WWW.IJRPR.COM

International Journal of Research Publication and Reviews

Journal homepage: www.ijrpr.com ISSN 2582-7421

Review on an Ayurvedic Polyherbal Formulation for Rheumatoid Arthritis

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ABSTRACT:

A complex autoimmune illness with an unclear etiology, rheumatoid arthritis (RA) primarily affects the joints, though it can also appear extra-articularly. A multidisciplinary approach is necessary for effective RA care because of its complexity, which stems from a pathophysiological process that is still poorly understood. It is a chronic, debilitating autoimmune disease that damages the bone and cartilage surrounding joints and causes systemic inflammation. Being a systemic disease, it can impact internal organs including the heart, lungs, and eyes as well as the entire body. Despite the fact that many synthetic medications are utilized as standard treatments for rheumatoid arthritis, their side effects can jeopardize the effectiveness of the therapeutic approach. Unfortunately, there is now no proven medication that can effectively treat rheumatoid arthritis; instead, contemporary medicine can only address its symptoms, which include joint discomfort and inflammation. The pain and inflammation in the joints can be reduced by using the plants and herbs in different ways. Traditional medicine uses a wide variety of herbal medications, and fundamental scientific study has shown that these treatments are effective in treating arthritis. Through the suppression of inflammatory biomarkers with fewer adverse effects and the provision of information for future research into natural drug therapy for rheumatoid arthritis, the current review has elaborated on a selection of traditionally used herbal medicinal plants with phytoconstituents that possess anti-inflammatory activity.

Keywords: Rheumatoid Arthritis, Herbal Plants, Joints.

INTRODUCTION:

Joint synovial inflammation and progressive bone and cartilage degradation leading to progressive immobility are hallmarks of rheumatoid arthritis (RA), a chronic inflammatory illness with an unclear etiology [1]. Inflammation of the joints, or arthritis, is one of the oldest known disorders that affect people of all ages. Approximately 20% of the population in India suffers from arthritis [2].

There is no hereditary component to rheumatoid arthritis. Scientists think that certain individuals are predisposed to the illness due to their genetic makeup. Rheumatoid arthritis does not always develop in people with these genes. Usually, an illness or environmental factor acts as a "trigger," activating the genes. When this trigger is present in the body, the immune system responds incorrectly. The immune system begins to produce substances that harm the joint instead of protecting it. As a result, rheumatoid arthritis may develop. It is an autoimmune disease, which means that the body's immune system unintentionally attacks healthy tissues. Normal joints have a relatively thin lining with few blood vessels, but rheumatoid arthritis joints have a thick lining filled with white blood cells. Chemicals such as interleukin-1 (IL-1) and tumor necrosis factor alpha (TNF-alpha), which cause pain, joint swelling, and joint destruction, are secreted by white blood cells. New cytokines such as IL-17 and IL-182 have been found recently. These cytokines cause the chondrocytes and synovial fibroblasts in the surrounding articular cartilage to release enzymes that break down collagen and proteoglycans, causing tissue degradation and the involvement of RANK ligand (RANKL) in the pathophysiology of chronic arthritis [3,4]. Although arthritis may impact anyone at any age, it is most common between the ages of 25 and 50, peaking between the ages of 40 and 50. There are roughly 100 different kinds of arthritis, but the most prevalent ones include juvenile arthritis, osteoarthritis, rheumatoid arthritis, ankylosing spondylitis (AIS), and systemic lupus erythematous [5].

Herbal Therapy for the Treatment of Arthritis:

Since ancient times, herbal medicines have been used to treat a wide range of illnesses, and it is not hyperbole to claim that the usage of herbal medications predates humanity [6]. Herbal remedies are created using the therapeutic expertise of generations of doctors who have been working in the traditional medical system for hundreds of years [7]. Because the medications that are now on the market are either very costly or have specific negative effects, researchers are now very interested in therapeutic substances that are produced from plants [8]. Herbal plants, which are abundant worldwide and provide therapeutic compounds for the prevention and treatment of a wide range of illnesses, are a gift from nature [9]. Over 80 percent of people worldwide utilize herbal medications for their basic medical needs, according to the WHO. Since the beginning of civilization, herbal remedies have been a part of human society's efforts to fight disease [10]. These herbal plants' medicinally significant components are chemical components that give the body the required physiological effect [11].

