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HERBAL APPROACHES TO ARTHRITIS: A REVIEW OF THERAPEUTIC PLANTS AND THEIR BIOACTIVE COMPOUNDS

Abhijeet Balasaheb Pawar

Department of Pharmacology, YSPM's YTC, Satara - 415011, Maharashtra, India.

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Correspondence to Author:

Abhijeet Balasaheb Pawar

Department of Pharmacology,
YSPM's YTC, Satara - 415011,
Maharashtra, India.

E-mail: abhijeetpawar9291@gmail.com

ABSTRACT: Arthritis, a chronic inflammatory condition, significantly impacts the quality of life by causing pain, swelling, and joint degeneration. Conventional therapies, including non-steroidal anti-inflammatory drugs (NSAIDs), corticosteroids, and disease-modifying antirheumatic drugs (DMARDs), often present severe side effects, necessitating alternative approaches. This review explores the therapeutic potential of traditionally used anti-arthritic plants and their bioactive phytochemical constituents. Several medicinal plants, such as *Ocimum sanctum* (Tulsi), *Cissus quadrangularis* (Devil's Backbone), *Zingiber officinale* (Ginger), and *Withania somnifera* (Ashwagandha), have demonstrated promising anti-inflammatory and immunomodulatory effects. The primary active constituents include quercetin, kaempferol, luteolin, gallic acid, β -sitosterol, and caffeic acid, which exhibit potent antioxidant and anti-inflammatory properties by targeting key inflammatory pathways, such as NF- κ B, COX-2, and cytokines like TNF- α and IL-1 β . This study provides an extensive compilation of anti-arthritic plants, categorizing them by botanical names, families, parts used, and major phytochemicals. Plants such as *Merremia tridentata*, *Justicia gendarussa*, and *Salacia reticulata* are also highlighted for their traditional usage and emerging scientific validation in arthritis management. Furthermore, the mechanisms of action of key phytochemicals, such as their role in reducing oxidative stress, modulating immune responses, and preventing cartilage degradation, are discussed. By synthesizing traditional knowledge with scientific evidence, this review emphasizes the potential of these medicinal plants as safer, cost-effective, and sustainable alternatives or complementary therapies for arthritis. Future research directions include clinical trials, standardization of plant extracts, and the development of novel formulations to enhance bioavailability and therapeutic efficacy.

INTRODUCTION: The immune system in the body is essential because an overactive immune system can result in a number of deadly diseases due to hypersensitive or allergic reactions that can cause numerous abnormalities.

Autoimmune diseases are caused by a loss of the body's natural ability to distinguish between self and non-self, which causes immune responses against our own cells and tissues.

Due to their unclear etiology, several prevalent autoimmune disorders, such as myasthenia gravis, serum sickness, pernicious anemia, reactive arthritis, etc., pose serious problems for the medical and pharmaceutical communities¹. Women are three times more inclined than men to develop rheumatoid arthritis (RA), which affects 0.3–1% of the world's population, according to the WHO².

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RA is a systemic autoimmune disease that is inflammatory and chronic³. Pain, swelling, and bone and cartilage degradation are the main signs of RA, which leads to long-term disability. While the precise cause is unknown, a number of theories suggest that exposure to environmental factors, such as viruses, and genetic predisposition work together to cause it⁴.

Although the exact mechanism of disease is yet unclear, some free radicals, such as superoxide and nitrous oxide, are released as byproducts of cellular metabolism. Such free radicals may cause T-cells to produce interleukins (IL) and tumor necrosis factor (TNF- α), which in turn may affect immune cells' production of growth factors, cytokines, and adhesive molecules because these substances may lead to inflammation and tissue destruction⁵. The pathological alterations in RA include neovascularization, infiltration of inflammatory cells, and hyperplasia of the synovial membrane, which lead to articular degeneration and cartilage erosion.

