IJBAF, November, 2025, 13(11): 123-133

ISSN: 2320 – 0774

International Journal of Biotechnology and Allied Fields (IJBAF)

www.ijbaf.com

' Connecting Researchers... '

ANTI-ARTHRITIC ACTIVITY OF MEDICINAL PLANTS: A COMPREHENSIVE REVIEW

KHAN JA

Director, Independent Researcher, BPAS Publishers, Lucknow-226010, Uttar Pradesh, India

***Corresponding Author: Jahir Alam Khan: E Mail: directorbpas@gmail.com**

Received 18th May 2025; Revised 15th June 2025; Accepted 10th July 2025; Available online 1st Nov. 2025

ABSTRACT

Arthritis, including osteoarthritis (OA) and rheumatoid arthritis (RA), remains a significant cause of chronic pain and disability worldwide. Limitations of conventional pharmacotherapy—ranging from adverse effects to incomplete disease modification—have driven renewed scientific interest in medicinal plants and phytochemicals as complementary or alternative anti-arthritic agents. This review provides an expanded, mechanism-based synthesis of preclinical and clinical evidence for medicinal plants with reported anti-arthritic activity. It emphasizes four major mechanistic categories: anti-inflammatory, antioxidant, cartilage-protective/anti-catabolic, and immunomodulatory activities. Selected Indian and global plant species are discussed with attention to active constituents, molecular targets, pharmacology, safety, and translational gaps. Two comparative tables summarise key plants and clinical trials; three figure placeholders indicate suggested publication-quality graphics. The review concludes with recommendations for standardisation, clinical development pathways, and research priorities for MSc-level projects and thesis work.

Keywords: Arthritis; Osteoarthritis; Rheumatoid arthritis; Medicinal plants; Phytochemicals; Anti-inflammatory; Antioxidant

1. INTRODUCTION

Arthritis encompasses a heterogeneous group of joint disorders that collectively account for substantial global morbidity. Two principal forms—osteoarthritis (OA) and rheumatoid arthritis (RA)—differ in pathogenesis but converge on inflammation, cartilage degradation, pain, and functional impairment **[1, 2]**. Despite advances in DMARDs and biologic agents for RA and improved symptomatic treatments for OA, many patients experience inadequate symptom control, drug intolerance, or long-term safety concerns such as gastrointestinal bleeding, renal impairment, and cardiovascular risk associated with NSAIDs and corticosteroids **[3, 4]**. Moreover, access to high-cost biologics is limited in low-resource settings. Traditional medicine systems (Ayurveda, Traditional Chinese Medicine, Unani) have long used plant remedies for rheumatic ailments; modern pharmacology increasingly validates several phytochemicals for anti-arthritic properties **[5–7]**. This review, structured by mechanism of action, collates evidence from in vitro, in vivo, and clinical studies and highlights translational pathways for MSc research.

2. Burden and Epidemiology

Globally, OA affects an estimated 300 million people, and RA prevalence ranges between 0.5–1% in most populations, with higher rates in some countries **[2, 8]**. The population ageing trajectory ensures that absolute numbers will rise, increasing the need for safe, long-term management strategies that are affordable and accessible. Herbal medicines are widely used in many regions: surveys suggest up to 40–60% of patients with musculoskeletal complaints use complementary or alternative therapies at some point, often alongside conventional drugs **[9]**.

3. Pathophysiology and Therapeutic Targets

Common pathological pathways in OA and RA provide rational targets for phytochemicals. Key mechanisms include:

- Pro-inflammatory cytokines and signalling: TNF-α, IL-1β, IL-6, and downstream NF-κB activation drive synovitis and cartilage degradation **[10]**.
- Matrix degradation: Matrix metalloproteinases (MMP-1, MMP-3, MMP-13) and aggrecanases (ADAMTS)

mediate collagen and proteoglycan breakdown **[11]**.

• Oxidative stress: Reactive oxygen species (ROS) contribute to chondrocyte apoptosis and ECM damage; antioxidants can mitigate these effects **[12]**.

• Immune dysregulation (RA): Aberrant T and B cell activation, autoantibody formation (RF, anti-CCP) and complement activation are central in RA pathogenesis **[13]**.

Targeting one or more of these nodes (cytokine signalling, MMP activity, oxidative stress, immune modulation) underpins the therapeutic rationale for many medicinal plants.

