Based on the provided sources, the following herbs and natural compounds are categorized by their theoretical efficacy in treating Rheumatoid Arthritis (RA), sorted by their primary bioactive agents, synergistic potential, and specific pathological targets.

1. Primary Bioactive Compounds (Monotherapy)

These compounds are frequently cited as having robust theoretical efficacy through multi-target mechanisms, acting similarly to Disease-Modifying Antirheumatic Drugs (DMARDs).

- **Triptolide & Celastrol (from *Tripterygium wilfordii*):** Theoretically among the most potent natural agents, these are often termed "natural DMARDs" 1, 2. They function by inhibiting NF-κB, angiogenesis, and pro-inflammatory chemokines, though their utility is theoretically limited by a narrow therapeutic window and potential toxicity 3-5.
- **Sinomenine (from *Sinomenium acutum*):** Theoretically effective for "wind-dampness" phenotypes, sinomenine mediates anti-inflammatory and immunosuppressive effects by regulating cytokine secretion and inhibiting synovial hyperplasia 6-9. It is theorized to protect bone from destruction via the RANK/RANKL/OPG pathway and Nrf2 signaling 10, 11.
- **Curcumin (from *Curcuma longa*):** Theorized to act as an anti-tumor necrosis factor (TNF) agent 12. It targets the JAK/STAT signaling pathway to modulate immune cell function (Th1/Th17/Treg balance) and inhibits NF-κB and MAPK pathways to reduce inflammation 13, 14. It is also noted for improving morning stiffness and joint swelling 15.
- **Resveratrol:** Proposed to ameliorate RA via the STAT3/HIF-1/VEGF molecular pathway 16, 17. It theoretically reduces oxidative stress (increasing SOD, decreasing MDA) and pro-inflammatory cytokines like TNF-α and IL-6 18, 19.
- **Quercetin:** Theoretically inhibits neutrophil activities and infiltration 20. It is proposed to downregulate the JAK1/STAT3 pathway and inhibit the NLRP3 inflammasome, thereby reducing synovial inflammation and cartilage injury 21, 22.
- **Berberine:** An alkaloid that theoretically manages RA by modulating the gut microbiota and inhibiting the TLR4/NF-κB signaling pathway, which suppresses the inflammatory response in synovial cells 23, 24.
- **Paeoniflorin (from *Paeonia lactiflora*):** Acts by regulating G protein-coupled receptor kinase 2 (GRK2) and inhibiting the NF-κB signaling pathway, theoretically restoring the abnormal signaling in synoviocytes 25, 26.

2. Synergistic Combinations

The sources highlight several combinations where compounds theoretically enhance efficacy or reduce toxicity compared to single-agent use.

Herb-Herb Synergies

- **Triptolide + Curcumin:** This combination is theorized to have a synergistic effect on inhibiting cell proliferation and inducing apoptosis in inflamed cells via the IL-17/NF-κB signaling pathway, improving symptoms more effectively than either agent alone 27, 28.
- **Rosmanol + Carnosol:** Two diterpenoids from *Callicarpa longissima* that, when combined, theoretically block the TLR4/NF-κB/MAPK pathway more significantly than when used individually 29.

- **Imperatorin + Beta-sitosterol:** Predicted to act synergistically to alleviate arthritis severity by regulating LTA, CD83, and SREBF1 targets, a dynamic not observed when administered separately 30, 31.
- **Sinomenine + Berberine:** Integrated into a microneedle patch, this combination theoretically realizes both anti-inflammation and anti-angiogenesis effects synergistically 32.
- **Epicatechin + Procyanidin A2:** Found in *Xanthocerais lignum*, this pair demonstrated a synergistic inhibitory effect on the proliferation of rheumatoid arthritis fibroblast-like synoviocytes (RA-FLS) 33.

Herb-Drug Synergies

- **Curcumin + Leflunomide:** Co-loaded phytosomes of these two agents theoretically resulted in significant reduction of paw swelling and inflammatory markers compared to the free drugs or their physical mixture 34, 35.
- **Total Glucosides of Paeony (TGP) + Methotrexate (MTX):** TGP is theoretically posited to enhance the efficacy of MTX while simultaneously reducing its hepatotoxicity 36.
- **Tetrandrine + Methotrexate:** This combination is noted to theoretically improve clinical outcomes such as erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) more effectively than monotherapy 37.
- **Chrysin + Mesenchymal Stem Cells:** Combined administration was found theoretically more effective in treating arthritis due to strong antioxidant and anti-inflammatory properties than either treatment alone 38, 39.

3. Compounds Sorted by Theoretical Pathological Target

The sources categorize certain herbs by their specific ability to interrupt distinct RA pathological processes.

Inhibition of Angiogenesis (Pannus Formation)

- **Geniposide (from *Gardenia jasminoides*):** Theorized to inhibit synovial angiogenesis by regulating metabolic pathways 40 and the VEGF pathway 41.
- **Ardisia crispa (Quinone-rich fraction):** Proposed to alter the angiogenic cascade by targeting VEGF-A, PI3K, and STAT3 proteins 42.
- **Shikonin:** Theoretically decreases immature blood vessels in the synovial membrane 43.

Inhibition of Bone & Cartilage Destruction

- **Icariin (from *Epimedium*):** Proposed to inhibit osteoclast differentiation and bone resorption, potentially offering dual action against inflammation and bone loss 44, 45.
- **Calycosin:** Shown to offer superior bone protection compared to methotrexate in theoretical models by retarding osteoclastogenesis 46.
- **Ginsenoside Rg3:** Evaluated for beneficial effects on immunosuppression and joint protection 47.

Regulation of Macrophage Polarization

- **Koumine (from *Gelsemium elegans*):** Theorized to alleviate RA by regulating the polarization of macrophages (shifting from inflammatory M1 to restorative M2 phenotype) 48.

- **Cepharanthine:** Proposed to attenuate joint inflammation by blocking macrophage M1 polarization and suppressing monocyte chemotaxis 49, 50.

4. Complex Formulations (Multi-Target Efficacy)

These formulations are theorized to work via "multi-component, multi-target" mechanisms.

- **Wutou Decoction (WTD):** A classic formula theorized to reduce inflammation and regulate immune response by targeting TNF, IL-6, and PTGS2 51-53.
- **Guizhi-Shaoyao-Zhimu Decoction (GSZD):** Proposed to inhibit the migration and invasion of synovial fibroblasts and induce apoptosis via the PI3K-Akt and MAPK pathways 54, 55.
- **Xinfeng Capsule (XFC):** Theorized to improve coagulation and platelet abnormalities in RA patients while reducing inflammation 56, 57.
- **Shentong-Zhuyu Decoction:** Proposed to treat RA by promoting blood circulation and resolving stasis, targeting VEGF and TNF signaling pathways 58, 59.
- **Ermiao San:** Its theoretical mechanism involves inhibiting the PI3K/AKT/mTOR signaling pathway to activate HIF-1α induced glycolysis 60.