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Integrative Medicine for Rheumatoid Arthritis: Assessing the Impact of Naturopathic Interventions on Inflammatory Markers: A Narrative Review

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Abstract
Rheumatoid arthritis is one of the crisis now a days faced from young adults to the old age ,due to uneven lifestyle and routines in common individuals .By addressing lifestyle factors and encouraging the body's natural healing processes, naturopathy offers a variety of therapies that can help control the symptoms of rheumatoid arthritis (RA), such as pain and inflammation. Dietary changes, hydrotherapy, acupuncture, and herbal remedies are examples of naturopathic treatments that can lower inflammatory markers and disease activity. So this study goes to the insights and summarise all the naturopathic interventions done towards the inflammatory markers in the field of integrative medicine.

Keywords: Rheumatoid Arthritis, Saunabath, Hydrotherapy, Diet, Fasting, Herbaltherapy.

Abbreviations

RA	Rheumatoid arthritis
NET's	Neutrophil extracellular traps
MHC	Major Histocompatibility Complex
FLS	Fibroblast-like synoviocytes
TG	Triglyceride
DMARDs	Disease-Modifying Anti-Rheumatic Drugs
ACPA	Autoantibody in clinical practice
DGE	(Deutsche Gesellschaft für Ernährung e.V.)German Nutrition Society
ADIRA	Anti-inflammatory Diet In Rheumatoid Arthritis
MMP	Matrix metalloproteinase
MAPK	Mitogen-activated protein kinase
RA-FLS	Rheumatoid arthritis fibroblast-like synoviocyte
FCA	Focal Cerebral Arteriopathy
EGF	Epidermal Growth Factor
DAS28	Disease Activity Score in 28 joints

