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HERBAL PLANT USED IN THE TREATMENT OF RHEUMATOID ARTHRITIS: A SYSTEMATIC REVIEW

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ABSTRACT

Rheumatoid arthritis (RA) is a chronic autoimmune disease characterized by persistent synovial inflammation, progressive cartilage and bone destruction, and functional impairment. The pathogenesis of RA involves complex immune dysregulation, including autoantibody production, abnormal T- and B-cell activation, and excessive release of pro-inflammatory cytokines such as TNF-α, IL-1, and IL-6. Although conventional therapies such as disease-modifying anti-rheumatic drugs, non-steroidal anti-inflammatory drugs, and corticosteroids are effective in symptom management, their long-term use is associated with adverse effects and limited disease remission. Growing interest in herbal medicines has highlighted their potential as alternative or complementary therapies for RA due to their multi-targeted pharmacological actions and improved safety profiles. Various medicinal plants and their bioactive

constituents have demonstrated anti-inflammatory, immunomodulatory, and antioxidant activities in experimental arthritis models. This review summarizes the pathogenesis of RA, limitations of current treatments, and the therapeutic potential of herbal medicines, emphasizing the need for further clinical validation.

KEYWORDS: Rheumatoid arthritis, autoimmune disease, Inflammation.

KEY MESSAGES

Rheumatoid arthritis (RA) is a chronic autoimmune disease driven by immune dysregulation and pro-inflammatory cytokines, leading to joint damage and disability. Current therapies, while effective in symptom control, are limited by adverse effects and incomplete remission. Herbal medicines, with their multi-targeted anti-inflammatory, immunomodulatory, and antioxidant properties, show promise as complementary approaches. However, rigorous clinical validation is essential to establish their therapeutic role in RA management.

INTRODUCTION

Rheumatoid arthritis (RA) is a chronic autoimmune disorder characterized by persistent synovial inflammation, leading to joint destruction and systemic manifestations.[1] The pathophysiology of RA involves complex immune mechanisms including activation of T and B lymphocytes and macrophages, which secrete pro-inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α), interleukin-1 (IL-1), and interleukin-6 (IL-6). These cytokines promote pannus formation, an invasive granulation tissue that erodes cartilage and bone, driving progressive joint damage through cycles of immune activation and synovial hyperplasia. Genetic predisposition and environmental factors, including smoking and infections, contribute to disease initiation and persistence.[2] Histopathologically, RA is marked by synovial membrane hyperplasia with infiltration of lymphocytes and plasma cells. The synovium transforms into pannus tissue that invades adjacent cartilage and subchondral bone, resulting in cartilage degradation and bone erosion. Microscopically, this is characterized by synovial lining thickening, neovascularization, and lymphoid aggregate formation. Cartilage exhibits fibrillation and proteoglycan loss, while bone resorption is mediated by osteoclast activation. These cellular and structural alterations underlie clinical manifestations such as joint swelling, pain, and deformity.[3,4] Several herbal plants have been traditionally employed in RA treatment, utilizing their anti-inflammatory and immunomodulatory properties to alleviate symptoms and modulate disease progression.[5]

EPIDEMIOLOGY

Arthritis has a global prevalence of approximately 1–2% and may present at any age, with a higher incidence observed in females. The condition is most prevalent among individuals aged 15–45 years, with a female-to-male ratio of nearly 6:1. In contrast, sex distribution is relatively balanced during early childhood and in individuals over 60 years of age. Epidemiological studies indicate that genetic predisposition, in combination with environmental triggers, contributes significantly to disease manifestation. The etiology of arthritis varies among its forms; osteoarthritis is primarily associated with advancing age, female sex, prior joint injury, and obesity. Furthermore, genetic abnormalities, including mutations in collagen-related genes, have been implicated in disease progression.[6]

Signs, Symptoms, and Diagnosis

Rheumatoid arthritis presents with joint pain, swelling, prolonged morning stiffness, fatigue, and systemic symptoms. Diagnosis relies on clinical assessment supported by serological markers such as rheumatoid factor and imaging findings, as recommended by the American College of Rheumatology.[7, 8]

