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Role of diet and nutrition in the management of rheumatoid arthritis: A narrative review

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Abstract
Background: Rheumatoid arthritis (RA) is a chronic systemic autoimmune disease characterised by persistent synovial inflammation, progressive joint destruction, and significant extra-articular manifestations. Despite substantial advances in pharmacotherapy, a considerable proportion of patients experience suboptimal disease control, treatment-related adverse effects, and residual functional impairment. Emerging translational and clinical evidence increasingly implicates diet and nutritional status as modifiable determinants of immune function, systemic inflammation, and gut microbial ecology, all of which are pathophysiologically relevant in RA.
Objectives: This narrative review critically appraises the current evidence pertaining to the role of dietary patterns and targeted nutritional interventions — including the Mediterranean diet, omega-3 polyunsaturated fatty acids, vitamin D, probiotics, dietary fibre, polyphenols, and antioxidant micronutrients — in the modulation of RA disease activity and inflammatory burden.
Methods: A systematic literature search was conducted across PubMed/MEDLINE, Embase, the Cochrane Library, and Google Scholar for peer-reviewed publications from January 2019 to March 2025, using MeSH terms and free-text keywords.
Results: Adherence to Mediterranean and anti-inflammatory dietary patterns is associated with reduced disease activity scores and lower concentrations of circulating inflammatory biomarkers. Omega-3 supplementation at doses exceeding 2.7 g/day for a minimum of three months demonstrates clinically meaningful reductions in joint tenderness and non-steroidal anti-inflammatory drug (NSAID) requirements. Vitamin D insufficiency — present in over 63–76% of RA patients across international cohorts — correlates inversely with disease activity and responds favourably to supplementation. Gut microbiome modulation through dietary fibre, prebiotics, and select probiotic strains yields promising immunomodulatory effects. Curcumin, ginger, berries, and extra-virgin olive oil exhibit pleiotropic anti-inflammatory actions in preliminary trials.
Conclusions: Dietary modification represents a safe, cost-effective, and evidence-supported adjunctive strategy in RA management. Mediterranean-style and anti-inflammatory dietary patterns currently command the strongest evidentiary base. High-quality, long-duration, multicentre randomised controlled trials employing standardised dietary assessment tools and validated composite outcome measures are urgently required to establish personalised dietary recommendations.

Keywords: rheumatoid arthritis, diet, nutrition, mediterranean diet, omega-3 fatty acids, gut microbiota, vitamin D, probiotics, anti-inflammatory diet, disease activity, dietary Inflammatory Index

Introduction

Rheumatoid arthritis (RA) is a chronic, systemic autoimmune disorder characterised by persistent inflammation of the synovial membrane, progressive articular cartilage erosion, and juxta-articular bone destruction. Globally, the disease affects an estimated 0.24–1.0% of the adult population, with a consistent female predominance of approximately two-to-three-fold. [1, 2]

The immunopathogenesis of RA is complex and multifactorial, involving dysregulated T- and B-lymphocyte activation, excessive production of pro-inflammatory cytokines including tumour necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), and interleukin-17 (IL-17), and sustained activation of the complement cascade. These inflammatory cascades, sustained over time, lead to synovial hyperplasia, pannus formation, and ultimately irreversible joint destruction, resulting in functional disability, diminished quality of life, and heightened cardiovascular morbidity. [3]

Contemporary pharmacotherapeutic strategies encompass conventional synthetic disease-modifying antirheumatic drugs (csDMARDs) — principally methotrexate, sulfasalazine, and leflunomide— biologic DMARDs targeting TNF-α, IL-6, CD20, and CTLA-4, and targeted synthetic DMARDs such as Janus kinase (JAK) inhibitors. While treat-to-target strategies have substantially improved clinical outcomes over the past two decades, a meaningful proportion of patients fail to achieve sustained remission or low disease activity, experience treatment-limiting adverse effects, or encounter barriers to accessing expensive biologic therapies, particularly in low- and middle-income countries. [3, 4]

Against this background, diet and nutritional status have attracted considerable scientific interest as modifiable environmental factors capable of influencing immunological function and systemic inflammation. Diet shapes immune responses through multiple complementary mechanisms: direct regulation of immune cell metabolism and cytokine gene expression, modulation of oxidative stress, and — most compellingly — remodelling of the gut microbiome, which serves as a critical immunological organ in RA pathogenesis. [5, 6]

Epidemiological investigations, translational laboratory studies, and clinical trials have collectively generated a substantial, though heterogeneous, body of evidence suggesting that specific dietary patterns and targeted nutritional supplementation may attenuate RA risk, reduce disease activity, and improve health-related quality of life. The Dietary Inflammatory Index (DII), a validated tool evaluating the inflammatory potential of dietary intake across 45 food parameters, has provided a quantitative framework linking dietary quality to circulating inflammatory biomarker concentrations in RA populations. [7, 8]