For patients with rheumatoid arthritis, treatment aims to reduce symptoms, delay the course of the condition, and enhance quality of life⁶. Since, a series of processes are involved in the pathophysiology of both RA and OA, managing arthritis requires a multifaceted strategy. Steroid anti-inflammatory drugs (SAIDs), non-steroidal anti-inflammatory drugs (NSAIDs), and immunosuppressant medications are the conventional anti-inflammatory therapy used to treat arthritis. These medications work by inhibiting inflammatory factors, proteins, and enzymes to suppress inflammatory pathways. Non-steroidal anti-inflammatory medicines (NSAIDs) and disease-modifying antirheumatic drugs (DMARDs), which were once the mainstays of RA treatment, have given way to new biological agents like TNF monoclonal antibody. There are five clinical approaches to treating RA. The most effective strategy is to reduce inflammatory symptoms and disease development by using NSAIDs first, then little amounts of glucocorticoids. DMARDs such as methotrexate, sulfasalazine, gold salts, or D-penicillamine may be used in the treatment of chronic patients. In some situations, medications that block T-cell activation, such as abatacept, IL-1 neutralizing agents, such as

anakinra, and TNF- α neutralizing agents, such as infliximab, etanercept, etc., can also be used to treat chronic conditions. Lastly, chronic patients are treated with immunosuppressive and cytotoxic medications such as cyclosporine, azathioprine, and cyclophosphamide^{7, 8}. Although the long-term hazards of the aforementioned therapeutic medicines are still understood, they minimize inflammation and joint damage. Gastrointestinal ulcers, cardiovascular problems, hematologic toxicity, nephrotoxicity, pulmonary toxicity, myelosuppression, hepatic fibrosis, stomatitis, cirrhosis, diarrhea, immunological reactions, and local injection-site reactions are among the long-term dangers associated with medicines. Furthermore, ongoing monitoring is necessary due to increased expenses and adverse effects, which include elevated risks of infections and malignancies.

Types of Arthritis:

Rheumatoid Arthritis⁹:

1. Multiple joints experiencing joint pain, stiffness, and edema.
2. Symptoms that are symmetrical and impact both sides of the body.
3. Stiffness in the morning that lasts more than half an hour.
4. Other symptoms, such as malaise, fever, and exhaustion.

Osteoarthritis Symptoms⁹:

1. Usually affecting the hands, fingers, or knees, joint pain and stiffness.
2. One side's joints were more severely damaged than the other.
3. The joints on one side suffered more damage than the other.
4. Potential hip and back pain as well.

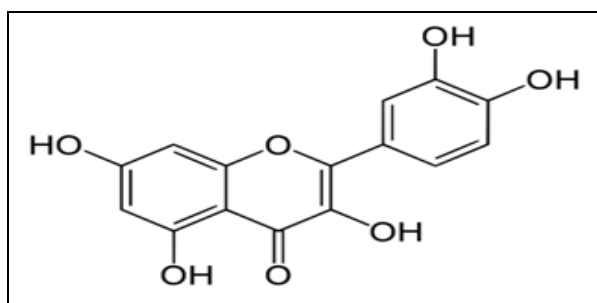
Both illnesses are chronic, which means they last for a long time. There are no recognized treatments for either illness. Since osteoarthritis is degenerative, it will only get worse over time. The prognosis for osteoarthritis can usually be

favourable with the right care. Rheumatoid arthritis is considerably more erratic. The autoimmune disease can lead to a number of problems. Other ailments, such as cardiovascular and respiratory disorders, may occasionally arise. Patients with rheumatoid arthritis are also susceptible to conditions like lupus and lymphoma. There is no universal prognosis for rheumatoid arthritis because each patient's condition can develop differently.

Essential Phytochemical and Structure:

Quercetin:

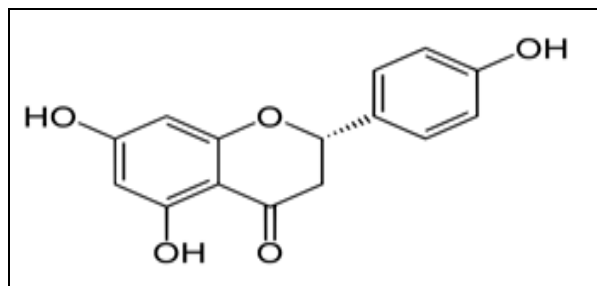
Structure:



In this study, mice treated with quercetin showed less infiltration of inflammatory cells and less hyperplasia because the flavonoids reduced the levels of TNF alpha, IL1beta, IL17, and MCP-1 in the blood ^{10, 11}. It has also been demonstrated that ouercitin treatment lowers the plasma level of IL1 beta ¹². The phosphorylation of ERK-1/2, p38, and JNK; the production and proliferation of MMPs, COX-2, and PGE2; the activation of NF-kB; and the reduced propagation of fibroblasts all impacted the level of IL-1b ¹³. The low cytokine levels following quercetin treatment seem to have been in line with the low cellularity observed in the synovial compartment. Additionally, quercetin is known to influence NF-kB activation, which lowers cytokine expression ¹⁴.