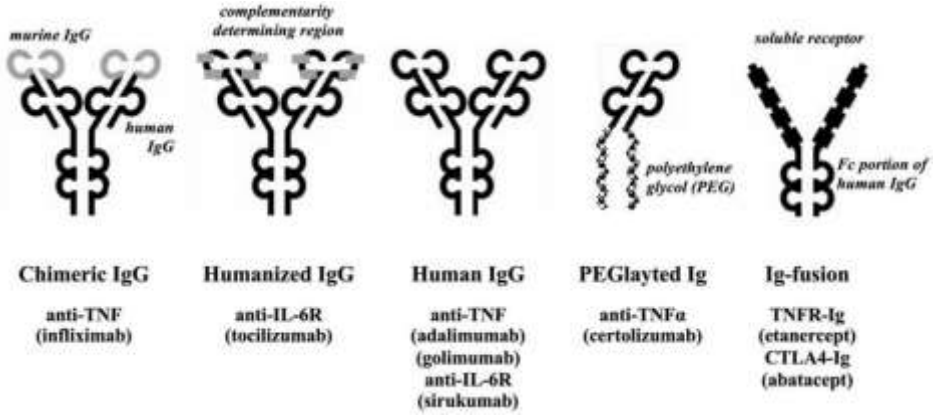
Introduction

Rheumatoid arthritis (RA) is a chronic, systemic autoimmune disease primarily affecting synovial joints. It is characterized by persistent inflammatory arthritis that typically begins in small peripheral joints—such as those of the hands and feet—and usually presents symmetrically. If left untreated, RA can progress to involve larger proximal joints and result in significant joint damage.[1] Globally, an estimated 17.6 million (95% uncertainty interval) persons suffered from rheumatoid arthritis in 2020. The global age-standardized prevalence rate increased by 14·1% from 1990 to 208·8 instances per 100,000 people. The age-standardized female-to-male prevalence ratio was 2·45, indicating a higher prevalence in females. Between 1990 and 2020, the age-standardized death rate decreased by 23·8% to 0·47 fatalities per 100,000 people (38, 300 deaths worldwide).[2] Immunological abnormalities says like, The most common autoantibody in clinical practice is ACPA, which can be seen in RA patients years before symptoms appear. One of the main causes of RA patients' decreased immunological tolerance is peptididyl arginine deiminase (PADI), which triggers the posttranslational modification process that turns arginine or glycine residues of healthy proteins into citrulline .[3] Compared to native proteins, citrullinated proteins are more immunogenic and have a higher affinity for the antigen-binding groove of human leukocyte antigen (HLA)-DR. One significant source of citrullinated proteins is neutrophil extracellular traps, or NETs. NETs were more prevalent in RA patients' circulation and synovial fluids than in healthy controls. The development of NETs causes the joints' inflammatory milieu to persist.[4] Inflammatory pathways like, One of the main proinflammatory cytokines in RA, TNF-α, has been shown to increase inflammation by upregulating TNFR II, activating the nuclear factor-kappa B (NF-κB) pathway, and causing RA-FLS to secrete receptor activator of NF-κB ligand (RANKL) for osteoclast formation. The viability of cytokine-targeted therapy for RA has been validated by the effective clinical use of TNF-α and IL-6R antagonists, and research into further cytokine-targeted medications is still ongoing.[5] NF-κB is substantially expressed in RA patients' synovial tissue. NF-κB pathway hyperactivation can trigger the production of inflammatory cytokines such TNF-α, IL-1β, and IL-6, which in turn intensify NF-κB signalling to create a vicious cycle.[6] By seeing genetically ,With an estimated 53–68% heritability, RA has a significant genetic component, particularly in patients who test positive for ACPAs.The MHC region contains the most attributable HLA-DRB1 alleles, accounting for approximately 20% of the genetic risk for RA.[7] In RA, the DNA methylation pattern is also changed, which impacts FLS activity and immune response, ultimately leading to the development of the illness. In fact, RA peripheral blood mononuclear cells (PBMCs) and RA FLS have worldwide DNA hypomethylation. In contrast to FLS produced from OA, Nakano et al. showed that 1859 genes related to cell mobility, adhesion, and trafficking were differently methylated in RA FLS. This suggests that distinct methylation profiles may change gene expression in FLS and have a role in the pathophysiology of RA.[8] Metabolic disorders and RA relation is like,Hyperglycemia and increased TG were linked to an increased risk of incident RA. By altering DNA methylation, elevated TG may trigger circulating immune cells, which could impact RA and other inflammatory conditions. There are two possible reasons why hyperglycemia can raise the risk of RA. First, hyperglycemia may have an impact on the connective tissue matrix's constituents and raise reactive oxygen species production, which can weaken antioxidant defences and cause inflammation and tissue damage. Second, prolonged hyperglycemia may encourage the production of advanced glycation end products, which can trigger autoimmune reactions due to their antigenicity.[9]

Recent pharmacological findings and DMARDs advicing

The Pharmacological Therapies in RA are by lowering inflammation, NSAIDs (naproxen, ibuprofen, and coxibs) are used to lessen pain during the acute phase response. By blocking cyclooxygenase (COX), particularly COX-2, which is elevated during inflammation, NSAIDs produce their pharmacological effects. However, as prostaglandin inhibition can result in severe side effects such bleeding, gastrointestinal ulcers, heart failure, renal failure, rashes, disorientation, confusion, seizures, etc., the risk of injury should be taken into account.[10]

Fig.1 - For the treatment of RA there are five TNF-targeting drugs, two IL-6 receptor-targeting drugs, one B-cell antigen CD20-targeting antibody and one selective T-cell costimulatory modulator.[11]



(Fig. 1) Biological DMARDs approved for RA For the treatment of RA there are five TNF-targeting drugs, two IL-6 ...