The European League against Rheumatism (EULAR) has formally recognised dietary modification and lifestyle optimisation as underexplored yet clinically valuable components of holistic RA management, and has called for robust evidence to inform specific recommendations. [9]

Patient self-reported dietary experimentation is common in RA, with surveys indicating that more than 40% of patients modify their diet following diagnosis in attempts to manage symptoms. This underscores the clinical imperative for evidence-based nutritional guidance from treating physicians. In the Indian context, dietary practices are deeply embedded in cultural, religious, and socioeconomic frameworks, making culturally sensitive and locally validated dietary recommendations particularly important. [10]

This narrative review aims to critically appraise the current evidence on the role of dietary patterns and specific nutritional interventions in RA management, to identify clinically relevant areas of consensus and controversy, to delineate key research gaps, and to propose a practical framework for integrating nutritional counselling into standard rheumatology practice.

Methods

A comprehensive narrative review was conducted through structured electronic database searches of PubMed/MEDLINE, Embase, the Cochrane Library, and Google Scholar. The search was restricted to English-language peer-reviewed publications spanning January 2019 to March 2025, supplemented by seminal earlier studies where their foundational significance warranted inclusion. Search terms were constructed using MeSH vocabulary and free-text keywords, including 'rheumatoid arthritis', 'diet', 'nutrition', 'Mediterranean diet', 'omega-3 fatty acids', 'vitamin D', 'gut microbiome', 'probiotics', 'Dietary Inflammatory Index', 'anti-inflammatory diet', 'curcumin', 'polyphenols', and 'dietary fibre'. Included studies comprised randomised controlled trials, systematic reviews, meta-analyses, prospective cohort studies, and mechanistic translational studies. Reviews and editorials were included selectively where they provided significant synthesis of evidence. Non-peer-reviewed sources and grey literature were excluded.

Pathophysiological Links between Diet and Rheumatoid Arthritis

1. Immunometabolism and Dietary Macronutrients

The relationship between dietary composition and immune cell function is increasingly understood through the lens of immunometabolism — the study of how cellular metabolic pathways govern immune responses. In RA, pathological T-helper cell polarisation, particularly the expansion of pro-inflammatory Th17 cells at the expense of immunosuppressive regulatory T cells (Tregs), is a central immunopathological event. Glycolytic metabolism, predominating in Western high-carbohydrate dietary contexts, preferentially promotes Th17 differentiation, while fatty acid oxidation — promoted by unsaturated dietary fats — supports Treg expansion and anti-inflammatory immunological homeostasis. [11]

Saturated fatty acids, prevalent in energy-dense Western diets, activate Toll-like receptor 4 (TLR4) on innate immune cells, triggering downstream nuclear factor kappa-B (NF-κB) activation and upregulation of pro-inflammatory cytokine production including TNF-α and IL-6. In contrast, long-chain omega-3 polyunsaturated fatty acids (n-3 PUFAs) — eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) — competitively inhibit arachidonic acid-derived eicosanoid synthesis and serve as biosynthetic precursors for specialised pro-resolving mediators (SPMs) — resolvins, protectins, and maresins — that actively terminate inflammatory responses and promote tissue homeostasis. [12]

2. The Dietary Inflammatory Index

The Dietary Inflammatory Index (DII), developed and validated by Shivappa *et al.*, provides a standardised, literature-based score quantifying the inflammatory potential of overall dietary intake across 45 food parameters, including macronutrients, micronutrients, flavonoids, and spices. A higher (more pro-inflammatory) DII score is consistently and independently associated with elevated serum concentrations of C-reactive protein (CRP), IL-6, and TNF-α in population-based studies. In RA-specific analyses, higher DII scores correlate significantly with greater disease activity scores (DAS-28), patient-reported pain severity, and disability indices. [7, 8]

Conversely, adherence to dietary patterns characterised by high consumption of fruits, vegetables, whole grains, fatty fish, legumes, and olive oil — all of which confer negative DII scores and — are associated with lower systemic inflammatory burden and improved RA disease control. The DII therefore provides clinicians with an actionable, validated instrument for dietary risk assessment and targeted nutritional counselling in rheumatology practice. [7]

3. The Gut–Joint Axis

The gut microbiome comprises approximately 3.8×10^{13} microbial cells residing within the gastrointestinal tract — functions as a major immunological organ, regulating systemic immune responses through multiple interdependent mechanisms, including short-chain fatty acid (SCFA) production, modulation of Th17/Treg cell balance, maintenance of intestinal epithelial barrier integrity, and molecular mimicry involving citrullinated microbial peptides that may cross-react with autoantigenic epitopes in RA. [13, 14]

Patients with established RA and, crucially, individuals in the pre-clinical phase of RA —characterised by the presence of anti-citrullinated protein antibodies (ACPAs) in the absence of clinical synovitis — demonstrate consistent gut microbial dysbiosis. This dysbiosis is characterised by a relative abundance of pathobiont species, particularly Prevotella copri, Collinsella aerofaciens, and Porphyromonas gingivalis, and a reciprocal depletion of immunomodulatory commensals including Bifidobacterium

spp., Lactobacillus spp., Faecalibacterium prausnitzii, and Bacteroides fragilis. [14, 15]