Herbal remedies have been used in India's officially recognized alternative health systems, including Ayurveda, Unani, Sidha, Homeopathy, and Naturopathy, since ancient times [12]. More than 2500 plant species are now utilized as herbal medicines in India. Herbal remedies have been utilized for almost 3,000 years, either directly as traditional medicine or indirectly in the manufacturing of modern pharmaceuticals [13]. Therefore, one may be able to find novel, more affordable, and effective medications by using the knowledge of traditional botanicals [14].

Polyherbal Formulations for Arthritis:

In patients with acute or chronic RA, analgesics and NSAIDs can help reduce pain and inflammation [15]. Even though there are treatments for RA, new strategies are being developed to balance the known advantages and possible hazards because of the possibility of irreversible organ damage or negative effects [16]. Currently, safer and more effective medications are being produced from eastern sources to treat RA. To lessen these negative effects and boost the positive ones, a wide range of herbal extracts and medicines, including polyherbal combinations, are made [15].

The polyherbal combination known as Rheum off Gold is frequently suggested by Ayurvedic physicians to cure arthritis. Using a complete Freund's adjuvant (CFA)-induced arthritis model in Wistar rats, the anti-arthritic activity was verified. The arthritis index, paw thickness, and inflammatory indicators such as C-reactive protein, serum rheumatoid factor, and erythrocyte sedimentation rate (ESR) all significantly decreased in comparison to dexamethasone. The formulation might therefore have anti-arthritic qualities [17].

Rats were used to test an Unani polyherbal mixture for antiarthritic effects. Models of formaldehyde and CFA-induced arthritis were used to assess Manjoon Suranjan's anti-arthritic effectiveness. The information gathered indicated that the formulation has anti-arthritic properties [18]. Sudard was evaluated as a potent anti-arthritic polyherbal compound using Wistar rat models of arthritis caused by formaldehyde and adjuvants. At 150 mg/kg and 300 mg/kg p.o. doses, the formulation has anti-inflammatory and anti-arthritic effects [19].

Tongbiling (TBL-II) possesses anti-arthritic qualities and was created by altering the Chinese herbal formulation of TBL. The formulation's anti-arthritic efficacy was examined in Wistar rats using a collagen-induced arthritis model. The TNF-α and IL-1β levels were observed to be significantly reduced at 100 and 300 mg/kg p.o. Consequently, the anti-arthritic potential of the formulation was established [20].

For the past century, the Chinese herbal combination HLXL has been utilized to treat arthritis and inflammation. Additionally, utilizing a CFA model in rats, the anti-arthritic properties of HLXL herbal formulation were assessed following some adjustments. The polyherbal preparation was found to have anti-arthritic activity by dramatically lowering TNF-α and IL-β levels as well as paw oedema [21].

Ganghwaljetongyeum's ability to treat RA in rabbit knee synovial membranes was investigated. HG82 cell growth was found to be significantly inhibited, indicating that the polyherbal formulation had anti-arthritic properties. Additionally, TNF-α, IL-10, and NO species were significantly reduced [15].

Epidemiology:

The World Health Organization (WHO) estimated that between 0.5 and 2% of adults worldwide suffer from RA, with a higher risk of developing the disease in North America and Europe than in Asia [22–24]. In India, between 0.28 and 0.7% of people have RA, and the prevalence is higher in rural areas than in urban areas [25–26]. The risk of having RA in adults is 1.7% (1 in 59) for men and 3.6% (1 in 28) for women. Patients with RA have a threefold higher overall mortality rate than the general population [26,27]. Accordingly, the survey data indicated that RA is more common in women than in men, meaning that women are two to three times more likely to get RA than men because of the drop in estrogen levels that occurs following menopause [27]. Additionally, depending on the severity of the disease and other genetic factors, the lifespan expectancy with RA disease might be lowered by up to 5–10 years [28, 29].