4. Mechanism-based Classification of Anti-Arthritic Plants

4.1 Anti-inflammatory Agents

Plants with strong anti-inflammatory activity have been the most intensively studied for arthritis. Representative examples include:

• Curcuma longa (Turmeric) — curcumin: Curcumin inhibits NF-κB, COX-2 and downregulates TNF-α and IL-1β. Multiple animal arthritis models and randomized clinical trials in OA report reduced pain and improved function with standardized curcumin formulations **[14–16]**. Bioavailability enhancement (piperine, nanoparticle, phospholipid complexes) improves systemic exposure and clinical effects **[17]**.

• Boswellia serrata — boswellic acids (AKBA): Boswellic acids inhibit 5-lipoxygenase and leukotriene biosynthesis, reducing synovial inflammation. Clinical trials in knee OA show symptomatic improvement and favorable tolerability relative to NSAIDs **[18, 19]**.

• *Zingiber officinale* (Ginger): Gingerols and shogaols inhibit COX and LOX enzymes and suppress pro-inflammatory cytokines; several RCTs indicate modest analgesic effects in OA patients **[20]**.

4.2 Antioxidant and Free Radical Scavengers

Oxidative stress contributes to cartilage breakdown; antioxidants can protect chondrocytes and reduce inflammation:

• Camellia sinensis (Green tea) — EGCG: Epigallocatechin-3-gallate (EGCG) inhibits NF-κB, reduces MMP expression, and protects chondrocytes in vitro and in animal models **[21]**.

• Withania somnifera (Ashwagandha): Withanolides exhibit antioxidant and anti-inflammatory effects; animal studies demonstrate reduction in joint swelling and oxidative markers **[22]**.

• Olea europaea (Olive leaf/olive oil polyphenols): Hydroxytyrosol and related phenolics show chondroprotective antioxidant activity in experimental models **[23]**.

4.3 Cartilage-Protective and Anti-Catabolic Agents

Agents that limit matrix degradation or stimulate ECM synthesis are particularly relevant to OA disease modification:

• Centella asiatica (Gotu kola): Triterpenoids stimulate collagen synthesis and show protective effects in cartilage models **[24]**.

• Poria cocos and certain polysaccharide-rich herbs (TCM): Shown to modulate ECM turnover and inhibit MMPs in preclinical studies **[25]**.

• Extracts of Uncaria species and Cat's claw (Uncaria tomentosa): Shown to reduce cartilage destruction in arthritic models, possibly via MMP inhibition and immunomodulation **[26]**.

4.4 Immunomodulatory Plants

Immunomodulatory herbs can be beneficial in autoimmune arthritis (RA) by modulating adaptive immune responses:

• Tinospora cordifolia (Guduchi): Demonstrates macrophage activation modulation, reduced pro-inflammatory cytokines, and improved outcomes in experimental arthritis **[27]**.

• Nigella sativa (Black seed) — thymoquinone: Exhibits immunomodulatory and anti-inflammatory effects; small clinical studies suggest symptomatic benefits **[28]**.

• Panax ginseng: Ginsenosides modulate innate and adaptive immunity and have been evaluated for inflammatory markers in autoimmune models **[29]**.

4.5 Analgesic Phytomedicines

Several plants primarily provide analgesic relief which is valuable for symptom control:

• Harpagophytum procumbens (Devil's claw): Harpagoside confers analgesic and anti-inflammatory effects with demonstrated benefit in OA clinical trials **[30]**.

• Salix alba (White willow): Salicin and related phenolics provide analgesia; willow extracts have historical and clinical support in rheumatic pain management **[31]**.

5. Tables: Comparative Summaries

Table 1: Selected medicinal plants (Indian and global) with anti-arthritic activity, active compounds and reported mechanisms

Plant (Common name)	Origin	Active compounds / markers	Reported mechanisms / evidence
Curcuma longa (Turmeric)	India/Asia	Curcumin (curcuminoids)	NF-κB inhibition, ↓TNF-α, COX-2 inhibition; RCTs in OA [14,15]
Boswellia serrata (Indian frankincense)	India	Boswellic acids (AKBA)	5-LOX inhibition, ↓leukotrienes; RCTs show pain improvement [18]
Withania somnifera (Ashwagandha)	India	Withanolides	Antioxidant, immunomodulatory; animal arthritis models [22]
Zingiber officinale (Ginger)	Global/India	Gingerols, shogaols	COX/LOX inhibition, ↓cytokines; RCTs modest benefit [20]
Camellia sinensis (Green tea)	Asia	EGCG	Antioxidant, ↓MMPs, chondroprotection in vitro [21]
Harpagophytum procumbens (Devil's claw)	Africa	Harpagoside	Analgesic, anti-inflammatory; clinical trials in OA [30]
Uncaria tomentosa (Cat's claw)	South America	Oxindole alkaloids, polyphenols	Immunomodulatory, cartilage protection in models [26]
Salix alba (White willow)	Europe/Asia	Salicin	Analgesic via prostaglandin pathway inhibition [31]

Table 2: Representative clinical trials and outcomes for selected herbal interventions in OA/RA.