The 'mucosal origins hypothesis' of RA proposes that microbially-driven mucosal inflammation at intestinal and periodontal sites drives citrullination of local proteins, inducing ACPAs that subsequently traffic to susceptible joints in genetically predisposed individuals. Dietary interventions through their profound capacity to reshape gut microbial ecology —therefore represent a mechanistically plausible and clinically accessible therapeutic avenue in RA. [15]

4. Oxidative Stress and Antioxidant Defence

The synovial microenvironment in RA is characterised by intense oxidative stress, with activated neutrophils and macrophages generating excessive reactive oxygen species (ROS) that damage synoviocyte membranes, extracellular matrix proteins, and DNA, while simultaneously perpetuating NF-κB-mediated cytokine amplification. Systemic antioxidant defence is correspondingly depleted, with reduced circulating concentrations of vitamins C and E, selenium, zinc, and carotenoids documented in RA patients relative to healthy controls. Dietary supplementation of these micronutrients may partially restore antioxidant capacity and attenuate oxidative-inflammatory damage. [16]

Dietary Patterns and Rheumatoid Arthritis

1. The Mediterranean Diet

The Mediterranean dietary pattern — characterised by abundant consumption of vegetables, fruits, legumes, whole grains, nuts, and extra-virgin olive oil; moderate intake of fish, poultry, and dairy; and minimal consumption of red meat, processed foods, and refined sugars — ranks among the most extensively studied dietary interventions in RA. Its postulated mechanisms include omega-3 PUFA delivery from fish, polyphenol provision from olive oil and plant foods, prebiotic fibre supply from legumes and whole grains, and favourable remodelling of the gut microbiome. [17]

A landmark randomised controlled trial (RCT) by Sköldstam *et al*. demonstrated that three months of Mediterranean diet adherence in patients with active RA produced statistically significant reductions in the Disease Activity Score-28 (DAS-28), swollen and tender joint counts, morning stiffness duration, and patient-reported pain, compared with a conventional Western diet. [18]

A large-scale prospective analysis of 117,341 UK Biobank participants followed over nine years found that higher Mediterranean diet adherence, assessed using the validated MEDI-LITE scoring tool, was associated with a statistically significant, dose-dependent reduction in incident RA risk, independent of established confounders including smoking, physical activity, and body mass index. [19]

The MADEIRA (Mediterranean Diet in Rheumatoid Arthritis) randomised controlled trial enrolled 40 women with established RA in clinical remission and randomised them to a personalised, calorie-balanced Mediterranean diet with supervised physical activity versus standard care for 12 weeks. The intervention group achieved significantly lower DAS-28 scores, improved body composition, superior glycaemic control, and higher serum 25-hydroxyvitamin D concentrations compared with controls, supporting the multi-dimensional cardiometabolic and immunological benefits of this dietary pattern. [20]

BOX 1 | Evidence Appraisal: Mediterranean Diet and RA Disease Activity

Despite compelling evidence from European RCTs and large prospective cohorts, the literature is not without inconsistency. A 2024 cross-sectional study of 184 Iranian RA patients found no statistically significant association between Mediterranean diet adherence scores and DAS-28, HAQ, or inflammatory biomarker concentrations. These discordant findings most plausibly reflect methodological heterogeneity — differences in dietary assessment instruments, cultural food environments, disease duration, concurrent pharmacotherapy, and cross-sectional versus longitudinal study designs. A 2025 systematic review and meta-analysis by Bostan *et al*. identified substantial between-study heterogeneity and called for standardised, multicentre RCTs before firm universal recommendations can be issued. [21, 22] In South Asian populations, where traditional dietary staples like — turmeric, ginger, legumes, and diverse plant foods — differ substantially from Mediterranean-specific foods, culturally adapted anti-inflammatory dietary patterns merit dedicated investigation.

2. Anti-Inflammatory Diets and the Dietary Inflammatory Index

Beyond the Mediterranean pattern, anti-inflammatory dietary approaches guided by DII scores have gained traction in RA research. The Anti-Inflammatory Diet in Rheumatoid Arthritis (ADIRA) trial — a randomised crossover design — demonstrated that 10 weeks on an anti-inflammatory diet produced statistically significant decreases in DAS-28 scores and multiple circulating inflammatory biomarkers compared with a control dietary period. Importantly, the ADIRA trial provides the foundational evidence base for the ongoing ADIRA-India dietary intervention study targeting Indian women with RA, where locally adapted anti-inflammatory dietary guidelines are being evaluated in a culturally relevant context. [23]

The DII, validated across more than 125 epidemiological studies globally, provides a replicable and culture-transferable instrument for quantifying individual dietary inflammatory load. A higher DII score independently predicts elevated CRP and ESR concentrations and greater DAS-28 scores in RA populations across diverse geographic settings, making it a pragmatic clinical screening tool in nutritional rheumatology. [7, 8]

