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Boswellia serrata in osteoarthritis and rheumatoid arthritis: effects, mechanisms, and clinical evidence

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Abstract

Background. Osteoarthritis (OA) and rheumatoid arthritis (RA) carry a large global burden, and standard pharmacotherapy is limited by gastrointestinal, renal and cardiovascular toxicity. Boswellia serrata oleogum resin has re-emerged as an anti-inflammatory adjunct.

Aim. To summarise current evidence on the phytochemistry, mechanisms, pharmacokinetics, clinical efficacy, and safety of Boswellia serrata in OA and RA.

Materials and methods. A structured search of PubMed, bioRxiv, Scopus and Google Scholar (1990–2026, with seminal pre-1990 records retained) combined Boswellia serrata, boswellic acid, AKBA, 5-LOX, NF-κB, osteoarthritis, rheumatoid arthritis, pharmacokinetics and clinical trial.

Results. Boswellic acids, primarily AKBA, act through allosteric 5-LOX inhibition, IKK–NF-κB suppression, anti-cytokine and anti-MMP effects, cathepsin G inhibition and Nrf2/HO-1 antioxidant activity. Standardised extracts produce clinically meaningful reductions in WOMAC and VAS scores in knee OA, and a Cochrane synthesis reports a 17-point VAS reduction at 90 days (NNT 2). RA evidence is sparser and provisional. Short-term safety is favourable; rare hepatic signals and CYP3A4-mediated interactions warrant attention.

Conclusions. Boswellia serrata is a multi-target adjunct with a reproducible mechanism, consistent symptomatic benefit in OA, an evidence-light role in RA and acceptable short-term safety; manufacturer-independent multi-centre RCTs are the priority.

Keywords: Boswellia serrata; Boswellic acids; Osteoarthritis; Rheumatoid arthritis; 5-Lipoxygenase; Phytotherapy.

1. Introduction

Osteoarthritis (OA) and Rheumatoid Arthritis (RA) account for the largest share of chronic musculoskeletal disability worldwide. They create a sustained burden on health-care systems, working-age populations, and aging societies [1–3]. OA is the most prevalent joint disease, affecting hundreds of millions. Its age-standardised prevalence has risen steeply over the last three decades, driven by population aging, rising body-mass index, and prior joint injury [1,2]. RA is less common but more uniformly disabling. Its chronic synovitis leads to articular destruction, extra-articular complications, and excess cardiovascular mortality unless suppressed early with a Disease-Modifying Antirheumatic Drug (DMARD) [3,4]. Both conditions impair

mobility, sleep, and mental health. Their cumulative costs to direct medical care and lost productivity continue to grow [1,4].

Conventional pharmacotherapy for symptomatic relief in OA and for adjunctive analgesia in RA still relies on Non-Steroidal Anti-Inflammatory Drug (NSAID) therapy. This is recommended as a core option by current OA guidelines, but these contain explicit caveats about long-term use [5,6]. Chronic NSAID usage is related to peptic ulcer disease, upper-gastrointestinal bleeding, renal impairment, and adverse cardiovascular events. These risks are especially pronounced in elderly and multimorbid patients, in whom OA prevalence is highest [7]. Paracetamol is now thought to have only a modest, clinically uncertain effect on OA pain. Opioid use is discouraged because of dependence and limited durable benefit [5,6]. In RA, methotrexate-anchored DMARD strategies and biologic agents have transformed the prognosis. Still, a substantial fraction of patients show incomplete response, intolerance, or treatment fatigue. Back-up options acting through anti-inflammatory pathways remain clinically attractive [3,4].

These limitations have renewed interest in plant-derived anti-inflammatories with credible mechanisms and favourable tolerability profiles. Examples include curcuminoids, ginger, and Boswellia serrata resin extracts [8–10]. Boswellia serrata (Indian frankincense) has been used for centuries in Ayurvedic and Unani medicine to treat inflammatory and rheumatic conditions. Its main active triterpenoids, the boswellic acids, have since been characterised as inhibitors of 5-lipoxygenase and modulators of NF-κB signalling [10,11]. Modern standardised extracts and improved bioavailability formulations have generated a clinically relevant body of randomised trials and pharmacokinetic data, particularly in knee OA. The rheumatoid evidence base is older and thinner, but coherent [9,12]. A structured synthesis of this literature is therefore timely.

This review summarises the current evidence on Boswellia serrata for the management of OA and RA across 10 sections. Following the literature search, Section 3 discusses the botany and phytochemistry of the resin and its principal boswellic acids. Section 4 presents mechanistic data on 5-lipoxygenase inhibition, NF-κB suppression, anti-cytokine and anti-matrix metalloproteinase actions, and redox modulation. Section 5 examines pharmacokinetics and the rationale for standardised commercial extracts. Sections 6 and 7 rate controlled clinical evidence in OA and RA, respectively. Section 8 reviews safety, drug interactions, and regulatory status, while Section 9 addresses strengths, weaknesses, and key considerations for clinicians regarding Boswellia serrata as adjunct therapy in joint disease.

2. Materials and methods

A structured literature search was conducted to identify primary studies, randomised controlled trials, and quantitative syntheses on Boswellia serrata and its constituent boswellic acids in Osteoarthritis (OA) and Rheumatoid Arthritis (RA). PubMed served as the primary database. BioRxiv, Scopus, and Google Scholar were also used. The primary date window was 1990–2026, with seminal pre-1990 records retained when judged foundational. Notably, the Sailer/Ammon characterisation of boswellic acids as 5-lipoxygenase inhibitors was included [13].