Etiological factors of rheumatoid arthritis:

Environmental factors, sex hormones, and genetic factors combine to trigger the immunological response [30]. The evolution of the current condition is believed to have been influenced by a number of variables. (1) Effects of sex and sex hormones: elderly women are three to four times more likely than men to develop RA [27].In addition to its anti-inflammatory effects, estrogen suppresses NF-jB signaling by upregulating the expression of jB-Ras2, an NF-jB inhibitor that further suppresses the genes of inflammatory mediators and lowers TNF-alpha, interleukins (IL-6), and other chemicals linked to arthritis [31].Rheumatoid arthritis was caused by women's decreasing estrogen levels during the onset of menopause [32]. Oestrogen regulates metabolism, has anti-inflammatory effects, and aids in fat maintenance. The menopause decrease in oestrogen levels often causes arthritis. Pregnancy, the postpartum period, and the perimenopausal era are therefore times when RA is more prevalent in women [33]. Serum levels of BND cells, IL-4, and IL-6 are also elevated by the hyperprolactinemia that causes auto-antibodies [34]. (2) Genetic factors: RA was 12–15% more common among monozygotic twins than in the general population, which is about 2–5% higher. This implies that STAT% and signalling transducer polymorphisms exist in siblings [35, 36]. Rheumatoid arthritis is approximately 40–65% heritable, or genetically linked, due to mutations in the human leucocyte antigen (HLA) gene and other immune- and inflammatory-like interleukin (IL-4, IL-10) genes [35,37]. (3) The environment and additional elements: Numerous bacteria found in the environment, including Salmonella, Campylobacter, Porphyromonas gingivalis, Proteus mirabilis, and Mycoplasma organism, have caused RA [38]. Moreover, viruses including the rubella virus and Epstein-Barr virus (EBV) are the cause of RA [39]. Additionally, rheumatoid arthritis has been linked to changes in the makeup and function of the intestinal microbiome, meaning that patients with the disease have a less diverse gut microbiota than healthy people [40]. Because it causes the formation of RF factor, which is up to three times more prevalent in smokers than non-smokers, smoking is also one of the risk factors for rheumatoid arthritis [41, 42].

SYMPTOMS:

Joint discomfort and swelling, stiffness in the morning, sleep issues, exhaustion, weight loss, and a feeling of having the flu are some of the symptoms. Rheumatoid arthritis is diagnosed by looking for abnormal antibodies (IgG) in the blood, known as rheumatoid factor. An antigen-antibody complex is created when these react with antigen, resulting in pain and inflammation of the synovial membrane. The diagnosis must be confirmed by at least four of the seven criteria listed below, according to the American College of Rheumatology [43, 44].

- Morning stiffness around the joint that lasts at least 1 hour
- Arthritis of three or more joints for at least 6 weeks
- Arthritis of hand joints for at least 6 weeks
- Arthritis on both sides of the body for at least 6 weeks
- Rheumatoid nodules under the skin
- Rheumatoid factor presents in blood testing
- Evidence of rheumatoid arthritis on X-rays

The widespread occurrence of arthritis symptoms and their impact on social, professional, and physical activities can be depressing, leading to psychological aftereffects as anxiety, melancholy, and a sense of powerlessness [45]. Because RA might start with mild symptoms like achy joints or mild stiffness, it can be challenging to diagnose. When RA is active, the stiffness is usually at its worst in the morning. It may go on for an hour or two, or possibly the entire day. Since few other arthritic diseases exhibit stiffness for an extended period of time in the morning, this is a sign that a person may have RA [46].

In 1958, the American Rheumatism Association (ARA) initially proposed classification criteria for RA [47]. The American College of Rheumatology (ACR) updated the 1958 ARA criteria in 1987 [48].

Table 1: The 1987 revised ARA/ACR criteria for classification off rheumatoid arthritis.