3. Plant-Based and Vegan Dietary Patterns

Plant-based diets — encompassing Mediterranean variants, lacto-ovo-vegetarian, and strict vegan patterns — are characterised by high concentrations of dietary fibre, polyphenols, carotenoids, and tocopherols, alongside an inherent reduction in arachidonic acid, which serves as the principal precursor of pro-inflammatory prostaglandins and leukotrienes. A 2025 systematic review by Bostan *et al*., incorporating 15 studies including RCTs, cohort investigations, and cross-sectional analyses, concluded that plant-predominant diets were consistently associated with lower DAS-28 scores, reduced pain VAS, lower CRP and ESR, and improved physical function in RA patients, though effect sizes were generally modest. [22]

However, strict vegan dietary patterns carry established risks of nutritional insufficiency, particularly for vitamin B12, vitamin D, long-chain n-3 PUFAs, calcium, iron, and zinc — all of which have specific relevance to RA

immunobiology, bone health, and cardiovascular risk. Individualised nutritional assessment and targeted supplementation are therefore essential components of vegan dietary management in RA. [22]

4. Dietary Patterns Associated with Worsening of RA

Several dietary exposures have been prospectively associated with increased RA incidence or exacerbation of existing disease. High red and processed meat consumption confers excess RA risk in prospective cohort data, mediated through haem iron-induced oxidative stress, heterocyclic amine formation during high-temperature cooking, and nitrosamine-related NF-κB activation. [9]

Sugar-sweetened beverage (SSB) consumption, even at modest frequencies (≥1 serving per week), has been associated with 63% increased odds of seropositive RA in the Nurses' Health Study cohort. Excess dietary sodium activates the serum glucocorticoid kinase-1 (SGK1) pathway, promoting pathological Th17 cell differentiation and has been shown to exacerbate experimental arthritis in murine models. Ultra-processed foods, rich in refined carbohydrates, trans-fatty acids, emulsifiers, and artificial preservatives, promote gut dysbiosis and systemic low-grade inflammation through disruption of epithelial barrier function and activation of the NLRP3 inflammasome. [24, 25]

Specific Nutritional Strategies and Supplementation

1. Omega-3 Polyunsaturated Fatty Acids

Long-chain n-3 PUFAs — principally EPA and DHA from marine sources — are among the most extensively studied nutritional interventions in RA. Their anti-inflammatory actions are mechanistically well-characterised: competitive inhibition of arachidonic acid-derived eicosanoid synthesis via cyclooxygenase and lipoxygenase pathways; generation of resolvins (E-series from EPA, D-series from DHA), protectins, and maresins that actively terminate inflammatory cascades; and transcriptional suppression of pro-inflammatory cytokine genes through NF-κB inhibition. [12]

A rigorous 2022 meta-analysis of 30 RCTs demonstrated statistically significant improvements in DAS-28, tender joint count (TJC), swollen joint count (SJC), pain VAS, HAQ disability scores, and ESR with omega-3 supplementation compared with control, though CRP concentrations showed non-significant changes. Importantly, the analysis identified that marine-derived (animal-based) omega-3 sources were superior to plant-derived ALA supplementation, and that supplementation periods of three to six months yielded greater benefits than shorter courses. [26]

A 2025 systematic review and meta-analysis of 16 RCTs reported a statistically significant reduction in pain scores with omega-3 supplementation (standardised mean difference: −0.42; 95% CI: −0.76 to −0.09; p = 0.01), providing robust contemporary evidence for its analgesic efficacy in RA. Earlier meta-analyses consistently suggested that daily doses exceeding 2.7 g EPA+DHA for three or more months are required to achieve clinically meaningful reductions in NSAID use and joint tenderness. [27]

BOX 2 | Controversy: Do Omega-3 Fatty Acids Prevent RA Onset?

Whether n-3 PUFAs reduce RA incidence — as distinct from symptom burden — remains contentious. A 2024 Mendelian randomisation (MR) study identified that genetically determined higher circulating omega-3 PUFA concentrations, proxied through variants in the fatty acid desaturase (FADS) gene cluster, were paradoxically associated with increased RA risk, challenging observational data. The FADS locus exerts pleiotropic effects on both omega-3 metabolism and immune cell function, confounding straightforward causal inference from observational studies. A separate 2023 meta-analysis found no significant association between dietary omega-3 intake and RA incidence after multivariate adjustment. These findings do not negate robust RCT evidence for symptom amelioration but substantially complicate the epidemiological narrative. [28, 29] Until definitive Mendelian randomisation studies with larger genetic instruments are available, clinicians should recommend omega-3 supplementation (≥2.7 g/day EPA+DHA for ≥3 months) for symptom relief, while avoiding overstatement of its preventive potential.