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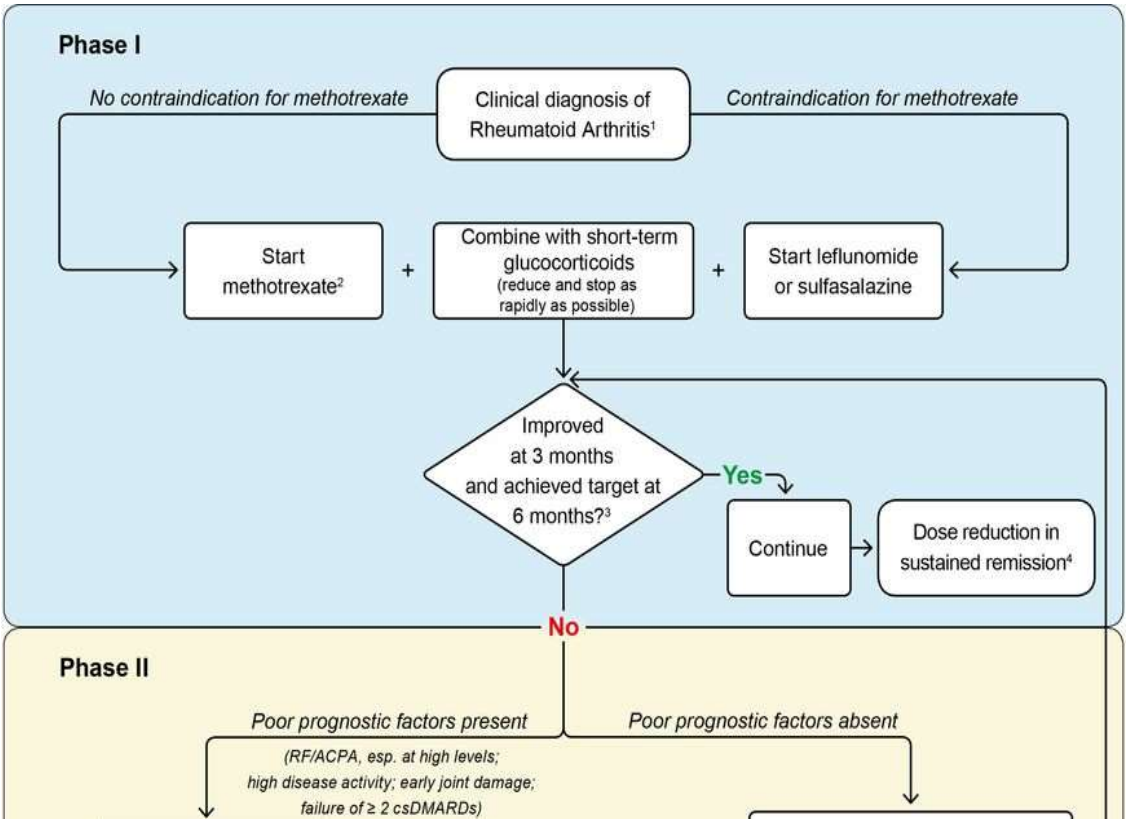
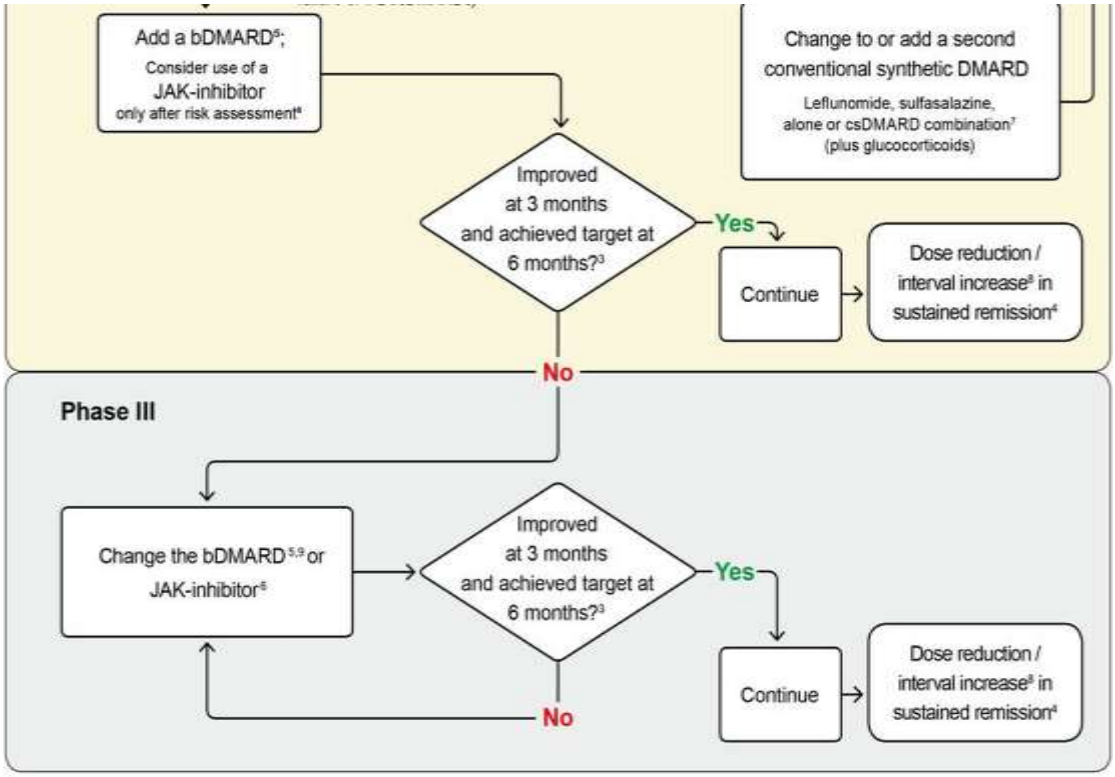




