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REVIEW

Managing Cardiovascular Risk in Patients with Autoimmune Diseases: Insights from a Nutritional Perspective

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Abstract

Purpose of Review Autoimmune diseases manifest as an immune system response directed against endogenous antigens, exerting a significant influence on a substantial portion of the population. Notably, a leading contributor to morbidity and mortality in this context is cardiovascular disease (CVD). Intriguingly, individuals with autoimmune disorders exhibit a heightened prevalence of CVD compared to the general population. The meticulous management of CV risk factors assumes paramount importance, given the current absence of a standardized solution to this perplexity. This review endeavors to address this challenge from a nutritional perspective.

Recent Findings Emerging evidence suggests that inflammation, a common thread in autoimmune diseases, also plays a pivotal role in the pathogenesis of CVD. Nutritional interventions aimed at reducing inflammation have shown promise in mitigating cardiovascular risk.

Summary The integration of nutritional strategies into the management plans for patients with autoimmune diseases offers a holistic approach to reducing cardiovascular risk. While conventional pharmacological treatments remain foundational, the addition of targeted dietary interventions can provide a complementary pathway to improve cardiovascular outcomes.

Keywords Autoimmune Diseases · Cardiovascular Disease · Risk Factors · Diet · Nutritional Components

Introduction

The human immune system constitutes a sophisticated network of diverse cells expressing a broad spectrum of receptors, synergistically collaborating to mount responses to infections and uphold metabolic well-being. Autoimmune diseases manifest when this intricate system undergoes misdirection, targeting the body's own tissues. Conditions such as rheumatoid arthritis (RA), systemic lupus erythematosus (SLE), antiphospholipid syndrome (APS), systemic sclerosis (SS), and systemic vasculitis are among the disorders encompassed by this aberrant immune response. Approximately 5–8% of the global population grapples with autoimmune diseases, imposing considerable suffering on afflicted individuals and posing a significant socioeconomic challenge on a global scale [1]. Notably, cardiovascular diseases (CVD) are markedly prevalent among individuals with autoimmune disorders and emerge as the principal contributors to increased morbidity and mortality in these patients [2, 3], it is critical to control the CV risk in autoimmune diseases.

A multifaceted interplay of traditional CV risk factors including obesity, dyslipidemia, smoking, insulin resistance, metabolic syndrome, and hypertension and non-traditional risk factors such as chronic systemic inflammation, oxidative stress and the use of therapeutic drugs, is implicated in the heightened prevalence of CVD among patients with autoimmune diseases [4]. In the realm of CV risk management, lifestyle, particularly diet, stands out as an indispensable factor. Significantly, the 2016 European League Against Rheumatism (EULAR), which provides guidance on managing cardiovascular disease risk for individuals with immune system disorders, recognized the pivotal role of a healthy diet. Nevertheless, the guidelines did not furnish

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explicit and detailed dietary recommendations, creating an opportunity for additional investigation and refinement in this critical dimension of cardiovascular risk mitigation for patients grappling with autoimmune diseases [5].

Within this review, we have elucidated the impact of diet on CV risk associated with autoimmune diseases and provided a detailed overview of potential nutritional strategies aimed at mitigating this risk. Furthermore, we have introduced a spectrum of beneficial nutrients implicated in the modulation of these conditions.

