Naveen Shodh Sansar (An International Refereed / Peer Review Multidisciplinary Research Journal)
RNI No.- MPHIN/2013/60638, ISSN 2320-8767, E- ISSN 2394-3793, Scientific Journal Impact Factor (SJIF)- 8.054,
July to September 2026, E-Journal, Vol. I, Issue LV (55), ISO 9001:2015 - E2024049304 (QMS)

Phytochemical Profiling and Anti-Osteoarthritic Potential of Selected Indian Medicinal Plants: An In Vitro Evaluation

Dr. Ragini Sikarwar*

***Assistant Professor and HOD (Botany) Govt. Home Science PG Lead College, Narmadapuram (M.P.) INDIA**

Abstract: Osteoarthritis (OA) is, honestly, the most common joint disorder worldwide, and right now pharmacological care depends quite a lot on non-steroidal anti-inflammatory drugs (NSAIDs), which come with GI, renal and cardiovascular risks that are very well known especially when they are taken for a long time. Because of that, researchers have shown renewed interest in medicinal plants that were used for joint problems in Indian traditional medicine for a long while. This narrative review tries to bring together what in vitro work says about the anti-osteoarthritic capacity of six frequently studied Indian medicinal plants: Curcuma longa, Boswelliaserrata, Zingiberofficinale, Withaniasomnifera, Tinosporacordifolia, and Trigonellafoenum-graecum. A structured search across Scopus, PubMed, and Google Scholar was done to capture in vitro pharmacological studies. In the material that was reviewed, phytochemicals linked with these plants tended to reduce pro-inflammatory signals like interleukin-1β, interleukin-6, tumour necrosis factor-alpha and prostaglandin E2. They also blocked cartilage-wear enzymes such as cyclooxygenase-2, 5-lipoxygenase, and matrix metalloproteinases, plus in multiple chondrocyte and cartilage-explant setups, they kept cartilage matrix integrity markers. Still, the mechanistic strength doesn't look the same across all plants, and only a small number of studies compare standardized extracts, under basically the same assay conditions. Overall, this review lays out the evidence as it stands, points to methodological shortcomings, and considers whether these botanicals could be used in translation, either as add-ons or as an alternative to routine pharmacotherapy for osteoarthritis.
Keywords: Indian medicinal plants, in vitro, anti-inflammatory,osteoarthritis, phytochemicals.

Introduction - Walk into any orthopaedic outpatient clinic on a busy morning, and a good share of the people waiting will be there for the same reason knees, hips, or hands that ache more than they used to. Osteoarthritis has quietly become one of the largest contributors to disability in ageing populations, too. A 2024 analysis from the Global Burden of Disease Study 2021 put the worldwide prevalence of osteoarthritis at roughly 607 million people, and disability-adjusted life years have kept climbing since 1990.[1] The knee remains the joint that is most often affected, and the burden falls disproportionately on women, also on older adults. [1,2] India, with its large and rapidly ageing population, is not exempt from this trend. Joint pain also ranks among the most frequent complaints seen in both allopathic and Ayurvedic clinical practice.

At the tissue level, osteoarthritis is no longer viewed just as "wear and tear" not even close. Cartilage breakdown is now considered an active inflammatory process, and not only passive degeneration. Inflammatory mediators, including pro-inflammatory cytokines like interleukin-1β (IL-1β) and tumour necrosis factor-alpha (TNF-α), can drive the release of matrix metalloproteinases (MMPs) and aggrecanases. Those then degrade collagen, and proteoglycans in the joint. [3,4] This inflammatory framing matters because it opens the door to pharmacological strategies that go beyond pain relief, and instead target the biochemical drivers behind cartilage loss.

The trouble is that the drugs people most commonly reach for, non-steroidal anti-inflammatory drugs (NSAIDs), well they're kind of a blunt tool. They help, but not without some kind of price. If you use NSAIDs for a long time it's linked with gastric ulceration, and bleeding, also cyclo-oxygenase-2 (COX-2) selective agents seem to come with a higher risk of myocardial infarction and stroke.[5] So clinicians treating older patients (and they are the majority in the osteoarthritis crowd) end up sort of stuck between symptom control and drug safety, like juggling two uneven weights. This push-pull, is exactly why researchers have kept circling plant derived alternatives, the kind that could give anti-inflammatory activity but with a gentler side-effect profile.

