit, and placebo responses in OA are substantial. For clinicians and readers, the most defensible interpretation is that selected characterised formulations may provide modest symptom support; evidence remains insufficient to infer the same magnitude of pain, stiffness, or mobility benefit for the Biotex finished product without direct testing. In addition, outcome interpretation should consider the minimum clinically important difference, treatment duration, baseline symptom severity, concomitant analgesic use, and whether analyses were intention-to-treat or per-protocol. Short studies can detect analgesic or anti-inflammatory effects but are not designed to establish long-term structural benefit. Likewise, reductions in CRP, cytokines, or matrix markers can strengthen biological plausibility but should not replace validated patient-reported and performance outcomes. This distinction is especially relevant to a product review because mechanistic sophistication can make an intervention appear more clinically established than the underlying trial evidence warrants.

19. Safety and Tolerability

Across randomised Curcuma/curcumin OA trials, short-term tolerability is generally favourable, and adverse-event rates are often comparable with placebo; gastrointestinal complaints are among the more frequently reported events[64,65]. Rare idiosyncratic liver injury associated with turmeric/curcuminoid supplements is nevertheless documented. The U.S. DILIN surveillance series identified 10 adjudicated turmeric-associated liver-injury cases, mostly with a hepatocellular pattern; five required hospitalisation and one was fatal[60]. Italian phytovigilance data and a systematic review added further reports[61], and a 2025 clinicopathological series described 11 supplement-associated hepatitis cases[63]. A 2026 United States Pharmacopoeia review placed these reports in the context of widespread use and numerous clinical trials without organ toxicity; it characterised clinically apparent hepatic events as uncommon and recommended precautionary labelling for users with a history of liver problems[62]. This balance is important for readers: a rare safety signal warrants awareness without overturning the generally favourable

short-term tolerability seen in clinical studies. Boswellia is also usually well tolerated, with gastrointestinal and hypersensitivity-type events among the more commonly described adverse reactions[12,85]. Product-specific safety surveillance for the marketed Biotex combination would further strengthen the evidence base.

20. Herb–Drug Interactions

Herb–drug interaction claims require similar discipline. Curcuminoids inhibit several CYP enzymes and transporters in vitro, and pharmacokinetic interactions have been reported with selected drugs, but the human database remains limited, and effects are dose/formulation dependent[86,87]. Importantly, a small clinical study of Meriva® in patients receiving stable antiplatelet, anticoagulant, or thyroid-replacement therapy did not find significant changes in bleeding time, INR, or thyroid-function control[88]. Thus, it is inappropriate to state broadly that curcumin 'causes bleeding' with anticoagulants; the more accurate message is that interaction potential exists and warrants clinical review in high-risk settings. Boswellia extracts inhibit CYP3A4/5 and CYP2C9 in microsomal systems, while more physiologic hepatocyte studies suggest lower interaction potential[89,90]. No interaction study of the Biotex combination was identified. Readers managing polypharmacy should therefore apply precaution proportional to drug therapeutic index, comorbidity, and formulation exposure rather than extrapolating every in vitro interaction into a clinical contraindication.

21. Dose and Formulation Translation

The 300 mg total botanical mass of the Biotex product cannot be compared directly with doses in published trials because active standardisation and delivery systems differ. The broader pharmacokinetic literature nevertheless supports the central premise behind Nanocurcumin®: improved curcumin delivery can produce much higher systemic exposure than native curcumin and can make lower nominal doses pharmacologically meaningful[8,9,10,11]. Conventional curcuminoid trials often used gram-range daily doses, whereas several enhanced-delivery preparations achieved measurable pharmacokinetic or clinical effects at substantially lower doses[10,44,72,73]. The scientifically appropriate conclusion is therefore not that 150 mg of Nanocurcumin® must equal a particular gram dose of conventional curcumin, but that nanoformulation can materially improve dose efficiency. Exact equivalence requires the curcumin payload and pharmacokinetics of Nanocurcumin® to be quantified. Boswellia remains a separate translational issue because the Biotex label specifies 150 mg *B. serrata* bark while most positive trials used standardised resin extracts with defined boswellic-acid profiles[31,33,75]. For clinicians and formulators, the Curcuma evidence supports a delivery-enhanced curcumin rationale; the Boswellia arm still requires constituent-level characterisation for dose bridging.

22. Evidence-to-Product Appraisal

For the Biotex finished product, the literature provides a favourable and biologically coherent rationale for combining *C. longa* standardised for Nanocurcumin® with *B. serrata*. Nanocurcumin® retains curcumin as the active molecule, so the extensive curcumin mechanism and ingredient-level clinical literature is directly informative for its active moiety. Human pharmacokinetic evidence also demonstrates that advanced delivery systems can markedly increase curcumin exposure compared with unformulated curcumin[8,9,10,11], making a lower-dose delivery-enhanced strategy scientifically credible and potentially more dose-efficient. What remains to be quantified for Nanocurcumin® is the formulation-specific magnitude of that enhancement, not the relevance of curcumin pharmacology itself. The Boswellia evidence transfer is less direct because the current Biotex label specifies bark, while most OA studies used standardised resin-derived preparations. Overall, the product is supported by a strong Curcuma/Nanocurcumin® active-moiety rationale and a complementary Boswellia concept; analytical characterisation of the bark constituent profile and direct finished-product studies would refine, rather than create, this scientific rationale.

Table 5: Evidence-to-claim appraisal for the Biotex formulation.