(FIG-2)Flow chart. ACR, American College of Rheumatology; bDMARDs, biological disease-modifying antirheumatic drugs; csDMARDs, conventional synthetic DMARDs; FDA, Food and Drug Administration; JAK, Janus kinase; MTX, methotrexate;NMSC, non-melanoma skin caner; tsDMARDs, targeted synthetic DMARDs.[12]

Naturopathy, which treats such diseases by combining herbal, nutritional, and other complementary approaches, is one of the most popular complementary and alternative medicine (CAM) modalities. This review looks at the data about the elements that naturopaths consider to be major causes of rheumatoid arthritis and, as a result, are the primary focus of therapeutic care. Food allergies, elevated oxidative stress, elevated circulating immune complexes, increased gut permeability, and excessive inflammatory processes are some of these reasons. By changing these variables through dietary changes, dietary fat modulation, and the use of antioxidants and proteolytic enzymes, naturopathic treatment aims to reduce symptoms.[13]

METHODS

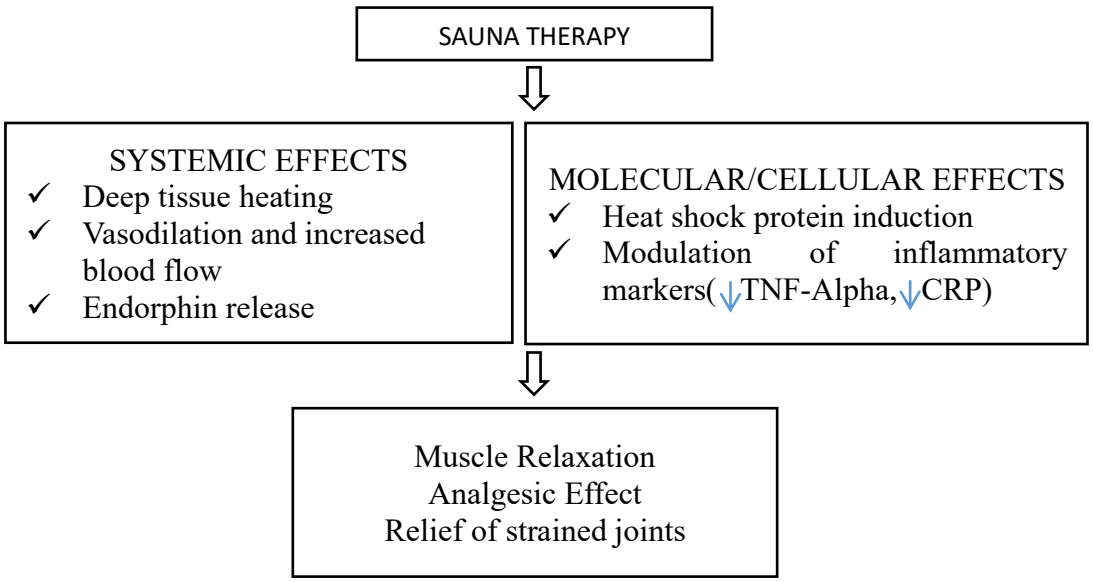
All necessary information is derived from articles published in journals on PubMed and Google Scholar until 2025. Search terms pertaining to the management of rheumatoid arthritis, naturopathic approaches, and all treatments under this system were included. We eliminated editorial and other websites' opinions on this paper and added from all trial and review papers.

From Heat to Healing : Sauna Therapy's Role in Rheumatoid Arthritis Care

Sauna therapy effects by decreasing pro-inflammatory substances (TNF-α, CRP, PGE2, LTB4) and increasing IL-10-mediated anti-inflammatory effects, heat exposure modifies inflammatory pathways. Sauna therapy also improves neuroendocrine function and reduces oxidative stress, which helps the body as a whole. Research shows that sauna therapy helps people with rheumatoid arthritis (RA) by lowering stiffness, reducing discomfort, and increasing mobility.[14] During the IR sauna therapy, pain and stiffness

decreased by 5 to 24 points on the VAS in all patients, indicating a clinically meaningful improvement. For RA patients, pain decreased by about 40% and 60%, and stiffness decreased by about 50% and 60%. delayed effects on primary outcomes during and four weeks following therapy. During the 4-week course of treatment, RA patients' pain, stiffness, and exhaustion all slightly improved; stiffness nearly attained statistical significance (p=0.06).[15] Sauna was thought to relieve pain and swelling by promoting analgesia and sweating, and it has been demonstrated to have an anti-inflammatory impact.[16]

