

Nutritional Interventions and Inflammatory Modulation in Autoimmune Diseases: A Narrative Review

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Abstract: Autoimmune diseases arise from the interaction between genetic susceptibility, environmental exposures, and loss of self-tolerance, involving chronic inflammation, oxidative stress, and impaired intestinal barrier function. This narrative review examined dietary patterns, nutrients, bioactive compounds, and microbiota-targeted interventions as adjuncts in rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis, ulcerative colitis, and Hashimoto thyroiditis. PubMed/MEDLINE and the Cochrane Library were searched, prioritizing systematic reviews, meta-analyses, and clinical trials published from 2021 to September 2026, together with relevant seminal studies. The most consistent evidence supports nutrient-dense dietary patterns, particularly the Mediterranean pattern, as adjunctive strategies to improve diet quality and selected clinical and inflammatory outcomes. Potential benefits have also been reported for unsaturated fatty acids, prebiotic fibers, strain-specific probiotics, and some bioactive compounds, although findings remain heterogeneous and disease-, intervention-, and nutritional-status dependent. Exclusion diets, supplements, and restrictive metabolic strategies should not be generalized. Nutritional care should therefore be individualized, integrated with medical therapy, and centered on overall diet quality, adequate energy and protein intake, and correction of documented deficiencies [1-5].

Keywords: Autoimmune Diseases; Inflammation; Diet Therapy; Mediterranean Diet; Gut Microbiota; Clinical Nutrition.

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I. INTRODUCTION

Autoimmune diseases comprise a heterogeneous group of conditions in which mechanisms of immune tolerance become insufficient and allow responses against the body's own components. Their clinical expression depends on the interaction of genetic susceptibility, biological sex, environmental exposures, infections, smoking, body composition, medications, and other elements of the exposome. The increasing frequency of autoimmunity observed across different populations highlights the need to investigate modifiable factors without reducing complex diseases to a single environmental or dietary cause [1,2].

The higher prevalence of several autoimmune diseases among women involves hormonal, chromosomal, epigenetic, and immunological factors. Estrogens, androgens, and X chromosome-linked genes influence B- and T-cell activation, cytokine production, and responses to self-antigens, but the magnitude and direction of these effects vary by disease and stage of life. Therefore, sex differences should not be explained solely by a single hormone or immune pathway [2].

Diet may interact with autoimmunity through multiple pathways, including the availability of substrates for lipid mediator synthesis, regulation of redox status, body composition, insulin sensitivity, epithelial integrity, microbial metabolite production, and micronutrient adequacy. Biological plausibility, however, does not automatically translate into clinical benefit; interpretation must consider study design, sample size, intervention duration, concomitant pharmacological therapy, and the clinical relevance of the outcomes assessed [3-5].

Against this background, this review aimed to critically synthesize evidence on dietary patterns, nutrients, bioactive compounds, and microbiota-modulating strategies in the adjunctive management of autoimmune diseases in adults, with emphasis on rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis, ulcerative colitis, and Hashimoto thyroiditis [3-5].

II. METHODS

A narrative review with a structured literature search was conducted. This design was selected because it enables the integration of mechanistic evidence, observational studies, clinical trials, and systematic reviews in a broad and clinically heterogeneous field. The writing and critical appraisal followed the principles of the Scale for the Assessment of Narrative Review Articles, which emphasizes topic relevance, explicit objectives, description of the search process, evidence-based statements, and balanced presentation of the findings [6].

Searches were conducted in PubMed/MEDLINE and the Cochrane Library and included publications available through September 2026. Medical Subject Headings and free-text terms related to autoimmune diseases, rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis, inflammatory bowel disease, ulcerative colitis, and Hashimoto disease were combined with diet therapy, dietary patterns, Mediterranean diet, anti-inflammatory diet, nutrition, inflammation, microbiota, probiotics, prebiotics, and bioactive compounds. Reference lists of selected studies were also hand-searched [6].

Systematic reviews with or without meta-analysis, clinical trials, and prospective studies in adults published between 2021 and 2026 were prioritized. Earlier studies were retained when necessary for conceptual understanding or when they represented key evidence. Studies conducted exclusively in vitro or in animals, case reports, publications without a direct relationship to a nutritional exposure or intervention, and reports with insufficient information for appraisal were excluded. Because this was a narrative review, no PRISMA flow diagram, pooled effect estimate, or formal risk-of-bias assessment was produced; findings were organized by mechanisms, diseases, and clinical applicability [6].