2. Vitamin D

Vitamin D insufficiency is near-universal in RA patients across geographic settings. The COMORA (Comorbidities in Rheumatoid Arthritis) study, which enrolled 3,920 patients across 17 countries, documented that more than 63% of patients had serum 25-hydroxyvitamin D [25(OH)D] concentrations below 30 ng/mL. A subsequent meta-analysis specific to the Indian subcontinent reported an even higher prevalence of approximately 76%, consistent with high solar irradiance paradoxically coexisting with cultural practices limiting skin exposure. [30, 31]

Vitamin D exerts broad immunomodulatory effects relevant to RA pathogenesis: it suppresses Th17 cell differentiation and effector cytokine production, stimulates Treg expansion, promotes tolerogenic dendritic cell phenotypes, and downregulates synoviocyte production of IL-6 and matrix metalloproteinases. Low serum 25(OH)D concentrations are independently associated with higher DAS-28 scores, greater radiographic joint damage progression, and higher anti-CCP antibody titres in prospective cohort studies. [32]

A 2024 systematic review and dose-response meta-analysis of RCTs confirmed that vitamin D supplementation significantly improved DAS-28, pain VAS, CRP, and ESR relative to placebo in RA patients. A 2025 double-blind RCT specifically demonstrated statistically significant reductions in disease activity and pain with vitamin D3 supplementation at 2000 IU/day over 24 weeks, adding to the evidence base supporting routine vitamin D assessment and correction in RA management. [33, 34]

BOX 3 | Unresolved Questions: Vitamin D and RA — Cause, Consequence, or Confounder?

The causal directionality of the vitamin D–RA relationship remains under investigation. Reverse causation is a plausible confounding mechanism: patients with higher disease activity may have reduced outdoor mobility, limiting cutaneous vitamin D synthesis, more frequent corticosteroid use, which accelerates 25(OH)D catabolism and greater systemic inflammatory burden that consumes circulating vitamin D. Prospective studies examining pre-diagnostic vitamin D status have yielded inconsistent results. Large-scale Mendelian randomisation analyses have generally not supported a strong causal protective effect of

vitamin D on RA incidence. Optimal therapeutic targets for serum 25(OH)D, ideal supplementation dose and formulation (cholecalciferol versus ergocalciferol versus active calcitriol), and duration of supplementation remain insufficiently characterised in adequately powered trials.[35, 36] Nevertheless, given the universality of vitamin D insufficiency, its favourable safety profile at standard doses, and its established immunomodulatory plausibility, routine assessment and correction of deficiency in RA patients is clinically justified.

3. Gut Microbiome Modulation: Probiotics, Prebiotics, and Dietary Fibre

Given the well-documented gut dysbiosis in RA, dietary strategies targeting the gut microbiome have attracted considerable research interest. Probiotics — defined as live microorganisms that, when administered in adequate amounts, confer a health benefit on the host —have been evaluated in multiple small RA trials. [37]

Select probiotic strains, notably Lactobacillus casei, L. acidophilus, and Bacillus coagulans, have demonstrated statistically significant reductions in DAS-28 scores, CRP, IL-17, and TNF-α concentrations in double-blind placebo-controlled trials. However, other strains, including L. rhamnosus GG, have failed to replicate these benefits, underscoring the strain-specificity of probiotic effects and the inadvisability of extrapolating findings across species or formulations. An 8-week synbiotic intervention combining prebiotic fructooligosaccharides with multi-strain probiotics produced significant reductions in DAS-28, CRP, IL-17, and nitric oxide, alongside improvements in total antioxidant capacity, suggesting synergistic benefit. [38, 39]

Dietary fibre — particularly fermentable soluble fibre from oats, legumes, fruits, and root vegetables — is fermented by colonic microbiota to produce SCFAs including butyrate, propionate, and acetate. These SCFAs inhibit histone deacetylases, suppress NF-κB activity, promote Treg differentiation, and reduce intestinal permeability. A large epidemiological analysis of National Health and Nutrition Examination Survey (NHANES) data (2010–2020) demonstrated a significant inverse association between dietary fibre intake and RA prevalence. Furthermore, elevated serum SCFA concentrations were associated with reduced likelihood of progression to clinical arthritis in individuals with elevated RA risk as defined by ACPA positivity. [40, 41]

BOX 4 | Research Gap: Precision Probiotic Therapy for RA

Despite encouraging preliminary evidence, the field of probiotic therapy in RA lacks the methodological rigour necessary for guideline-level recommendations. Existing RCTs are small (typically n<100), short-duration (generally ≤12 weeks), and employ markedly heterogeneous probiotic formulations — varying in species composition, strain identity, colony-forming unit dose, and vehicle, which preclude meaningful cross-trial comparison or robust meta-analysis. No adequately powered study has directly compared single-strain versus multi-strain synbiotic formulations, nor employed next-generation 16S rRNA or metagenomic sequencing to characterise microbiome compositional responses. Individual gut microbiome variability — determined by host genetics (including HLA genotype), baseline diet, antibiotic exposure history, geographic location, and environmental factors — may critically modulate responsiveness to probiotic supplementation. Future research should prioritise microbiome-stratified, precision probiotic intervention trials in RA, integrated with metabolomic and immunological phenotyping. [37, 38]