Search strings combined botanical and chemical terms, including ‘’Boswellia serrata’’, “boswellic acid”, “acetyl-11-keto- β -boswellic acid” or “AKBA.” Commercial preparations searched included 5-Loxin, Aflapin, AprèsFlex, and Casperome. Disease and mechanism descriptors such as “osteoarthritis”, “rheumatoid arthritis”, “5-lipoxygenase”, “NF-κB”, “matrix metalloproteinase”, “clinical trial”, “randomized”, “pharmacokinetics”, and “safety” were joined by Boolean operators. Medical Subject Headings (MeSH) were used in PubMed, where available. Authoritative narrative reviews were screened for additional primary references [10,12].

Inclusion was restricted to peer-reviewed primary research, randomised controlled trials, systematic reviews, and meta-analyses on Boswellia serrata or boswellic acids in OA or RA. Mechanistic in vitro and animal studies directly informative for joint inflammation, as well as primary pharmacokinetic and safety data, were also included. Records were excluded if they were duplicates, conference abstracts without an accompanying full-text publication, or isolated case reports unless of landmark mechanistic value. Non-English records without an authoritative translation, or those deemed off-topic after full-text inspection, were also excluded. Citations found in each search were exported to a reference manager and screened in two stages. The first was a title-and-abstract pass against the eligibility criteria, followed by a full-text screen of retained records.

3. Boswellia serrata: botanical and phytochemical background

Boswellia serrata Roxb. ex Colebr. (family Burseraceae) is a deciduous, moderate-sized tree native to the dry hill forests of central and northern India. Its oleo-gum resin, traditionally known as salai guggal, has been used in Ayurvedic medicine to treat joint pain, asthma, and chronic diarrhea for more than two millennia [11,14]. The resin is harvested by making incisions in the trunk and collecting the milky exudate. This exudate hardens on contact with air, forming yellow-to-brown tears. Closely related species - B. sacra, B. carterii, B. papyrifera and B. frereana - yield resins of broadly similar but quantitatively distinct composition. B. serrata is the principal source of the standardised extracts evaluated in arthritis research, and most clinical trials in Osteoarthritis (OA) and Rheumatoid Arthritis (RA) refer specifically to this species [15,16].

The crude oleo-gum resin is a heterogeneous matrix that can be divided, by solubility, into three fractions of comparable mass: a water-soluble polysaccharide gum (approximately 30–60%), a small volatile-oil fraction rich in mono- and sesquiterpenes (5–10%), and a non-water-soluble resin fraction (30–60%) dominated by pentacyclic triterpene acids [11,14]. Pharmacological activity tracks the resin fraction, and within it, a family of pentacyclic triterpene acids of the β-amyrin and α-amyrin (ursane and oleanane) skeletons known collectively as boswellic acids [16,17]. Six principal congeners are routinely quantified: α-boswellic acid (α-BA), β-boswellic acid (β-BA), the corresponding 3-O-acetyl derivatives (acetyl-α-BA and acetyl-β-BA), 11-keto- β -Boswellic Acid (KBA), and 3-O-Acetyl-11-keto- β -Boswellic Acid (AKBA). Of these, AKBA carries a disproportionate share of the in vitro pharmacology and is the molecule against which standardised commercial extracts are titrated; KBA contributes complementary activity and, because it accumulates to

higher plasma concentrations than AKBA, is widely used as a Pharmacokinetic (PK) marker [12,16]. The resin also contains tetracyclic triterpene acids, lupane- and oleanane-type triterpenes, and minor lipophilic constituents, such as incensole acetate; the latter has demonstrable activity in central nervous system models but is not relevant to the arthritis literature considered here [11,15].

A practical result of this chemistry is that, in modern clinical research, "Boswellia serrata extract" almost never refers to crude resin. Quantitative high-performance liquid chromatography analyses of commercial products document up to 100-fold variation in AKBA and KBA content between nominally equivalent supplements, with several preparations falling well short of their label claims [18]. To address this heterogeneity, manufacturers introduced standardised preparations from the late 1990s onwards, titrated to defined percentages of AKBA and total boswellic acids and, in some cases, formulated with phospholipid or self-emulsifying carriers to enhance oral bioavailability [12]. The detailed characterisation of these preparations - 5-Loxin, Aflapin, AprèsFlex, and Casperome - and of their PK behaviour in humans is presented in Section 5 and Table 2; the present section establishes only that boswellic acids, and AKBA in particular, are the molecular entities whose pharmacology determines clinical effect. The mechanisms by which boswellic acids - and AKBA in particular - modulate the inflammatory cascade are detailed in Section 4.

4. Mechanisms of action

The pharmacology of boswellic acids has, over three decades, evolved from a single-target enzyme-inhibition story into an account of multi-target modulation of the inflammatory and proteolytic cascades relevant to Osteoarthritis (OA) and Rheumatoid Arthritis (RA). Principal molecular targets are summarised in Table 1; the present section integrates the biochemical, structural, and animal-model evidence that those entries condense.

Principal pentacyclic triterpene acids of Boswellia serrata, their molecular targets, type of evidence, and key references. AKBA = acetyl-11-keto-β-boswellic acid; KBA = 11-keto- β -boswellic acid; β -BA = β -boswellic acid; PGES-1 = microsomal prostaglandin E synthase-1; IKK = IκB kinase; NF-κB = nuclear factor κB; MMP = matrix metalloproteinase; TLR4 = Toll-like receptor 4; IL-1R = interleukin-1 receptor; HO-1 = haem oxygenase-1; Nrf2 = nuclear factor erythroid 2-related factor 2; COX = cyclo-oxygenase; ADAMTS = a disintegrin and metalloproteinase with thrombospondin motifs.