Naringenin:

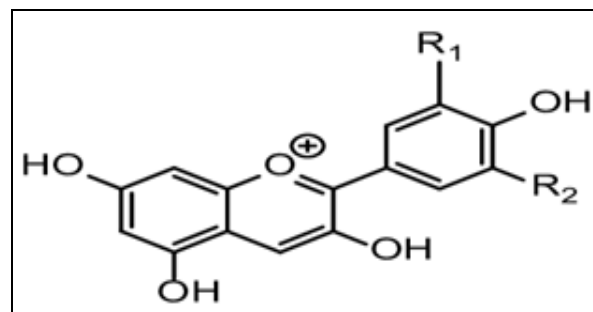
Structure:



Recent research has shown that naringenin inhibits the generation of inflammatory cytokines, which are key players in RA and are generated by T cells and macrophages. Naringenin has been shown to decrease the generation of inflammatory cytokines by T cells and macrophages by disrupting the signalling pathway controlled by the Toll-like receptor (TLR), changing the stability of cytokine mRNA or its translation. Naringenin promotes lysozyme degradation, which in turn accelerates the breakdown of intracellular cytokines ¹⁵. Naringenin taken orally increased the rate of demyelination in the spinal cord tissue and decreased immune cell infiltration, which worsened the symptoms. According to the findings, naringenin causes Th2 cells to polarize by decreasing the production of the primary polarization-inducing components, hence decreasing the Th1, Th9, and Th17 lymphocyte subtypes.

Anthocyanin:

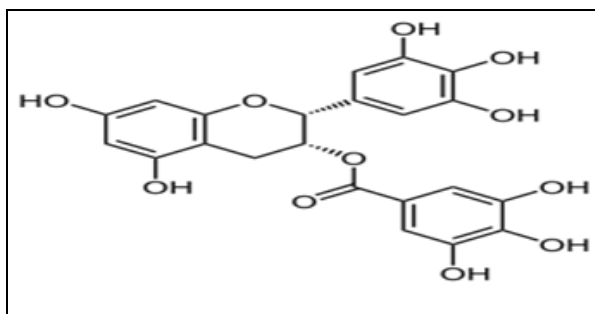
Structure:



The benefits of anthocyanin on autoimmune arthritis were made clear in this study by Th17 suppression, a reduction in proinflammatory cytokines through NF-kB signalling modulation, and, for the first time, osteoclast inhibition. Th17 cells have been recognized as unique subtypes of CD4+ cells within the Th1 and Th2 cell groups throughout the last ten or so years. Th17 cells are important in the pathophysiology of several autoimmune disorders, including RA, and are distinguished by the production of several proinflammatory cytokines, such as IL-6, IL-17, IL-21, and TNF- α ¹⁶. These cytokines are in charge of Th17 differentiation as well as the systemic inflammation and structural damage linked to RA ¹⁷.

Epigallocatechin Gallate (EGCG):

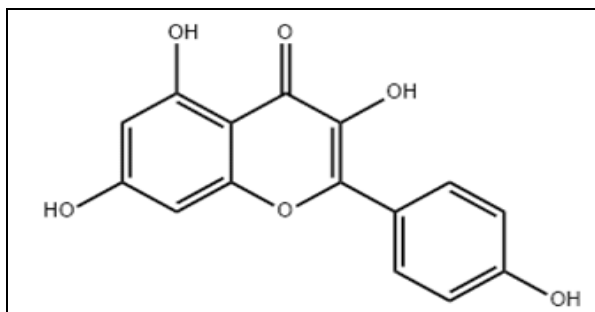
Structure:



