

Multi-Target Actions Of Medicinal Plants In Rheumatoid Arthritis: Current Evidence And Future Perspectives

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ABSTRACT

Rheumatoid arthritis (RA) is a chronic autoimmune inflammatory condition characterized by synovitis, cartilage destruction, bone erosion, systemic inflammation, and extra-articular manifestations. Although disease-modifying antirheumatic drugs (DMARDs), biologics, and Janus kinase (JAK) inhibitors have improved clinical outcomes, long-term management remains challenged by systemic adverse effects, high financial burdens, and incomplete disease responses. Medicinal plants and their bioactive phytochemicals present promising multi-target therapeutic alternatives that simultaneously regulate immune dysfunction, oxidative stress, inflammatory signaling, angiogenesis, and osteoclastogenesis. Key regulated signaling pathways include NF- κ B, MAPK, JAK/STAT, PI3K/Akt, NLRP3 inflammasome, Nrf2, TGF- β , RANKL, and HIF-1 α . Plants with substantial evidence include *Curcuma longa*, *Boswellia serrata*, *Withania somnifera*, *Tripterygium wilfordii*, *Camellia sinensis*, *Zingiber officinale*, *Nigella sativa*, *Tinospora cordifolia*, *Panax ginseng*, and *Glycyrrhiza glabra*. Advanced computational approaches such as transcriptomics, proteomics, network pharmacology, molecular docking, AI-driven drug discovery, and nanotechnology-based delivery systems are enhancing our understanding of these multi-component therapies. However, challenges like phytochemical variability, low aqueous solubility, limited standardization, and herb drug interactions must be resolved through rigorous multi-center clinical trials. Medicinal plants hold significant potential as adjunctive or complementary therapies, offering enhanced efficacy with fewer adverse effects.

Keywords: Medicinal plants, MAPK, Natural products, NF- κ B, Phytochemicals, Rheumatoid arthritis.

INTRODUCTION

Rheumatoid arthritis (RA) is a systemic, multi-factorial autoimmune disorder characterized by persistent synovial membrane inflammation, progressive cartilage degradation, and severe bone erosion [1–3]. Despite major therapeutic breakthroughs with synthetic and biological disease-modifying antirheumatic drugs (DMARDs), a significant proportion of patients experience treatment refractoriness, severe adverse reactions, or disease relapses upon drug withdrawal [4–7].

Medicinal plants and their isolated phytochemicals have gained clinical interest due to their multi-target pharmacological actions [8–10]. Unlike conventional synthetic drugs designed for single molecular targets, natural secondary metabolites (such as polyphenols, alkaloids, flavonoids, and terpenoids) exert

synergistic activities across interconnected cellular networks [11–13]. This review provides a comprehensive analysis of the molecular targets, clinical efficacy, network pharmacology interactions and safety profiles of medicinal plants in RA management.

Immunopathogenesis of Rheumatoid Arthritis

Rheumatoid arthritis develops through complex interactions between genetic factors (such as HLA-DRB1 shared epitope alleles) and environmental triggers, including cigarette smoking, periodontal pathogen exposure, and intestinal dysbiosis [1,14]. These factors promote post-translational modifications (primarily citrullination and carbamylation), leading to the breakdown of immune tolerance and autoantibody generation [2,15].