Criterion	Simple title	Definition
1.	Morning stiffness	Stiffness in and around the joints in the morning that lasts for at least an hour before getting better. Three joints or more.
2.	3 or more joint regions with arthritis	A doctor has noticed simultaneous soft tissue swelling or fluid in certain areas (not only bone enlargement). Right or left PIP, MCP, wrist, elbow, knee, ankle and MTP joints are among the 14 potential locations.
3.	Arthritis of hand Joints	At least one wrist, MCP, PIP joint area that is inflamed (as previously described)
4.	Symmetric Arthritis	Joint areas [as specified in (2)] on both sides of the body may be involved simultaneously (bilateral involvement of PIPs, MCPs, or MTPs is permissible without total symmetry).
5.	Rheumatoid Nodules	A doctor's observation of subcutaneous nodules, over bony prominences, or extensor in juxta articular regions.
6.	Serum rheumatoid factor	Serum rheumatoid factor levels that are abnormal, or any test that yields a positive result in less than 5% of healthy control subjects.
7.	Radiographic Changes	Rheumatoid arthritis radiographic abnormalities on posteroanterior hand and wrist radiographs must include erosions or clear bone decalcification that is localised in or most noticeable around the affected joints (changes from osteoarthritis alone do not qualify).

RISK FACTORS FOR RA:

Genetic, environmental, and stochastic factors all contribute to RA, which is a complex disease [49]. According to scientific studies, the estimated hereditary risk for RA is almost 50% [50,51]. RA can be classified as either seropositive or seronegative based on the presence or absence of rheumatoid factor (RF) and ACPAs. There are also variations in the risk variables that are implicated [52,53]. The primary genetic factors linked to an ACPA-positive subtype are tyrosine phosphatase non-receptor type 22 (PTPN22) risk alleles [54,55], human leukocyte antigen D-related (HLA-DR) alleles [52], and genes related to tumor necrosis factor-receptor associated factor 1 and complement component 5 (TRAF1/C5) [56], whereas interferon regulatory factor 5 (IRF-5) is exclusive to the ACPA-negative subtype [57]. Environmental risk factors are crucial in managing RA since they are major contributors to population health. Smoking has been connected to the onset or aggravation of RA, just as other diseases. By chance, a study with a different goal discovered the first evidence linking smoking to an increased risk of RA [58]. It has since emerged as the most well-characterized risk factor for RA. Numerous studies have examined the dangerous compounds found in tobacco products, and the findings indicate that smoking sends out a certain signal. A specific subtype of RA may be triggered by smoking, which may be linked to a genetic context [59]. According to reports, smoking has no or very little influence on ACPA-negative RA [61] but has an impact on RF- or ACPA-positive RA [60]. Furthermore, smokers with HLA-DR Beta 1 shared epitope alleles have a significantly increased chance of developing ACPA-positive RA [62]. Passive smokers and the risk of having RA have not been found to be associated in any way [63].

One occupational exposure that affects RA is silica dust exposure. Patients with ACPA-positive RA are primarily affected by the reported correlation between silicosis and RA [64]. Rheumatoid pneumoconiosis, sometimes referred to as Caplan's syndrome, is an uncommon condition that occurs in RA patients who have developed silicosis as a result of prolonged exposure to silica [65].

Consumption patterns and dietary components have also been evaluated throughout time. RA is influenced by dietary factors, and studies have shown that vegetarian diets and fasting periods can help reduce the disease's progression. Additionally, limiting red meat and eating more fruit and oily fish may reduce the incidence of RA [66,67]. Coffee consumption may be a risk factor for RA because of its possible involvement in the development of RF [68]. Although further research is required to confirm this notion, a case-control study revealed that alcohol use may improve RA by lowering the risk of developing ACPA-positive RA [59]. A customized diet for each individual should therefore be taken into account.

One biological risk factor that could lead to the onset of RA is infection. According to a comparative cohort research, people with RA have a much higher incidence of joint, skin, and bone infections than those with non-inflammatory rheumatic disorders [69]. Furthermore, Lyme arthritis, a condition that shares many characteristics with RA, has also been linked to bacterial triggers [70].

A harmful bacterium called Porphyromonas gingivalis is the cause of periodontal disease. Due to its role in promoting osteoclast development and citrullination stimulation, there has been evidence of a connection between RA and periodontal disease [71].

The foundation for comprehending the intricacy of the biomolecular mechanisms that coordinate RA may be a thorough description of the interplay among environment, genes, and stochastic events.

