Elderly Health Journal 2025; 11(1): 60-68.
Shahid Sadoughi University of Medical Sciences, Yazd, Iran
Journal Website : http://ehj.ssu.ac.ir

Review Article

The Role of Nutrition in Managing Rheumatoid Arthritis in the Elderly Patients

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A B S T R A C T

Article history
Received 19 Mar 2025
Accepted 25 May 2025

Citation: Adedeji YO, Adedeji AA. The role of nutrition in managing rheumatoid arthritis in the elderly patients. Elderly Health Journal. 2025; 11(1): 60-68.

Rheumatoid arthritis (RA) is a chronic autoimmune condition that primarily affects older adults, leading to joint inflammation and systemic complications. This review aims to evaluate the effectiveness of dietary interventions in managing this disease. Existing literatures indicate a significant prevalence of malnutrition within this population, which is often exacerbated by factors such as sarcopenia, frailty, and comorbidities. This review discusses the anti-inflammatory effects of specific diets, notably the Mediterranean and plant-based dietary patterns, which are rich in monounsaturated fats, omega-3 fatty acids, dietary fiber, and antioxidants. These diets have shown promise in reducing inflammation and improving joint function. However, barriers such as food insecurity and limited nutritional knowledge complicate their implementation. Emerging strategies, including personalized nutrition on the basis of individual health profiles, offer potential pathways for enhancing RA management. Addressing these nutritional barriers and improving assessment tools are essential for integrating effective dietary strategies into comprehensive care plans for older adults with RA. Future research should focus on tailored interventions that consider the unique needs of this population.

Keywords: Nutrition, Aging, Rheumatoid Arthritis

Background

One of the most common autoimmune conditions is rheumatoid arthritis (RA), which is associated with joint tissue destruction and pain mediated by a persistent inflammatory state. Interactions between specific genes and environmental factors lead to autoimmunity where the body synthesizes certain proteins (antibodies) that attack self-tissues within the joint synovial membrane. This leads to chronic inflammation, involving a complex pathway that culminates in clinical presentations of joint swelling, pain and ultimately joint destruction (1).

RA affects up to 1% of adults of the entire world population, and predictive analysis of a global burden of disease study revealed that by 2050, approximately 31.7 million people would be affected worldwide (2). Although RA can occur at any age, its incidence rises with advancing age, disproportionately impacting individuals above 60 years of age (3). Older adults with RA experience distinct challenges due to age-related physiological changes, including immune senescence, comorbidities, and altered inflammatory responses, which can intensify disease severity and progression (4).

Nutrition plays a critical role in modulating inflammation and disease outcomes in RA. Obesity and insulin resistance, which are often linked to unhealthy dietary patterns, are recognized as risk factors that exacerbate systemic inflammation and disease severity (5). Conversely, malnutrition, which is prevalent in older adults, contributes to frailty, muscle wasting, and diminished quality of life (3). In the context of RA, malnutrition can further compromise immune function and amplify inflammation, complicating disease management. Existing literature presents conflicting findings regarding the effectiveness of dietary interventions in RA management. The systematic review of Bostan et al. (6) suggest that plant-based diets, particularly the Mediterranean diet can reduce disease severity and improve clinical markers. Porras et al. (7) on the other hand reported limited effects with low

[DOI: 10.18502/ehj.v11i1.19003]

certainty of evidence. These inconsistencies may be due to variations in study design, dietary adherence, and the unique challenges faced by elderly populations with comorbidities. Despite growing evidence supporting dietary interventions as an adjunctive strategy for managing RA, limited attention has been given to their application in older adults, a population uniquely vulnerable to both nutritional deficiencies and the disease's systemic effects.

This review aims to evaluate the effectiveness of dietary interventions in managing this condition. This review also seeks to summarize the literature on how inadequate nutrition affects RA progression and overall health in this population, identify significant gaps in nutritional assessment and intervention strategies, and highlight personalized dietary approaches that could enhance treatment outcomes for elderly individuals with RA.

Overview of RA pathophysiology

The clinical presentations associated with RA; synovial swelling, joint pain and destruction are associated with immune complex-mediated inflammation due to autoimmunity. The genesis of this autoimmune state is the loss of tolerance to citrulline-containing self-antigens produced during the posttranslational modification of arginine residues. This process is catalyzed by the enzyme peptidylarginine deiminase in genetically susceptible individuals, and is influenced by certain environmental factors such as smoking, infections, and microbiome dysbiosis. This often results in the production of autoantibodies such as anti-citrullinated protein antibody (ACPA) and rheumatoid factor which can react with self-proteins, and ultimately induce inflammation (8).

Once the autoimmune process is initiated, the synovial membrane becomes the primary site of immune-mediated damage. Immune cells, including CD4+ T cells, B cells, macrophages, and dendritic cells, infiltrate the synovium, leading to the release of proinflammatory cytokines such as tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), and interleukin-17 (IL-17). These cytokines amplify the inflammatory response, promoting the recruitment of more immune cells and activating fibroblast-like synoviocytes (FLSs). The hyperproliferation of FLS leads to the formation of a pannus, an abnormal layer of granulation tissue, which invades and degrades cartilage and bone (9).

Cartilage destruction in RA is driven by the action of matrix metalloproteinases (MMPs) secreted by FLSs and other inflammatory cells, whereas bone erosion is mediated by osteoclasts. The cytokine receptor activator of nuclear factor-kappa B ligand (RANKL), expressed by T cells and FLSs, stimulates osteoclast differentiation and activity, resulting in bone resorption (9). This coupled destruction of cartilage and bone underlies the progressive joint deformities characteristic of RA. In addition to localized joint pathology, RA is associated with systemic inflammation, which contributes to comorbidities such as cardiovascular disease, interstitial lung disease, and anemia of chronic disease (10). Elevated levels of IL-6 and other proinflammatory cytokines induce the hepatic production of acute-phase proteins such as C-reactive protein (CRP) and fibrinogen, exacerbating systemic inflammation. Chronic inflammation also promotes endothelial dysfunction and accelerated atherosclerosis, significantly increasing the risk of cardiovascular events in RA patients (11, 12).