A whole-body hyperthermia that warms the muscles, raises the temperature of the tissues, and encourages body adaptability is created after completing a sauna.Based on these results saunayoga is found beneficial and relaxing form of exercise and can be recommended to be tried for people with rheumatic diseases.[17]



Unveiling The Therapeutic Potential Of Acupuncture In RA Management

Typically, manipulation occurs after the acupuncture needle is inserted into an acupoint, causing the subcutaneous collagen fibres to twist around the needle. Through the mechanosensitive transient receptor potential vanniloid-2 (TRPV2) channel protein on their membrane, this manipulation causes mast cells to degranulate . A cascade of analgesic and anti-inflammatory processes ensues, with mediators including histamine, serotonin (5-HT), adenosine, and adenosine triphosphate (ATP) being released. The processes of mast cell activation by manual acupuncture, moxibustion, and electroacupuncture may be based on the fact that TRPV2 channels can also be activated by mechanical, heat, and laser stimulation.[18] The extra-meridian odd points on the back of the hand between the roots of the first and fifth fingers and at the junction—a total of eight points—are the Baxie acupoints utilised in this study.[19] By referring to eight Xie-pathogenic factors—wind, cold, heat, dampness, hunger, satiety, labour, and rest—the Baxie acupoints also represent more expansive traditional meanings.These harmful elements can be released by activating Baxie acupoints, which will lead to a notable improvement or recovery. According to recent studies, acupuncture at the Baxie acupoints can manage RA activity by reducing the release of associated inflammatory factors and bidirectionally regulating the immune system.[20] By re-establishing the balance of Th1/Th2, Th17/Treg, and other cytokines and restoring the function of the T lymphocyte population, acupuncture can prevent the onset of RA. Additional benefits of acupuncture include lowering the expression of inflammatory cytokines such as IL-1, IL-17, IL-23, and TNF-α, preventing synovial cells from activating and working in concert with IL-1 and TNF-α,

reducing inflammatory cell infiltration to maintain the body's immune balance, preventing the development of local synovial pannus, delaying the breakdown of joint synovial tissue, and protecting the bone joint and articular cartilage.[21] The following criteria were used to choose the acupuncture points: UB-11 is a crucial point for bones, GB-39 is a crucial point for bone marrow, LI-4 and ST-44 are analgesic points for pain, and K-3 is the kidney meridian's source point. It is commonly known that acupuncture alters the local environment and systemic immunity. When the needle is inserted, there is inflammation but no infection. In a variety of inflammatory disease models, acupuncture reduces the toll-like receptor (TLR)-initiated inflammatory signalling pathway and increases anti-inflammatory cytokines, especially interleukin (IL)-10, through M1–M2 macrophage transformation, which leads to the resolution of inflammation. Acupuncture treats immunological dysfunction by balancing the helper T cell (Th)1/Th2 population. The hypothalamic-pituitary-adrenal (HPA) axis, a brain circuit that mediates systemic immunity, is also influenced by acupuncture.[22] Fire needles can effectively control the inflammatory response of RA, with an effect similar to methotrexate, which is related to the down-regulation of ACPA and TNF-α levels. Warm needles can reduce limb swelling of RA and effectively control the progression of the disease, which may be related to lowering serum immunoglobulin, IL-1, and TNF-α levels and alleviating the response. Its effectiveness is linked to anti-inflammation, anti-oxidation, immune system function regulation, and endorphins or serotonin.[23] When there is a substantial muscle spasm, electrical acupuncture may be the most effective treatment. Five research have been found by Bhatt-Sanders to support the idea that acupuncture treatments significantly reduce pain, whereas two investigations found no discernible difference between actual and placebo acupuncture.[24] It is hypothesised that calcitonine gene-related peptide has vasodilative and anti-inflammatory properties once neuropeptides are released from nerve terminals under acupuncture control. The intricate relationships with substance P, the analgesic effect of β-endorphin, and the equilibrium between pro-inflammatory and anti-inflammatory cytokines specific to cells, such as interleukin-10 and tumour necrosis factor-α .[25]