➤ *Biological Basis of the Diet-Autoimmunity Interface*

Autoimmune inflammation is sustained by interconnected innate and adaptive immune pathways involving antigen presentation, differentiation of T-cell subsets, autoantibody production, complement activation, and cytokine release. Dietary components may modify the metabolic environment in which these responses occur, but they do not replace immunomodulatory therapies. Clinically, diet should be considered part of integrated care that may address cardiovascular risk, body composition, symptoms, and quality of life rather than an isolated treatment for the autoimmune process itself [1,3,4].

Dietary fat quality is one of the most extensively studied mechanisms. Polyunsaturated fatty acids contribute to membrane composition and to the production of eicosanoids and specialized pro-resolving mediators, whereas patterns rich in saturated fat and ultra-processed foods may coexist with lower intakes of fiber and antioxidant compounds. In systemic lupus erythematosus, a systematic review with meta-analyses found associations between unsaturated fatty

acid-rich diets and selected disease outcomes, although study heterogeneity limits universal prescriptions [3].

Oxidative stress also contributes to the pathophysiology of several autoimmune diseases. Fruits, vegetables, legumes, whole grains, nuts, olive oil, and fish provide compounds that may support antioxidant defenses and the resolution of inflammation. Nevertheless, the response to an isolated nutrient does not necessarily reproduce the effect of a complete dietary pattern, and intermediate biochemical markers should not be interpreted as equivalent to clinical remission [7-10].

III. ANTI-INFLAMMATORY DIETARY PATTERNS

➤ *Mediterranean Diet and Rheumatoid Arthritis*

Among the dietary patterns evaluated, the Mediterranean diet has the most consistent clinical evidence. It combines a high intake of minimally processed plant foods, olive oil, fiber, and phenolic compounds with moderate fish consumption and a lower contribution of processed meats and products rich in added sugars and saturated fat. In rheumatoid arthritis, systematic reviews and meta-analyses suggest a modest reduction in pain and possible improvement in disease activity markers, although certainty of evidence varies and protocols are not uniform [7].

In the MADEIRA trial, an isocaloric Mediterranean intervention combined with physical activity promotion improved adherence to the dietary pattern and was accompanied by a reduction in the Disease Activity Score 28 and improvement in cardiometabolic indicators among women with rheumatoid arthritis in remission. Because diet and physical activity were modified simultaneously, the effects cannot be attributed exclusively to food intake; however, the study demonstrates the feasibility of a structured lifestyle intervention [8].

Recent observational data have also associated greater adherence to the Mediterranean diet with a lower risk of rheumatoid arthritis, although prospective associations do not establish causality. In parallel, a small open-label study found that an anti-inflammatory diet modified serum oxylipin profiles in patients with active disease; the small sample and lack of blinding require cautious interpretation. Collectively, these findings support the Mediterranean pattern as a plausible adjunctive strategy, not as a single protocol or replacement for pharmacotherapy [9,10].

Beyond inflammatory activity, nutritional care should address muscle mass, adiposity, cardiovascular risk, and adequate energy and protein intake. A systematic review in rheumatoid arthritis and spondyloarthritis showed that dietary interventions may alter body weight, body composition, and cardiometabolic factors, but outcomes depend on the type and intensity of the intervention. In patients with weight loss, sarcopenia, or poor intake, restrictive diets may increase nutritional risk [11].

➤ *Systemic Lupus Erythematosus*

In systemic lupus erythematosus, the nutrition literature is less extensive and includes highly diverse interventions. The review by Wang et al. identified potential benefits of dietary patterns rich in unsaturated fatty acids, particularly for inflammation and cardiometabolic risk; however, the authors emphasized methodological heterogeneity and the limited number of trials. Practical recommendations should therefore prioritize overall dietary adequacy and individual risk factors rather than standardized doses of a single lipid [3].

The interface between nutrition and epigenetics was explored in a randomized trial of folic acid and vitamin B12 supplementation in women with inactive lupus. Changes in adipose tissue DNA methylation depended on nutritional status, suggesting that obesity and the metabolic context may modify the response. The finding is mechanistically relevant but does not support indiscriminate supplementation with methyl donors, particularly because epigenetic outcomes do not, by themselves, establish sustained clinical improvement [12].

➤ *Multiple Sclerosis*

In multiple sclerosis, a systematic review with network meta-analysis found signals of improvement in fatigue and quality of life with some dietary interventions but rated overall confidence in the evidence as low or very low because of small samples, risk of bias, and heterogeneity. Current evidence is therefore insufficient to establish the definitive superiority of a specific diet for modifying the disease course [13].