PATHOPHYSIOLOGY OF RA:

Numerous theories have been proposed, despite the fact that the pathophysiological mechanisms behind RA remain incompletely understood. According to reports, immunological processes might take place years before joint inflammation symptoms appear; this is known as the "pre-RA phase" [72]. Modified self-antigens, including as immunoglobulin G (IgG), type 2 collagen, and vimentin, might result from the interplay between environmental influences and epigenetic changes on the genomic structure. Citrullination is a post-translational alteration that occurs when peptidyl arginine deiminases transform these proteins with arginine residues into citrulline [73,74]. Furthermore, cytokine production from joint conditions such as synovial hyperplasia or infections can result in joint inflammation and altered self-antigens [75].

The immune system can no longer identify citrullinated proteins (vimentin, type II collagen, histones, fibrin, fibronectin, Epstein-Barr nuclear antigen 1, α-enolase) as self-structures because of the susceptibility genes HLA-DR1 and HLA-DR4 [76]. Antigen-presenting cells (APCs) are activated dendritic cells that take up antigens in order to trigger an immune response. The entire complex moves to the lymph node, where CD4+ helper T cell activation occurs. Additionally, B cells in the lymph node's germinal Centre are stimulated by sequential and reciprocal impulses with T cells, a process known as costimulation in immunology.

The relationship between CD28 and CD80/86 is an example of costimulation [77,78]. At this stage, B cells develop into plasma cells that make autoantibodies based on the receptors of the precursor cells, undergo somatic hypermutation, or class-switch recombination, and begin to multiply [79]. Self-tissues and organs are unintentionally targeted by autoantibodies, which are proteins generated by an immune system that is no longer able to distinguish between self and non-self-components. RF and ACPA are the most extensively studied autoantibodies linked to RA. With an 85% testing specificity in RA patients, RF is an IgM antibody that targets the constant region, or Fc portion of IgG [80]. It also forms an immune complex that can pass through synovial fluid by joining forces with complement protein and IgG. By binding to citrullinated proteins and forming immune complexes that build up in the synovial fluid, ACPA more precisely targets RA [81].

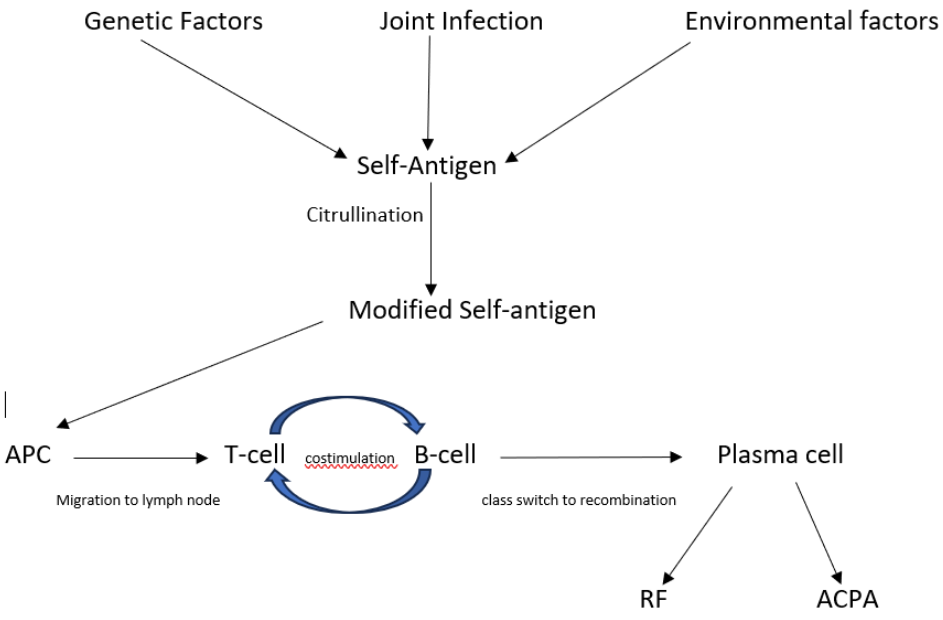

Figure 2. Immunological processes in the pre-RA phase. ACPA, anti-citrullinated protein antibodies; APC, antigen-presenting cells; RF, rheumatoid factor.