Therapeutic Efficacy of Hydrotherapy in Rheumatoid Arthritis

Therapeutic benefits of hydrotherapy, with warm water immersion. According to the Arthritis Impact Measurement Scales 2 questionnaire, every patient saw an improvement in their physical and emotional well-being. There was a decline in the belief that pain was controlled by random events, indicating progress. Additionally, individuals who received hydrotherapy demonstrated noticeably better improvements in knee range of motion and joint discomfort.Patients who had hydrotherapy continued to see improvements in their emotional and psychological states. [26] Temperature, resistance, immersion, and buoyancy are all significant factors. The gate idea suggests that the water's temperature and pressure on the skin may be the cause of the pain reduction. Immersion in water raised plasma levels of methionine-encephalin while decreasing levels of prolactin, corticotropin, and β endorphin. Reduced joint swelling and relaxed muscles might also be important. Significant reductions in tension and mood could be a factor in the outcomes. Reilly and Bird discovered that group therapy in a public pool was more successful than one-on-one therapy in a hospital pool. This was most likely brought about by promoting social connection and focussing on health and well-being rather than illness.[27] The program of aquatic exercise showed moderate benefits on depression, significant impacts on pain, and negligible effects on physical fitness. The indirect relationship between pain and the reduction in depression among participants in the aquatic exercise program was validated by the mediation model.[28] Simple habits like

taking cold showers may be used as alternative therapies to increase resilience and immunity, according to new research on the health advantages of cold exposure.[29] Balneotherapy and mud therapy have been shown to lower serum levels of pro-inflammatory cytokines TNF-α and IL-1β, as well as the regulatory cytokine IL-6, and to raise levels of the anti-inflammatory growth factor IGF-1 in a number of low-grade inflammation-related pathologies, particularly rheumatic diseases. Balneotherapy, the modification of these cytokines and adipokines results in anti-inflammatory-mediated chondroprotective effects that may be helpful in rheumatic disorders because they are all significant mediators of inflammation and cartilage metabolism.[30] The immune system can function more quickly and effectively by increasing the circulation of white blood cells through hot water hydrotherapy, which speeds up blood flow throughout the body. Additionally, the body produces more endorphins after a hot soak. Endorphins are the body's "pain killers" and are linked to joyful or exuberant emotions. Endorphins also help tissues repair more quickly, reduce pain, and boost the immune system.[31]

Metabolic Modulation in RA : Insights in to Intermittent Fasting

In fasting , mainly shows that an 8:16 intermittent fasting schedule significantly improved a number of oxidative stress and inflammatory indicators when compared to recommended dietary intake.[32] According to studies, Intermittent Fasting during Ramadan reduces the body's inflammatory status by lowering inflammatory markers such as leptin, interleukin 1 beta (IL-1β), interleukin 6 (IL-6), tumour necrosis factor-alpha (TNF-α), C-reactive protein (CRP), and interferon-gamma (INF-γ).[33] Fasting, as reported in earlier trials, allowed us to see improvements in RA disease activity. Day 7 showed a noticeable change from the anti-inflammatory DGE diet, highlighting how quickly the benefits of fasting start to manifest. This feature raises the possibility that fasting could be a useful component of a short-term treatment for i) acute RA flare-ups and ii) patients who would rather make temporary dietary adjustments than long-term ones. Our findings align with those of other dietary therapies for RA, like the ADIRA diet, which have produced comparable mid-term effects above minimal important differences (MID).[34]

Nutritional Approaches to Modulating Rheumatoid Arthritis Severity

In the diet therapy Mediterranean diet emphasises eating a lot of plant-based meals and avoiding a lot of meat. Olive oil is the primary sources of fats. Omega-3 polyunsaturated fatty acids and vitamins have anti-inflammatory and protective qualities, which contribute to the Mediterranean diet's function in rheumatic disorders. Natural anti-inflammatory qualities are possessed by components of olive oil, including oleic acid and olecanthal.[35] The main active ingredient in soybeans, genistein, has qualities that include anti-inflammatory, anti-angiogenesis, anti-proliferative, antioxidant, immunomodulatory, pain alleviation, and joint protection. Studies conducted both in vitro and in vivo indicate that genistein may be a useful treatment for RA. In fibroblast-like synoviocytes of RA, genistein has been demonstrated to suppress MMP-9 production and proliferation produced by IL-1β, TNF-α, or EGF.[35,36] TNF-alpha-R2 and IL-6 plasma levels are linked to a high-fiber diet. Dietary fibre intakes were negatively correlated with TNF-alpha-R2 (P values for trend were 0.002 for total, 0.02 for soluble, and <0.001 for insoluble fibres) and IL-6 (P values for trend were 0.01 for total fibre, 0.004 for soluble fibre, and 0.001 for insoluble fibre) after controlling for covariates.[37] By lowering adhesion molecule expression, lowering leucocyte chemotactic response, and promoting the synthesis of the anti-inflammatory lipid mediators protectins and resolvins, omega-3 FA has been demonstrated to suppress