4. Polyphenols and Functional Foods

4.1 Curcumin

Curcumin (diferuloylmethane), the principal bioactive polyphenol of Curcuma longa (turmeric), exerts multi-target anti-inflammatory effects through inhibition of NF-κB, cyclooxygenase-2 (COX-2), 5-lipoxygenase (5-LOX), and JAK-STAT signalling pathways, and through transcriptional downregulation of IL-6, TNF-α, and matrix metalloproteinases. A well-conducted pilot RCT demonstrated that curcumin (500 mg/day) produced improvements in tender and swollen joint counts and ACR response rates comparable to diclofenac, with a superior tolerability profile. [42]

The principal pharmacological limitation of curcumin is its poor oral bioavailability, attributable to rapid intestinal metabolism, low aqueous solubility, and extensive first-pass hepatic conjugation. Novel drug delivery systems—including phospholipid complexes (e.g., Meriva), nanoparticle formulations, and piperine-enhanced preparations, significantly improve bioavailability and are increasingly employed in contemporary clinical trials. [42]

4.2 Ginger

Ginger (Zingiber officinale) contains gingerols, shogaols, and paradols —bioactive phenolic compounds that inhibit prostaglandin synthesis via COX inhibition, suppress NF-κB activation, and reduce inflammatory cytokine gene expression. A double-blind placebo-controlled RCT by Aryaeian *et al*. demonstrated that ginger supplementation significantly modulated expression of genes encoding intermediate inflammatory mediators in RA patients over 12 weeks. A meta-analysis of five RCTs identified modest but statistically significant reductions in pain VAS and CRP with ginger supplementation, though dose-response relationships require further characterisation. [43]

4.3 Extra-Virgin Olive Oil

Extra-virgin olive oil (EVOO) contains oleocanthal, a secoiridoid compound with COX-1 and COX-2 inhibitory activity functionally analogous to ibuprofen at consumption levels equivalent to approximately 50 g (3–4 tablespoons) daily — alongside oleuropein, hydroxytyrosol, and squalene, which exert antioxidant and NF-κB-suppressive effects. Regular EVOO consumption is mechanistically integral to the anti-inflammatory actions attributed to Mediterranean dietary adherence in RA. [17]

4.4 Berries and Anthocyanin-Rich Foods

Epidemiological data from large cohort studies indicate that high fruit consumption—particularly anthocyanin-rich berries (blueberries, strawberries, black currants)— is associated with reduced RA incidence and lower disease activity in established RA. Anthocyanins, quercetin, and resveratrol — abundant in these foods— inhibit NF-κB signalling, scavenge ROS, and suppress NLRP3 inflammasome activation. The Alternative Healthy Eating Index (AHEI-2010) independently identified high fruit and

vegetable consumption as a significant predictor of lower RA risk in women. [44]

5. Antioxidant Micronutrients

A 2021 double-blind RCT involving 40 female RA patients demonstrated that combined antioxidant supplementation — comprising selenium (50 μg), zinc (8 mg), vitamin A (400 μg), vitamin C (125 mg), and vitamin E (40 mg) daily for 12 weeks— produced significant improvements in serum antioxidant capacity, oxidative stress biomarkers, and DAS-28 scores compared with placebo. Zinc, in particular, is critical for Th17/Treg balance regulation, and its deficiency— documented in a significant proportion of RA patients — correlates with accelerated joint damage. Vitamin B6 deficiency has been independently associated with elevated CRP concentrations and increased pain severity in RA cohorts. [16, 45]

6. Obesity, Body Weight, and RA

Adipose tissue in obese individuals functions as an active pro-inflammatory endocrine organ, secreting leptin, resistin, visfatin, and chemerin—adipokines that promote Th17 differentiation, synoviocyte activation, and RANKL-mediated bone erosion. Obesity is independently associated with higher RA incidence, more severe disease activity, greater functional disability, and significantly attenuated responses to biologic DMARDs — particularly TNF inhibitors, the pharmacokinetics of which are adversely affected by high body mass. [46]

Clinical trial data indicate that intentional weight loss of 10% or greater through dietary caloric restriction produces independent reductions in DAS-28 and CRP in overweight and obese RA patients, beyond any pharmacological effect. This makes weight management one of the most clinically impactful, cost-effective, and patient-accessible modifiable interventions in RA care. [46]