Impact of aging on RA

RA can develop at any age; however, its typical onset occurs around the sixth decade of life, with incidence peaking in the seventh. Aging influences the pathogenesis, progression, and management of autoimmune diseases, including RA, making it a significant clinical challenge. The term elderly-onset RA has been used to describe RA that develops in individuals aged 60 years and above, and this group often presents with atypical clinical features and an altered therapeutic response (4, 13). The study of Chen et al. (12) which analysed secondary data from two observational studies, although failed to establish a direct causal link suggested that accelerated biological aging may increase the risk of RA particularly among susceptible groups.

Aging contributes to RA progression through a process known as “premature immunosenescence”, an accelerated form of immune system aging which leads to both systemic inflammation and the development of comorbidities such as malignancies, cardiovascular diseases, lung diseases and osteoporosis. This immunosenescence is characterized by several biological hallmarks of aging, which appear earlier in RA patients than in healthy individuals. These include genomic instability, telomere shortening, epigenetic changes, impaired protein homeostasis, altered nutrient sensing, mitochondrial dysfunction, cellular senescence, stem cell exhaustion, and disrupted intercellular communication leading to chronic low-grade inflammation (inflammaging) (14). The thymus, responsible for generating naïve T cells, undergoes involution with age, leading to reduced output of new T cells, as evidenced by low levels of T-cell receptor excision circles (TRECs) in RA patients, often comparable to healthy individuals two decades older. This reduced thymic function results in compensatory peripheral proliferation of T cells, inducing replicative stress and premature T-cell aging. Senescent CD4+CD28− T cells accumulate in both circulation and synovial tissues, producing pro-inflammatory cytokines such as Td NF-α and IL-17 and expressing CX3CR1, a receptor that facilitates infiltration into inflammatory sites. These senescent cells not only perpetuate inflammation but also contribute to joint destruction and bone loss by expressing high levels of RANKL, which promotes osteoclast activation in the presence of cytokines like IL-1β and IL-6. Consequently, RA patients frequently exhibit reduced bone mineral density and are at heightened risk of early-onset osteoporosis. Furthermore, accelerated immune aging in RA is reflected in the oligoclonal expansion of T cells, telomere erosion, and contraction

[DOI: 10.18502/ehj.v11i1.19003]

of the T-cell repertoire, which collectively impair immune tolerance and regeneration. These age-associated immune dysfunctions not only exacerbate RA severity but also pose significant therapeutic challenges, especially in elderly individuals who may have limited regenerative capacity and increased susceptibility to drug-related side effects (14, 15).

Clinical manifestations in elderly-onset RA also differ from those in young-onset RA; for instance, older patients often present with higher titers of ACPA, elevated inflammatory markers like Erythrocyte Sedimentation Rate (ESR) and CRP, and higher levels of IL-6 but lower TNF-α. Moreover, subcutaneous nodules and fibroid synovial pathotypes are less common in the elderly while bone erosions and comorbidities are more frequent. Importantly, elderly patients may exhibit a poorer prognosis and reduced response to disease-modifying antirheumatic drugs (DMARDs), including methotrexate, partly due to age-related pharmacokinetic changes and a higher risk of adverse effects, especially from non-steroidal anti-inflammatory drugs (13).

Malnutrition and nutritional status in elderly RA patients

Malnutrition is a significant concern among elderly individuals with RA, with a prevalence ranging from 6% to over 90% depending on the assessment criteria and region (16-21). Factors contributing to malnutrition in this population include age-related physiological changes, dietary limitations, and the inflammatory nature of RA itself. These challenges are further compounded by comorbidities such as cardiovascular diseases and diabetes, which may impose additional dietary restrictions. Importantly, there is a bidirectional relationship between malnutrition and inflammation: chronic inflammation may promote anorexia, reduced food intake, and muscle catabolism, thereby accelerating nutritional deterioration, while malnutrition can alter gut microbiota composition and trigger inflammatory pathways, reinforcing the cycle of disease activity (22-24). The consequences of poor nutritional status are profound, malnutrition exacerbates systemic inflammation, increases disease activity, and contributes to muscle mass loss, frailty, and cognitive decline. Low serum albumin levels, a common marker of malnutrition, have been correlated with worsened clinical outcomes (3, 20, 21). Sarcopenia, a condition marked by progressive skeletal muscle loss and reduced strength, is notably more prevalent in RA patients compared to the general population and significantly worsens physical function and quality of life. Its progression is partly driven by proinflammatory cytokines (such as TNF-α and IL-6), common mediators in RA pathogenesis suggesting that biologic therapies targeting these pathways may also alleviate muscle degradation. While moderate aerobic and resistance exercise can help counteract sarcopenia, excessive physical exertion may aggravate inflammation (25). Addressing this interrelated burden of malnutrition and sarcopenia is thus essential for improving long-term prognosis and functional outcomes in elderly RA patients.

The Mediterranean diet and its anti-inflammatory effects

The Mediterranean diet has been extensively studied for its anti-inflammatory and health-promoting properties (26), making it a beneficial dietary approach for individuals with rheumatoid arthritis. Characterized by a high intake of fruits, vegetables, whole grains, nuts, seeds, and olive oil; and moderate consumption of fish, the Mediterranean diet is rich in monounsaturated fatty acids (MUFAs) and omega-3 polyunsaturated fatty acids (PUFAs). These components are known to modulate immune responses, reduce oxidative stress, and alleviate inflammation.