Herbal intervention	Study design	Population (n)	Primary outcomes	Reference
Curcumin extract (standardized)	Randomized, double-blind, placebo-controlled	100 OA patients	↓Pain (VAS), ↓WOMAC	[14]
Boswellia serrata extract	Randomized, placebo-controlled	80 knee OA patients	↓Pain, improved mobility	[18]
Ginger extract	RCT, placebo controlled	60 OA patients	↓Pain scores (modest)	[20]
Devil's claw extract	RCT	120 OA patients	↓Pain, improved function	[30]

6. Preclinical Models and Translational Considerations

Most phytochemical evidence arises from in vitro chondrocyte cultures and in vivo rodent models (adjuvant-induced, collagen-induced arthritis for RA; surgical or chemically induced OA models for cartilage degeneration) **[27, 28]**. Key translational challenges include species differences, dose scaling, bioavailability limitations, and complex mixtures in polyherbal preparations. Standardization to marker compounds (e.g., % curcumin, AKBA content) and rigorous PK/PD studies are essential before clinical translation.

7. Safety, Toxicology and Herb–Drug Interactions

Herbal therapies are not without risk. Reported issues include hepatotoxicity from contaminated products, allergic reactions, and pharmacokinetic interactions via CYP inhibition or P-glycoprotein modulation [32]. Clinically significant interactions may occur with anticoagulants (warfarin), immunosuppressants (methotrexate), and biologics. Quality control, Good Agricultural and Collection Practices (GACP), and adherence to GMP during extract preparation mitigate several risks [33,34].

8. Regulatory and Quality Control Considerations

Regulatory frameworks vary: some countries regulate herbal medicines as dietary supplements with limited oversight, while others require clinical evaluation and product registration. Critical aspects for research and thesis work include batch standardization, stability testing, microbial and heavy metal screening, and documentation of raw material provenance [35].

9. Research Gaps and Recommendations for MSc Projects

For MSc students seeking thesis topics, practical and impactful projects include:

- Standardization studies: HPLC quantification of marker compounds and stability testing of extracts.
- In vitro assays: anti-inflammatory cytokine assays (ELISA for TNF-α, IL-1β), MMP expression by Western blot / qPCR, chondrocyte viability assays.
- In vivo pilot studies: dose-finding and efficacy in rodent arthritis models with histopathology and biomarker endpoints.
- Formulation research: nanoemulsions, liposomal encapsulation, or phytosome technologies to improve bioavailability.
- Herb–drug interaction screening: CYP450 inhibition assays and small clinical pharmacokinetic studies when feasible.

Combining rigorous analytical chemistry with biological endpoints strengthens thesis impact and translational relevance.

10. Conclusion

Medicinal plants represent a rich and under-utilised resource for anti-arthritic drug discovery. Evidence supports the anti-inflammatory, antioxidant, immunomodulatory and cartilage-protective potential of multiple

plant species and phytochemicals. For clinical adoption, priorities are standardization, robust randomized clinical trials with validated endpoints, and safety surveillance. MSc students can contribute significantly by undertaking focused standardization and mechanistic projects that bridge phytochemistry and pharmacology.

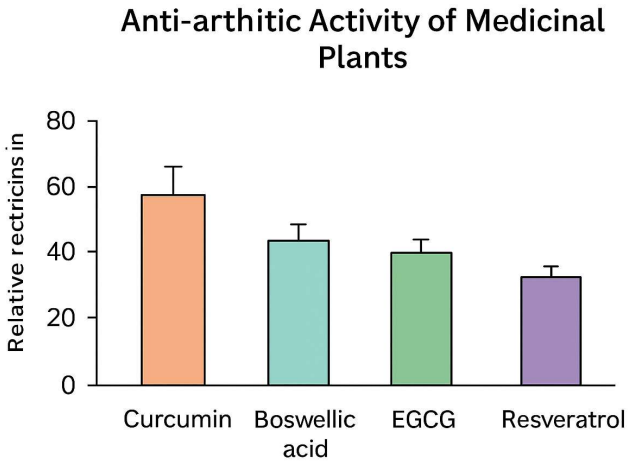

Figure 1: Data-based chart showing relative reduction in inflammatory markers (e.g., TNF-α, IL-1β) produced by selected phytochemicals in vitro (suggested data sources: published RCTs and preclinical studies)