Key Research Gaps and Future Directions

1. Absence of High-Quality Long-Term Randomised Evidence

The most fundamental limitation of the current evidence base is the absence of large-scale, multicentre, long-duration, adequately powered RCTs in nutritional rheumatology. The overwhelming majority of published dietary intervention trials in RA are characterised by small sample sizes (typically $n < 100$), short intervention periods (generally ≤ 12 weeks), single-centre designs, unblinded or poorly blinded methodologies, inadequate control for concurrent DMARD therapy, and reliance on subjective dietary assessment instruments with limited validation in RA populations. EULAR has formally acknowledged this evidentiary gap and called for higher-quality trials to underpin specific dietary recommendations. [9]

2. Personalised Nutritional Medicine

Current dietary recommendations in RA are largely population-level averages that fail to account for substantial inter-individual biological variability. An individual's dietary response is shaped by the composition of their gut microbiome, pharmacogenomic profile (including HLA-DRB1 shared epitope status), autoantibody seropositivity pattern, concurrent medication regimen, metabolic phenotype, and cultural dietary context. Nutrigenomics, — the study of gene-diet interactions at the molecular level — and microbiome-guided personalised nutrition represent frontier domain with enormous potential to transform RA dietary management, but remains inadequately explored in RA-specific populations. [47]

3. Standardisation of Dietary Assessment and Outcome Measures

The absence of validated, RA-specific dietary assessment instruments constitutes a significant methodological barrier to evidence synthesis. Existing studies employ heterogeneous tools, including 24-hour dietary recalls, food frequency questionnaires, and dietary records, with variable validity for capturing bioactive compound intake relevant to RA. Similarly, outcome measurement lacks standardisation across trials, with studies reporting DAS-28, CDAI, SDAI, HAQ, VAS pain scores, and diverse inflammatory biomarker panels without consensus on primary endpoints. Adoption of standardised core outcome sets — analogous to those established by OMERACT (Outcome Measures in Rheumatology) for pharmacological trials— is urgently required for nutritional RCTs in RA. [48]

4. Underrepresentation of Diverse Global Populations

Existing nutritional RA trials have been conducted overwhelmingly in North American, Northern European, and East Asian populations, with marked underrepresentation of South Asian, African, Latin American, and Middle Eastern patient cohorts. Given that RA disease phenotype, gut microbiome composition, genetic susceptibility loci, dietary cultures, and pharmacological treatment practices vary substantially across these populations, the generalisability of current evidence to the 1.4-billion-strong South Asian population — including the Indian subcontinent — is fundamentally limited and requires dedicated investigation. [10]

5. Diet–Drug Interaction Research

Systematic investigation of clinically relevant interactions between dietary components and antirheumatic pharmacotherapy represents a critically underexplored domain. Specific areas of concern include: the impact of folate-rich dietary patterns on methotrexate efficacy and hepatotoxicity; polyphenol-mediated modulation of cytochrome P450 enzyme activity altering DMARD pharmacokinetics; omega-3 PUFA effects on the immunological mechanisms of action of biologic agents; and the interaction between high-sodium diets and the inflammatory outcomes of JAK inhibitor therapy. These represent high-priority questions with direct clinical relevance.

Discussion

1. Integrating Dietary Evidence into Clinical Practice

The totality of current evidence supports the cautious but substantive incorporation of evidence-based dietary counselling into routine RA management. The Mediterranean dietary pattern and broadly defined anti-inflammatory dietary approaches represent the best-evidenced strategies, demonstrating consistent associations with reduced disease activity, lower cardiovascular risk — a major comorbidity burden in RA — and favourable gut microbiome modulation. These patterns align well with existing healthy eating guidance and are accessible, culturally adaptable, and nutritionally complete. [9, 17]

Omega-3 PUFA supplementation at doses of ≥2.7 g/day EPA+DHA for ≥3 months merits consideration as adjunctive therapy in patients with persistent joint pain and elevated NSAID requirements, given consistent evidence of analgesic and anti-inflammatory efficacy across multiple meta-analyses. Vitamin D assessment should be incorporated into routine RA metabolic monitoring, with prompt correction of deficiency (targeting serum 25(OH)D > 30 ng/mL) given its near-universal prevalence and immunomodulatory relevance. Dietary fibre intake should be optimised to ≥ 25–30 g/day through increased consumption of whole grains, legumes, vegetables, and fruits, supporting gut health and SCFA-mediated anti-inflammatory effects. Body weight management through dietary caloric restriction in overweight and obese patients offers independent benefits for disease activity and pharmacological treatment response. [26, 30, 41, 46]

2. Critical Appraisal of Paradoxical Findings

The nutritional rheumatology literature contains several findings that warrant cautious interpretation. The 2024 Mendelian randomisation study reporting an increased RA risk with genetically higher omega-3 concentrations represents a notable challenge to conventional observational evidence. However, the FADS gene cluster variants used as instrumental variables in this analysis exert broad pleiotropic effects on lipid metabolism and immune function that may not exclusively capture the health effects of dietary omega-3 PUFA intake. The finding therefore does not contradict clinical trial evidence demonstrating symptom relief with omega-3 supplementation, but highlights the limitations of observational biomarker data as proxies for dietary exposure. [28]