Risk Factors of Rheumatoid Arthritis

- **Genetic predisposition:** A positive family history increases susceptibility to rheumatoid arthritis.
- **Age and sex:** Risk increases with age, and females are affected more frequently than males.
- **Joint injury:** Previous trauma to joints may contribute to the later development of arthritis.
- **Obesity:** Excess body weight places mechanical stress on joints and promotes inflammation.
- **Smoking:** Cigarette smoking significantly elevates the risk, especially in genetically predisposed individuals.[9]

PATHOGENESIS OF RA

Rheumatoid arthritis (RA) is a chronic inflammatory autoimmune disorder with a multifactorial etiology involving genetic susceptibility and environmental triggers. Its pathogenesis is characterized by immune dysregulation, activation of inflammatory pathways, and associated genetic, epigenetic, and metabolic alterations that collectively drive persistent synovial inflammation and joint damage.[10]

Immunological Basis of Rheumatoid Arthritis

AUTOANTIBODIES

Autoantibodies frequently precede the clinical onset of rheumatoid arthritis (RA), with anti-citrullinated protein antibodies (ACPAs) serving as the most specific diagnostic markers. These antibodies arise from citrullination of proteins by peptidyl arginine deiminase enzymes, enhancing antigen presentation via HLA-DR molecules and promoting autoimmunity. Neutrophil extracellular traps contribute to the pool of citrullinated antigens and sustain synovial inflammation. ACPAs, along with rheumatoid factor and other anti-modified protein antibodies, actively participate in synovial inflammation, bone erosion, and systemic complications.

T- AND B-CELL–MEDIATED IMMUNITY

Rheumatoid arthritis is driven by persistent immune activation within the synovium, characterized by lymphoid aggregates and ectopic germinal centers. $CD4^+$ T cells dominate the inflammatory infiltrate and preferentially differentiate into Th1 and Th17 subsets, while regulatory T-cell function is impaired. $CD8^+$ T cells further amplify inflammation through cytokine production. B cells contribute to disease progression by producing autoantibodies, secreting pro-inflammatory cytokines, and activating fibroblast-like synoviocytes, there by perpetuating chronic synovial inflammation.

Signaling Pathways Involved in the Pathogenesis of Rheumatoid Arthritis

Multiple intracellular signaling pathways play a central role in the pathogenesis of rheumatoid arthritis (RA) by regulating immune activation, inflammation, and joint destruction. Dysregulation of these pathways represents a major target for therapeutic intervention.

JAK–STAT PATHWAY

The JAK–STAT pathway mediates cytokine-driven inflammatory responses in RA. Pro-inflammatory cytokines such as IFN-γ and IL-6 activate JAK1/JAK2, leading to STAT1 and STAT3 phosphorylation and nuclear translocation, which promotes macrophage activation, acute-phase responses, and synovial inflammation. Pharmacological inhibition of JAKs has emerged as an effective therapeutic strategy in RA.

P38 MAPK PATHWAY

Activation of the p38 MAPK pathway by inflammatory cytokines enhances the production of TNF-α, IL-1β, and IL-6 and induces chondrocyte apoptosis, contributing to cartilage degradation and joint damage. Inhibition of p38 MAPK suppresses cytokine release and mitigates inflammatory and degenerative processes in RA.

NF-KB PATHWAY

NF-κB signaling is a key regulator of inflammatory gene expression in RA. Its activation in synovial immune cells and fibroblasts induces cytokines, chemokines, and matrix-degrading enzymes, thereby promoting chronic synovial inflammation and cartilage destruction. Suppression of NF-κB activity reduces inflammatory mediator production and joint damage.

RANKL–RANK PATHWAY

The RANKL–RANK signaling axis drives osteoclast differentiation and activation, leading to bone erosion in RA. Engagement of RANK activates downstream pathways, including NF-κB, PI3K/Akt, and MAPKs, resulting in enhanced osteoclastogenesis and bone loss. Targeting this pathway helps prevent bone destruction and joint deterioration.[11,12]

Table 1: Experimental Model Used In RA.