KBA	5-LOX	Allosteric, non-redox, non-competitive inhibition; structure-activity established	[13,21,22]
AKBA	5-LOX (most potent boswellic acid); IKK / NF-κB	Allosteric 5-LOX site (crystal structure); direct IKK-α/β binding and inhibition	[19,22,24]
β-BA	Microsomal PGES-1; TLR4 / IL-1R signalling in articular cells	Most plasma-bioavailable boswellic acid modulates innate-immune signalling in chondrocytes, osteoblasts, synoviocytes (in silico + in vitro)	[12,35]
AKBA, acetyl-α-BA	Cathepsin G	Reversible, competitive inhibitor; $IC_{50} \sim 600$ nM (within plasma-achievable range)	[37]
Boswellic acids (composite)	MMP-3, MMP-9, MMP-10, MMP-12, MMP-13; ADAMTS / aggrecanase	Reduced expression and activity in microvascular endothelium, OA synovial explants, and articular cartilage	[32,33,36]
AKBA	Nrf2 / HO-1 antioxidant axis	Antioxidant and anti-synovitis effect (rat OA, in vivo); Nrf2 inhibition reverses phenotype	[38]
AKBA	Cyclo-oxygenase pathway	Not a COX inhibitor at pharmacologically relevant concentrations (orthogonal to NSAIDs)	[13]

4.1 5-Lipoxygenase inhibition

The core mechanism, established by Safayhi, Ammon and colleagues, is explicit, specific, non-redox inhibition of 5-Lipoxygenase (5-LOX), the iron-dependent enzyme that converts arachidonic acid to leukotriene A_4 and controls the supply of pro-inflammatory leukotrienes to joint tissue. In intact rat peritoneal neutrophils, AKBA inhibited leukotriene-B_4 formation with a half-maximal Inhibitory Concentration (IC_{50}) of approximately 1.5 μM, while triterpene acids lacking the 11-keto / 3-O-acetyl pattern were inactive; AKBA did not interfere with cyclo-oxygenase or 12-lipoxygenase at comparable concentrations, distinguishing the boswellic acids from Non-Steroidal Anti-Inflammatory Drugs (NSAIDs) at their primary target [13]. Work on purified human 5-LOX showed non-competitive inhibition with respect to arachidonate ($IC_{50} \approx 16$ μM) at a site distinct from the substrate pocket [19]; photoaffinity labelling localised the binding to a calcium-dependent regulatory region [20]; and structure–activity analysis confirmed that the 11-keto group plus a hydrophilic ring-A substituent are required [21]. Gilber et al. [22] resolved the mechanism crystallographically, showing AKBA wedged between the membrane-binding and catalytic domains of stable 5-LOX, approximately 30 Å from the catalytic iron - the structural basis for true allosteric, non-redox inhibition.

A revisionist literature has tempered enthusiasm for 5-LOX as the dominant in vivo target. Siemoneit et al. [23] showed that boswellic acid binding to plasma albumin (95%) sharply attenuates 5-LOX inhibition in whole blood, and that a single 800 mg oral dose of frankincense did not lower plasma leukotriene B_4 in healthy volunteers. Abdel-Tawab et al. [12] concluded that plasma AKBA after standardised extracts rarely exceeds the low-nanomolar range, well below the in vitro IC_{50}, while β-BA, a weaker 5-LOX inhibitor, reaches the highest plasma concentrations of the family. These observations motivated the search for additional plasma-accessible targets, as discussed below.

4.2 NF-κB and the IκB-kinase pathway

A second well-characterised axis represents direct interference with Nuclear Factor-κB (NF-κB) signalling. Syrovets et al. [24] showed that acetyl-α-BA and AKBA bind recombinant IκB-Kinase (IKK)-α and IKK-β, inhibit IKK catalytic activity, prevent phosphorylation of IκBα and Ser-536 of the p65 subunit, and thereby suppress lipopolysaccharide-induced Tumour Necrosis Factor-α (TNF-α) release from human monocytes. The same target operates in vivo: AKBA suppresses tumour growth in androgen-independent prostate cancer xenografts [25], reduces atherosclerotic lesion area and vascular cytokines in lipopolysaccharide-challenged ApoE $^{-/-}$ mice [26], and resolves psoriasiform skin inflammation in CD18-hypomorphic mice when given systemically or topically [27]. Because NF-κB controls the transcription of TNF-α, Interleukin (IL)-1β, IL-6, cyclo-oxygenase-2, inducible nitric-oxide synthase, and Matrix Metalloproteinase (MMP)-3, MMP-9 and MMP-13, a single upstream block at IKK is sufficient to explain a substantial fraction of the downstream anti-inflammatory and chondroprotective effects [17,24].