the inflammatory process.[38] Vitamin D is an essential compounent, When circulating 25-hydroxyvitamin D3, a metabolite that indicates vitamin D status, is locally converted to 1,25D by extrarenal CYP27B1, vitamin D also triggers a variety of intracrine actions. These actions include the promotion of immunoregulation, antimicrobial defence, xenobiotic detoxification, anti-inflammatory/anticancer actions, and cardiovascular benefits.[39] The primary phytochemical found in green tea, epigallocatechin-3-gallate (EGCG), has been shown to have preventive effects against several cancers as well as cardiovascular, inflammatory, and neurological diseases. In RA, EGCG can inhibit synovial fibroblasts' expression of Mcl-1, an anti-apoptotic protein, which in turn promotes the apoptosis of these cells. Additionally, by downregulating MMP-1, MMP-2, and MMP-3 in synovial fibroblasts, ECGC prevents bone and cartilage degradation and suppresses IL-1B-induced IL-6 synthesis by synovial fibroblasts.[40]

Herbal Approaches to Modulating Rheumatoid Arthritis : Key Ingredients and Effects

CURCUMIN - A growing number of randomised clinical trials have been carried out in recent years to evaluate the safety and effectiveness of curcumin treatment in RA patients. Compared to individuals in the diclofenac sodium group, patients who received 500 mg of curcumin twice daily for eight weeks experienced a significant improvement in their Disease Activity Score 28 (DAS-28) and American College of Rheumatology (ACR) scores (ACR-20, 50, and 70).[41] In RA patients, curcumin was found to dramatically reduce joint oedema, walking time, and morning stiffness. There have also been reports of a number of processes, such as the suppression of AP-1, ERK1/2, MAPK, and NF-κB. All things considered, the findings point to curcumin being a safe and efficient medication for RA treatment.[42] Herbal remedies or phytopharmaceuticals may be useful adjuvants in the treatment of RA, according to the systematic review and meta-analysis. When used as an adjuvant, curcumin improves DAS-28, VAS, rheumatoid factor, CRP, and ESR; however, it has no effect on the number of tender joints or swollen tenders. Garlic, ginger, and cinnamon have promise, but research on them is currently lacking.[43] Curcumin increased cell death and reduced TNF-α-induced MH7A and RA-FLS cell invasion, migration, and proliferation.[44] Numerous investigations have documented that at 1, 2, 3, and 4 hours following a single oral dose of 500–8000 mg in humans, as well as following a prolonged oral administration of 440–2200 mg of curcumin or curcuma extracts daily, curcumin concentrations are incredibly low or nonexistent in serum and tissues.This is linked to curcumin's poor stability in aqueous solution at physiological pH; in just 30 minutes, curcumin breaks down into a variety of dimerisation end products, including trans-6-(4′-hydroxy-3′-methoxyphenyl)-2,4-dioxo-5-hexenal, ferulic aldehyde, ferulic acid, feruloyl methane, vanillin, and vanillic acid.Following curcumin ingestion, the metabolites of curcumin are found in the bloodstream in significant amounts. The anti-inflammatory and antioxidant properties of these curcumin metabolites may be what lessen the symptoms of metabolic disorders like arthritis.[45] 259 RA patients with a duration of 6–12 weeks were included in 6 studies. American College of Rheumatology (ACR-20) scores, visual analogue scale (VAS), and 28 joints (DAS-28) were used to measure disease activity. Curcumin treatment considerably raised ACR-20 scores in all three studies where it was examined, dramatically decreased DAS-28 scores in four of the five studies, and significantly raised VAS scores for pain in all three studies. Six and five investigations, respectively, measured the circulating C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR), and four of these studies found that curcumin administration significantly decreased these parameters.In all three pertinent investigations, curcumin ingestion resulted in a significant decrease in rheumatoid factor (RF).