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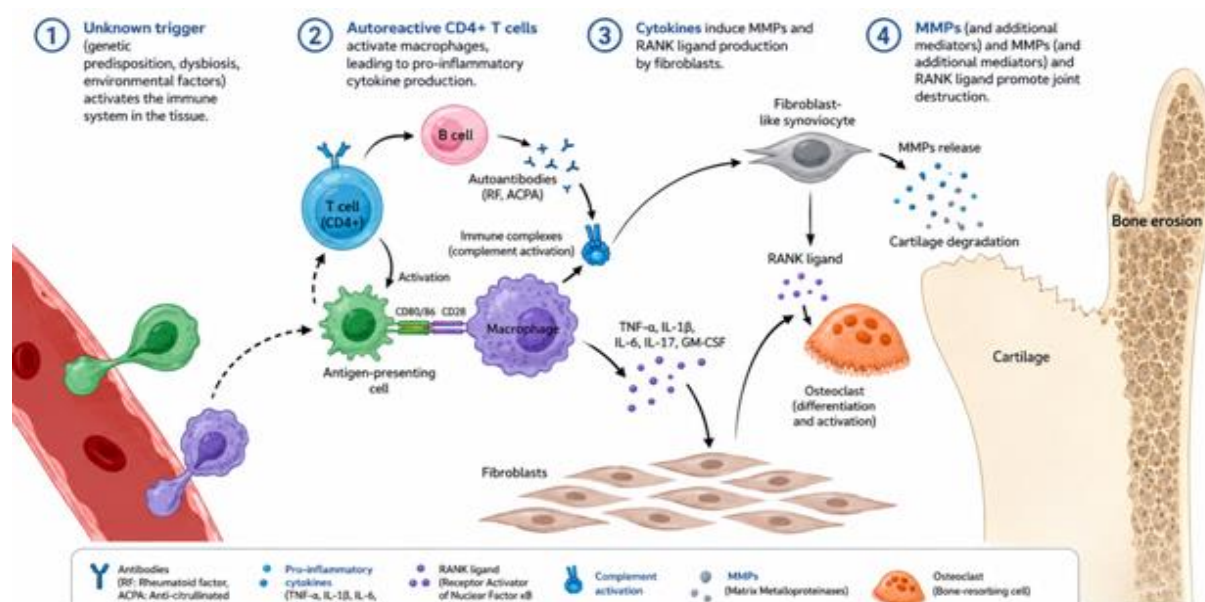


Fig. 1. Pathogenesis of rheumatoid arthritis.

Antigen-presenting cells (APCs) present citrullinated self-antigens to naïve CD4⁺ T cells, driving their differentiation into helper T cell subsets (Th1 and Th17) [3,16]. Activated B cells produce autoantibodies, including rheumatoid factor (RF) and anti-citrullinated protein antibodies (ACPAs), which form immune complexes that activate classical and alternative complement pathways [17,18]. Macrophages, neutrophils, and fibroblast-like synoviocytes (FLSs) undergo rapid recruitment and activation within the synovial microenvironment [19,20]. Activated FLSs display a tumor-like hyperproliferative phenotype, producing excessive matrix metalloproteinases (MMPs) and pro-inflammatory cytokines such as TNF- α , IL-1 β , IL-6, IL-17, and GM-CSF [21–23].

Intracellular signaling networks modulate these cellular responses. The nuclear factor-kappa B (NF- κ B) cascade amplifies transcription of inflammatory enzymes like cyclooxygenase-2 (COX-2) and inducible nitric oxide synthase (iNOS) [24,25]. The mitogen-activated protein kinase (MAPK) cascades (p38, ERK, and JNK) regulate FLS survival and cytokine production [26,27], while Janus kinase/signal transducer and activator of transcription (JAK/STAT) signaling mediates downstream cytokine transcription [28,29]. Concurrently, activation of the RANK/RANKL signaling axis drives osteoclast maturation, leading to irreversible periarticular bone erosion [30,31].

Current Therapeutic Strategies and Their Limitations

The modern management of RA follows a treat-to-target strategy aimed at achieving clinical remission or low disease activity [1,32]. Pharmacotherapy relies on nonsteroidal anti-inflammatory drugs (NSAIDs), systemic glucocorticoids, conventional synthetic DMARDs (csDMARDs, e.g., methotrexate, leflunomide), biological DMARDs (bDMARDs targeting TNF- α , IL-6R, CD20, or CD80/86), and targeted synthetic DMARDs (tsDMARDs, such as JAK inhibitors) [7,33,34].