4.3 Anti-cytokine and immune effects

Cytokine suppression shows both transcriptional and post-translational mechanisms. In a whole-genome screen of TNF-α stimulated human microvascular endothelial cells, Roy et al. [28] found that 113 of 522 induced transcripts - including those encoding vascular cell-adhesion molecule-1, intercellular adhesion molecule-1 and several MMPs — were sensitive to a 30% AKBA-enriched extract. Ammon, 2016, 2010 [10,29] compiled the wider immunomodulatory profile: lymphocyte proliferation, macrophage phagocytosis, Th1/Th17 cytokine output, complement C3 convertase, and antibody-dependent responses are each modulated, with effects directed towards immune dampening rather than suppression of host defence. In rat collagen-induced arthritis, gum-resin extract reduced serum TNF-α, IL-1β, IL-6 and oxidative-stress markers in parallel with reduced paw inflammation [30], with analogous findings in adjuvant-induced rat arthritis using bioenhanced (micellar) preparations [31].

4.4 Anti-cartilage and anti-protease effects

The translation of the anti-inflammatory mechanism into cartilage biology is the most directly OA-relevant axis. Roy et al. [32] showed that 5-Loxin (BE-30, 30% AKBA) reduces TNF-αinduced MMP-3, MMP-10 and MMP-12 expression in microvascular endothelial cells. In mouse destabilised medial meniscus OA, oral and topical boswellic acid attenuated cartilage damage and synovial inflammation, and in vitro,

the same preparation suppressed IL-1-β and Toll-Like Receptor 4 (TLR4) induced inflammatory mediators in OA synovial explants [33]. In a rat monosodium-iodoacetate OA model, standardised gum-resin extract dose dependently reduced joint swelling and restored type-II collagen and aggrecan expression while suppressing MMP-3 and MMP-13 transcription, prostaglandin E_2 and leukotriene B_4 [34]. Franco-Trepat et al. [35] dissected the molecular associations of β-BA in articular cells and showed, by combined in silico docking and in vitro signalling readouts, that it blocks TLR4, IL-1 receptor, NLRP3 inflammasome, NF-κB and MAPK activation in chondrocytes, osteoblasts, and synoviocytes - notable because β-BA reaches the highest plasma concentrations after oral dosing. Direct chondroprotection has been demonstrated in IL-1α/oncostatin-M stimulated bovine cartilage explants, in which a related species (Boswellia frereana) suppressed MMP-9, MMP-13, nitric oxide and prostaglandin E_2 [36]. Beyond MMP transcription, Tausch et al.[37] identified Cathepsin G (catG) - a neutrophil-derived serine protease implicated in joint Extracellular Matrix degradation - as a direct, reversible, competitive target of boswellic acids, with IC_{50} values of approximately 600 nM, well within plasma concentrations achievable after standardised dosing.

4.5 Antioxidant, Nrf2 and additional targets

Boswellic acids additionally engage the cellular antioxidant machinery. Zhou et al. [38] showed that AKBA restrains synovitis in a rat anterior cruciate ligament transection plus destabilised medial meniscus OA model through activation of the nuclear factor erythroid 2-related factor 2 (Nrf2) / haem oxygenase-1 (HO-1) axis, with Nrf2 inhibition (ML385) abolishing the protective phenotype - a causal experiment placing the antioxidant pathway upstream rather than alongside the cytokine effects. Reactive Oxygen Species (ROS) suppression and induction of superoxide dismutase and catalase have been reported in synoviocytes and chondrocytes, as well as in adjuvant arthritis tissue [10,30,31]. Additional protein targets include human leukocyte elastase [29], microsomal prostaglandin-E synthase-1 [12], and topoisomerases I and II [10,17].

4.6 Synthesis: a multi-target framework

Taken together, the mechanistic literature describes boswellic acids - and AKBA in particular - not as selective inhibitors of any single enzyme but as multi-target modulators acting on upstream signalling nodes (5-LOX, IKK–NF-κB, TLR4/IL-1R, Nrf2) and on functionally relevant proteases (catG, MMPs, human leukocyte elastase), as summarised in Table 1. This profile contrasts with the single-target action of NSAIDs (cyclo-oxygenase inhibition); AKBA does not inhibit cyclo-oxygenase at pharmacologically relevant concentrations [8,13], which underlies the boswellia clinical safety profile's difference from that of NSAIDs. The principal residual problem is the gap between the micromolar IC_{50} values defining these mechanisms in cell-free systems and the nanomolar to low micromolar plasma concentrations achieved in humans after oral standardised extracts [12,23]; cathepsin G inhibition (sub-micromolar) and Nrf2/IKK signalling are the most plausible plasma-accessible mechanisms in vivo, while 5-LOX inhibition probably requires tissue concentrations attainable only with bioenhanced formulations.

5. Pharmacokinetics and standardised formulations

The clinical pharmacology of boswellic acids is dominated by poor oral bioavailability. Boswellic acids are highly lipophilic pentacyclic triterpenes (logP>5) of low aqueous solubility, are substrates for intestinal P-glycoprotein (P-gp) efflux, and undergo extensive first-pass metabolism through the cytochrome P450 (CYP) system, so that only a small fraction of an oral dose reaches systemic circulation [12,39]. In healthy volunteers given a standardised Boswellia serrata extract, plasma β-boswellic acid (β-BA) reaches the highest concentrations among the four principal congeners, while AKBA accumulates approximately an order of magnitude below KBA; the reported Time to Maximum Plasma Concentration (Tmax) is 4-8 h, with multiple-hour elimination half-lives [12,40]. The food effect is large: in a randomised cross-over PK study, dosing after a standardised high-fat meal substantially increased the KBA Area Under the Curve (AUC) and Maximum Plasma Concentration (Cmax) relative to fasted dosing, supporting a recommendation that supplements be taken with food [40].