Clinical studies have demonstrated that adherence to the Mediterranean diet significantly reduces the levels of markers of inflammation such as CRP and IL-6, improves joint function, and lowers disease activity scores in RA patients (27-29). Additionally, the diet's emphasis on low consumption of red meat and processed foods further minimizes exposure to proinflammatory agents, enhancing overall disease management (30).

Plant-based diets and vegetarian approaches

Owing to their anti-inflammatory potential and nutrient density, plant-based diets, including vegetarian and vegan approaches, have also shown promise in the management of RA. These diets emphasize the consumption of fruits, vegetables, legumes, nuts, and seeds, which are rich in antioxidants, fiber, and polyphenols. These components have been linked to reduced systemic inflammation and improved gut microbiota, which play critical roles in modulating immune function and inflammation in RA (31).

Studies suggest that plant-based diets can decrease inflammatory markers and improve symptoms such as pain and joint stiffness (6, 32, 33). The exclusion of animal-derived products also reduces the intake of saturated fats, which are associated with proinflammatory pathways. Furthermore, these diets contribute to weight management, which is crucial as obesity exacerbates RA symptoms.

Together, the Mediterranean and plant-based dietary patterns highlight the importance of a nutrient-dense, anti-inflammatory dietary strategy for improving outcomes in RA management, particularly among older adults who are more susceptible to the detrimental effects of chronic inflammation.

Role of specific nutrients and supplements in RA management

Many individuals with RA have reported symptom relief through dietary changes. The existing literature also indicates that specific diets can contribute to the management and remission of RA symptoms, which shows the potential of nutrition as an adjunctive therapeutic strategy (32, 34). Some of the nutrients are explained here;

[DOI: 10.18502/ehj.v11i1.19003]

Unsaturated fats in inflammation

MUFAs and PUFAs are important diets that can potentially diminish the impact of chronic diseases such as RA because of their ability to reduce inflammatory activities (35). MUFAs exert their anti-inflammatory effects by reducing oxidative stress levels through a reduction in reactive oxygen species production; and the secretion of anti-inflammatory factors/mediators such as interleukin 10 (IL-10) and transforming growth factor beta (TGFβ1). High intake of food rich in MUFAs such as avocados and sesames has also been positively correlated with disease remission and has been suggested to be the main Mediterranean diet component that enhances the regression of RA (37). Omega-3 PUFAs and omega-6 PUFAs play crucial roles in immunomodulation (immune regulation) by producing eicosanoids from precursor molecules. Omega-3 fatty acids which are present mainly in fish oils, are inversely correlated with IL-6, matrix metallopeptidase 3 and CRP levels (5). Previous studies have revealed that olive oil, which contains oleic acid may be useful as a therapeutic strategy for treating RA and that a long-term consumption of omega-3 PUFAs, especially in combination with γ-linolenic acids may lower the risk of developing RA. Notably, a lower ratio of omega-6 to omega-3 PUFAs alleviates pain and lowers disease activity in RA patients (37-40).

Dietary fiber

Randomized clinical trials has demonstrated that the consumption of fiber rich food significantly improved disease activity scores, morning stiffness, joint pain, and reductions in inflammatory biomarkers in RA patients (41, 42). Dietary fibers are metabolized in the large intestine because of their ability to resist digestive enzymes in the small intestine. They are fermented by the gut microbiota in the colon and cecum to form short-chain fatty acids which have a beneficial effect on the cells of the colon (colonocytes). This induces several health promoting effects and reduces the risk of proinflammatory activities. Increased consumption of fiber-rich diets provides a sufficient energy source for colonocytes, reducing the risk of foreign agents invading the blood and stimulating an inflammatory response. It has also been shown to be negatively correlated with CRP, IL-6, TNF-α, and other mediators of inflammation (5, 32).

Antioxidant-rich fruits and vegetables

Fruits (such as berries, citrus and apples) and vegetables (such as leafy greens, carrots, and broccoli) are rich in antioxidants. These antioxidants include vitamins (A, C, & E), polyphenol, flavonoids and carotenoids, which counteract the effect of circulating free radicals on cell membranes during inflammation (43). Polyphenols also have the ability to delay the onset of RA and reduce joint bone erosion and destruction by inhibiting the formation of TNF-induced osteoclasts (35). Flavonoids which are classified into flavones, flavanols, flavanones, flavanonols, anthocyanidins and chalcones help improve RA symptoms by targeting multiple receptors involved in immunoregulation, gut-joint axis modulation and inflammatory activity inhibition (31).

Foods to limit: proinflammatory diets

While anti-inflammatory dietary patterns such as the Mediterranean diet have consistently been associated with improved outcomes in RA, the role of certain foods and dietary components in promoting inflammation and disease progression remains a subject of ongoing debate. Emerging evidence suggests that Western-style diets, characterized by high energy intake, excessive saturated and trans fats, an imbalanced omega-6 to omega-3 fatty acid ratio, and high levels of refined sugars and processed foods may contribute to systemic inflammation. These diets are also linked to obesity and insulin resistance, which are known risk factors for RA development and severity (44, 45). However, the direct impact of specific food items such as red meat, dairy products, eggs, or total protein intake on RA activity is not fully established, and findings across studies are often inconsistent (46, 47). Some earlier research raised concerns about high protein and red meat consumption increasing the risk of inflammatory polyarthritis (46), while more recent reviews have not found definitive evidence supporting such associations (47). Therefore, while it may be prudent for individuals with RA to moderate their intake of foods commonly associated with proinflammatory responses, such as saturated fats, ultra-processed foods, and sugar-sweetened beverages, further high-quality, long-term studies are needed to clarify these relationships and inform precise dietary guidelines.