Table 2: Traditionally used anti-arthritic plants

Scientific name	Family	Common name	Disease Treated	Part used	Dosage form	References
Abrus precatorius Linn.	Papilionaceae	Indian liquorice, chirmiti	Menopausal symptoms, cough	L	Oil	[82]
Acacia catechu Willd.	Fabaceae	Mimosa catechu	Diarrhea, Osteoarthritis	R	Extract	[83]
Acalypha indica Linn.	Euphorbiaceae	Kuppu, Arittmanjarie	Asthma, Pneumonia	L	Juice	[84]
Acanthus illicifolius Linn.	Acanthaceae	Sea holly, Moranna harikusa	Paralysis, wounds	L	Extract	[82]
Achillea millefolium Linn.	Compositae	Rojmari, bloodwort, arrow-root	Parkinson's disease	H	Extract	[85]
Achyranthus aspera Linn.	Amaranthaceae	Chirchitta, aghada, prickly chaff-flower	Piles, kidney stone	R	Infusion	[86]
Actaea spicata Linn.	Ranunculaceae	Baneberry, grapewort	Inflammation, lumbago	R	Powder	[82]
Adansonia digitata Linn.	Malvaceae	Gorakh amli	Antitussive, antipyretic	L	Poultices	[84]
Aegel marmolosa Corr.	Rutaceae	Stone apple, bael	Anticancer	F	Juice	[88]
Ailanthus excels Roxb.	Simaroubaceae	Indian tree of heaven	Bronchitis	L	Oil, extract	[83]
Allium cepa Linn.	Liliaceae	Onion, palandu	Asthma, inflammatory disorders	Bu	Paste	[84]
Allium sativum Linn.	Liliaceae	Garlic, lasun	Rheumatism	S	Oil	[87]
Alstonia scholaris R.Br.	Apocynaceae	Datyuni, saptaparna	Malaria, jaundice	Mj	Juice	[89]
Althaea rosea Cav.	Malvaceae	Hollyhock, round dock	Inflammation, constipation	Fl	Oil	[82]
Ammannia baccifera Linn.	Lythraceae	Dadmari, agni-garva	Typhoid fever	L	Blisters	[90]
Amorphophallus campanulatus Roxb.	Aracea	Zamikand, kandula kandvardhana	Liver disease, piles	S	Oil	[82]
Apium graveolens Linn.	Umbellifeae	Ajmoda, celery	Gout	R	Decoction	[85]
Argyreia speciosa Sweet.	Convulvulaceae	Elephant creeper	Ulcers, diabetes	R	Powder	[84]
Aristolochia bracteata Linn.	Aristolochiaceae	Birthworts, pipevines	Tumors, malaria	Wh	Extract	[92]
Aristolochia serpentaria Linn.	Aristolochiaceae	Virginian snake root	Diuresis	R	Infusion	[91]
Asparagus racemosus Willd.	Liliaceae	Shatavari	Infertility	R	Oil	[87]
Atropa belladonna Linn.	Solanaceae	Black cherry, sagangur	Stomach ulcer	R, L	Extract	[82]
Azadirachta indica A. Juss.	Meliaceae	Bakayan, Indian lilac, balnimb	Anti-fungal	L	Decoction	[82]
Azima tetracantha Lam.	Salvadroraceae	Kundali	Rheumatism	L, R	Decoction	[84]
Bacopa monnieri Penell.	Plantaginaceae	Brahmi	Constipation	Wh	Extract	[93]
Barosma crenulata Hook.	Rutaceae	Bucchu, buku	Rheumatism	L	Powder	[91]
Bauhinia racemosa Lam.	Fabaceae	Bidi leaf tree, kachnal	Diabetes	B	Extract	[88]
Bauhinia tomentosa Linn.	Fabaceae	Yellow bell orchid	Liver inflammation	L	Infusion	[94]
Bidens pilosa Linn.	Compositae	Black jack, phutium	Cancers, wounds	Sh	Young shoots	[82]
Blumea ripens DC.	Asteraceae	Red stink wood	Skin wound	S	Oil	[83]
Bula alba Linn.	Cupuliferae	White birch bark	Rheumatism	L	Extraction	[87]
Boerhaavia diffusa Linn.	Nyctagineae	Punarnava, thikri	Anti dengue	R	Paste	[95]
Boucerosia aucheriana Dcne.	Asclepiadaceae	Charungli, chungi pamanke	Skin disease	St, Wh	Juice	[82]
Borassus flabellifer Linn.	Arecaceae	Toody palm, sugar palm	Gastritis disorders	F	Juice	[88]
Calotropis procera R.Br.	Asclepiadaceae	Madar	Helminthiasis	R-B	Extract	[91]
Cardiospermum halicacabum Linn.	Sapindaceae	Balloon vine, winter cherry	Chronic bronchitis	R, L	Decoction	[98]
Carissa carandas Linn.	Apocynaceae	Karamardaka	Constipation	S	Extract	[83]
Carissa spinarum Linn.	Apocynaceae	Karaunda, garna	Epilepsy	R	Extract	[82]
Carthamus tinctorius Linn.	Compositae	Wild saffron, kamalottara	Stroke	Fl	Hot infusion	[87]
Cassia fistula Linn.	Caesalpiniceae	Sonhali, nripadruma	Edema	B, L	Paste	[84]
Cassia sophera Linn.	Caesalpiniceae	Bas-ki-kasunda	Cough	L	Infusion	[87]
Cassia tora Linn.	Fabaceae	Charota, taga	Flatulence	L	Infusion	[83]
Cadreia toona Roxb.	Meliaceae	Toona, khusing	Menstrual Disorders	B	Infusion	[87]
Cedrus deodara Lou Don.	Coniferae	Deodar, kilan, geyar	Microbial Infection	W	Oil	[82]
Cedrus libani Barrel	Coniferae	Deodar, devadaru	Toothache	Gm	Gum	[87]
Celastrus paniculata Willd.	Calastraceae	Malakanguni, vanhiruchi	Epilepsy	S	Decoction	[84]