These constraints generate a quantitative gap between the in vitro mechanism and the in vivo exposure. Inhibition of 5-LOX by AKBA in cell-free and whole-cell assays is in the low-range, whereas plasma AKBA after standardised oral dosing remains in the low-nanomolar range and is further reduced by >95% albumin binding [12]. Part of the gap is closed by alternative targets accessible at achievable plasma concentrations: β -BA and AKBA inhibit microsomal prostaglandin E synthase-1 and human cathepsin G with sub-micromolar potency [12,37], and chronic dosing rapidly reaches steady-state. The active pharmacophore in vivo is therefore unlikely to be AKBA acting as a 5-LOX inhibitor alone, but a portfolio of boswellic acids acting on multiple inflammatory checkpoints.

Modern clinical trials accordingly use standardised commercial preparations rather than crude resin (Table 2). 5-Loxin (Sabinsa) is enriched to ≥ 30% AKBA and is the proprietary extract used in the pivotal 90-day knee-OA trial of Sengupta et al. [41]; its preclinical dossier supports doses of 100–250 mg/day [42]. Aflapin (Sabinsa) couples 5-Loxin with the non-volatile oil fraction of B. serrata and shows approximately 52% higher systemic AKBA exposure than 5-Loxin in rats [43]; 100 mg/day produces clinically detectable benefit within 5-7 days [44–46]. AprèsFlex (5-Loxin AF) is a second-generation ≥ 20% AKBA standardisation used in biomarker-rich trials [47]. Casperome (Indena) is a lecithin-based phytosome formulation that increases plasma KBA AUC up to 7-fold and tissue concentrations up to 35-fold relative to the unformulated extract in animal PK [48,49]. Boswellin Super (Sabinsa), a ≥ 30% AKBA preparation, was recently evaluated at 150–300 mg twice daily [50]. Cross-trial dosing comparisons must therefore specify the extract and its standardisation, given the well-documented inter-product heterogeneity in AKBA content discussed in Section 8.

The PK-PD gap is thus partly closed by lipid- and phospholipid-based bioavailability enhancement, by tissue accumulation of lipophilic boswellic acids in synovial and other lipid-rich compartments, and by chronic dosing to steady state.

Standardised Boswellia serrata commercial extracts evaluated in arthritis trials, with composition, typical daily dose, and main references. AKBA standardisation percentages are taken from manufacturer monographs and the cited validation studies; product-to-product variation in AKBA content has been documented [18].

5-Loxin (Sabinsa)	≥ 30% AKBA-enriched B. serrata extract; pivotal proprietary preparation in modern OA trials.	100–250 mg/day	[41,42]
Aflapin (Sabinsa)	5-Loxin synergised with the non-volatile oil fraction of B. serrata; ~52% higher systemic AKBA exposure than 5-Loxin in rats.	100 mg/day	[43–46]
AprèsFlex / 5-Loxin AF (Sabinsa)	Second-generation ≥ 20% AKBA standardisation; used in biomarker-rich knee-OA RCTs.	100 mg/day	[47]
Casperome (Indena)	Lecithin-based phytosome of B. serrata extract; up to seven-fold higher plasma KBA AUC and up to thirty-five-fold higher tissue concentrations versus unformulated extract in animal PK.	250–500 mg/day	[48,49]
Boswellin Super (Sabinsa)	≥30% AKBA-enriched extract evaluated in three-arm dose-ranging knee-OA RCT.	150–300 mg twice daily	[50]
Generic / non-proprietary B. serrata extract	Variable AKBA content; HPLC quantification across commercial supplements has shown up to 100-fold variation between products.	333 mg three times daily (Kimmatkar 2003 historical)	[18,52]

6. Clinical evidence in osteoarthritis

OA is the most thoroughly studied indication for Boswellia serrata. The evidence base is dominated by knee-OA randomised controlled trials (RCTs) and a small but converging set of meta-analyses; quality varies, but larger and more recent studies are well-controlled, and meta-analytic syntheses place Boswellia among the better-supported nutraceuticals for symptomatic OA [9,51]. The principal outcome instruments are the Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC) and the Visual Analogue Scale (VAS) for pain, supplemented by the Lequesne functional index, walking distance, and biomarkers or imaging in a minority of trials.

Foundational evidence comes from Kimmatkar et al. [52], whose 30-patient crossover trial of a generic B. Serrata extract (333 mg three times daily for 8 weeks) reported reduced pain, increased flexion, and longer walking distance compared with placebo. The pivotal modern trial is the 75-patient parallel RCT of 5-Loxin at 100 or 250 mg/day for 90 days, which showed considerable improvements in WOMAC, VAS and Lequesne for both doses, with the higher dose reaching significance within 7 days; synovial fluid Matrix Metalloproteinase-3 (MMP-3) was reduced in the 5-Loxin arms, the first human tissue mechanistic readout in this literature [41]. Vishal et al. [44]

extended the rapid-onset signal: Aflapin 100 mg/day for 30 days in 60 patients produced detectable improvements in WOMAC and VAS by day 5. A head-to-head 90-day trial of 5-Loxin versus Aflapin versus placebo (n = 60) confirmed activity for both, with Aflapin numerically outperforming 5-Loxin, consistent with the higher AKBA exposure documented in Section 5 [45]. The biomarker-rich Aflapin trial of Karlapudi et al. [47] (n = 70, 30 days) added serum reductions in MMP-3, tumour necrosis factor-α (TNF-α), high-sensitivity C-reactive protein (hsCRP), Cartilage Oligomeric Matrix Protein (COMP) and the type-II collagen degradation marker C2C. An 180-day RCT of the standardised SN13108F extract in 80 patients went further by reporting magnetic resonance imaging evidence of preserved cartilage volume and thickness, and improved joint-space width, versus placebo - the first disease-modifying signal in a Boswellia trial [53]. The recent Boswellin Super RCT (150 or 300 mg twice daily, 90 days, n = 105) again reported improvement within five days [50], and a multicentre trial of a curcumin + B. serrata combination in 162 patients with hand OA published in Osteoarthritis and Cartilage extends the indication beyond the knee [54].