India's traditional pharmacopoeia is, honestly, a pretty

obvious place to look. Ayurvedic writings mention dozens of plants for joint pain and swelling, and quite a few of them, like turmeric, boswellia, ginger, ashwagandha, guduchi, and fenugreek—these, have gotten serious pharmacological attention over time. What's shifted in roughly the last two decades is that there are now cell-based and tissue-based in vitro models that let researchers ask a more precise question than "does this herb help arthritis." Instead, they can ask: which specific cytokines, enzymes and signalling pathways are involved, and in what concentration? This review pulls together the in vitro evidence for six of the most heavily studied Indian medicinal plants, with the goal of giving readers a clear mechanism-level view of what has actually been shown in the lab, rather than relying on folklore or anecdote.

Search Methodology: A literature search was conducted across Scopus PubMed/ MEDLINE, and Google Scholar for studies published up to 2026. The search terms combined the common and botanical names of six plants (Curcuma longa / turmeric Boswelliaserrata / salaiguggul, Zingiberofficinale / ginger Withaniasomnifera / ashwagandha, Tinosporacordifolia / guduchi, Trigonellafoenum-graecum / fenugreek) along with terms such as "osteoarthritis", "chondrocyte", "in vitro", "anti-inflammatory", "cartilage", and "cytokine". Studies were included if they reported an in vitro pharmacological evaluation like a cell line, primary chondrocyte, cartilage explant, or an enzyme/protein assay, that related to osteoarthritis-associated inflammation or cartilage catabolism. In vivo animal studies and clinical trials were included only when they were necessary to contextualise the in vitro findings, and they were clearly marked as such within the text. Reviews meta-analyses, and epidemiological reports were also used for background information and burden-of-disease data. Finally, reference lists from the retrieved articles were hand searched, for extra relevant primary studies.

Osteoarthritis: Pathophysiology and the Limits of Current Pharmacotherapy: Cartilage inside a healthy joint stays in like this quiet equilibrium between making new material and breaking it down. The chondrocytes, the one cell type that really lives there, keep producing type II collagen and aggrecan , while at low levels catabolic activity quietly clears matrix that has seen better days. Osteoarthritis however tilts that balance. Mechanical loading, ageing, extra body weight, and any joint injury nudge chondrocytes along with synovial macrophages to release IL-1β and TNF-α , which then promote cyclo-oxygenase-2 (COX-2), inducible nitric oxide synthase, and a whole cascade of matrix metalloproteinases, especially MMP-13 and MMP-3, plus aggrecanases like ADAMTS-5.[3,4,6] The overall outcome is gradual loss of collagen and proteoglycan, joint space getting smaller, and the pain plus stiffness that patients really, actually feel.

In terms of guideline- recommended pharmacotherapy, most of it leans on oral and topical NSAIDs, sometimes paired with intra-articular corticosteroids or hyaluronic acid. NSAIDs work for pain, pretty effectively in the short run, but the safety picture is not great. Both selective and non-selective NSAIDs have a higher chance of gastrointestinal ulceration, and COX-2 inhibitors in particular link to increased cardiovascular risk, so strong that regulators have limited their use in people who already have cardiovascular disease.[5] Since osteoarthritis tends to affect older adults, who also more often carry cardiovascular comorbidities, there's a genuine shortage of safer long-term options for disease management. It's that shortage, more than mere tradition, that has pushed renewed interest in botanical preparations that can tone down similar inflammatory pathways without the same organ-system downsides.