Epidemiology of CVD in Autoimmune Disease

Autoimmune diseases are characterized by a notably elevated prevalence of CVD, which stands as the foremost contributor to morbidity and mortality within this patient population [2, 3]. Numerous observational studies conducted on cohorts of RA patients with varying characteristics have reported a broad spectrum of incidence rates for CV events and mortality. CV mortality constitutes 40–50% of all fatalities in individuals with RA [6]. The mortality rate in patients with SLE is three times higher than that in the general population, primarily due to premature coronary atherosclerosis. Autopsy result have revealed the presence of coronary atherosclerosis in up to 40% of SLE patients [7]. Mortality in SLE patients presents a bimodal pattern, with an initial peak owing to clinical disease activity and a late peak attributable to the development of atherosclerosis and CVD. A cohort study involving 865 individuals revealed that patients with SS experienced a higher incidence of myocardial infarction and stroke, with rates of 4.4 and 4.8 per 1000 person-years, respectively, in contrast to 2.5 per 1000 person-years for both myocardial infarction and stroke in a matched healthy control group. Furthermore, upon adjustment for covariates, the corresponding Hazard Ratios were calculated as 1.80 (95% CI: 1.07–3.05) for myocardial infarction and 2.61 (95% CI: 1.54–4.44) for stroke [8]. Moreover, CVD tends to manifest at a younger age compared to the general population, frequently remaining asymptomatic, especially in its early stages.

CV Risk Factors in Autoimmune Disease

Traditional Risk Factors in Autoimmune Disease

Obesity

Obesity is a chronic inflammatory condition often associated with CVD both in the general population and among individuals with autoimmune diseases [9]. Substantial epidemiological data demonstrates a robust correlation between excess body weight or obesity and the susceptibility to autoimmune diseases [10]. The prevalence of obesity in many autoimmune diseases is slightly higher than in the general population [11]. The underlying pathophysiology involves a complex interplay between adiposity-induced systemic inflammation and autoimmune-mediated processes. In obesity, the expansion of adipose tissue leads to a dysregulated secretion of adipokines and pro-inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6). These inflammatory mediators contribute to endothelial dysfunction, atherosclerosis, and other CV complications [12]. This synergistic effect of obesity-related inflammation and autoimmune pathology accentuates the risk of developing CVD. Furthermore, obesity can exacerbate the inflammatory milieu in autoimmune conditions, thereby potentiating CV risk [13].

Hypertension

Hypertension is a contributing factor to subclinical atherosclerosis and CV events in autoimmune disease [14]. Several studies have provided evidence that the prevalence of hypertension is higher in patients with autoimmune diseases than in the general population [15]. For example, a large population-based study reported a 31% prevalence of hypertension in patients with RA, compared to 23% in the general population [16]. Similarly, various case-control studies, including those conducted on large cohorts, consistently showed a significantly higher prevalence (30–38%) of hypertension in patients with primary Sjögren's syndrome [17–19]. Furthermore, a meta-analysis encompassing 32 studies and 17,187 patients with SLE found that 42.6% of these patients had high blood pressure [20]. Additional research indicated that the prevalence of hypertension can be as high as 40% in SLE patients under 40 years of age [21, 22]. Immune-mediated mechanisms and chronic inflammation have been demonstrated to have a relevant role in the pathogenesis of hypertension [23]. Additionally, certain medications used in the management of autoimmune diseases, such as corticosteroids, can induce or exacerbate hypertension, complicating the clinical scenario [24].

Dyslipidemia

In individuals with autoimmune diseases, the chronic systemic inflammation disrupts normal lipid metabolism. Pro-inflammatory cytokines, especially IL-6 and TNF-α, are elevated in autoimmune conditions and play a pivotal role in altering lipid profiles. IL-6, for instance, has been shown to increase hepatic synthesis of triglycerides and very

low-density lipoprotein (VLDL), leading to hypertriglyceridemia. It can also inhibit lipoprotein lipase (LPL) activity, reducing the clearance of triglyceride-rich lipoproteins [25]. TNF-α, on the other hand, influences the expression of apolipoprotein B, which is essential for the assembly and secretion of VLDL and LDL particles.