Claim	Supporting evidence tier	Verdict

"Supports joint comfort/pain relief"	Ingredient-level meta-analyses[65,66,67,68,69,78,79] and combination RCTs[6,80,82,84]	Supported by the Curcuma/curcumin and Boswellia ingredient evidence and by related combination trials; direct Biotex trials would quantify the product-specific effect size.
"Multi-target, mechanistically convergent action"	Curcumin OA mechanisms[28,29,37,38,39], Boswellia mechanisms[16,23,24,25,41,42,43], and enhanced-delivery evidence[8,9,10,11,44]	Supported at the active-moiety level. Curcumin mechanisms apply to the Nanocurcumin® payload, while Boswellia evidence is predominantly resin-derived and therefore requires constituent confirmation for the labelled bark component.
"Nanocurcumin® provides high bioavailability"	Human pharmacokinetic studies and delivery-system reviews[8,9,10,11]	Strongly supported as a delivery principle: advanced curcumin formulations can produce substantially greater systemic exposure than native curcumin. The exact fold enhancement of Biotex Nanocurcumin® remains product-specific.
"Synergistic combination"	Formal dose–effect synergy has not been established; quantitative combination methodology is discussed in[54]	Not demonstrated; use "potentially additive" / "complementary"
"Cartilage regeneration/disease modification"	No robust human structural disease-modification evidence in the current OA evidence base[65,70]	Unsupported by human evidence
"Product-specific efficacy and safety"	Curcumin active-moiety efficacy is supported broadly; no finished-product RCT or direct Nanocurcumin® PK bridge located.	Curcumin mechanisms/ingredient evidence are applicable to Nanocurcumin®; finished-product effect size, dose equivalence, and Boswellia-bark contribution remain product-specific

23. Strengths and Limitations

Strengths of the evidence base include multiple randomized trials and meta-analyses of Curcuma/curcumin[65,66,67,68,69], standardized Boswellia extracts[78,79], molecular and cellular OA studies[24,28,41,42,43], human curcumin pharmacokinetic studies[8,10,11], a systematic review of advanced oral curcumin delivery[9], combination trials across knee and hand OA[6,80,81,82,83,84], and an updated 2026 network meta-analysis[70]. The literature therefore spans mechanism, exposure, patient symptoms, and comparative clinical outcomes. For Nanocurcumin®, an important strength is pharmacological continuity: the active molecule remains curcumin, allowing the established curcumin mechanism and efficacy literature to inform its biological rationale, while nanoformulation addresses a recognised oral-delivery limitation. The principal uncertainties are quantitative and product-specific—the exact curcuminoid payload, particle characteristics, and human exposure achieved by Nanocurcumin® were not independently retrievable. Trial quality and duration also vary, and formulations, comparators, OA phenotype, disease severity, and endpoints are heterogeneous. Formal synergy has not been demonstrated[54], and robust human structural disease-modification data are lacking. For the Boswellia arm, the label's bark declaration remains the main translational limitation because most clinical evidence is based on resin-derived extracts.

24. Conclusion

The combination of *Curcuma longa* and *Boswellia serrata* has a favourable, biologically plausible rationale for supporting OA-related joint comfort, pain, stiffness, and function, backed by a growing

clinical literature on the individual ingredients and on other Curcuma–Boswellia combinations. Nanocurcumin® is curcumin delivered through a nano-enabled formulation rather than a different active molecule; the established molecular actions and ingredient-level clinical evidence for curcumin therefore remain relevant to its active payload. Advanced delivery systems have produced large gains in human curcumin exposure—approximately 27-fold for Theracurmin® and about 185-fold for a liquid micellar formulation in representative studies—and systematic review evidence supports improved oral bioavailability across many advanced systems[8,9,10,11]. These data provide strong support for the principle that nanocurcumin can be substantially more bioavailable and potentially more dose-efficient than conventional unformulated curcumin, although the exact fold enhancement of Biotex Nanocurcumin® requires direct characterisation. Resin-derived Boswellia constituents offer complementary anti-inflammatory and lipid-mediator mechanisms[26,30,50,51,52,53], while the marketed label specifies *B. serrata* bark, making constituent-level confirmation important for precise dose bridging. Trials of other Curcuma + Boswellia preparations have reported symptom improvements[6,80,81,82,83,84]; the most recent network meta-analysis nevertheless found the mixed-formulation evidence base too limited for a definitive combined-treatment estimate[70]. Overall, the Biotex capsule has a positive, evidence-aligned scientific rationale for joint-support positioning, with the Nanocurcumin® component benefiting from the broad curcumin evidence base and enhanced-delivery principle; direct finished-product studies would quantify the magnitude of benefit rather than determine whether the formulation has biological plausibility.

Declarations

Author affiliation and relationship to the product. All authors are affiliated with the Department of Research and Development, Biotex Life Solutions Pvt. Ltd. The reviewed Biotex Curcuma + Boswellia formulation and the Nanocurcumin® ingredient are associated with the company's research and development activities.

Scientific approach and evidence boundaries. The review distinguishes evidence generated with the marketed product from evidence concerning other Curcuma–Boswellia combinations, individual ingredients, and preclinical mechanisms. Where product-specific analytical, pharmacokinetic, or clinical information was not independently retrievable, this is stated explicitly; ingredient-level evidence is not represented as direct proof of the finished formulation.

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Use of AI-assisted tools. AI-assisted tools were used for literature searching, information organisation, language correction, and formatting support. Scientific interpretation, claim assessment, reference verification, and final approval were performed by the authors, who take responsibility for the manuscript.

Trademark. Nanocurcumin® is a registered trademark of Biotex Life Solutions Pvt. Ltd.

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