Indian Medicinal Plants Studied for Anti-Osteoarthritic Potential

Curcuma longa (Turmeric): Turmeric's active curcuminoid, curcumin, is probably the most studied phytochemical across this whole area, and the in vitro results are accordingly plentiful. In a transcriptomic study on primary human osteoarthritic chondrocytes, grown from seventeen patients, curcumin down-regulated more than a hundred genes tied to the inflammatory response, including NF-kB1/2, multiple chemokines, and both COX-1 and COX-2 , while also suppressing MMP-13, MMP-1 , and ADAMTS-5 expression.[7] The same report also described curcumin activating the Nrf2 antioxidant pathway, effectively turning on genes tied to detoxification and cytoprotection within only six hours after dosing.[7] Earlier work from Mathy-Hartert and coworkers already suggested that curcumin curbed pro-inflammatory messenger release, and reduced MMP-3 production in cultured chondrocytes, which gives some mechanistic support to what clinical trials were later, and separately, observing.[8]

That clinical story is still worth saying, just a bit, because it lines up with the in vitro findings, not really standing off by itself. Systematic reviews and randomized trials have described meaningful decreases in knee pain and joint effusion when using curcumin or Curcuma longa extracts, and in a few trials the effect size came close to standard NSAIDs. [9,10,11,12,13] Of course, none of this is proof that curcumin produces the exact same mechanism seen in cell culture, but the overlap between what happens in cells and what happens in patients is, overall, more reassuring than trusting either type of evidence alone.

Boswelliaserrata (Indian Frankincense, SalaiGuggul) Boswellia pharmacology kind of turns around boswellic acids, it's a family of pentacyclic triterpenes, where 3-O-acetyl-11-keto-β-boswellic acid (AKBA) is the one that gets studied the most. Where NSAIDs are usually redox oriented in their behavior, boswellic acids work as non-redox inhibitors of 5-lipoxygenase (5-LOX) , this enzyme is involved in making pro-inflammatory leukotrienes, and they don't just block cyclo-oxygenase in a straight, direct

way.[14,15] In a test with human blood cells, a standardized Boswellia extract inhibited 5-LOX with an IC50 of about 43 µg/mL, and it toned down leukotriene B4 plus prostaglandin E2 production at concentrations that were even lower.[16] The same extract also shielded SW1353 chondrosarcoma cells from IL-1β-triggered loss of SOX-9, which is a transcription factor pretty important for keeping chondrocyte identity and cartilage matrix production, at doses as low as 1 to 5 µg/mL.[16]

Mechanistically not the same as curcumin, but still sort of complementary, boswellic acids additionally inhibit human cathepsin G, a protease associated with cartilage matrix breakdown, and they suppress NF-kB activation.[17,18] A transcriptomic check comparing Curcuma longa versus Boswelliaserrata extracts on the same batch of osteoarthritic chondrocytes suggested that both treatments reduced inflammatory and catabolic gene expression, but through mostly different routes. Curcumin seemed to run via Nrf2 and arachidonic acid metabolism, while boswelia leaned more toward Toll-like receptor down-regulation and autophagy induction, which gives a reasonable biologic logic for putting the two together.[7]

Zingiberofficinale (Ginger): Ginger's pungent phenolic substances—mostly 6-gingerol and its dehydration byproduct 6-shogaol—have been tried across a wider spread of in vitro joint tissue models than maybe any other plant mentioned in this review. A rather thorough 2024 review listed in vitro work involving fibroblast-like synoviocytes and chondrocytes, and it reported that ginger extracts and isolated single compounds consistently dampened TNF-α, COX-2, and prostaglandin E2 output in synovial cells from both osteoarthritis and rheumatoid arthritis people.[19] In one of the papers gathered there, zingerone suppressed MMP-13 levels in both SW1353 cells and in explanted pig cartilage, which really pins the compound to cartilage preserving effects rather than just symptom easing, pain relief if you will.[19]

Also, when ginger extract was applied to C28I2 human chondrocytes it lowered reactive oxygen species and lipid peroxidation triggered by IL-1β, and it also increased the cells' own antioxidant enzymes like superoxide dismutase, glutathione peroxidase, and catalase.[19] Separately, 6-shogaol decreased nitric oxide, MCP-1, and IL-6 release from human chondrocyte cultures.[19] Overall, these observations place ginger in the role of doing multiple things at once: cytokine downshifting, blocking enzyme activity, and providing direct antioxidant fortification inside the chondrocyte itself.