Celosia argentia Linn.	Amaranthaceae	Paanai keerai	Eye problems	L	Decoction	[94]
Centella asiatica Urban.	Mackinlayaceae	Gotu kola	Atopic dermatitis	St	Extract	[99]
Cephaelis ipecacuanha A.Rich.	Rubiaceae	Poaya	Cancer	R	Extract	[91]
Ephedra vulgaris Rich.	Ephedraceae	Khanda, ma-hung	Nasal condestion	Be	Decoction	[102]
Euphorbia antiquorum Linn.	Euphorbiaceae	Triangular spurge, Tidhara, vajratundi	Dropsy	Br	Gum, milky juice	[103]
Ficus bengalensis Linn.	Urticaceae	Banyan tree, sriksha	Dysentery	S, F	Juice	[104]
Ficus religosa Linn.	Urticaceae	Pippala, peepul tree	Gonorrhea	B	Decoction, oil	[87]
flendarussa vulgaris Nees.	Acanthaceae	Nili-nargandi, kala-bashimb	Chronic rheumatism	L	Infusion	[87]
Jatropha curcas Linn.	Euphorbiaceae	Jangli-erandi, angula-leaved physic nut	Rhematic and muscular pain	S	Oil	[106]
Justica gendaruusa Burm.	Acanthaceae	Nilinargandi, kapika, bhutakeshi	Hepatic injuries	L	Decoction	[107]
Lawsonia alba Linn.	Lythraceae	Heena, mendhi, mehndi	Ulcers, ophthalmic disorder	L	Paste	[108]
Leucas aspera Spreng.	Labiatae	Chotahalkusa, tamba,	Psoriasis	L	Juice	[109]
Linum usitatissimum Linn.	Linaceae	Lins, uma, tisi	Gout, obesity	S	Poultice	[110]
Merremia tridentate Hallier.	Convulvulaceae	Prasarini	Hemorrhoids	Wh	Extract	[111]
Murray koenigii Linn.	Rutaceae	Bristly bryoni	Edema	L	Powder	[94]
Myristica fragrans Houtt.	Myristaceae	Nutmeg, jati-phalam., jaiphal	Rheumatoid Arthritis	S	Oil	[84]
Myristica malabarica Lamk.	Myristaceae	Malabar nutmeg, malati, kamuk	Bronchitis	S	Embrocation	[87]
Myropyrum similacifolium Blume.	Oleaceae	Chatura-mallikei	Cough	L	Extract	[87]
Myrtus caryophyllus Linn.	Myrtaceae	Cloves, lavangaha, laung	Rheumatic pain	F	Oil	[87]
Myrtus communis Linn.	Myrtaceae	Myrtle, murad	Pulmonary and Skin disease	L	Oil	[87]
Naregamia alata W.& A.	Meliaceae	Goanese ipecacuanha, amlavalli	Eczema	Wh	Extract	[82]
Nicotiana tabacum Linn.	Solanaceae	Tobacco, tambaku, tamrakuta	Respiratory tract disease	L	Decoction	[87]
Nycthanthes arbor-tristis Linn.	Oleaceae	Night jasmine, siharu, parijata	Rheumatoid Arthritis	L	Infusion	[87]
Ocimum gratissimum Linn.	Labiatae	Shrubby basil, ramtulasi	Inflammation, diabetes	Wh	Fumigations	[84]