Comparator and combination trials situate Boswellia against active controls. The four-arm equivalence trial of Chopra et al. [55], in which 440 knee-OA patients received an Ayurvedic formulation containing Boswellia, a related Ayurvedic formulation, glucosamine sulfate or celecoxib for 24 weeks, found no clinically meaningful WOMAC difference between arms, with the caveat that the Boswellia-containing formulation produced a modest but statistically meaningful rise in serum alanine aminotransferase. Haroyan et al. [56] reported additive benefit of curcumin combined with boswellic acids over either component alone in 201 OA patients in a 12-week three-arm placebo-controlled trial. The MEBAGA trial randomised 120 patients with knee OA to methylsulfonylmethane plus boswellic acids or glucosamine sulfate for 60 days and reported non-inferior symptom relief [57].

Meta-analytic synthesis converges on the same conclusion. The Cochrane review of oral herbal therapies for OA judged the evidence for enriched Boswellia 100 mg/day over 90 days to be of high quality, with a 17-point pain VAS reduction relative to placebo, translating to a number needed to treat of 2 [9]. Yu et al. [51] pooled seven RCTs and 545 patients and reported weighted mean differences of 8.33 on VAS, 14.22 on WOMAC pain, 10.75 on WOMAC function and 2.27 on Lequesne, all favouring Boswellia. Bannuru et al. [58] extended the case to curcumin + Boswellia, and the 2025 network meta-analysis of 39 RCTs and 4,599 knee-OA patients ranked Boswellia highest in probability of being most effective for both pain and stiffness [59]. The British Journal of Sports Medicine systematic review of dietary supplements for OA included Boswellia among a small group with large short-term effect sizes on pain [60].

Across roughly a dozen RCTs and several meta-analyses, standardised Boswellia extracts produce a clinically meaningful, statistically significant reduction in OA pain and improvement in function relative to placebo, with onset within five to seven days for AKBA-enriched and synergised preparations and effects sustained over twelve

weeks; the signal is strongest for AKBA-enriched (5-Loxin, Aflapin, Boswellin Super, SN13108F) and bioavailability-enhanced (phytosome) formulations.

7. Clinical evidence in rheumatoid arthritis

RA is an immune-mediated synovitis whose pathogenic core - TNF-α, IL-1β and IL-6 cytokine drive, NF-κB dependent transcription, leukotriene mediated neutrophil recruitment, and serine-protease mediated matrix degradation maps closely onto the multi-target pharmacology of boswellic acids surveyed in Section 4 [3,10]. AKBA inhibits IKK-NF-κB upstream of the principal RA cytokines; the boswellic acids inhibit cathepsin G with sub-micromolar potency in the plasma-accessible range [37], and 5-LOX inhibition addresses the leukotriene-driven neutrophil chemoinvasion implicated in chronic synovitis. The human controlled-trial base, however, is sparse, dated and contradictory, and the standard of care - methotrexate-anchored disease-modifying therapy, layered with biologics and Janus-Kinase inhibitors - has been transformed since the first Boswellia–RA trials were performed [3,4,61].

Two studies anchor the human RA literature. Etzel, 1996 [62] pooled outcomes from more than 260 patients exposed to the H15 standardised Boswellia serrata extract and reported reductions in joint swelling, pain and erythrocyte sedimentation rate; the design is best characterised as a case-collection summary and carries the methodological weaknesses of its era. The definitive controlled study is the 12-week double-blind RCT of Sander et al. [63], in which 78 patients with chronic polyarthritis received H15 at 3,600 mg per day or placebo as an add-on to existing therapy: no significant improvement was seen on the Ritchie articular index, erythrocyte sedimentation rate, C-reactive protein or NSAID consumption, despite acceptable tolerability. In addition to these data, the herbomineral crossover RCT of Kulkarni et al. [64] (Articulin-F, combining Withania somnifera, Boswellia serrata, Curcuma longa, and a zinc complex; n = 42) showed significant reductions in pain and disability and is frequently cited in the RA-adjacent literature, even though the index population was osteoarthritic. No adequately powered modern RCT of a standardised AKBA-enriched extract in RA has yet been published.

Preclinical RA models keep the mechanistic case alive. In rat collagen-induced arthritis, oral B. serrata extract reduced paw oedema, serum TNF-α, IL-1β, and IL-6 levels, oxidative stress markers, and attenuated cartilage erosion [30]. Adjuvant-induced arthritis in rats responded to a micellar curcumin/boswellic acid combination with an anti-inflammatory effect comparable to that of diclofenac [31]. Choudhary et al. [65] tested an amino analogue of β-boswellic acid (BA-25) in combination with low-dose methotrexate in collagen-induced arthritis and reported additive cytokine suppression and improved arthritic-score reduction, articulating the contemporary preclinical rationale for combining standardised boswellic-acid preparations with conventional Disease-Modifying Antirheumatic Drug (DMARD) therapy rather than substituting for it.