Withaniasomnifera (Ashwagandha): Ashwagandha root is traditionally prescribed in Ayurveda for joint pain, and its withanolide rich extracts have been tested directly on human osteoarthritic cartilage, rather than on immortalized cell lines, which kind of gives the resulting data a different flavor all together. Sumantran and colleagues used a validated explant model of cartilage damage , exposing knee cartilage taken from osteoarthritis patients to an aqueous root extract, then measuring nitric oxide and glycosaminoglycan release as markers of inflammation and matrix breakdown.[20] The extract significantly reduced nitric oxide release in roughly half of the patient samples tested, and the authors spell this out pretty candidly as patient dependent rather than universal, which honestly is refreshingly rare across this kind of literature.[20]

That patient-to-patient variability is, on its own, a useful signal. It implies that whatever mechanism ashwagandha is driving through, might be tied to the baseline inflammatory state or to genetic factors that shift from one person to another. It is a nuance that is easy to lose when studies report only group averages. It also helps explain why in vitro explant models, messy as they can be, sometimes tell us more about real world variation than a tidier immortalized cell assay would.

Tinosporacordifolia (Guduchi): Guduchi gets used in a bunch of traditional Indian arthritis formulations, and its in vitro anti-inflammatory effects are shown in a clearer way mostly in immune-, and macrophage-based settings, not so much in chondrocyte cultures by themselves. For example in THP-1 monocytic cells, a chloroform extract from Tinosporacordifolia was reported to suppress lipopolysaccharide induced NF-kB activation , and then it reduced downstream pro inflammatory cytokine output.[21] Separately, in a rat autoimmune arthritis model, giving the extract brought down joint and cartilage harm, and the authors also noted changes in immune mediator patterns, they then tied those outcomes back to the in vitro immunomodulatory activity of the same extract on cultured immune cells.[22]

Because the evidence for guduchi seems to sit more heavily on immune cell assays and adjuvant arthritis models, rather than on direct chondrocyte work or cartilage explant experiments, its anti-osteoarthritic promise is better framed as plausible. It fits the mechanistic picture that matches NF-kB driven inflammation, rather than being shown directly inside cartilage tissue. So yeah, this is a real evidence gap, not a small footnote. A more targeted chondrocyte study would really help the argument a lot.

Trigonellafoenum-graecum (Fenugreek) and Other Supporting Botanicals: Fenugreek seed extracts have shown anti-inflammatory, anti-arthritic activity in petroleum ether and mucilage fractions, mostly due to linolenic and linoleic acid content.[23] A lot of the mechanistic work that exists though, was done in adjuvant-induced arthritis models in rats, rather than in isolated chondrocyte systems, so, like guduchi, fenugreek's evidence base for osteoarthritis, is still kind of indirect. Vitexnegundo (nirgundi) shows up a lot in Ayurvedic joint-care combinations and it's traditionally associated with anti-inflammatory effects in rheumatic as well as osteoarthritic contexts, however, strong in vitro data at the chondrocyte-level that is specific to this plant, seems comparatively limited in the peer-reviewed literature. This

is still a spot where more basic primary research would really help, and it kind of stands out.

In Vitro Methods Commonly Used to Evaluate Anti-Osteoarthritic Activity: Fenugreek seed extracts show anti-inflammatory, anti-arthritic activity in petroleum ether and mucilage fractions , mainly because of linolenic and linoleic acid content. [23] A bunch of the mechanistic work that exists though, was done in adjuvant induced arthritis models in rats, not really in isolated chondrocyte systems so, like guduchi, fenugreek's evidence base for osteoarthritis is still sort of indirect.

Vitexnegundo (nirgundi) keeps appearing in Ayurvedic joint care combinations and it's historically tied to anti-inflammatory effects in rheumatic settings as well as osteoarthritic ones, but the stronger in vitro data at the chondrocyte level that is directly specific to this plant, seems a bit less robust in the peer reviewed literature. This remains one of those areas where more basic primary research could genuinely clarify things, and it kind of stands out.