Activation of epigallocatechin gallate is essential in causing tissue damage in a number of chronic inflammatory and degenerative conditions, such as rheumatoid arthritis (RA) synovial inflammation, diabetic at hero sclerosis, amyloidosis, arterial restenosis, and osteoarthritis¹⁸. Additionally, it was shown that the early onset and progression of OA are closely associated with cartilage inflammation. Additionally, excessive ROS in cells might worsen the deterioration of articular cartilage and trigger inflammatory pathways. One polyphenol, EGCG, is the main bioactive ingredient. It has anti-oxidative and anti-inflammatory properties, making it a promising treatment option for chronic and degenerative illnesses¹⁹.

Kaempferol:

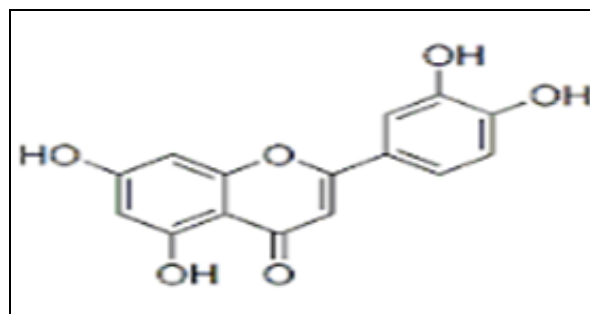
Structure:



According to earlier research, kaempferol reduces the development of osteoclasts and the migration and invasion of fibroblast-like synoviocytes *in-vitro*, as well as alleviates the symptoms of arthritis in mice²⁰. In this study, we confirmed that oral treatment with kaempferol significantly ameliorated arthritis symptoms and decreased inflammatory cytokine levels in CIA mice. Unexpectedly, the intraperitoneal injection of kaempferol showed marginal efficacy in the CIA model mice.

Luteolin:

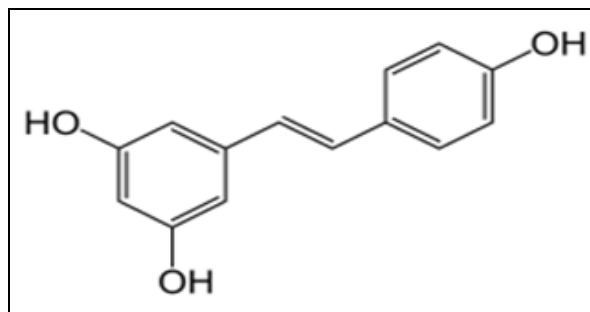
Structure:



The Anti arthritis activity rats' cytokines were measured 24 days following the injection. Arthritis rats' production of IL-6, IL-17, IL-1, and TNF- α was elevated, and the administration of Luteolin. After administration of Luteolin no of inflammatory cell are increased. P2X4, NLRP1, ASC, and Caspase-1p10 where production is increased. The most prevalent flavonoid in dandelions, lutein, has been shown to have potent anti-oxidative and anti-inflammatory properties^{21, 22}.

Resveratrol:

Structure:



The natural phytohormone resveratrol has garnered a lot of interest lately because of its potential as a treatment for RA. Resveratrol has been shown to suppress nuclear factor κ B (NF- κ B), mitogen-activated protein kinase (MAPK), and other cellular signaling pathways.

This suppresses the synthesis and release of matrix metalloenzymes, proinflammatory cytokines, and interleukins. Resveratrol can also control macrophage differentiation and function, trigger the death of MH7A, FLS, and inflammatory cells in RA, and preserve the regular redox dynamics of mitochondria. Additionally, Resveratrol is an effective treatment for RA that reduces cartilage degradation, proliferation, and synovial inflammation²³.