Insulin Resistance

The mechanistic nexus between insulin resistance and CV risk is multifactorial and interlinked with the inflammatory milieu characteristic of autoimmune disorders. Insulin resistance may precipitate both microangiopathy and macroangiopathy, contributing to peripheral arterial dysfunction. This state can impede blood flow and elevate blood pressure. Furthermore, it may disrupt the functional integrity of cardiomyocytes and endothelial cells [26]. In the context of autoimmune diseases, chronic systemic inflammation exacerbates insulin resistance [27]. The prevalence of insulin resistance in individuals with autoimmune diseases varies across different conditions and studies, several studies have shown a higher prevalence of insulin resistance in certain autoimmune diseases compared to the general population. A study by El-Magadmi et al. found that insulin resistance was present in 36.5% of SLE patients, significantly higher than in controls [28]. the prevalence of insulin resistance in RA patients was 41%, considerably higher than in matched controls [29]. In patients with psoriasis, a meta-analysis by Armstrong et al. showed that the prevalence of insulin resistance was approximately 40.7% [30].

Smoking

Tobacco smoking is widely acknowledged as a modifiable risk factor for CVD, and its impact on individuals with autoimmune diseases is particularly deleterious. Smoking has a direct impact on endothelial function, reducing the bioavailability of nitric oxide, a key molecule in maintaining vascular homeostasis, and contributing to endothelial dysfunction, an early marker of cardiovascular pathology [31]. Smoking promotes the oxidative modification of lipoproteins, particularly LDL, enhancing their atherogenic potential [32]. In autoimmune diseases, smoking has been consistently linked to CVD and markers of subclinical atherosclerosis [33].

Sedentary Lifestyle

A sedentary lifestyle, characterized by prolonged periods of physical inactivity, constitutes a significant risk factor for the development of CVD, particularly in individuals with autoimmune diseases [34, 35]. Inactivity contributes to the dysfunction of the endothelium, a harbinger of atherosclerosis [36]. Furthermore, a sedentary lifestyle is associated with the development of traditional cardiovascular risk factors such as insulin resistance, hypertension, and dyslipidemia—all of which compound the CVD risk in autoimmune patients [37].

No-Traditional Risk Factors in Autoimmune Disease

Chronic Systemic Inflammation

In autoimmune diseases, persistent systemic inflammation is a key non-traditional factor that increases CV risk [38]. Pro-inflammatory cytokines such as TNF-α, IL-1, IL-6, and interferons(IFNs), which are chronically elevated in autoimmune conditions, play a direct role in endothelial dysfunction, a precursor to atherosclerosis [39]. This dysfunction results from the upregulation of adhesion mole cules, recruitment of inflammatory cells to the endothelium, and alteration of the endothelial response to vasodilatory stimuli [40]. Moreover, inflammation enhances the oxidative modification of LDL cholesterol, making it more atherogenic, and stimulates a pro-thrombotic state by increasing the expression of tissue factors on the endothelial surface, thus promoting thrombus formation and atherosclerotic plaque development [41].

Oxidative Stress

Oxidative stress, characterized by an imbalance between reactive oxygen species (ROS) production and antioxidant defenses, is a significant non-traditional factor contributing to CVD in autoimmune diseases. ROS, including superoxide anions, hydrogen peroxide, and hydroxyl radicals, inflict direct damage to the vascular endothelium, leading to endothelial dysfunction, a key early event in atherogenesis [42]. This dysfunction is marked by reduced nitric oxide availability, promoting vasoconstriction and platelet aggregation. Additionally, oxidative stress facilitates the oxidative modification of lipoproteins, such as LDL, enhancing their atherogenic potential [43]. Chronic inflammation inherent in autoimmune diseases exacerbates ROS production, further amplifying oxidative stress and accelerating cardiovascular pathology [44].

Autoimmunity and Autoantibodies

Autoimmunity contributes to cardiovascular disease through the activity of autoantibodies such as antiphospholipid antibodies (aPL), rheumatoid factor (RF), anti-citrullinated protein antibodies (ACPAs), and anti-nuclear antibodies (ANAs) [45]. These autoantibodies interact with endothelial cells, platelets, and coagulation factors, tilting

the hemostatic balance toward thrombosis [46]. Autoantibodies can also form circulating immune complexes, which deposit in the vascular endothelium, activate complement, and stimulate local inflammation, thereby contributing to the atherosclerotic process [47]. Additionally, antibodies against components such as oxidized LDL or cardiolipin can directly participate in the formation and progression of atherosclerotic plaques, fostering localized inflammation and lipid accumulation within the vascular wall [48]. Binding of autoantibodies to their targets often results in the activation of immune cells, particularly monocytes and macrophages. These activated cells release cytokines and chemokines, further exacerbating vascular inflammation and contributing to plaque formation and instability [49].