Animal Model	Method of Induction (Brief)	Key RA Features Mimicked	Parameters Measured	Relevance to Herbal Studies
Collagen-Induced Arthritis (CIA)	Immunization with type II collagen	Autoimmune synovitis, autoantibody production, cartilage and bone erosion	Paw swelling, arthritis score, TNF-α, histology	Widely used to evaluate immunomodulatory and disease-modifying effects of herbal compounds[13,14]
Freund's Complete Adjuvant (FCA)-Induced Arthritis	Injection of mycobacterial adjuvant	Chronic inflammation, joint swelling, systemic immune activation	Paw edema, arthritis index, hematology	Suitable for assessing anti-inflammatory and analgesic potential of plant extracts[15,16]
Antigen-Induced Arthritis (AIA)	Local antigen challenge in sensitized animals	Acute immune-mediated synovial inflammation	Joint swelling, cytokines, mobility, histopathology	Useful for studying inflammatory mechanisms and short-term efficacy of herbal agents[17]

General Treatment for Rheumatoid Arthritis and Its Limitations

Management of rheumatoid arthritis (RA) primarily focuses on reducing pain, inflammation, and preventing joint damage. Conventional therapies include disease-modifying anti-rheumatic drugs (DMARDs), non-steroidal anti-inflammatory drugs (NSAIDs), and corticosteroids. While these treatments alleviate symptoms, they do not cure RA or halt disease progression. Long-term use of these drugs can cause serious side effects, including liver, kidney, and heart issues, as well as short-term effects like nausea, infections, and allergic reactions. Thus, current treatments are limited to symptom management rather than addressing the root cause of RA.[18]

Needs For Natural Remedies

Natural remedies have been used for centuries to manage various diseases, including rheumatoid arthritis. According to the WHO, around 80% of the global population still relies on herbal treatments. Traditional use of plants, whether as infusions, raw forms, or juices, has shown beneficial effects, highlighting the potential of phytoconstituents in developing effective therapies for RA.[19]

Possible Mechanism of Action of Herbal Drugs

Herbal drugs exert multi-targeted effects in rheumatoid arthritis (RA) by modulating key inflammatory pathways. Toll-like receptors (TLRs) activate NF-κB via MyD88-dependent signaling, leading to transcription of pro-inflammatory genes such as iNOS, COX-2, and IL-6. This promotes prostaglandin synthesis, leukocyte proliferation, and pannus formation. Pro-inflammatory cytokines, including TNF-α, IL-1β, and IL-6, further drive cartilage degradation by activating collagenases and proteases, while enhancing infiltration of T cells, B cells, and macrophages into synovial tissue. Herbal agents have been shown to inhibit NF-κB activation, downregulate pro-inflammatory cytokines, and reduce oxidative stress, thereby mitigating inflammation and joint damage in RA.[20]

Table 2: Possible Mechanism of Action of some Herbal Drugs.

Herbal Plant (Scientific Name)	Active Compound / Phytochemicals	Experimental Model / Study Type	Mechanism of Action	Part used	Reference
Curcuma longa	Curcumin	CIA, FCA	NF-κB inhibition, anti-inflammatory, antioxidant	Rhizome	[21,22,23]
Withania somnifera	Withanolides	CIA, AIA	Immunomodulatory, anti-inflammatory,	Roots	[24,25,26]