Herbal View:

Xanthium Strumarium Linn. (XS) (Family-Compositae): XS, often referred to as burweed, burdock, or cochlebur, is native to tropical regions of India. XS is a one-meter-tall annual plant with short, robust, hairy stems that typically grows along riverbanks in warmer climates, along roadsides, and in waste areas. In addition to improving appetite, voice, complexion, and memory, it has long been used as a laxative, anthelmintic, tonic, digestive aid, and antipyretic. Additionally, XS is used to treat fever, salivation, leukoderma, biliousness, insect poisoning, and epilepsy. Ayurvedic and Chinese medicine systems have utilized plant infusions to treat rheumatism. Alkaloids, sesquiterpene lactones, xanthinin, xanthumin, and xanthatin; sulphated glycosides, including xanthostrumarin, attractyloside, and carboxyatractyloside; phytosterols, xanthanol, isoxanthanol, xanthosin, 4-oxo-bedfordia acid, hydroquinone, xanthanolides, and deacetyl xanthumin are the active ingredients of XS's aerial parts²⁴. But according to new research, XS has an antibacterial²⁵. Anti tumor²⁶, anti-cancer²⁷, anti-tussive²⁸, anti-fungal^{28, 30}, anti-inflammatory^{31, 32}, vasorelaxant³³, hypoglycaemic³⁴, antimitotic³⁵, anti-malarial³⁶.

Vitex negundo Linn. (VN) (Family-Verbenaceae): VN is known as a five-leaved chaste tree, a big aromatic shrub, or occasionally a smaller, thin tree with tomentose branches that are thickly whitish and quadrangular. Burma and Southern India are the birthplaces of VN³⁷. VN has long been used to treat cholera, syphilis, enlarged liver, rheumatism, headaches, and diarrhea. Leaves, rice, gul, and garlic are used as a rheumatism cure. VN leaf extract was used to cure rheumatism and joint inflammation in Ayurvedic, Unani, and Chinese medical systems. The plant was used to treat rheumatism by the Konkan people of Maharashtra³⁸.

Fishindine, flavones, luteolin-7-glucoside, casticin, iridoid glycosides, vitamin C, β -sitosterol, and phthalic acid are the main chemical components³⁹. VN has several pharmacological properties, including as analgesic and anti-inflammatory⁴⁰. The reported activities of this plant anticonvulsant, antioxidant, insecticidal, and antirheumatic. Agnusidei, the active chemical extracted from the

ethanolic extract of leaves, reduced the high levels of ESR, leukotriene B₄, PGE₂, cytokines, IL-17, TNF- α , and interferon gamma when given orally at doses of 1.56 mg/10 ml, 3.12 mg/10 ml, 6.25 mg/10 ml, and 1.25 mg/10 ml. Therefore, it may be said that the VN has anti-arthritis properties⁴¹.

Sida rhombifolia Linn. (SR) (Family-Malvaceae): SR is a tiny, upright shrub that grows in arid regions like India and Ceylon. It has rough branches with stellate hairs⁴². The herb has long been used as a nutritious, tonic, diuretic, aphrodisiac, and to cure rheumatism, piles, and gonorrhea. Rheumatism is treated with dosages of $\frac{1}{4}$ seer, which is the juice of a whole plant that has been crushed with a small amount of water in the medical systems of Indonesia and Johore. The plant has yielded the following compounds: β -phenethylamine, N-methyl- β -phenethylamine, S-(β) N- β -methyl tryptophan methyl ester, vasicinol, vasicinone, vasicine, choline, hypaphorine methyl ester, hypaphorine, and betaine. Among the plant's documented properties are cytotoxic⁴³. The reported activities of this plant are Antimicrobial, antibacterial anti-inflammatory, antipyretic Paw edema brought on by the CFA technique was lessened by the aqueous and ethanol extract of the SR's aerial parts at doses of 30 and 100 mg/kg. Therefore, it may be said that the plant has strong anti-arthritis properties⁴⁴.

Terminalia chebula Retz. (TC) (Family-Combretaceae): TC is the most commonly utilized herb in ayurveda and a well-known traditional plant in the Indian traditional medical system. The TC, also referred to as Kadukkai among the Tamil Nadu tribe in India, was used to cure a number of illnesses, including fever, cough, diarrhea, gastroenteritis, skin conditions, candidiasis, UTIs, and wound infections³⁷. TC is a medium-sized deciduous tree with a diverse look. Its cylindrical bole is typically short, measuring 5–10 m in length and 60–80 cm in diameter. Tannins, flavonoids, resins, fixed oil, fructose, amino acids, and sterols are the phytoconstituents of TC. Additionally, gallic acid, ellagic acid, chebulagic acid, chebulinic acid, and chebulic acid are the active ingredients of tannins. In the traditional Thai system, TC was employed as an antioxidant, carminative, and expectorant. Chronic constipation, detoxification, poor digestion, and body rejuvenation are frequent