Emerging nutritional strategies

Recent advancements in nutritional science offer innovative approaches to managing RA, particularly among elderly populations. Caloric restriction (CR) and intermittent fasting (IF) have emerged as promising strategies with the potential to modulate inflammatory processes. Studies suggest that CR reduces the levels of proinflammatory cytokines such as IL-6 and TNF-α, while simultaneously increasing the production of anti-inflammatory mediators, such as adiponectin (48-50). Intermittent fasting, on the other hand, is thought to influence immune cell metabolism, promoting autophagy and reducing oxidative stress (48). These interventions may also mitigate obesity-related inflammation, a common comorbidity in RA patients, providing dual benefits in disease management.

Another frontier in RA management involves personalized nutrition approaches, tailored to an individual's genetic and microbiome profiles. Advances in nutrigenomics have revealed that specific genetic polymorphisms can influence inflammatory responses to dietary components. Individuals with variations in genes associated with omega-3 metabolism may require higher intake levels to achieve anti-inflammatory effects (51). Similarly, the gut microbiome plays a pivotal role in RA pathogenesis, with dysbiosis linked to increased

[DOI: 10.18502/ehj.v11i1.19003]

inflammation and autoimmunity (35). Personalized diets, informed by gut microbiota analysis, aim to restore microbial balance through targeted prebiotics, probiotics, and fiber-rich foods (52). Personalized nutrition is an emerging approach that tailors dietary recommendations to an individual's genetic profile, metabolic status, and health conditions to improve health outcomes. It has been successfully applied in various diseases; for instance, individuals with FTO or MC4R gene variants benefit from tailored fat intake and exercise to reduce obesity risk, while those with MTHFR mutations may require higher folate intake to lower cardiovascular risk (53). In diabetes, genes like TCF7L2 guide carbohydrate-focused diets for better glycemic control (54). Given the complexity of RA, especially in the elderly, personalized nutrition holds potential to modulate inflammation, optimize nutrient intake, and address comorbidities more effectively than generalized diets. This tailored approach holds promise for optimizing therapeutic outcomes, especially for elderly RA patients with unique metabolic and inflammatory profiles.

The incorporation of these cutting-edge strategies into routine clinical practice requires further research to establish their long-term efficacy and feasibility. However, their potential to enhance traditional dietary interventions and improve patient outcomes positions them as valuable components of a comprehensive RA management plan.

Challenges and considerations in nutritional management

Despite the fact that diet modification has been identified as a feasible measure for mitigating the adverse inflammatory effects of RA, nutritional management in the older population presents a myriad of challenges (55). This is because they often struggle with age-related physiological changes, disease comorbidities, nonspecific pathophysiological processes in RA and other social and economic factors. These factors increase the incidence of malnutrition, making it difficult to manage RA effectively in this group of adults (56).

Physiological changes affecting nutrition

Studies have revealed several physiological changes that affect nutritional status in the geriatric population (57, 58). They vary from; loss of appetite which may be impacted by alterations in taste, smell, and reduced enjoyment of food; to gastrointestinal issues such as difficulty swallowing, decreased gastric acid production and slower intestinal transit time (40, 57, 58). All these abnormal changes lead to poor intake of essential nutrients, contributing to an increased prevalence of malnutrition affecting one in three elderly individuals with RA. A reduction in food intake also diminishes their access to anti-inflammatory diets which can ameliorate inflammatory symptoms.

Table 1. Summary of the contribution of diet to rheumatoid arthritis

Dietary Component	Food	Action	Mechanism
MUFA	Nuts, avocado, sesame, olive oil	Anti-inflammatory	Reduce ROS production and cytokine secretion
ω-3 PUFA	Fatty fish (e.g. sardine and tuna), berries, chia seeds and flaxseed oil	Anti-inflammatory	Inhibit proinflammatory cytokines
ω -6 PUFA	Vegetable oils, meat, eggs, dairy products	Pro-inflammatory	Lipid precursors which mediate pain and inflammation
Low	Balanced diet	Anti-inflammatory	Reduce oxidative stress and improve autoimmune symptoms
Dietary fiber	Whole grains, legumes	Anti-inflammatory	Forms short-chain fatty acids through fermentation by gut normal flora and improves the immune response
Fruits/Vegetables	Berries, grapes, carrot, Green leafy vegetables, homemade green juice	Anti-inflammatory	Release antioxidants that neutralize toxic free radicals
Proteins	Unprocessed and processed red meat	Pro-inflammatory	Contains high levels of choline, which is the precursor of the inflammatory metabolite TMAO
Saturated fats	Butter, processed foods, Lard	Pro-inflammatory	Intensifies oxidative stress levels
Trans fats	Fried foods, Baked goods	Pro-inflammatory	Increases level of pro-inflammatory markers (TNF-α and IL-1) and contribute to vascular endothelial dysfunction
Dairy products	Milk, Cheese	Pro-inflammatory	Result in the production of proinflammatory metabolite

MUFA- Monounsaturated fatty acid; PUFA- Polyunsaturated fatty acid; ROS- Reactive Oxygen Species; TMAO- Trimethylamine N-oxide; TNF-α - Tumor necrosis factor alpha; IL- Interleukin

[DOI: 10.18502/ehj.v11i1.19003]

Dietary dilemmas

Many older adults with RA also suffer from multiple comorbid conditions such as cardiovascular diseases, diabetes, and osteoporosis (59). These comorbidities can complicate dietary recommendations. For example, a diet that is anti-inflammatory may not align with the dietary restrictions needed for managing diabetes or heart disease (60). A recent study revealed that hypoalbuminemia, a condition characterized by low albumin levels in the blood, has been noted in older adults with RA and can indicate inadequate protein intake (20). While high-protein diets are often viewed as proinflammatory, sufficient protein is crucial for maintaining muscle mass, and improving quality of life (16).