Treatment-related CV Effects

Pharmacotherapy for autoimmune diseases, while crucial for managing these conditions, can inadvertently elevate CVD risk, representing a significant non-traditional factor. Several classes of drugs commonly used in the treatment of autoimmune disorders, including glucocorticoids, non-steroidal anti-inflammatory drugs (NSAIDs), and certain immunosuppressive agents, have been implicated in this increased risk [50, 51]. Glucocorticoids can induce hypertension, a well-known risk factor for CVD, by increasing sodium reabsorption in the renal tubules and enhancing vascular sensitivity to catecholamines [52]. They also promote dyslipidemia by elevating levels of triglycerides and LDL cholesterol while reducing HDL cholesterol. Furthermore, glucocorticoids exacerbate insulin resistance and can precipitate diabetes mellitus, another key risk factor for CVD [53]. The cumulative impact of these metabolic derangements substantially increases the risk of atherosclerosis and cardiovascular events. NSAIDs inhibit the cyclooxygenase (COX) enzymes, which play a critical role in prostaglandin synthesis [54]. The inhibition of COX-2, in particular, leads to a reduction in prostacyclin, while sparing thromboxane [54]. This imbalance increases the risk of thrombotic events such as myocardial infarction and stroke. Additionally, NSAIDs can lead to fluid retention and exacerbate hypertension, further elevating CVD risk [55]. Methotrexate can interfere with folate metabolism and lead to elevated homocysteine, increasing the risk of atherosclerosis [56].

CV Risk Prediction

The assessment of CVD risk in autoimmune disease patients presents a challenge. We briefly summarize the tools of CV risk prediction for several typical autoimmune disease as shown in Table 1. CV risk prediction tools for most autoimmune diseases typically align with those used for the general population. However, it is evident that these tools, which do not consider disease-specific attributes, may introduce bias. Some predictive models do incorporate disease characteristics for adjustment, but their accuracy lacks sufficient

Table 1 CV risk prediction tools for autoimmune diseases

Disease	Tool	Description	Reference
RA	QRISK 2 risk score	RA is a factor to be taken into account The evidence about the accuracy and validity is scarce.	[57]
	Adapted SCORE	SCORE CVD risk prediction tool with a 1.5 multiplication factor.	[58]
SLE	FRS	Tends to underestimate CVD risk	[59]
	mFRS	FRS with a 2.0 multiplier significantly improved the measure's sensitivity from 0.13 to 0.31 A commendable level of specificity when identifying patients at moderate to high risk of coronary artery disease	[60]
	QRISK 3 risk score	SLE is a factor to be taken into account The validation studies pertaining specifically to SLE populations have not yet been undertaken	[61]
	SLE-specific risk score	Incorporates disease-related variables including the a SLE disease activity index, the presence of lupus anticoagulant, and low levels of C3 complement, in addition to conventional CV risk factors Higher estimated risks for CV events compared to ACC/AHA risk equation.	[62]
APS	aGAPSS	A clinical scoring system that incorporates the three major antiphospholipid antibodies, hypertension, and dyslipidemia, initially developed to predict thrombotic events Data regarding CV events were not separately documented	[63]
	Modified version of aGAPSS	Achieved by incorporating additional scoring elements for diabetes mellitus, smoking, and obesity This modification has shown improved discriminatory and predictive capabilities, further validation remains imperative	[64]
SS	Same as for the general population	No studies have explored the accuracy of CV risk prediction tools in patients with SS	[57]