uses for the polyherbal combination "Triphala," which contains TC, *Terminalia bellerica*, and *Embolica officinalis* 45. TC possesses an anti-bacterial, anti-viral, anthelmintic, anti-fungal, anti-ameobic, anti-neoplastic, anti-plasmodial, antioxidant, anti-diabetic and anti-ulcerogenic activity. The TC reported to have an immunomodulatory radioprotective, cytoprotective, cardioprotective, and hepatoprotective activity. Furthermore, in models of formaldehyde-induced arthritis and CFA-induced arthritis, the hydroalcoholic extract of TC significantly reduces joint swelling. The extract's anti-arthritic properties resulted from a notable decrease in TNF- α , IL-6, and IL-1 β levels 46.

***Trigonella foenum-graecum* Linn (TF) (Family Papilionaceae):** TF, often referred to as fenugreek, is a herbaceous plant that has been used extensively as a traditional medicinal, food additive, and food. TF contains ash, woody fibers, soluble

carbohydrates, and albuminoids 47. In traditional medicine, the plant is used to cure a variety of conditions, including diabetes, high cholesterol, wounds, inflammation, and gastrointestinal disorders. Methimodaka, Svalpamethimodaka, and other TF confections are used to treat rheumatism 37.

Choline, trigonelline, and mucilage are all abundant in fenugreek seeds. Research on its extract has revealed anti-hyperglycemic and estrogenic, antioxidant, anticancer, anti-inflammatory, and antirheumatic activities. At a dose of 75 mg/kg, the fenugreek mucilage extracted from TF seeds exhibited anti-arthritic properties and reduced increased SGOT, SGPT, CRP, nitrites, ESR, and white blood cell count levels. The TF may work by reducing the release of arthritis-related mediators, oxidative stress, and cell influx. Finally, TF demonstrated anti-arthritic properties 48.

Phytochemical Constituents of Anti-Arthritic Plants:

TABLE 1: PHYTOCHEMICAL CONSTITUENTS OF ANTI-ARTHRITIC PLANTS

Botanical name	Family	Common name	Part used	Chemical constituent	Ref.
<i>Acalypha indica</i> Linn.	Euphorbiaceae	Kuppu, Aritmanjarie	Leaves	Quercetin, kaempferol, Ellagic acid, Beta-sitosterol	49
<i>Berberis asiatica</i> Roxb.	Berberidaceae	Kilmora	Steam	Berberine, Palmatine, Quercetin, kaempferol, Ellagic acid	37
<i>Cissus quadrangularis</i> Linn.	Vitaceae	Devil's backbone	Whole plant	Quercetin, kaempferol, Beta-sitosterol, Ascorbic acid	50
<i>Datura alba</i> Nees.	Solanaceae	Thornapple, tattur	Leaves	Quercetin, kaempferol, Beta-sitosterol, Stigmasterol	37
<i>Embolica officinalis</i> Gaertn.	Euphorbiaceae	Amla	Fruits	Quercetin, kaempferol, Rutin, Ellagic acid, Beta-sitosterol, Ascorbic acid, Gallic acid, Betulinic Acid	51
<i>Ferula narthex</i> Boiss.	Umbelliferae	Hingra, bhutari, devil's dung	Leaves	Quercetin, kaempferol, Ferulic Acid, Vanillic Acid, Ferutinin	52
<i>Gelsemium nitidum</i> Michaux.	Loganiaceae	Wild yellow jessamine	Root	Quercetin, kaempferol, Gelsemine, Gelsemicine, Koumine, Humantenine, oleanolic acid	53
<i>Hiptage benghalensis</i> Linn.	Malpighiaceae	Hutimukta, kampti	Leaves	Quercetin, kaempferol, Lupeol, Betulinic Acid, Gallic Acid, Ellagic Acid, Catechins	52
<i>Indigofera oblongifolia</i> Forsk.	Papilionaceae	Jhilla, mridupatraka	Root	Quercetin, kaempferol, Ellagic acid, Gallic acid, Beta-sitosterol	52
<i>Justicagendaruusa</i> Burm.	Acanthaceae	Nilinargandi, kapika, bhutakeshi	Leaves	Apigenin, luteolin, quercetin, endarusan A and B, caffeic acid, chlorogenic acid	54
<i>Koelpinia linearis</i> Pallas.	Asteraceae	Koelpinia	Whole plant	Quercetin, kaempferol, Gallic acid, caffeic acid	50
<i>Merremia tridentate</i> Hallier.	Convolvulaceae	Prasarini	Whole plant	Quercetin, kaempferol, luteolin, Gallic acid, ferulic acid, β -sitosterol, stigmasterol	55
<i>Naregamia alata</i>	Meliaceae	Goanese	Whole plant	Quercetin, kaempferol,	52