The pragmatic clinical position is therefore conservative. Standardised Boswellia serrata extracts can reasonably be considered as adjuncts in RA for symptomatic relief and NSAID-sparing, and as a mechanistically rational accompaniment to methotrexate or biologics; they cannot, on present evidence, be considered substitutes

for DMARD therapy, and they have not been shown to alter radiographic progression, disability accrual or cardiovascular risk [3,4,61]. The most consequential gap is methodological: adequately powered, multi-centre, manufacturer-independent RCTs of AKBA-enriched or phytosomal extracts as an add-on to methotrexate, evaluated against the American College of Rheumatology (ACR) response criteria (ACR-20/50/70), the Disease Activity Score-28 (DAS28), the Health Assessment Questionnaire-Disability Index (HAQ-DI) and radiographic progression, are urgently needed before any firmer recommendation can be made.

8. Safety, drug interactions plus regulatory status

Across the RCT literature reviewed in Sections 6 and 7, standardised Boswellia serrata extracts have been generally well tolerated, with Adverse Event (AE) rates not significantly different from placebo and no serious AE causally attributed to the extract [41,44,47,50,52]. The most frequently reported events are mild, dose-related upper gastrointestinal symptoms (nausea, epigastric discomfort, diarrhoea), occasional headache, and rare allergic skin reactions [9,66,67]. Tolerability of chronic dosing in the range used for joint disease is also supported by the inflammatory bowel disease literature, where 6-week courses of Boswellia gum resin (350 mg t.i.d.) achieved remission rates comparable to those of sulfasalazine in active ulcerative colitis, without excess adverse events [68].

A weak hepatic signal has emerged from one large pragmatic study and isolated case reports. In the 24-week, 440-patient Ayurvedic-equivalence trial by Chopra et al. [55], asymptomatic and reversible elevation of serum alanine aminotransferase was documented in 26 patients in the Boswellia-containing arms, leading to 7 protocol-mandated withdrawals; transaminases normalised on discontinuation. Finsterer, 2024 [69] reported a single case of acute Boswellia-associated syndrome of inappropriate antidiuretic hormone secretion with seizure and rhabdomyolysis after self-medication. Periodic liver function monitoring on chronic high-dose therapy and avoidance in pre-existing severe hepatic disease are reasonable precautions.

Drug-interaction risk centres on modulation of Cytochrome P450 (CYP) and efflux transporters. Boswellic acids inhibit several CYP isoforms in vitro; in diabetic rats, Samala et al. [70] showed that boswellic acids increased the plasma exposure and hypoglycaemic effect of glyburide, kinetics consistent with CYP3A-mediated interaction. Boswellic acids also modulate P-glycoprotein function in vitro [39], raising a theoretical interaction with narrow-therapeutic-index P-glycoprotein substrates. Patients on calcineurin inhibitors, certain statins, or other narrow-therapeutic-index CYP3A4 substrates should therefore be flagged for closer monitoring during initiation; warfarin, principally a CYP2C9 substrate, also warrants caution given reports of boswellic-acid effects on multiple CYP isoforms. Combination with NSAIDs, by contrast, is pragmatic rather than additively toxic: pooled and meta-analytic data show that Boswellia tends to reduce concomitant NSAID consumption rather than potentiate gastric or renal injury [9,58].

Data in special populations are limited: pregnancy and lactation use is conventionally avoided for want of adequate human safety data, paediatric data are scarce, and no formal renal or hepatic dose adjustment is established. Manufacturer-sponsored

subchronic toxicology of 5-Loxin and Aflapin in rats supports a no-observed-adverse-effect level several-fold above the human therapeutic dose [42,46].

The regulatory status of Boswellia serrata reflects this uneven evidence base. In the United States and the European Union, Boswellia preparations are sold as dietary or food supplements; no major regulator has approved Boswellia as a prescription medicine for an arthritis indication, and no European Medicines Agency Committee on Herbal Medicinal Products monograph for arthritis use exists at the time of submission. ESCOP has issued a herbal monograph, and the AYUSH ministry of India classifies Boswellia as a recognised Ayurvedic drug [11,14,15]. A persistent quality-control concern is between-product heterogeneity: Miscioscia et al. [18] documented up to roughly 100-fold variation in AKBA and KBA content between commercial products labelled as Boswellia, including some preparations effectively devoid of the AKBA pharmacophore. Clinicians and trialists should prefer products with third-party verified standardisation, ideally matching the proprietary preparations validated in published RCTs.

9. Discussion

The evidence assembled in this review converges on a coherent characterisation of Boswellia serrata, and AKBA in particular, as a multi-target plant-derived anti-inflammatory whose clinical signal is most consistent in symptomatic OA and provisional in RA. The mechanistic argument is reproducible across in vitro, animal and limited human pharmacokinetic data: 5-LOX inhibition resolved at atomic resolution [22], IKK–NF- κ B suppression and downstream cytokine attenuation [10,24], anti-MMP and anti-cathepsin-G activity bridging inflammation to matrix degradation [32,37], and Nrf2/HO-1-mediated antioxidant rescue in synovitis [38]. This multi-axis modulation is precisely the profile expected to generate effects on pain and function exceeding those of single-target agents, and the standardised-extract era (5-Loxin, Aflapin, AprèsFlex, Casperome, Boswellin Super) has improved the reproducibility of clinical outcomes relative to the heterogeneous, early, crude-resin trials [9,10,22].