Metabolic strategies are also being investigated. In a randomized trial, intermittent calorie restriction was feasible and produced favorable metabolic and immunological changes in people with multiple sclerosis, whereas an open-label ketogenic diet study reported reduced fatigue in only 16 participants. A feasibility trial of the MIND dietary pattern demonstrated good adherence and improved diet quality but was not powered to confirm cognitive benefit. These findings are promising but remain preliminary and cannot yet be generalized [14-16].

In practice, ketogenic diets and calorie restriction require nutritional screening, assessment of contraindications, clinical supervision, and monitoring of fiber, micronutrient, and protein intake. Potential symptom relief should be balanced against the sustainability of the intervention and risks such as loss of lean mass, constipation, lipid abnormalities, and poor adherence, particularly among patients with fatigue or functional limitations [13-16].

➤ *Ulcerative Colitis*

In ulcerative colitis, the relationship among diet, symptoms, and inflammation is particularly complex because food tolerance may vary according to disease activity, extent of intestinal involvement, and current therapies. A systematic review with meta-analysis found heterogeneous results across dietary interventions and did not support a single pattern capable of inducing or maintaining remission in all patients.

Preservation of nutritional status and prevention of unnecessary restrictions should accompany every dietary strategy [17].

A multicenter trial evaluated the Mediterranean diet with or without curcumin or resveratrol in mild-to-moderately active disease and found improvement in clinical and inflammatory outcomes over eight weeks. However, the multiple intervention arms and components make it difficult to isolate the contribution of each supplement. The evidence favors an approach centered on the dietary pattern and individual tolerance, with supplementation reserved for specific, monitored decisions [18].

➤ *Hashimoto Thyroiditis and Gluten Exclusion*

Gluten exclusion in Hashimoto thyroiditis without celiac disease remains controversial. A recent meta-analysis identified changes in selected thyroid markers and autoantibodies, but the included studies were few, small, and heterogeneous. An earlier meta-analysis had already concluded that evidence was insufficient to recommend a gluten-free diet to all individuals with autoimmune thyroiditis. Celiac disease and other gluten-related disorders should therefore be investigated when clinically indicated, without turning exclusion into a general rule [19,20].

In a pilot trial of 45 euthyroid patients, the Mediterranean diet improved markers of antioxidant defense, whereas the gluten-free diet did not significantly change autoantibodies, thyroid function, or oxidative stress. These findings reinforce that overall diet quality may be more relevant than isolated gluten withdrawal in patients without celiac disease [21].

The same caution applies to rheumatoid arthritis: a focused review found no consistent basis for excluding gluten in adults without celiac disease or non-celiac gluten sensitivity. Restrictions without a diagnosis may reduce dietary variety, fiber, and micronutrient intake, increase costs, and impair adherence. Counseling should distinguish a formal medical indication, a supervised therapeutic trial, and personal preference [22].

➤ *Gut Microbiota, Epithelial Barrier and Immunity*

The gut is a major site of interaction among antigens, the microbiota, and the immune system. Dysbiosis, altered microbial metabolites, and impaired epithelial barrier function have been reported in several autoimmune diseases; however, it remains difficult to determine whether these changes are causes, consequences of disease, medication effects, or reflections of dietary intake. The term leaky gut should not be used as a nonspecific diagnosis or as an automatic justification for exclusion diets [5,23].

Fermentable fibers support the production of short-chain fatty acids, including butyrate, which contribute to colonocyte metabolism, epithelial integrity, and immune-cell regulation. This mechanistic basis supports diets rich in plant foods and explains the interest in prebiotics. Nevertheless, metabolite production depends on the microbiota, substrate, dose, and individual tolerance; direct extrapolation from

mechanism to clinical response should therefore be avoided [5,23].

In rheumatoid arthritis, a meta-analysis of probiotic trials suggested possible improvement in selected inflammatory markers but found substantial variability in strains, doses, and duration. A triple-blind trial of 10 g/day of inulin for eight weeks reported improvements in some inflammatory and clinical indices, although not all outcomes differed between groups. These findings do not support assuming that all probiotics or prebiotics have equivalent effects; recommendations should consider gastrointestinal symptoms, tolerance, and product-specific evidence [24,25].

In Hashimoto thyroiditis, nutritional education combined with *Lactiplantibacillus plantarum* 299v improved more quality-of-life domains than nutritional education with placebo, but it did not reduce anti-thyroid peroxidase antibodies or body mass index. The study suggests symptomatic and quality-of-life benefits, not proven restoration of the intestinal barrier or suppression of autoimmunity. This distinction is essential for responsible communication of results [26].