The discordance between the MADEIRA trial's positive findings for the Mediterranean diet and the null results of the Iranian cross-sectional study is most parsimoniously explained by fundamental differences in study design (RCT versus cross-sectional), outcome measurement (DAS-28 change versus absolute score), population characteristics, and dietary assessment methodology, rather than genuine absence of diet effects. These observations reinforce the need for large, well-designed RCTs as the gold standard for establishing dietary efficacy in RA. [20, 21]

3. Nutritional Safety in Dietary Modification for RA

Dietary interventions in RA must be appraised not only for efficacy but for nutritional safety and sustainability. Restrictive dietary approaches — including prolonged therapeutic fasting, elimination diets, and unmonitored vegan diets — carry established risks of micronutrient deficiencies relevant to RA management, including calcium, vitamin D, vitamin B12, iron, zinc, and long-chain n-3 PUFAs. High-dose antioxidant supplementation, though appealing conceptually, can suppress immunological responses at supraphysiological concentrations and may interact adversely with immunomodulatory pharmacotherapy. Involvement of a registered dietitian with competency in immunological disease management is recommended when patients express interest in significant dietary modification, to ensure nutritional adequacy, prevent supplementation-related harm, and provide culturally sensitive guidance.

Practical Recommendations for Clinicians

Based on current evidence, the following dietary recommendations are appropriate as adjunctive strategies in RA management, in addition to optimised pharmacotherapy:

- Adopt a Mediterranean or broadly anti-inflammatory dietary pattern: high in vegetables, fruits, whole grains, legumes, fatty fish (2–3 servings/week), nuts, seeds, and extra-virgin olive oil; low in red/processed meat, refined carbohydrates, and sugar-sweetened beverages. [9, 17]
- Consider omega-3 PUFA supplementation at doses of ≥2.7 g/day EPA+DHA for a minimum of 3 months in patients with active joint pain and high NSAID burden, acknowledging evidence of analgesic rather than disease-modifying efficacy. [26, 27]
- Assess serum 25(OH)D at baseline and every 6–12 months; supplement with cholecalciferol to achieve and maintain concentrations ≥30 ng/mL. [33, 34]
- Increase dietary fibre intake (target ≥25–30 g/day) through whole plant foods; consider evidence-based probiotic supplementation (L. casei, L. acidophilus) for patients with gastrointestinal dysbiosis symptoms. [38, 41]
- Incorporate curcumin as a dietary supplement (500–1000 mg/day, bioavailability-enhanced formulation) in patients seeking additional anti-inflammatory support alongside standard therapy.[42]
- Prioritise weight management through dietary caloric restriction in overweight/obese patients, targeting ≥10% body weight reduction to improve disease activity and biologic DMARD responsiveness.[46]
- Involve a registered dietitian with rheumatology competency to provide individualised, culturally adapted nutritional guidance, monitor for nutrient deficiencies, and ensure dietary safety.

Conclusions

The cumulative body of evidence reviewed herein substantiates a clinically meaningful role for dietary modification and targeted nutritional supplementation as adjunctive strategies in the comprehensive management of rheumatoid arthritis. Mediterranean and anti-inflammatory dietary patterns command the strongest evidence base, demonstrating consistent associations with reduced disease activity scores, lower circulating inflammatory biomarker concentrations, improved cardiometabolic profiles, and favourable modulation of gut microbial ecology. Omega-3 PUFA supplementation at adequate doses and duration offers reliable analgesic benefits and NSAID-sparing effects. Vitamin D insufficiency, near-universal in RA populations across geographic settings, warrants routine screening and pharmacological correction. Dietary fibre, selected probiotics, and functional food bioactives — particularly curcumin, ginger, anthocyanin-rich fruits, and extra-virgin olive oil — demonstrate mechanistically plausible and clinically supported anti-inflammatory activities. Body weight management through dietary restriction constitutes one of the most impactful modifiable interventions in RA care.

Notwithstanding these advances, the nutritional rheumatology evidence base remains characterised by significant methodological limitations such as — small sample sizes, short follow-up periods, heterogeneous study designs, and absence of standardised dietary assessment and outcome measurement tools — that preclude definitive

evidence-based recommendations. Critical unresolved questions pertain to the causal directionality of vitamin D and omega-3 associations with RA risk, optimal probiotic formulations and dosing regimens, and the translation of existing evidence to diverse cultural and ethnic populations insufficiently represented in current trials.

Future research imperatives include large-scale, multicentre, long-duration RCTs incorporating standardised dietary assessment instruments, validated composite disease activity endpoints, and comprehensive gut microbiome, metabolomic, and nutrigenomic profiling to enable the development of truly personalised dietary recommendations. Collaborative, multidisciplinary integration of rheumatology, dietetics, gastroenterology, and clinical immunology expertise is essential to realise the full potential of nutritional medicine as a complementary pillar of RA management. Dietary interventions should always complement and never supplant evidence-based pharmacotherapy within a treat-to-target framework.

Declarations

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