			antioxidant		
Boswellia serrata	Boswellic acids	FCA	Inhibits leukotriene synthesis, anti-inflammatory	Root & seed	[27,28]
Tinospora cordifolia	Alkaloids, Glycosides	CIA	Immunomodulatory, cytokine suppression	Whole plant	[29,30]
Zingiber officinale	Gingerols, Shogaols	CIA	Anti-inflammatory, NF-κB inhibition	Rhizome	[31,32]
Camellia sinensis	Catechins (EGCG)	CIA, FCA	Antioxidant, inhibits pro-inflammatory cytokines	Leaves	[33,34]
Glycyrrhiza glabra	Glycyrrhizin	CIA	Anti-inflammatory, inhibits COX-2, cytokine suppression	Roots	[35,36]
Aloe barbadensis	Anthraquinone, anthracene	FCA	Antioxidant, inhibits pro-inflammatory cytokines	Leaves	[37, 38]
Piper nigrum Linn.	alkaloid piperine	CIA	Anti- inflammatory	Seed	[39,40]
Calotropis procera Linn.	alkaloid piperine	CIA	Inhibit the inflammatory cell	Leaves	[41,42]
Ficus bengalensis Linn.	tannins, saponin	FCA	anti-rheumatic activity	Stem Bark	[43,44]
Saraca asoca Roxb.	alkaloid piperine	FCA	Anti- inflammatory	Seed	[45,46]
Coriandrum sativum Linn.	tannins, saponin	FCA	Inhibit the secretion of pro-inflammatory cytokines including TNF-α	Leaves	[47,48]
Ruta graveolens Linn.	Anthraquinone, anthracene	FCA	Reduces cell influx, release of mediators, lipid peroxidation and oxidative stre	Leaves	[49,50]
Terminalia chebula Retz.	gallic acid, resins, anthraquinone	FCA	modulatory effect on pro-inflammatory cytokine expression in the synovium	Fruits	[51,52,53]
Strychnos potatorum Linn	triterpenes sterols and mannogalactans	FCA	Inhibit the secretion of pro-inflammatory cytokines	Seed	[54]
Cleome gynandra	anthroquinones, flavonoids,	AIA	anti-rheumatic activity	Leaves, Seed	[55]

Linn					
Adhatoda vasica nees	Anthraquinones	CIA	Reduced pro-inflammatory cytokines in serum and synovial tissues	leaves	[56]
Clerodendrum serratum	Triterpene, tannin, glycosides	cotton pellet model	anti-inflammatory activity	Leaves	[57,58]
Costus speciosus:	costunolide	FCA	Inhibition of NF-Kβ and MAPkinase pathways	Leaves	[59]
Cedrus deodara (Roxb.) Loud	Triterpene, tannin, glycosides	carrageenan	Inhibition of exudative-proliferative and chronic phase of inflammation	Leaves	[60]
Callicarpa macropohylla	carbohydrate, steroids, flavonoids and tannins	Albumin injected paw edem	Inhibit the histamine, serotonin, PGs synthesis which plays the major role in inflammation	Leaves	[61]
Citrullus colocynthis	glycoside quercetin	carrageenan-injected paw edem	Reduce the production like IL-6 and IL-1β and COX-2	Leaves	[62]
Cinnamomum zeylanicum	gallic acid and polyphenols	FCA	inhibits the PGs synthesis	Bark	[63]
Celastrus paniculatus willd	Sesquiterpene, Celapanigin, palmitic acid	carrageenan induce paw edema	Anti-inflammatory action	Seed	[64]
Euphorbia tirucalli	Triterpenoids	CIA	Antioxidant, inhibits pro-inflammatory cytokines	Leaves	[65]
Lavandula stoechas L	Coumarins, Leucoanthocyans, Flavonoids, Mucilags,	CIA	Inhibits the PGs synthesis	leaves and flowers	[66]
Pinus lambertiana	Pinitol	AIA	Suppression of mediators such as PGE2 and Leukotriene B4 (LTB4)	Leaves	[67]
Premna serratifolia linn	alkaloids, steroids, flavonoids, phenolic compounds, tannins	FCA	anti-inflammatory, antioxidant	Leaves	[68]

CONCLUSION

Rheumatoid arthritis is a chronic autoimmune disorder characterized by joint inflammation, pain, and progressive deformities. Conventional treatments, including DMARDs, NSAIDs, and corticosteroids, provide temporary symptomatic relief but are associated with adverse effects and do not offer a definitive cure. In this context, herbal therapies represent a promising alternative approach for RA management. Traditional and indigenous medicinal systems contain extensive knowledge of plant-based remedies with minimal side effects. Numerous herbal formulations and phytochemicals with anti-arthritic potential have been scientifically validated, with over 450 plant species reported to exhibit anti-arthritic activity. This review highlights medicinal plants with therapeutic potential and aims to encourage further research in the development of safer and more effective treatments for rheumatoid arthritis.

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