CV: cardiovascular; RA: rheumatoid arthritis; QRISK: QRESEARCH risk; SCORE: coronary risk evaluation; CVD: cardiovascular diseases; FRS: Framingham Risk Score; mFRS: modified Framingham Risk Score; SLE: systemic lupus erythematosus; ACC/AHA: American Heart Association /American College of Cardiology; APS: antiphospholipid syndrome; aGAPSS: adjusted global APS score; SS: systemic sclerosis

clinical evidence. Therefore, the imminent challenge lies in the creation of precise and dependable CVD risk stratification tools tailored to individuals with autoimmune diseases. Addressing this unmet need warrants immediate attention in the near future.

Effects of Diet on CV Risk Associated with Autoimmune Diseases

Although we are now well aware of the significant impact of diet as a pivotal component of lifestyle on common CV factors, there are few direct studies on the effect of diet on CV risk factors related with autoimmune disease. In fact, it is evident that non-traditional CV risk factors are intricately associated with diseases, encompassing inflammatory responses, immune regulation, and more. As one of the important environmental factors, diet has been paid growing attention to its impact on autoimmune diseases. A 2017 review in the "American Journal of Autoimmunity" showed that western dietary patterns, characterized by high intake of red and processed meats, high-fat dairy products, refined grains, and sugars, are associated with a higher prevalence of autoimmune diseases like multiple sclerosis, rheumatoid arthritis, and inflammatory bowel diseases. This is contrasted with the mediterranean diet, known for its abundance of fruits, vegetables, whole grains, fish, and extra virgin olive oil, which has been associated with a reduced risk of these conditions [65]. A large-scale study involving over 100,000 participants revealed that those adhering closely to a mediterranean diet had a 21% lower risk of RA compared to those with lower adherence [66].

The complex mechanisms of diet's influence on disease are still being explored, with the most discussed being inflammatory responses, gut microbes, and molecular mimicry.

Inflammation and Oxidative Stress

Diet exerts a profound influence on cytokine production, which plays a crucial role in immune signaling. High consumption of saturated fats and refined sugars can augment the synthesis of pro-inflammatory cytokines, TNF-α, IL-1, and IL-6 [67]. These cytokines are central to the propagation of inflammatory responses and are directly implicated in autoimmune conditions like RA and SS. Dietary components can activate or inhibit various signaling pathways associated with inflammation [68]. For instance, the nuclear factor kappa-light-chain-enhancer of activated B cells (NF-κB) pathway, a key regulator of inflammation, can be activated by certain dietary fats and sugars, leading to the transcription of pro-inflammatory genes and exacerbating autoimmune pathology [69]. Conversely, omega-3 fatty acids found in fish and flaxseeds are known to inhibit this pathway [70]. Oxidative stress, is another mechanism through which diet impacts inflammation and autoimmunity [71]. Diets rich in antioxidants, such as those high in fruits and vegetables, can mitigate oxidative stress, thereby reducing inflammation. Conversely, diets low in antioxidants and high in processed foods can exacerbate oxidative stress, contributing to the pathogenesis of autoimmune diseases [72].

Gut Microbiome

The gastrointestinal tract harbors a complex and diverse gut microbiota, essential for sustaining immune equilibrium. This microbiota significantly influences the maturation and functional dynamics of the immune system, notably in sculpting the T-cell array and modulating the equilibrium of pro-inflammatory and regulatory T-cell groups [73]. Dietary patterns profoundly alter gut microbiome composition, with specific nutrients like fiber, polyphenols, and fatty acids fostering the proliferation of beneficial microbes and the synthesis of short-chain fatty acids (SCFAs), including butyrate, acetate, and propionate [74]. These SCFAs play a vital role in maintaining gut barrier integrity and exhibit anti-inflammatory properties. Conversely, diets high in saturated fats, simple sugars, and processed foods favor pathogenic bacterial growth, intensifying inflammation [75]. Such dietary choices can shift microbial populations, leading to enhanced intestinal permeability or "leaky gut". This condition allows microbial entities and antigens to infiltrate the systemic circulation, sparking inappropriate immune reactions and potentially triggering autoimmune diseases [76].