The class differs from NSAIDs in molecular target as well as in adverse event profile. AKBA does not inhibit cyclo-oxygenase at pharmacologically relevant concentrations [10,13], so the two classes act through partly orthogonal pathways. Comparator and combination trials in OA - the equivalence trial of Chopra et al. [55], the curcumin + boswellic acid versus celecoxib comparison of Haroyan et al. [56], and the meta-analytic synthesis of Bannuru et al. [58] - suggest comparable symptomatic relief with a more favourable gastrointestinal side-effect signature and lower NSAID consumption, against the background of established NSAID-related peptic ulcer and bleeding risk [7]. Boswellia therefore fits a clinical niche as an adjunct or alternative for OA patients with NSAID intolerance or contraindication, but it does not displace the established roles of paracetamol, topical NSAIDs or intra-articular corticosteroids set out in current OA guidelines [5,6].

By contrast, Boswellia is not a DMARD and does not arrest RA structural progression as methotrexate or anti-TNF biologics do [3,4,61]. Its probable RA role, based on present data, is adjunctive - symptomatic and NSAID-sparing - and any patient with

active RA must remain on appropriate disease-modifying therapy. The preclinical combination data from Choudhary et al. [65] for a boswellic acid analogue plus methotrexate support the rationale for a therapeutic approach of combination rather than substitution.

Several strengths support the OA case. A reproducible mechanistic story spans in vitro, animal and human PK data; multiple well-controlled RCTs of standardised extracts converge on the same direction of effect on WOMAC, VAS and Lequesne endpoints; biomarker-rich studies ([47,53]) link symptomatic improvement to reductions in serum MMP-3, hsCRP, TNF- and cartilage-turnover markers, and Kumar et al. [53] provides the first MRI-based disease-modifying signal at six months; and short-term safety is reassuring across the systematic-review literature [9,66,67].

Limitations temper these strengths. Most OA trials are small (n 100) and short (4–12 weeks), with only Chopra et al. [55] and Kumar et al. [53] extending to 24 weeks or six months; the RA RCT base is essentially limited to the methodologically dated case-collection of Etzel, 1996 [62] and the negative pilot RCT of Sander et al. [63]; manufacturer involvement is documented in many landmark RCTs and creates a credible bias signal even when individual trials are well-conducted; the in-vitro to in-vivo concentration gap - micromolar IC_{50} values for several mechanisms versus low-micromolar-to-nanomolar plasma boswellic-acid levels after standardised oral dosing [12,23] - is only partly closed by tissue accumulation and bioavailability enhanced formulations; no major regulator has approved an arthritis indication; and product heterogeneity, with up to roughly 100-fold variation in AKBA between commercial preparations [18], means that “Boswellia serrata” without an extract specification is not a defined intervention.

Five research priorities follow. First, an adequately powered, multi-centre, manufacturer-independent Phase III OA RCT with standardised endpoints (WOMAC, VAS, MRI-based cartilage volume and joint-space width, NSAID-sparing) would consolidate or refute the current effect size estimates and resolve the disease-modification question raised by Kumar et al. [53]. Second, modern RA RCTs of AKBA-enriched or phytosomal extracts as adjuncts to methotrexate, evaluated against ACR-20/50/70, DAS28, HAQ-DI and radiographic progression, are required before any RA recommendation can be firmed up beyond the conservative posture of Section 7. Third, head-to-head bioavailability comparisons of AKBA-enriched micellar and phytosome formulations in adequately powered crossover designs would translate the in vitro-to-in vivo gap into actionable dosing recommendations. Fourth, mechanistic clarification of the in vitro versus in vivo concentration gap - through tissue PK in synovial-fluid sampling, identification of active metabolites and quantification of plasma-protein binding - would close the residual PK–PD discordance flagged across this review [12,23]. Fifth, long-term safety surveillance, including registry-based capture of hepatotoxicity [55,69] and CYP-mediated drug interactions [39,70], is needed to convert short-term tolerability into a reliable chronic-use safety profile.

10. Conclusion

Boswellia serrata is regarded as a multi-target plant-derived anti-inflammatory whose pharmacology rests on a reproducible quartet of mechanisms - 5-LOX inhibition, IKK–NF-κB suppression, anti-cytokine and anti-MMP activity and Nrf2-mediated antioxidant rescue - with AKBA as the dominant bioactive across these axes. The practical clinical takeaway is that standardised AKBA-enriched extracts (5-Loxin, Aflapin, AprèsFlex, Boswellin Super) and bioavailability enhanced phytosome formulations (Casperome) deliver clinically meaningful symptomatic improvement in osteoarthritis with a favourable short-term safety profile [9], while in rheumatoid arthritis, the role is mechanistically attractive but evidence-light and strictly adjunctive to disease-modifying therapy [10]. Substantial limitations remain: the rheumatoid arthritis randomised controlled trial base is sparse and dated; long-term and disease-modifying effects are understudied; manufacturer involvement complicates multiple pivotal trials; the in vitro-to-in vivo pharmacokinetic gap is incompletely resolved; and between-product heterogeneity in AKBA content undermines comparability across the literature. Adequately powered, manufacturer-independent, multi-centre randomised trials of well-characterised, standardised extracts, with explicit reporting of extract composition and modern outcome instruments, are the priority for closing these gaps and translating mechanistic promise into firm clinical recommendations.

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During the preparation of this work, the authors used generative AI to assist with grammar and stylistic editing to ensure appropriate academic language and for translation into English. After using this tool, the authors reviewed and edited the content as needed and take full responsibility for the final content of the manuscript.

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