These two assays are pretty cheap, they are quick and widely used for the same reason, because they basically don't need any specialised cell culture facilities, and that's also why they show up so often in early-stage screening work on Indian medicinal plants. But honestly, they are also blunt instruments. They tell you next to nothing about which exact cytokine, or enzyme pathway is going on, and if you get a positive result in a protein-denaturation assay that doesn't automatically mean you'll see activity in a living chondrocyte. The more mechanistically informative studies mentioned earlier in this review, like RNA-sequencing on primary chondrocytes, cytokine immunoassays, enzyme-specific inhibition assays, are kind of at the other end of the spectrum compared to these screening tools. So readers looking through this literature should weigh the evidence accordingly, rather than treating each single "in vitro anti-inflammatory" statement as if it were the same as the others.

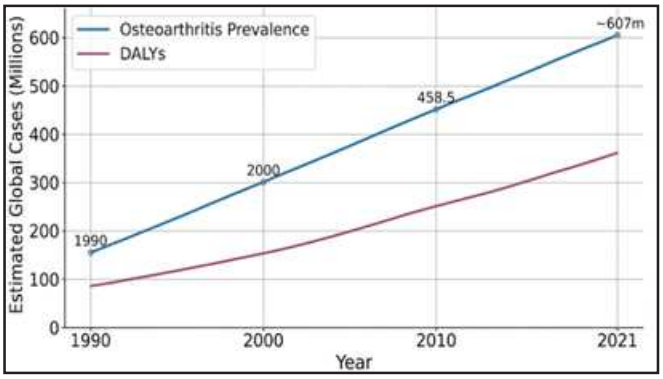


Figure 1: Global Burden of Osteoarthritis, 1990-2021

Thats chart kind of shows how global age-standardized osteoarthritis prevalence and disability-adjusted life years, (DALYs) kind of moved upward from 1990 to 2021. It uses Global Burden of Disease Study 2021 estimates, so thats the basis. On the x-axis you can see the years 1990, 2000, 2010, and 2021, and on the y-axis it shows the estimated worldwide cases in millions, and those numbers climb from a smaller starting point in 1990 to around 607 million cases by 2021. The main idea is, osteoarthritis prevalence keeps rising in step with population ageing, so it backs up the need for safer, sustainable, longer term management choices. The data comes from Li et al. Frontiers in Medicine 2024, (Global Burden of Disease Study 2021).[1]

Comparative Summary of In Vitro Findings

Table 1 summarises the plants their principal phytochemicals the in vitro models used to test them and the core anti-osteoarthritic findings reported in the literature reviewed above. If you read across the table, a sort of pattern shows up, nearly every plant seems to land on the same handful of targets, NF-êB signalling, COX/LOX enzymes, and matrix metalloproteinases. Even so, the specific phytochemicals are structurally not really related to each other. That overlap is arguably the most convincing part of this whole set of work, because it points to these plants not just doing some fuzzy, non-specific "antioxidant" thing, but rather nudging the same inflammatory machinery that conventional OA drug development already aims at.

Table 1 (see in last page)

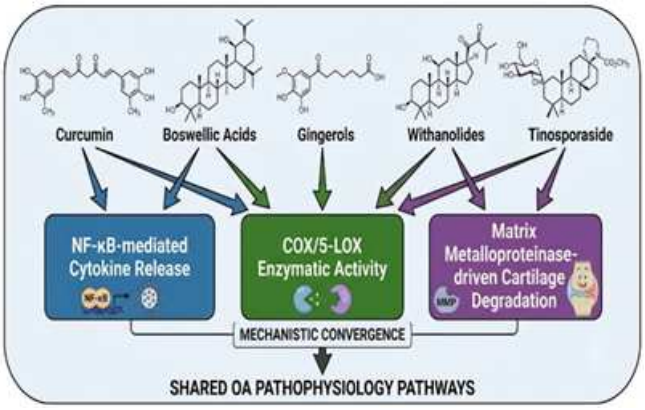


Figure 2: Convergent Molecular Targets of Indian Anti-Osteoarthritic Phytochemicals