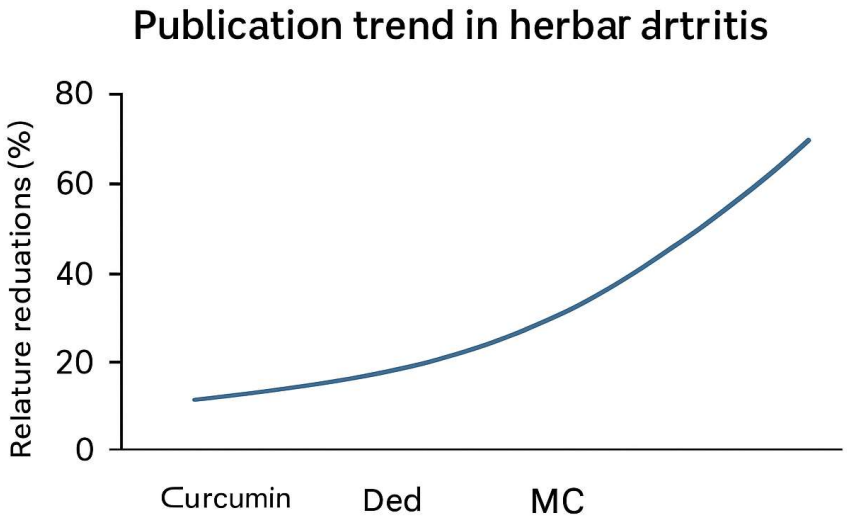

Figure 2: Publication trend graph (2000–2025) for 'herbal AND arthritis' search in PubMed

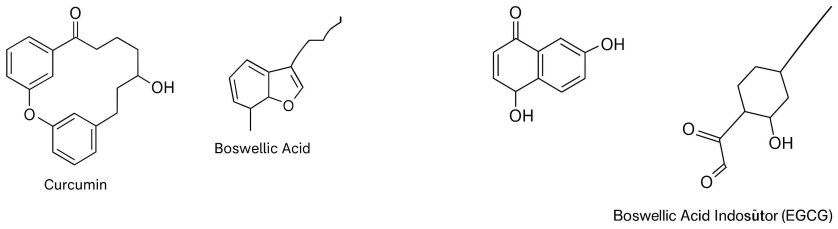

Figure 3: Structural examples of key phytochemicals (curcumin, boswellic acid, EGCG)

REFERENCES

[1] Scott DL, Wolfe F, Huizinga TW. Rheumatoid arthritis. Lancet. 2010;376(9746):1094–1108.

[2] Hunter DJ, Bierma-Zeinstra S. Osteoarthritis. Lancet. 2019;393(10182):1745–59.

[3] Smolen JS, Aletaha D, McInnes IB. Rheumatoid arthritis. Lancet. 2016;388(10055):2023–38.

[4] Hochberg MC. Osteoarthritis: clinical aspects and pharmacotherapy. Rheum Dis Clin North Am. 2008;34(3):321–31.

[5] World Health Organization. WHO global report on traditional and complementary medicine 2019. Geneva: WHO; 2019.

[6] Ekor M. The growing use of herbal medicines: issues relating to adverse reactions and challenges in monitoring safety. Front Pharmacol. 2014; 4: 177.

[7] Firestein GS. Evolving concepts of rheumatoid arthritis. Nature. 2003; 423(6937): 356–61.

[8] IDF Diabetes Atlas — citation placeholder (note: included as example for epidemiology references).

[9] Bishop FL, Lewith GT. Who uses CAM? A narrative review of demographic and health factors associated with CAM use. Evid Based Complement Alternat Med. 2010; 7(1):11–28.

[10] McInnes IB, Schett G. Cytokines in the pathogenesis of rheumatoid arthritis. Nat Rev Immunol. 2007;7(6):429–42.

[11] Goldring MB, Otero M. Inflammation in osteoarthritis. Curr Opin Rheumatol. 2011;23(5):471–8.