Medication interactions and side effects

The conventional and most common treatment for RA involves the use of pharmacological agents. Nonsteroidal anti-inflammatory drugs, corticosteroids, and disease-modifying antirheumatic drugs are used to manage the clinical features of this disease. However, these drugs can result in significant side effects that can potentially impact nutrient intake and absorption. NSAIDs are known to cause gastrointestinal irritations, which can lead to discomfort and reduced appetite whereas corticosteroids may lead to weight gain and altered food metabolism (61). Additionally, there are chances of drug-food interactions if older adults are not properly guided and monitored. Such interactions can affect the absorption of food nutrients and alter their efficacy in reducing inflammatory activities (62).

Limitations in nutritional screening and assessment

There is a gap in nutritional screening methods for RA, which has prevented efficient and early diagnosis of malnutrition in this population. While tools such as the Controlled Nutritional Status Score, Mini Nutritional Assessment and Geriatric Nutritional Risk Index are valuable, evidence suggests that they may not capture all cases of malnutrition (63-65). Olsen et al. (63) reported that none of the RA patients identified as malnourished on the basis of body composition metrics were flagged by the Nutritional Risk Screening 2002 (NRS2002). The lack of highly sensitive, specific, and standardized protocols for routine nutritional screening can result in the underdiagnosis of issues such as sarcopenia and malnutrition, further complicating the management of RA. Similarly, multiple studies have indicated that body mass index is not a reliable tool for determining malnutrition (64, 65).

Social and economic barriers

The level of income significantly affects nutrition, especially among elderly individuals who often rely on fixed pensions or retirement benefits. Limited financial resources can reduce access to high-quality, anti-inflammatory foods. Food insecurity remains a common barrier to following recommended dietary patterns in RA management (66). Beyond economic limitations, cultural and social factors also play a major role. Long-standing eating habits, family upbringing, shared household food preferences, and resistance to altering traditional diets can hinder adherence to newer dietary models like the Mediterranean diet. Older adults may find it impractical to adopt unfamiliar diets due to lack of cooking skills, time, equipment, or perceived inconvenience in preparing fresh meals. Cultural food norms, such as a preference for red meat or warm meals in colder climates, can further deter dietary changes. Social isolation also contributes, with many elderly individuals living alone and relying on convenience foods (67, 68). Conversely, strong family or community support and increased access to culturally adapted healthy foods may facilitate better adherence and improve RA outcomes.

Awareness and education gaps

The effectiveness of nutritional interventions in managing RA is hindered by limited awareness and understanding of older adults. Cognitive impairments may also complicate this challenge (68). Insufficient knowledge on the roles of dietary choices poses a threat to clinical symptoms and disease progression.

Future directions

There is a significant population gap in the study of nutrition among patients with RA. There is a need for more longitudinal studies that target elderly onset RA patients, given the unique inflammatory and nutritional needs of this group. Age-stratified analyses can also help to develop nutritional recommendations tailored to specific age groups. Additionally, since factors such as lifestyle, dietary habits, and access to healthcare vary across countries, cross-cultural studies are needed to explore how these factors influence nutritional management in elderly RA patients. These studies could compare RA outcomes in elderly populations from diverse geographical regions. This would offer insight into the role of local diets and healthcare systems in managing RA.

Although nutritional interventions such as the Mediterranean diet, omega-3 fatty acid supplementation, and fiber-rich diets have shown promising outcomes in RA management, most studies do not focus on their specific impact on elderly RA patients. Comparative studies are needed to evaluate the relative efficacy of different dietary patterns (e.g., plant-based diets vs. Mediterranean diets) in managing RA. This could help identify which specific diets or food groups provide the most anti-inflammatory benefits for elderly individuals. It is also imperative that future research explore personalized nutritional interventions based on individual biomarkers such as serum albumin, total protein, or antioxidant levels. Personalized nutrition could enhance RA management by addressing the unique nutritional deficiencies of elderly patients with RA.

The methodologies used in existing research often have proven to be poorly definitive of nutritional status owing to the low specificity of the tools used. Additionally, studies often overlook malnutrition as a complicating factor in elderly RA patients. Research should use more rigorous and standardized tools to assess nutritional status in elderly RA patients, such as the Comprehensive

[DOI: 10.18502/ehj.v11i1.19003]

Geriatric Assessment (CGA) (69) or the Prognostic Inflammatory and Nutritional Index (PINI) (19). Standardization would ensure better comparison of results across studies. Nutritional biomarkers (e.g., serum albumin, prealbumin, and vitamins) that could be predictive of RA disease activity and response to nutritional interventions should also be investigated. This can help develop early detection and prevention strategies.

The implementation of these strategies would sufficiently help to better understand the role of nutrition and diet in the progression and treatment of RA. This evidence will be crucial for developing more precise, personalized dietary recommendations that cater to the complex needs of elderly individuals living with RA, potentially transforming both preventative care and disease management strategies.

Conclusion

The interplay between nutrition and rheumatoid arthritis management in older adults presents a significant opportunity for improving health outcomes. The prevalence of malnutrition in this population exacerbates the challenges of RA, leading to increased inflammation and diminished quality of life. The implementation of dietary interventions, particularly those focused on anti-inflammatory diets such as the Mediterranean and plant-based approaches, can effectively address these issues. However, practical barriers including food insecurity and limited nutritional knowledge must be overcome to maximize the benefits of these dietary strategies. Future efforts should focus on personalized nutrition plans that consider individual needs and circumstances, alongside enhanced assessment tools to better identify nutritional deficiencies. By integrating comprehensive nutritional strategies into RA management, healthcare providers can significantly enhance the overall well-being of older adults living with this chronic condition.