This conceptual diagram kinda shows how phytochemicals from the six reviewed plants end up meeting at a shared set of molecular targets inside the osteoarthritic joint. Curcumin, boswellic acids, gingerols, withanolides, and tinosporaside seem to be feeding into three central nodes: NF-êB mediated cytokine release, COX/5-LOX enzymatic activity, and matrix metalloproteinase driven cartilage degradation. The key idea, as the diagram suggests, is that even if these compounds look chemically kinda different, they still largely land on the same core inflammatory and catabolic routes that are already linked to osteoarthritis pathophysiology. This is grounded in mechanistic findings put together from Sanchez et al. (2022), Alluri et al. (2020), and Szymczak et al. (2024). [7,16,19]

Discussion: Stepping back a bit from the plant-by-plant detail, a few broader observations sort of pop out. First, the quality of evidence is honestly uneven. Curcumin and

boswellic acids have been investigated in primary human osteoarthritic chondrocytes with modern transcriptomic tools, so you get a fairly granular mechanistic picture.[7] Ginger is pretty close behind, with a substantial body of synoviocyte and chondrocyte work.[19] Ashwagandha's evidence is real too, but it comes out of a single explant study, and patient response varied.[20] Guduchi and fenugreek more or less lean on immune-cell and whole-animal models rather than direct cartilage or chondrocyte data. So anyone citing this literature to claim that "Indian medicinal plants treat osteoarthritis" as one single monolithic thing, is kinda skipping over the fact that the proof is on very different footing, in a meaningful way.

Second, dose and bioavailability are still unresolved issues that in vitro data alone just cannot settle. The concentrations that work in cell culture—low single-digit micromolar for curcumin, tens of micrograms per millilitre for boswellic acids—do not automatically become achievable plasma concentrations after oral dosing, especially since curcumin has notoriously poor oral bioavailability.[7] Several studies included here tried to choose concentrations that line up with reported plasma levels after oral extract administration, which definitely helps translational relevance, but that approach is not yet consistent across the field.

Third, combination effects deserve more attention than they currently do, in my honest opinion. The one study in this review that really compared two plant extracts side by side—curcumin and boswellic acid on the same chondrocyte cultures— found that the combination outperformed either extract alone on a meaningful number of genes, also that the two extracts seemed to work through mostly non-overlapping pathways.[7] Since many traditional Indian formulations already stack multiple herbs, rather than using any single plant in isolation, this kind of head-to-head, mechanism-resolved comparison is exactly the sort of investigation the field needs more of. Not, you know, yet another single-extract screening paper with only partial context.

Finally, it is worth being clear about what in vitro data can and cannot tell us. Cell culture and explant models strip away the complexity of a living joint, systemic drug metabolism, immune cross-talk, mechanical loading. They also can't substitute for well-designed clinical trials, full stop. Where clinical data exist alongside the in vitro work, as with curcumin, the two streams of evidence broadly agree, which is reassuring. [9,10,11,12,13] Where clinical data are thin or missing, as with guduchi and fenugreek specifically for osteoarthritis, the in vitro findings should be read as hypothesis-generating, not confirmatory.

Conclusion: The in vitro evidence reviewed here makes a reasonably strong case that several Indian medicinal plants act on the same inflammatory an catabolic pathways implicated in osteoarthritis pathogenesis, chiefly NF-êB signalling, COX/5-LOX enzyme activity, an matrix metalloproteinase expression. Curcumin and boswellic acids currently have the most mechanistically detailed support, with ginger not far behind; ashwagandha, guduchi and fenugreek show promising but comparatively thinner direct chondrocyte-level evidence. None of this is a substitute for well-powered clinical trials. Still the field would benefit quite a lot from standardized extract concentrations, head-to-head comparisons, and more stress on primary chondrocyte and cartilage explant models, over less specific screening assays. With those caveats in place, these plants remain a scientifically credible, and also traditionally grounded, avenue for developing safer adjunctive therapies for osteoarthritis.