There were no significant negative effects linked to curcumin use in any of the investigations. Curcumin may be a safe treatment for RA, according to the current systematic review.[46]

CYNODON DACTYLON - In India, Cynodon dactylon is referred to as a creeper. It is also called "Bermuda grass" in English, "Doob" in Hindi, and "Duba" in Odia. It is a member of the Poaceae family.[47]

Carrageenan-induced acute inflammation and autacoids (carrageenan, serotonin, histamine, and dextran)-induced oedema have been demonstrated to be persuasively protected against by Cynodon dactylon. Due to an increase in prostaglandin synthesis, recent research has verified that adjuvant-induced arthritis is linked to both a decrease in bone formation and an increase in bone resorption.[48]

Anti-inflammatory Effects: Studies show that C. dactylon extracts contain anti-inflammatory properties that could aid in lowering inflammation brought on by diseases like inflammatory bowel disease, asthma, and arthritis.

Phytochemical constituents on a dry matter basis, greengrass has 11.75% total ash, 28.17% fibre, and 10.47% crude protein. There have also been reports of carotenoids including beta-carotene, neoxanthin, and violaxanthin, as well as flavonoids such apigenin, luteolin, orientin, and vitexin. Phenolics, phytosterols, glycosides, saponins, and volatile oils are other phyto-constituents.[49]

CINNAMON - Cassia cinnamonomum Presl is a tropical aromatic evergreen tree of the Lauraceae family that is frequently utilised in traditional Oriental medicine. It is also referred to as cassia or cinnamon. From C. cassia, more than 160 compounds have been discovered. Terpenoids, phenylpropanoids, and glycosides are the primary components. Among the many pharmacological actions of C. cassia's primary constituents include anti-platelet aggregation, antithrombotic, pro-angiogenesis, vasodilating, and microcirculation-improving properties. Cardiovascular protection, cytoprotection, neuroprotection, immunomodulation, anticancer, anti-inflammatory, analgesic, antibacterial, antiviral, and anti-tyrosinase properties are also present in C. cassia.[50]

INTEGRAL ROLE OF COLOR THERAPY ON RA

By using color therapy , lowering the activation energy needed and removing free radicals, red (644 nm) radiation boosts Superoxide dismutase (SOD) activity, which surely boosts immunity.[51] Blue light promotes the S-nitrosylation signalling pathway, which inhibits nuclear factor-kappa B kinase (NF-kB) and inhibitory kappa B kinase beta (IKK-β), reducing inflammation and oxidative stress. Light-emitting diode (LED) photobiomodulation has been demonstrated to produce peripheral neural block, inhibit central synaptic activity, alter neurotransmitters, lessen oedema and muscle spasm, and have anti-inflammatory properties. Furthermore, light has an analgesic effect on chronic illnesses like RA by increasing the production of opioid neurotransmitters, local blood circulation, and pain threshold.[52] Flavins, porphyrins, nitrosated proteins, and opsins absorb blue light at the molecular level, which causes the production of reactive oxygen species (ROS), the release of nitric oxide, and the activation of G protein coupled signalling. Reports on the impact of blue light-induced signalling on cells are few and inconsistent. Some studies reveal growth inhibition and apoptosis, while others indicate a manipulation of inflammatory markers or a regulation of proliferation and differentiation.[53]

Input (Color/Therapy)	Molecular/Cellular Effect	Signalling/Pathway Impact	Clinical/Physiological Outcome (Benefit for RA)

Input (Color/Therapy)	Molecular/Cellular Effect	Signalling/Pathway Impact	Clinical/Physiological Outcome (Benefit for RA)
Red Light (644 nm)	Lowers activation energy; Removes free radicals	Boosts Superoxide dismutase (SOD) activity	Boosts immunity
Blue Light	Absorbed by Flavins, Porphyrins, Nitrosated proteins, Opsins	Promotes S-nitrosylation signalling pathway	Reduces inflammation and oxidative stress
Blue Light	Leads to ROS production & Nitric oxide release	Activates G protein coupled signalling	Inconsistent Reports: Growth inhibition, Apoptosis, Manipulation of inflammatory markers, Regulation of proliferation/differentiation.
Light (LED Photobiomodulation)	N/A	Peripheral neural block, Inhibition of central synaptic activity, Alteration of neurotransmitters	Analgesic effect, Lessens oedema and muscle spasm, Anti-inflammatory properties
Light (General Analgesic Effect)	Increases production of opioid neurotransmitters	Increases local blood circulation	Increases pain threshold (Alleviates chronic pain in RA)

CONCLUSION

From this review culminate with a "deeper insight of information on Integrated approach of naturopathy on rheumatoid arthritis and how each therapies helps to get rid from inflamatory markers. The review includes as much detail as possible that is necessary based on topic.More specific papers are required for herbal therapies for RA.

Data availability - This article contains all of the data sets utilised in the review.

Declarations - There is no conflict of interest in this study

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