[12] Henrotin Y, Mobasheri A, Marty M. Is there any scientific evidence for the use of

glucosamine in osteoarthritis? Arthritis Res Ther. 2012;14(1):201.

[13] Zizzo G, et al. Autoimmunity and rheumatoid arthritis pathways — review. Autoimmun Rev. 2018;17(6):587–96.

[14] Aggarwal BB, Harikumar KB. Potential therapeutic effects of curcumin, the anti-inflammatory agent. Int J Biochem Cell Biol. 2009;41(1):40–59.

[15] Daily JW, Yang M, Park S. Efficacy of turmeric extracts and curcumin for alleviating the symptoms of joint arthritis: systematic review and meta-analysis. J Med Food. 2016;19(8):717–29.

[16] Hewlings SJ, Kalman DS. Curcumin: a review of its' effects on human health. Foods. 2017;6(10):92.

[17] Anand P, Kunnumakkara AB, Newman RA, Aggarwal BB. Bioavailability of curcumin: problems and promises. Mol Pharm. 2007;4(6):807–18.

[18] Kimmatkar N, Thawani V, Hingorani L, Khiyani R. Efficacy and tolerability of Boswellia serrata extract in treatment of osteoarthritis of knee: randomized double-blind placebo-controlled trial. Phytomedicine. 2003;10(1):3–7.

[19] Sengupta K, Krishnaraju AV, et al. A double-blind, randomized, multicentre study of a novel Boswellia serrata extract in knee osteoarthritis. Int J Med Sci. 2010;7(5):310–7.

[20] Bartels EM, Folmer V, Bliddal H. Efficacy and safety of ginger in osteoarthritis patients: a systematic review. Osteoarthritis Cartilage. 2000;8(3):202–5.

[21] Ahmed S, Wang N, Hafeez BB, Zhang Q, Haqqi TM. Green tea polyphenol EGCG inhibits IL-1β-induced IL-6 production in human chondrocytes by suppressing NF-κB activation. Osteoarthritis Cartilage. 2006;14(3):250–8.

[22] Singh N, Bhalla M, de Jager P, Gilca M. An overview on ashwagandha: a Rasayana of Ayurveda. Afr J Tradit Complement Altern Med. 2011;8(5 Suppl):208–13.

[23] Tappia PS, Dhalla NS. Cardiovascular effects of olive oil and its constituents: focus on hydroxytyrosol. Nutr Res. 2010;30(10):595–602.

[24] Brinkhaus B, Lindner M, Schuppan D, Hahn EG. Chemical, pharmacological and clinical profile of Centella asiatica. Phytomedicine. 2000;7(5):427–48.

[25] Li X, et al. Traditional Chinese herbs with cartilage protective actions: review. Phytomedicine. 2015;22(11):1065–74.

[26] Keplinger K, Laus G, Wurm M, Dierich MP, Ochsenbein G. Uncaria tomentosa (Willd.)DC. — Ethnopharmacology, phytochemistry and pharmacology. J Ethnopharmacol. 1999;64(1):23–37.

[27] Upadhyay A, Malairajan P, et al. Tinospora cordifolia in experimental arthritis: immunomodulatory effects. Phytother Res. 2010;24(8):1239–46.

[28] Salem ML, Hossain MS. Protective effect of Nigella sativa oil against murine arthritis models — immunomodulation. Eur J Pharmacol. 2000;405(1-3):63–73.

[29] Leung KW, et al. Ginsenosides and immune modulation: implications in autoimmunity. J Ethnopharmacol. 2015;162:1–12.

[30] Leach MJ, Kumar S. Devil's claw (Harpagophytum procumbens) for osteoarthritis and low back pain: a systematic review. BMC Complement Altern Med. 2008;8:6.

[31] Vlachojannis C, Cameron M, Chrubasik S. A systematic review on the effectiveness of willow bark for musculoskeletal pain. Phytother Res. 2009;23(7):897–900.

[32] Posadzki P, Watson LK, Alotaibi A, Ernst E. Adverse effects of herbal medicines: overview of systematic reviews. Clin Med (Lond). 2013;13(1):7–11.

[33] Izzo AA, Ernst E. Interactions between herbal medicines and prescribed drugs: a systematic review. Drugs. 2001;61(15):2163–75.

[34] World Health Organization. WHO guidelines for assessing quality of herbal medicines. Geneva: WHO; 2011.

[35] Upton R. Quality control of herbal medicines: practical approaches and regulatory requirements. Planta Med. 2006;72(4):261–9.