Despite high efficacy, long-term administration of conventional therapies presents notable clinical drawbacks. Extended NSAID and corticosteroid regimens cause severe gastrointestinal ulceration, cardiovascular mortality, osteoporosis, and renal dysfunction [1,35]. Primary non-responsiveness or secondary loss of efficacy occurs in approximately 30–40% of patients receiving csDMARDs or biologics [3,36]. Furthermore, biologics and tsDMARDs increase the incidence of serious opportunistic infections, tuberculosis reactivation, herpes zoster, and thromboembolic events [7,37]. The high cost of biologics also limits their accessibility in developing countries [38]. Critically, existing therapies suppress peripheral immune responses without restoring immunological self-tolerance [3,39].

Multi-Target Mechanisms of Bioactive Compounds

Phytochemicals modulate key nodes of the inflammatory, oxidative, and matrix degradation cascades involved in RA pathogenesis [8,40,41].

Medicinal Plant	Major Bioactive Compounds	Multi-Target Mechanisms Against RA	References
<i>Curcuma longa</i> (Turmeric)	Curcumin	Inhibits NF- κ B, MAPK, JAK/STAT; suppresses TNF- α , IL-1 β , IL-6, COX-2, iNOS; reduces ROS; inhibits RANKL-mediated osteoclastogenesis.	[8,10,42]
<i>Boswellia serrata</i>	Boswellic acids (AKBA)	Inhibits 5-LOX, NF- κ B, MMP-3/9; decreases leukotrienes, synovial inflammation, and cartilage degradation.	[41,43]
<i>Withania somnifera</i>	Withanolides	Modulates immune cell function; suppresses TNF- α , IL-6, IL-17; elevates SOD, CAT, GPx; attenuates oxidative damage.	[8,44]
<i>Zingiber officinale</i>	Gingerols, Shogaols	Inhibits COX-2, 5-LOX, NF- κ B; lowers prostaglandins, leukotrienes, TNF- α , IL-1 β ; reduces joint swelling and hyperalgesia.	[13,40]
<i>Tripterygium wilfordii</i>	Triptolide, Celastrol	Suppresses NF- κ B, JAK/STAT, PI3K/Akt; inhibits T/B cell activation, cytokine release, and FLS proliferation.	[11,12]
<i>Camellia sinensis</i>	Epigallocatechin gallate (EGCG)	Inhibits NF- κ B, STAT3, MAPKs; downregulates MMPs and RANKL expression; scavenges intracellular ROS.	[8,45]
<i>Tinospora cordifolia</i>	Berberine, Tinosporaside	Exerts immunomodulatory actions; suppresses TNF- α , IL-6, IL-17; enhances endogenous enzymatic antioxidant defense.	[8,46]
<i>Glycyrrhiza glabra</i>	Glycyrrhizin, Liquiritigenin	Inhibits HMGB1, NF- κ B, COX-2; suppresses pro-inflammatory cytokines; protects cartilage extracellular matrix.	[40,47]
<i>Paeonia lactiflora</i>	Paeoniflorin	Restores Treg/Th17 balance; blocks NF- κ B/MAPK signaling; reduces TNF- α , IL-6, IL-17 and synovial hyperplasia.	[14,48]
<i>Nigella sativa</i>	Thymoquinone	Blocks NF- κ B and NLRP3 inflammasome; decreases TNF- α , IL-1 β , IL-6; inhibits osteoclast differentiation.	[12,49]

Table 1: Multi-target molecular mechanisms of bioactive compounds of medicinal plants in rheumatoid arthritis

Clinical Evidence

Controlled clinical studies demonstrate that several medicinal plant formulations improve Disease Activity Score-28 (DAS28), tender/swollen joint counts, visual analogue scale (VAS) pain scores, ESR, and CRP [9,10,42].

***Curcuma longa*:** Standardized curcumin extract significantly reduced DAS28 scores and swollen joint counts compared to diclofenac sodium in active RA patients [10]. Modern bioavailable nanomicellar curcumin formulations achieved marked decreases in CRP, ESR, and pain indexes in randomized placebo-controlled trials [42].