Molecular Mimicry

Molecular mimicry describes the occurrence where exogenous antigens structurally resemble endogenous antigens, potentially inciting an autoimmune attack on the body's tissues [77]. Dietary elements can introduce these mimicking antigens [78]. A prime example is the link between gluten, present in wheat and other cereals, and celiac disease. The structural resemblance of gluten peptides to gut self-antigens may provoke an immune response, resulting in damage to intestinal tissue [79]. In type 1 diabetes, certain milk proteins are suspected of imitating pancreatic beta-cell antigens, possibly inciting an autoimmune response against these cells [80]. Furthermore, the role of the gut microbiome in molecular mimicry must be considered [81]. Dietary components influence the gut microbiota's composition and functionality, which may generate peptides mimicking host antigens. Epitope spreading, another critical aspect of

molecular mimicry in autoimmunity, involves the expansion of the autoimmune response due to chronic inflammation [82]. Initiated by molecular mimicry, this process leads to the release and recognition of new self-antigens by the immune system, further amplifying the autoimmune reaction [83].

Effects of Nutrients on CV Risk Factors in Autoimmune Diseases

Since diet has an important impact on CV risk factors, both for autoimmune diseases and the general population, we summarized the effects of several common nutrients on CV risk factors for autoimmune diseases, as shown in Fig. 1.

Omega-3 Fatty Acids

Omega-3 fatty acids, predominantly found in fish oils and certain plant oils, are polyunsaturated fatty acids (PUFAs) known for their significant role in improving health. The beneficial CV effects of Omega-3 fatty acids have been demonstrated through clinical studies and animal experiments [84]. The main mechanism involved modulating lipid metabolism [85], exerting anti-inflammatory effects, stabilizing cardiac rhythms [86] and more. Because of its powerful anti-inflammatory effect, its role in improving autoimmune has also been paid more attention. Omega-3 fatty acids exert anti-inflammatory effects through a variety of mechanisms mainly including shifting the arachidonic acid pathway to anti-inflammatory mediators and down regulation of proinflammatory cytokines [87]. What's more, Omega-3 fatty acids (eicosapentaenoic acid and docosahexaenoic acid) incorporate into the phospholipid bilayer of cell membranes. This incorporation alters the physical and chemical properties of the cell membrane, influencing cell signaling pathways, thus modulating inflammatory responses. Therefore, the improvement of Omega-3 fatty acids in CV risk factors of the autoimmune system not only reduces the traditional risk, but also the risk factors associated with the disease itself, such as disease activity. Here is a summary of some important studies on the effects of fatty acids on cardiovascular risk factors for RA and SLE in Table 2 (Table 2).

Antioxidants (Vitamin C, E, and Selenium)

Antioxidants like vitamin C, vitamin E, and selenium play critical roles in neutralizing free radicals and reactive oxygen species [92], which are elevated in autoimmune conditions