➤ *Bioactive Compounds and Supplementation*

Curcumin, resveratrol, and silymarin have been investigated for their effects on inflammatory signaling and redox status. A meta-analysis of curcumin studies in rheumatoid arthritis and ulcerative colitis found reductions in selected biomarkers, while a pilot trial of silymarin in active rheumatoid arthritis reported improvements in selected outcomes. Nevertheless, the small number of studies, differences in formulations and bioavailability, and short follow-up periods limit general recommendations [27,28].

Supplementation should be guided by a clear indication, assessment of dietary intake, and laboratory testing when appropriate. Pharmacological doses of bioactive compounds or micronutrients may interact with medications and cause adverse effects. Moreover, correction of a documented deficiency is distinct from using a supplement to modulate autoimmunity. Potential benefit should always be weighed against safety, cost, and quality of evidence [12,27,28].

Likewise, omega-3 fatty acids have an anti-inflammatory rationale, but response and dose vary with the disease, habitual fish intake, lipid profile, bleeding risk, and concomitant therapy. The clinical priority should be to incorporate food sources compatible with the overall dietary pattern; supplements may be considered individually without promising independent control of the disease [3,7].

IV. IMPLICATIONS FOR CLINICAL PRACTICE

Nutritional assessment in people with autoimmune diseases should include weight history, body composition when available, strength and functional status, food intake, gastrointestinal symptoms, fatigue, barriers to meal preparation, supplement use, and potential drug-nutrient interactions. Laboratory tests should address clinical

hypotheses and the treatment regimen rather than constitute indiscriminate panels. This approach helps distinguish disease-related inflammation, nutritional deficiency, medication adverse effects, and inadequate intake [11,13,17].

As a practical foundation, clinicians should prioritize a variety of vegetables, fruits, legumes, whole grains as tolerated, nuts and seeds, olive oil, and adequate sources of protein and unsaturated fats. The plan should be adapted to culture, income, preferences, comorbidities, disease activity, and functional capacity. In patients with poor intake or loss of muscle mass, adequate energy and protein may be more urgent than increasing dietary restriction [7-11].

Elimination of gluten, dairy products, or other food groups requires a clear indication, a measurable objective, and reassessment. When necessary, exclusion should include nutritionally equivalent substitutions and monitoring of adherence, symptoms, and quality of life. Restrictive strategies based solely on unvalidated tests, extensive lists of inflammatory foods, or promises of cure may lead to deficiencies, food-related guilt, and delays in effective care [19-22].

Interprofessional communication is also essential. Dietitians should document the nutrition diagnosis, objectives, intervention, and monitoring indicators and coordinate care with rheumatologists, neurologists, gastroenterologists, endocrinologists, and other professionals. Diet is an adjunct to medical therapy; changes in immunosuppressants, thyroid hormone replacement, or disease-modifying therapy should not be based solely on perceived dietary improvement [3,7,13,17].

V. LIMITATIONS OF THE EVIDENCE

Interpretation of the literature is limited by small samples, short interventions, varying diagnostic criteria, differences in pharmacological treatment, and multiple outcomes. In dietary studies, blinding is difficult, and adherence may coincide with changes in physical activity, sleep, and body weight. Furthermore, improvement in biomarkers does not necessarily indicate reduced clinical activity, prevention of organ damage, or a lower need for medication [7,13,17].

This review also has limitations inherent to the narrative design: study selection was not performed by two independent reviewers, and no formal risk-of-bias assessment or meta-analysis was conducted. The structured search and prioritization of recent systematic reviews and trials reduce, but do not eliminate, selection bias. The findings should inform clinical reasoning and identify evidence gaps rather than be interpreted as a prescriptive guideline [6].

VI. CONCLUSION

Available evidence supports nutrition as a relevant component of comprehensive autoimmune disease management, particularly through improvements in diet quality, nutritional status, cardiometabolic factors, and

selected symptoms or inflammatory markers. Among the approaches evaluated, nutrient-dense dietary patterns, especially the Mediterranean pattern, have the most coherent and applicable evidence, although effects differ across diseases and individuals.

Probiotics, prebiotics, bioactive compounds, calorie restriction, ketogenic diets, and gluten exclusion have more specific indications and remain supported by heterogeneous evidence. Nutritional care should be individualized, integrated with pharmacotherapy, monitored through clinical and nutritional outcomes, and revised according to response and tolerance. Future research requires larger samples, comparable protocols, longer follow-up, and clinically relevant outcomes.

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