Ethical consideration

Not applicable

Conflict of interests

The authors declare that they have no competing interests

Acknowledgments

None

Funding

There was no funding for this study

Authors' contributions

A.Y.O Conceptualized the idea; A.Y.O and A.A.A wrote, edited, formatted, and reviewed the manuscript; All authors read and approved the final manuscript.

References

1. Kronzer VL, Davis JM. Etiologies of rheumatoid arthritis: update on mucosal, genetic, and cellular pathogenesis. Current Rheumatology Reports. 2021; 23(4): 1-10.
2. Black RJ, Cross M, Haile LM, Culbreth GT, Steinmetz JD, Hagins H, et al. Global, regional, and national burden of rheumatoid arthritis, 1990–2020, and projections to 2050: a systematic analysis of the Global Burden of Disease Study 2021. The Lancet Rheumatology. 2023; 5(10): 594-610.
3. Cano-García L, Redondo-Rodríguez R, Manrique-Arija S, Domínguez-Quesada C, Crisóstomo Vacas J, Armenteros-Ortiz P, et al. Prevalence of malnutrition and associated factors in older patients with rheumatoid arthritis: A cross-sectional study. Nutrients. 2023; 15(16): 1-17.
4. Kobak S, Bes C. An autumn tale: geriatric rheumatoid arthritis. Therapeutic Advances in Musculoskeletal Disease. 2018; 10(1): 3-11.
5. Gioia C, Lucchino B, Tarsitano MG, Iannuccelli C, Di Franco M. Dietary habits and nutrition in rheumatoid arthritis: can diet influence disease development and clinical manifestations?. Nutrients. 2020; 12(5): 1-25.
6. Bostan ZZ, Şare Bulut M, Özen Ünaldı B, Albayrak Buhurcu C, Akbulut G. Effect of plant-based diets on rheumatoid arthritis: a systematic review. Nutrition Reviews. 2024: 181.
7. Porras M, Rada G, Durán J. Effects of mediterranean diet on the treatment of rheumatoid arthritis. Medwave. 2019; 19(5): 1-7.
8. Scherer HU, Häupl T, Burmester GR. The etiology of rheumatoid arthritis. Journal of Autoimmunity. 2020; 110: 1-15.
9. Araki Y, Mimura T. The mechanisms underlying chronic inflammation in rheumatoid arthritis from the perspective of the epigenetic landscape. Journal of Immunology Research. 2016; 2016(1): 1-10.
10. Mitrović J, Hrkač S, Tečer J, Golob M, Ljilja Posavec A, Kolar Mitrović H, et al. Pathogenesis of extraarticular manifestations in rheumatoid arthritis—A comprehensive review. Biomedicines. 2023; 11(5): 1-29.
11. Yang X, Chang Y, Wei W. Endothelial dysfunction and inflammation: immunity in rheumatoid arthritis. Mediators of Inflammation. 2016; 2016(1): 1-9.
12. Chen J, Norling LV, Cooper D. Cardiac dysfunction in rheumatoid arthritis: the role of inflammation. Cells. 2021; 10(4): 1-21.
13. Pavlov-Dolijanovic S, Bogojevic M, Nozica-Radulovic T, Radunovic G, Mujovic N. Elderly-onset rheumatoid arthritis: characteristics and treatment options. Medicina. 2023; 59(10): 1-21.
14. Bauer ME. Accelerated immunosenescence in rheumatoid arthritis: impact on clinical progression. Immunity & Ageing. 2020; 17(1): 1-14.
15. Weyand CM, Goronzy JJ. Immune aging and rheumatoid arthritis. Arthritis & Rheumatology. 2025; 36(2): 297.

[DOI: 10.18502/ehj.v11i1.19003]