***Tripterygium wilfordii*:** Standardized extracts containing triptolide demonstrated therapeutic response rates equal or superior to sulfasalazine and methotrexate in multicenter trials [11,12]. However, reproductive toxicity and hepatotoxicity require close dose monitoring.

***Nigella sativa* & *Zingiber officinale*:** Double-blind RCTs confirmed that *N. sativa* seed oil reduced DAS28 scores and serum TNF- α levels [49], while *Z. officinale* extract supplementation significantly reduced hs-CRP, TNF- α , and disease activity over a 12-week intervention period [13].

***Paeonia lactiflora* & *Boswellia serrata*:** Total Glucosides of Peony (TGP) combined with methotrexate achieved superior reductions in disease activity while reducing methotrexate-induced liver enzyme elevations [48]. *Boswellia serrata* extracts significantly reduced joint stiffness and improved physical function via dual inhibition of 5-LOX and NF- κ B [43].

Safety, Toxicity, and Herb–Drug Interactions

General Safety Profile

While plant extracts generally exhibit favorable tolerability, unstandardized preparations present clinical risks due to variable phytochemical profiles,

heavy metal contamination, pesticide residues, or microbial burden [16,17,50]. Mild adverse events include self-limiting nausea, dyspepsia, diarrhea, and transient headaches [16]. Strictly enforced Good Manufacturing Practices (GMP) are mandatory to ensure safety and clinical reproducibility [17].

Herbal Toxicity

Herbal toxicity can arise from intrinsic cytotoxic compounds, inappropriate dosages, prolonged exposure, or adulteration [17,18]. Hepatotoxicity (characterized by elevated aminotransferases) and nephrotoxicity (often associated with heavy metal exposure) have been reported for non-standardized preparations [18,19,51]. Comprehensive toxicological testing and rigorous quality assurance protocols are required [17,19].

Herb–Drug Interactions

Pharmacokinetic and pharmacodynamic interactions must be evaluated in RA patients receiving combination therapies [20,52]. Curcumin and ginger exert mild antiplatelet effects, which may elevate bleeding risks when combined with anticoagulants (e.g., warfarin) or antiplatelet agents [20,21,53]. *Boswellia serrata* can augment NSAID-induced gastrointestinal erosion [20]. *Withania somnifera* exhibits immunostimulatory actions that could interfere with immunosuppressive regimens [20,22,44]. Furthermore, phytochemicals that modulate Cytochrome P450 enzymes (e.g., CYP3A4, CYP2C9) or P-glycoprotein transporters can alter the systemic availability of co-administered DMARDs [20,22,54].

Network Pharmacology and Systems Biology

Network pharmacology has shifted natural product research from the traditional "one drug, one target" model to a "multi-component, multi-target, multi-pathway" paradigm [23,24,55]. Combining transcriptomics, proteomics, and metabolomics allows comprehensive mapping of complex biological networks [24,25].