References:-

1. Aidoo DB, Konja D, Henneh IT, Ekor M. Protective effect of bergapten against human erythrocyte hemolysis and protein denaturation in vitro. Int J Inflam. 2021;2021:1279359.
2. Allen KD, Thoma LM, Golightly YM. Epidemiology of osteoarthritis. Osteoarthritis Cartilage. 2022;30(2):184-195.
3. Alluri VK, et al. An anti-inflammatory composition of Boswelliaserrata resin extracts alleviates pain and protects cartilage in monoiodoacetate-induced osteoarthritis in rats. Evid Based Complement Alternat Med. 2020;2020:7381625.
4. Ammon HP. Boswellic acids and their role in chronic inflammatory diseases. AdvExp Med Biol. 2016;928:291-327.
5. Bannuru RR, Osani MC, Al-Eid F, Wang C. Efficacy of curcumin and Boswellia for knee osteoarthritis: systematic review and meta-analysis. Semin Arthritis Rheum. 2018;48(3):416-429.
6. Dajani EZ, Islam K. Cardiovascular and gastrointestinal toxicity of selective cyclo-oxygenase-2 inhibitors in man. J PhysiolPharmacol. 2008;59(Suppl 2):117-133.
7. Haroyan A, Mukuchyan V, Mkrtchyan N, Minasyan N, Gasparyan S, Sargsyan A, et al. Efficacy and safety of curcumin and its combination with boswellic acid in osteoarthritis: a comparative, randomized, double-blind, placebo-controlled study. BMC Complement Altern Med. 2018;18:7.
8. Henrotin Y, Malaise M, Wittoek R, de Vlam K, Brasseur JP, Luyten FP, et al. Bio-optimized Curcuma longa extract is efficient on knee osteoarthritis pain: a double-blind multicenter randomized placebo-controlled three-arm study. Arthritis Res Ther. 2019;21:179.
9. Hu Q, Ecker M. Overview of MMP-13 as a promising target for the treatment of osteoarthritis. Int J Mol Sci. 2021;22(4):1742.
10. Kumar N, et al. Tinosporacordifolia chloroform extract inhibits LPS-induced inflammation via NF-êB inactivation in THP-1 cells and improves survival in sepsis. BMC Complement Med Ther. 2021;21(1):73.
11. Li HZ, Liang XZ, Sun YQ, Jia HF, Li JC, Li G. Global, regional, and national burdens of osteoarthritis from 1990 to 2021: findings from the 2021 global burden of