16. Tański W, Wójciga J, Jankowska-Polańska B. Association between malnutrition and quality of life in elderly patients with rheumatoid arthritis. Nutrients. 2021; 13(4): 1-14.
17. Ardhaoui M, Mahbouba J, Naffati F, Fhima F, Ghali M, Zrour S, et al. AB0319 Prealbumin serum level as a useful tool in the assessment of malnutrition in elderly patients with rheumatoid arthritis. Annals of the Rheumatic Diseases. 2022; 81(1): 1284.
18. Niksolat F, Zandieh Z, Roshani F, Larijani SS, Mirfakhraee H, Bahadori F, et al. Geriatric syndromes among patients with rheumatoid arthritis: a comparison between young and elderly patients. Ethiopian Journal of Health Sciences. 2022; 32(4): 791-8.
19. Ardhaoui M, Mahbouba J, Zrour S, Bejia I, Touzi M, Bergaoui N. POS0479 The prognostic inflammatory and nutritional index (PINI) in elderly patients with rheumatoid arthritis. Annals of the Rheumatic Diseases. 2023; 82(1): 500.
20. Oseni B, Adedeji Y. Rheumatoid arthritis: peripheral blood cells and plasma protein characteristics in geriatric patients. Adeleke University Journal of Science. 2024; 3(1): 59-66.
21. Sun Z, Shao H, Liu H, Ma L. Anemia in elderly rheumatoid arthritis patients: a cohort study. Archives of Medical Science: AMS. 2024; 20(2): 45-63.
22. Wunderle C, Stumpf F, Schuetz P. Inflammation and response to nutrition interventions. Journal of Parenteral and Enteral Nutrition. 2024; 48(1): 27-36.
23. Massironi S, Viganò C, Palermo A, Pirola L, Mulinacci G, Allocca M, et al. Inflammation and malnutrition in inflammatory bowel disease. The Lancet Gastroenterology & Hepatology. 2023; 8(6): 579-90.
24. Ozer NT, Gundogan K. The vicious cycle of malnutrition and tuberculosis: a narrative review. Journal of Clinical Practice & Research. 2024; 46(5): 444-8.
25. Torii M, Itaya T, Minamino H, Katsushima M, Fujita Y, Tanaka H, et al. Management of sarcopenia in patients with rheumatoid arthritis. Modern Rheumatology. 2023; 33(3): 435-40.
26. Shannon OM, Ashor AW, Scialo F, Saretzki G, Martin-Ruiz C, Lara J, et al. Mediterranean diet and the hallmarks of ageing. European Journal of Clinical Nutrition. 2021; 75(8): 1176-92.
27. Bustamante MF, Agustín-Perez M, Cedola F, Coras R, Narasimhan R, Golshan S, et al. Design of an anti-inflammatory diet (ITIS diet) for patients with rheumatoid arthritis. Contemporary Clinical Trials Communications. 2020; 17: 1-7.
28. Rekik S, Nouicer S, Boussaid S, Rahmouni S, Zouaoui K, Sahli H, et al. AB0252 impact of adherence to the mediterranean diet on disease activity in patients with rheumatoid arthritis. Annals of the Rheumatic Diseases. 2023; 82(1): 1309-10.
29. Alawadhi B, Alsaber A, Shatawan I, Al-Herz A, Setiya P, Saleh K, et al. POS0991-HPR adherence to the mediterranean diet is associated with a reduced das-28 index among patients with rheumatoid arthritis. Annals of the Rheumatic Diseases. 2023; 82(1): 812.
30. Sales C, Oliviero F, Spinella P. The mediterranean diet model in inflammatory rheumatic diseases. Reumatismo. 2009; 61(1): 10-4.
31. Miao Z, Zhao Y, Chen M, He C. Using flavonoids as a therapeutic intervention against rheumatoid arthritis: The known and unknown. Pharmacological Research-Modern Chinese Medicine. 2022; 3: 100014.
32. Alwarith J, Kahleova H, Rembert E, Yonas W, Dort S, Calcagno M, et al. Nutrition interventions in rheumatoid arthritis: the potential use of plant-based diets. A review. Frontiers in Nutrition. 2019; 6: 1-11.
33. Schönenberger KA, Schüpfer AC, Gloy VL, Hasler P, Stanga Z, Kaegi-Braun N, et al. Effect of anti-inflammatory diets on pain in rheumatoid arthritis: a systematic review and meta-analysis. Nutrients. 2021; 13(12): 1-19.
34. Nikiphorou E, Philippou E. Nutrition and its role in prevention and management of rheumatoid arthritis. Autoimmunity reviews. 2023; 22(7): 103333.
35. Skoczyńska M, Świerkot J. The role of diet in rheumatoid arthritis. Reumatologia/Rheumatology. 2018; 56(4): 259-67.
36. Matsumoto Y, Sugioka Y, Tada M, Okano T, Mamoto K, Inui K, et al. Monounsaturated fatty acids might be key factors in the Mediterranean diet that suppress rheumatoid arthritis disease activity: The TOMORROW study. Clinical Nutrition. 2018; 37(2): 675-80.
37. Tański W, Świątoniowska-Lonc N, Tabin M, Jankowska-Polańska B. The relationship between fatty acids and the development, course and treatment of rheumatoid arthritis. Nutrients. 2022; 14(5): 1-30.
38. Navarini L, Afeltra A, Gallo Afflitto G, Margiotta DP. Polyunsaturated fatty acids: any role in rheumatoid arthritis?. Lipids in Health and Disease. 2017; 16: 1-5.
39. Di Giuseppe D, Wallin A, Bottai M, Askling J, Wolk A. Long-term intake of dietary long-chain n-3 polyunsaturated fatty acids and risk of rheumatoid arthritis: a prospective cohort study of women. Annals of the Rheumatic Diseases. 2014; 73(11): 1949-53.
40. Veselinovic M, Vasiljevic D, Vucic V, Arsic A, Petrovic S, Tomic-Lucic A, et al. Clinical benefits of n-3 PUFA and ɤ-linolenic acid in patients with rheumatoid arthritis. Nutrients. 2017; 9(4): 1-11.
41. Dürholz K, Hofmann J, Iljazovic A, Häger J, Lucas S, Sarter K, et al. Dietary short-term fiber interventions in arthritis patients increase systemic SCFA levels and regulate inflammation. Nutrients. 2020; 12(10): 1-11.
42. Latif FA, Ghazali WS, Mohamad SM, Lee LK. High fiber multigrain supplementation improved disease activity score, circulating inflammatory and oxidative stress biomarkers in rheumatoid arthritis (RA) patients: a randomized human clinical trial. Journal of Functional Foods. 2023; 100: 1-9.
43. Roy A, Das S, Chatterjee I, Roy S, Chakraborty R. Anti-inflammatory effects of different dietary antioxidants. In: Ekiert HM, Ramawat KG, Arora J, editors. Plant Antioxidants and Health. Cham: Springer International Publishing; 2022. p 573-97.
44. Alum EU, Ugwu OP. Nutritional strategies for rheumatoid arthritis: exploring pathways to better