L: Leaves, R: Roots, H: Herb, Wh: Whole plant, Rh: Rhizome, B: Bark, F: Fruit, Bu: Bulb, S: Seed, T: Tubers, Fl: Flowers, Mj: Milky juice, G: Grass, St: Stems, W: Wood, Be: Berries, Gm: Gum, K: Kernel, Sh: Shoot, C: Camphor, Rs: Resin, Br: Branches, Sg: Stigmas, Sp: Spores, Bt: Bracts, Sbe:Strawberries, Rbe: Ripeberries, Gr: Grain, C: Currants, O: Oil, P: Pulse, Sr: Sarocarp, Nt: Nuts, Al pt: Aerial parts, Ug pt: Underground parts-Ug pt

AYURVEDIC PRODUCTS:

1.Dabur India Ltd.

- -Product: Rheumartho
- -Formulation: Oil/Capsule
- -Use: Relieves joint pain, inflammation and stiffness

2.Patanjali Ayurved Ltd.

- -Product: Joint Care
- -Formulation: Oil/Capsule
- -Use: Reduces joint pain, inflammation and stiffness

3.Himalaya Wellness

- -Product: Rumalaya
- -Formulation: Tablet/Capsule
- -Use: Relieves joint pain, inflammation and stiffness

HERBAL SUPPLEMENT:

1.Nature's Bounty

- -Product: Turmeric Curcumin
- -Formulation: Capsule
- -Use: Anti-inflammatory, antioxidant properties

2.Gaia Herbs

- -Product: Turmeric Supreme
- -Formulation: Capsule
- -Use: Anti-inflammatory, antioxidant properties

CONCLUSION:

Many tribal and rural communities around the world use traditional medicines to treat arthritis. Currently, research into traditional medicine's anti-arthritic properties has produced and examined a wide range of herbal treatments used for this purpose. The data acquired from many sources aids in both the preservation of traditional indigenous knowledge and the identification of possible chemicals with encouraging anti-arthritic properties. The information obtained from the data gives details on the tested extracts' toxicity profile and mode of action. As a result, this review article was written to list the plants and their parts that have been traditionally used to cure arthritis up to 2013. Furthermore, to the best of our knowledge, preview reviews on arthritis therapy do not contain the most recent information on novel plant species or polyherbal formulations.

In order to achieve complete remission or at least a notable decrease in symptoms and clinical indicators, the ultimate goal of managing RA is to begin an aggressive medication regimen. The findings of the studies made it easier to comprehend the pathophysiological pathways and created novel treatment strategies, making RA a treatable condition. Many RA patients, nonetheless, are still not responding to the drugs that are now prescribed. There is still not enough information to fully control the illness, which emphasises the need for new medications and a stronger emphasis on personalised medicine.

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