W.& A.		ipecacuanha, amlavalli		Naregamieine, Lupeol, Betulinic Acid, Gallic acid	
<i>Ocimum sanctum</i> Linn.	Lamiaceae	Basil	Leaves	Quercetin, luteolin, Apigenin, Rosmarinic Acid, Caffeic Acid, Eugenol, Ursolic Acid, Oleanolic Acid	56
<i>Pergularisdaemia</i> Linn.	Apocynaceae	Utaran, akasan	Leaves	Quercetin, kaempferol, luteolin, β - sitosterol, stigmasterol, Lupeol, Betulinic Acid	50
<i>Rhamnus catharticus</i> Linn.	Rhamnaceae	Buckthorn	Ripeberries	Quercetin, kaempferol, Emodin, gallic acid, caffeic acid	57
<i>Salacia reticulate</i> Wight.	Celastraceae	Ekanayakam, koranti	Root & Bark	Quercetin, kaempferol, gallic acid, caffeic acid, Betulinic Acid, Oleanolic Acid	58
<i>Tinospora cordifolia</i> Miers.	Menispermaceae	Ambarvel, gharol, gulwel	Root, Stem	Quercetin, kaempferol, gallic acid, Magnoflorine, Palmatine, Berberine	52
<i>Urtica dioica</i> Linn.	Urticaceae	Common nettle, scaddie	Leaves	Quercetin, kaempferol, Rutin, Gallic acid, ferulic acid, β -sitosterol	52
<i>Vitex negundo</i> Linn.	Verbenaceae	Nirgundi, mewri, sawbhalu	Leaves	Vitexin, Isovitexin, Rutin, Oleanolic Acid, Betulinic Acid, Vitexine, Negundoside	59
<i>Withaniasomnifera</i> Du nal.	Solanaceae	Ashvagandha, winter cherry	Root	Withaferin A, Withanolide D, Withanone, Somniferine, Caffeic Acid, Chlorogenic	60
<i>Xylia dolabrififormis</i> Benth.	Papilionaceae	Schmsapa, irula	Bark	Quercetin, kaempferol, luteolin, Gallic Acid, Tannic Acid	37
<i>Zingiber officinale</i> Roscoe.	Taminaceae	Nagaram, ginger sonth	Rhizome	Zingiberene, Caffeic Acid, Ferulic Acid, β -Bisabolene	61

DISCUSSION: This review underscores the therapeutic potential of traditionally used medicinal plants in the management of arthritis. The bioactive phytochemicals identified in plants such as *Ocimum sanctum*, *Zingiber officinale*, *Cissus quadrangularis*, and *Withaniasomnifera* including quercetin, kaempferol, luteolin, gallic acid, and β -sitosterol exhibit significant anti-inflammatory, antioxidant, and immunomodulatory properties. These compounds target key inflammatory pathways, reduce oxidative stress, and protect joint tissues, offering a natural, holistic approach to arthritis management.

The study also highlights the need for further research to validate the efficacy of these plants through clinical trials, standardize extraction methods, and develop formulations that maximize bioavailability and therapeutic outcomes. Integrating these phytochemicals into modern medicine could lead to safer, more cost-effective alternatives or complementary therapies to conventional treatments, minimizing the adverse effects associated with synthetic drugs. In conclusion, leveraging the synergy between traditional knowledge and modern science provides

a promising path toward sustainable and effective solutions for arthritis and other inflammatory conditions.

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