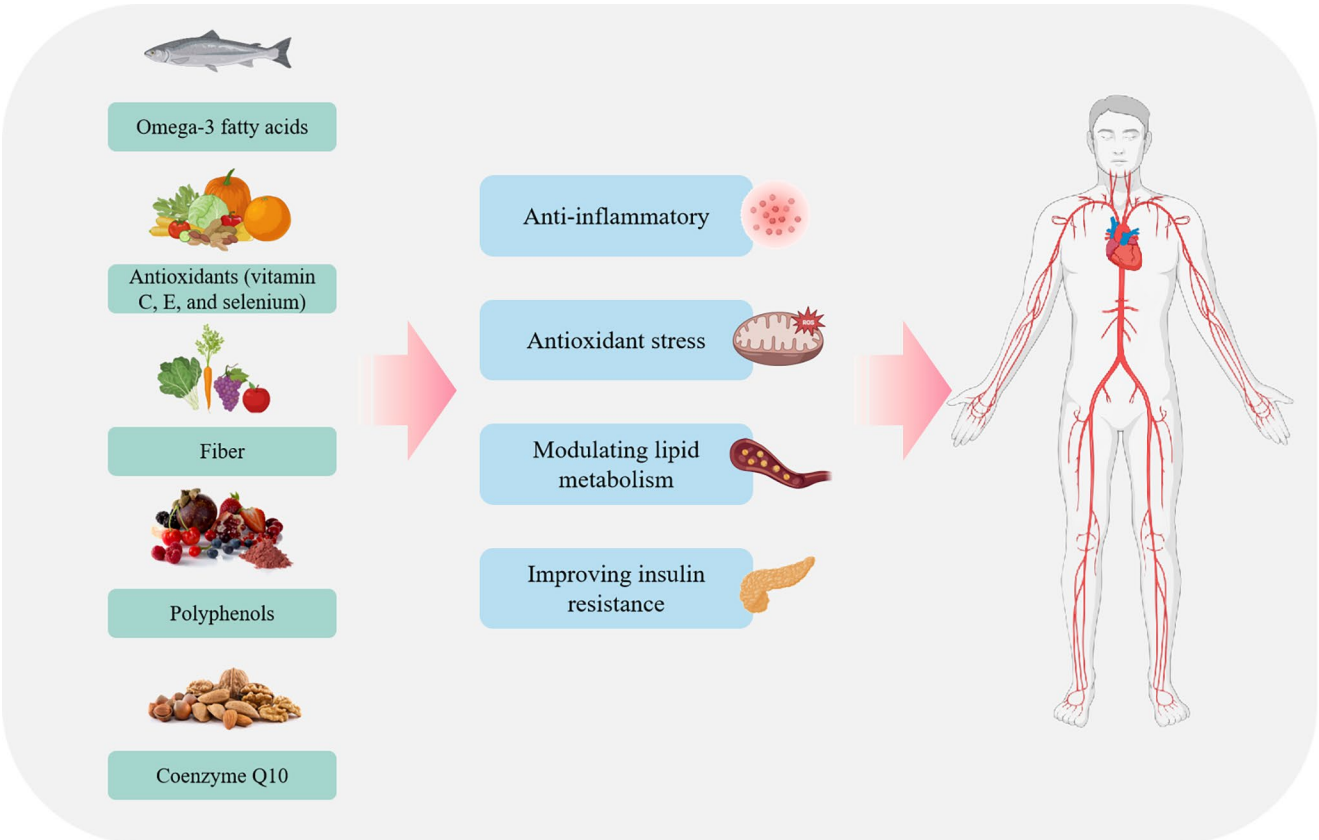

Fig. 1 Nutrients that modulate cardiovascular disease risk in autoimmune disease patients

Table 2 Effect of Omega-3 fatty acids on CVD risk in RA and SLE: evidence from clinical studies

Study type	Dose gr/daily	Duration	Autoimmune diseases	*N* patients	The expected outcome in CVD	Reference
A comparative observational study from Australia	4 to 4.5 g EPA plus DHA	3 yesrs	RA	31	AA was 30% lower in platelets and 40% lower in peripheral blood mononuclear cells; Favorable changes in fasting blood lipids.	[88]
Randomized interventional trial from Northern Ireland	3 g	24 weeks	SLE	69	Improved endothelial function by increasing flow-mediated dilatation from 3 to 5.7%	[89]
A comparative observational study from Brazil	3 g fish oil	4 monthes	SLE	62	Increased adiponectin levels and decreased leptin levels	[90]
Placebo-controlled randomized clinical trial from USA	6 capsules of fish oil/day equaling 2.25 g EPA and 2.25 g DHA metagenics	6 monthes	SLE	50	Erythrocyte sedimentation rate and serum IL-12 were reduced, while serum IL-13 was increased by fish oil supplementation	[91]