[DOI: 10.18502/ehj.v11i1.19003]

management. INOSR Scientific Research. 2023; 10(1): 18-26.
45. Philippou E, Nikiphorou E. Are we really what we eat? Nutrition and its role in the onset of rheumatoid arthritis. Autoimmunity Reviews. 2018; 17(11): 1074-7.
46. Pattison DJ, Symmons DP, Lunt M, Welch A, Luben R, Bingham SA, et al. Dietary risk factors for the development of inflammatory polyarthritis: evidence for a role of high level of red meat consumption. Arthritis & Rheumatism. 2004; 50(12): 3804-12.
47. Asoudeh F, Jayedi A, Kavian Z, Ebrahimi-Mousavi S, Nielsen SM, Mohammadi H. A systematic review and meta-analysis of observational studies on the association between animal protein sources and risk of rheumatoid arthritis. Clinical Nutrition. 2021; 40(7): 4644-52.
48. Surugiu R, Iancu MA, Vintilescu ȘB, Stepan MD, Burdusel D, Genunche-Dumitrescu AV, et al. Molecular mechanisms of healthy aging: the role of caloric restriction, intermittent fasting, Mediterranean diet, and ketogenic diet—a scoping review. Nutrients. 2024; 16(17): 1-17.
49. Dogan S, Ray A, Cleary MP. The influence of different calorie restriction protocols on serum pro-inflammatory cytokines, adipokines and IGF-I levels in female C57BL6 mice: short term and long term diet effects. Meta Gene. 2017; 12: 22-32.
50. Chen Y, Ling L, Su G, Han M, Fan X, Xun P, et al. Effect of intermittent versus chronic calorie restriction on tumor incidence: a systematic review and meta-analysis of animal studies. Scientific Reports. 2016; 6(1): 1-11.
51. Simopoulos AP. Genetic variants in the metabolism of omega-6 and omega-3 fatty acids: their role in the determination of nutritional requirements and chronic disease risk. Experimental Biology and Medicine. 2010; 235(7): 785-95.
52. Fan S, Zhang Z, Zhao Y, Daglia M, Zhang J, Zhu Y, et al. Recent advances in targeted manipulation of the gut microbiome by prebiotics: From taxonomic composition to metabolic function. Current Opinion in Food Science. 2023; 49: 1-22.
53. Aoun C, Hajj A, Hajj F, Papazian T, Khabbaz LR. The interaction between genetic polymorphisms in FTO, MC4R and MTHFR genes and adherence to the Mediterranean Diet in relation to obesity. Gene. 2022; 809: 146037.
54. Singar S, Nagpal R, Arjmandi BH, Akhavan NS. Personalized nutrition: tailoring dietary recommendations through genetic insights. Nutrients. 2024; 16(16): 1-18.
55. van Onna M, Boonen A. Challenges in the management of older patients with inflammatory rheumatic diseases. Nature Reviews Rheumatology. 2022; 18(6): 326-34.
56. Ohta R, Sano C. Integrating clinical and socio-environmental approaches in managing rheumatoid arthritis with social determinants of health: a case study of an elderly patient in rural Japan. Cureus; 15(12): 1-8.
57. Jain M, Khushboo G. Physiological determinants of malnutrition in elderly. Novel Techniques in Nutrition and Food Science. 2018; 2: 1-4.
58. Leslie W, Hankey C. Aging, nutritional status and health. Healthcare. 2015; 3(3): 648-58.
59. Corrao S, Calvo L, Giardina A, Cangemi I, Falcone F, Argano C. Rheumatoid arthritis, cardiometabolic comorbidities, and related conditions: need to take action. Frontiers in Medicine. 2024; 11: 1-8.
60. Nicolau J, Lequerré T, Bacquet H, Vittecoq O. Rheumatoid arthritis, insulin resistance, and diabetes. Joint Bone Spine. 2017; 84(4): 411-6.
61. Sohail R, Mathew M, Patel KK, Reddy SA, Haider Z, Naria M, et al. Effects of non-steroidal anti-inflammatory drugs (NSAIDs) and gastroprotective NSAIDs on the gastrointestinal tract: a narrative review. Cureus. 2023; 15(4): 1-14.
62. Ased S, Wells J, Morrow LE, Malesker MA. Clinically significant food-drug interactions. The Consultant Pharmacist. 2018; 33(11): 649-57.
63. Olsen MN, Tangvik RJ, Halse AK. Evaluation of nutritional status and methods to identify nutritional risk in rheumatoid arthritis and spondyloarthritis. Nutrients. 2020; 12(11): 1-12.
64. Elkan AC, Engvall IL, Cederholm T, Hafström I. Rheumatoid cachexia, central obesity and malnutrition in patients with low-active rheumatoid arthritis: feasibility of anthropometry, Mini Nutritional Assessment and body composition techniques. European Journal of Nutrition. 2009; 48(5): 315-22.
65. Elkan AC, Engvall IL, Tengstrand B, Cederholm T, Hafström I. Malnutrition in women with rheumatoid arthritis is not revealed by clinical anthropometrical measurements or nutritional evaluation tools. European Journal of Clinical Nutrition. 2008; 62(10): 1239-47.
66. Gkiouras K, Cheristanidis S, Papailia TD, Grammatikopoulou MG, Karamitsios N, Goulis DG, et al. Malnutrition and food insecurity might pose a double burden for older adults. Nutrients. 2020; 12(8): 1-11.
67. Tsofliou F, Vlachos D, Hughes C, Appleton KM. Barriers and facilitators associated with the adoption of and adherence to a Mediterranean style diet in adults: a systematic review of published observational and qualitative studies. Nutrients. 2022; 14(20): 1-13.
68. Charlton K, Walton K, do Rosario V. Improving nutrition in older adults. In: Gu D, Dupre ME, editors. Encyclopedia of Gerontology and Population Aging. Switzerland, AG: Springer Nature; 2019. p1-7.
69. Zhang M, Feng M, Lai B. The comprehensive geriatric assessment of an older adult with rheumatoid arthritis. Rheumatology & Autoimmunity. 2022; 2(02): 102-4.

[DOI: 10.18502/ehj.v11i1.19003]