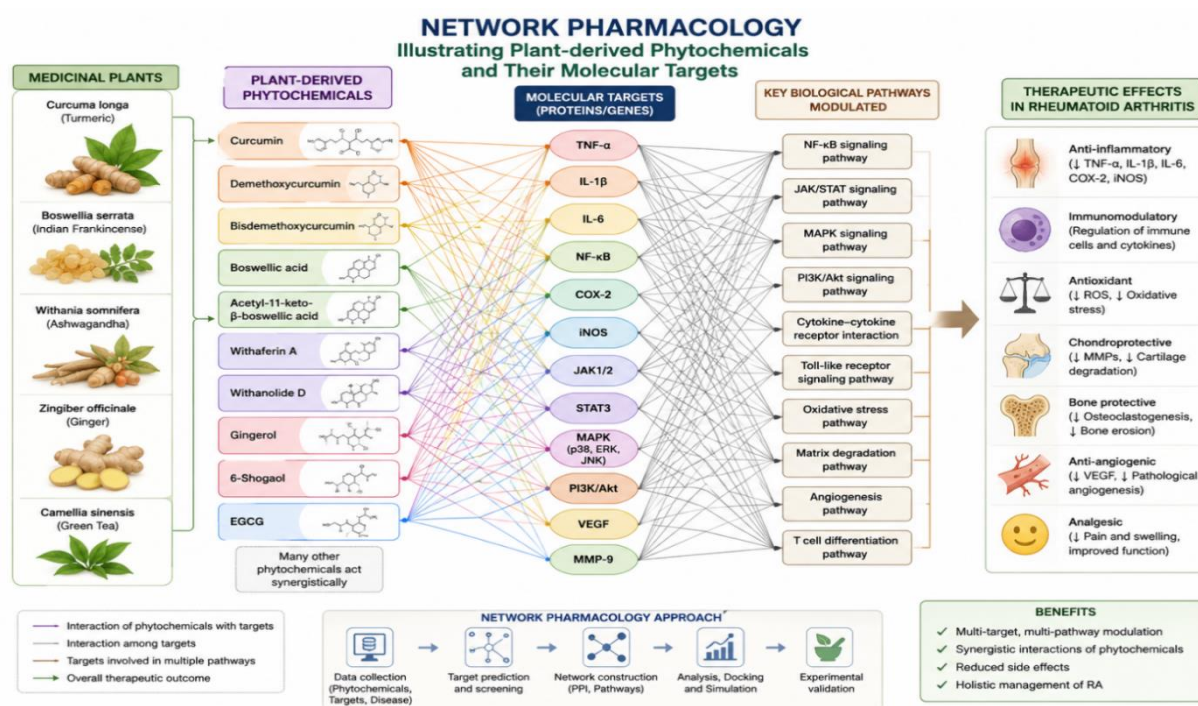


Fig. 2. The network pharmacology approach to illustrate plant-derived phytochemicals and their molecular targets for management of Rheumatoid arthritis.

Computational platforms leverage protein–protein interaction (PPI) networks, KEGG pathway analysis, Gene Ontology (GO) mapping, molecular docking, and molecular dynamics simulations to identify active compounds and candidate target genes [24,26,56]. Studies on *Curcuma longa*, *Boswellia serrata*, *Tripterygium wilfordii*, and *Withania somnifera* show that their components target interconnected signaling nodes, including TNF- α , IL-6, NF- κ B, Akt1, STAT3, COX-2, and MMP-9 [23,26,55]. Integrating artificial intelligence and multi-omics data accelerates target identification and aids the design of standardized polyherbal formulations [25,56].

Challenges and Future Perspectives

Translating preclinical findings into routine clinical practice requires addressing key hurdles:

Phytochemical Variation & Standardization: Natural variation in plant composition due to geographical, seasonal, and processing factors requires strict chemical profiling and standardization using marker compounds [4,16].

Poor Bioavailability: Low aqueous solubility, rapid hepatic metabolism, and limited systemic distribution limit the clinical efficacy of many phytochemicals [27,57]. Advanced nano-delivery platforms—

including liposomes, polymeric nanoparticles, phytosomes, and solid lipid nanoparticles—enhance targeted delivery to inflamed joints while extending systemic half-lives [27,57].

Rigorous Clinical Validation: High-quality, multi-center, double-blind RCTs adhering to international standards (e.g., CONSORT-Extension for Herbal Interventions) are essential to establish definitive safety profiles, pharmacokinetic parameters, and therapeutic efficacy [1,4,9].

CONCLUSION

Rheumatoid arthritis is a multi-factorial disease that presents ongoing therapeutic challenges. Medicinal plants offer a rich source of multi-target bioactive agents capable of attenuating inflammatory cascades, oxidative damage, autoantibody-mediated pathology, and joint destruction. When integrated with systems biology, network pharmacology, and targeted nano-delivery systems, standardized herbal formulations represent a viable approach toward safer, synergistic, and personalized treatment strategies for RA.

CONFLICTS OF INTEREST

The authors declare that there are no conflicts of interest.

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