disease study. Front Med (Lausanne). 2024;11: 1476853.
12. Liu X, Machado GC, Eyles JP, Ravi V, Hunter DJ. Dietary supplements for treating osteoarthritis: a systematic review and meta-analysis. Br J Sports Med. 2018;52(3):167-175.
13. Mathy-Hartert M, Jacquemond-Collet I, Priem F, Sanchez C, Lambert C, Henrotin Y. Curcumin inhibits pro-inflammatory mediators and metalloproteinase-3 production by chondrocytes. Inflamm Res. 2009;58(12):899-908.
14. Mobasheri A, van Spil WE, Budd E, Uzieliene I, Bernotiene E, Bay-Jensen AC, et al. Molecular taxonomy of osteoarthritis for patient stratification, disease management and drug development. CurrOpinRheumatol. 2019;31(1):80-89.
15. Sanchez C, Zappia J, Lambert C, Foguenne J, Dierckxsens Y, Dubuc JE, Delcour JP, Gothot A, Henrotin Y. Curcuma longa and Boswelliaserrata extracts modulate different and complementary pathways on human chondrocytes in vitro: deciphering of a transcriptomic study. Front Pharmacol. 2022;13: 931914.
16. Sannegowda KM, Venkatesha SH, Moudgil KD. Tinosporacordifolia inhibits autoimmune arthritis by regulating key immune mediators of inflammation and bone damage. Int J ImmunopatholPharmacol. 2015;28(2):171-179.
17. Sengupta K, Alluri KV, Satish AR, Mishra S, Golakoti T, Sarma KV, et al. A double blind, randomized, placebo controlled study of the efficacy and safety of 5-Loxin for treatment of osteoarthritis of the knee. Arthritis Res Ther. 2008;10(4):R85.
18. Singh GB, Atal CK. Pharmacology of an extract of salaiguggal ex-Boswelliaserrata, a new non-steroidal anti-inflammatory agent. Agents Actions. 1986;18(3-4):407-411.
19. Sohn D, Sokolove J, Sharpe O, et al. Plasma proteins present in osteoarthritic synovial fluid can stimulate cytokine production via toll-like receptor 4. Arthritis Res Ther. 2012;14(1):R7.
20. Sumantran VN, Kulkarni A, Boddul S, Chinchwade T, Koppikar SJ, Harsulkar A. Chondroprotective potential of root extracts of Withaniasomnifera in osteoarthritis. J Biosci. 2007;32(2):299-307.
21. Suresh V, Sruthi V, Padmaja B, Asha VV. In vitro anti-inflammatory activity of fenugreek (Trigonellafoenum -graecum) seed petroleum ether extract. Indian J Pharmacol. 2016;48(4):441-445.
22. Szymczak J, Grygiel-Górniak B, Cielecka-Piontek J. Zingiberofficinale Roscoe: the antiarthritic potential of a popular spice-preclinical and clinical evidence. Nutrients. 2024;16(5):741.
23. Tausch L, Henkel A, Siemoneit U, Poeckel D, Kather N, Franke L, et al. Identification of human cathepsin G as a functional target of boswellic acids from the anti-inflammatory remedy frankincense. J Immunol. 2009;183(5):3433-3442.
24. Wang Z, Jones G, Winzenberg T, Cai G, Laslett LL, Aitken D, et al. Effectiveness of Curcuma longa extract for the treatment of symptoms and effusion-synovitis of knee osteoarthritis: a randomized trial. Ann Intern Med. 2020;173(11):861-869.
25. Wojdasiewicz P, Poniatowski £A, Szukiewicz D. The role of inflammatory and anti-inflammatory cytokines in the pathogenesis of osteoarthritis. Mediators Inflamm. 2014;2014:561459.
26. Zeng L, Yu G, Hao W, Yang K, Chen H. The efficacy and safety of Curcuma longa extract and curcumin supplements on osteoarthritis: a systematic review and meta-analysis. Biosci Rep. 2021;41(6):BSR20210817.

Plant (Family)	Key Phytochemical(s)	In Vitro Model Used	Reported Anti-Osteoarthritic Finding
Curcuma longa (Zingiberaceae)	Curcumin, demethoxycurcumin	Primary human OA chondrocytes (alginate bead culture); IL-1β-stimulated chondrocytes	Down-regulated NF-κB-driven genes, COX-1/2, MMP-13, MMP-1, ADAMTS-5; reduced IL-6, CCL2 and nitric oxide release (Sanchez et al., 2022; Mathy-Hartert et al., 2009)
Boswelliaserrata (Burseraceae)	Boswellic acids (AKBA, KBA)	Human blood-derived cell 5-LOX/COX assay; SW1353 chondrosarcoma cells; primary OA chondrocytes	5-LOX inhibition (IC50 ~43 µg/mL); reduced leukotriene B4 and PGE2; protected SOX-9 expression from IL-1β depletion (Alluri et al., 2020; Sanchez et al., 2022)
Zingiberofficinale (Zingiberaceae)	6-Gingerol, 6-shogaol, zingerone	Fibroblast-like synoviocytes; C28I2 human chondrocytes; sow cartilage explants	Reduced TNF-α, COX-2, PGE2, MMP-13; lowered ROS and lipid peroxidation in chondrocytes (Szymczak et al., 2024)
Withaniasomnifera (Solanaceae)	Withanolides, withanolide glycosides	Human OA cartilage explant model	Reduced nitric oxide release and modulated glycosaminoglycan secretion in a subset of OA cartilage explants (Sumantran et al., 2007)
Tinosporacordifolia (Menispermaceae)	Tinosporaside, alkaloids, diterpenoid lactones	THP-1 cells; RAW 264.7 macrophages	Suppressed NF-κB activation and pro-inflammatory cytokine gene expression (TNF-α, IL-1β) (Sannegowda et al., 2015)