CVD: cardiovascular diseases; RA: rheumatoid arthritis; SLE: systemic lupus erythematosus; EPA: eicosapentaenoic acid; DHA: docosahexaenoic acid; AA: arachidonic acid; SLE: systemic lupus erythematosus; IL: interleukin

and contribute to endothelial dysfunction and atherosclerosis. Vitamin C and E, has been shown to protect LDL cholesterol from oxidation, a key step in the pathogenesis of atherosclerosis [93, 94]. Selenium, an essential micronutrient, is integral for the function of glutathione peroxidase, an enzyme that reduces peroxides and protects myocardial cell from oxidative damage [95]. A study by Lopes Virella et al. found that the use of antioxidants can reduce the risk of cardiovascular disease in people with type 1 diabetes [96]. A double-blind, placebo-controlled study from Hong Kong showed a decrease in lipid peroxidation after 3 months of combined vitamin C and E supplementation in SLE patients [97].

Fiber

Dietary fiber is an indigestible carbohydrate; According to its water solubility, it can be divided into soluble fiber and insoluble fiber. The role of dietary fiber in cardiovascular health has been extensively studied, with consistent findings indicating its protective role [98]. Dietary fiber contributes to cardiovascular health through multiple mechanisms. It aids in lowering LDL cholesterol levels and improving overall lipid profiles [99]. Fiber reduces insulin resistance [100], and impacts gut microbiota composition, fostering a beneficial microbial environment that has been linked to reduced systemic inflammation [101]. Low dietary fiber intake in adolescents with SLE is associated with increased abdominal obesity and therefore increased cardiovascular risk [102].

Polyphenols

Polyphenols are secondary metabolites in plants and to date > 8000 polyphenols have been identified [103]. They are found abundantly in fruits, vegetables, tea, and wine, exhibiting strong anti-inflammatory and antioxidant properties [104]. The antioxidant and anti-inflammatory effects of polyphenols potentially result in vasodilation, antithrombotic, hypolipidemic or anti-atherosclerosis, thus reduce cardiovascular risk [105–107]. In the realm of autoimmune diseases, polyphenols exert several beneficial effects: immunomodulatory effects, anti-inflammatory action, antioxidant properties [108]. At present, there is no direct study on the impact of polyphenols on the risk of cardiovascular diseases in people with autoimmune system diseases. However, from the protective effect of polyphenols on autoimmune system diseases and cardiovascular diseases, it is not difficult to infer that polyphenols, as a nutritional treatment, have a certain improvement effect on the CV risk of autoimmune system diseases.

Coenzyme Q10

Coenzyme Q10 (CoQ10), a lipophilic benzoquinone, plays a pivotal role in the mitochondrial electron transport chain. CoQ acts as a potent antioxidant by neutralizing ROS and inhibiting lipid peroxidation, thereby reducing oxidative stress within the cardiovascular system [109]. Another mechanism by which CoQ affects cardiovascular health is the enhancement of mitochondrial function [110]. Improved mitochondrial function can lead to enhanced myocardial contractility and improved overall cardiac performance. Emerging evidence underscores the potential metabolic benefits of CoQ10, including favorable alterations in lipid profiles and mitigation of insulin resistance. Consequently, CoQ10 has attracted much attention as a nutritional supplement for cardiovascular diseases [111].

Conclusion

Timely and meticulous intervention for the prevention and management of CV risk factors in the context of autoimmune diseases holds paramount significance for both disease trajectory and prognosis. This imperative remains steadfast, notwithstanding the existing categorization of CV risk factors specific to autoimmune diseases and the overarching emphasis on disease-associated risk factors. Regrettably, up to this point, the individualized prediction and nuanced management of CV risk factors within the autoimmune disease spectrum have been inadequately addressed.

In the realm of disease management, cost-effective lifestyle modifications are increasingly gaining traction, particularly those centered around dietary interventions aimed at enhancing overall health. Consequently, our investigation delves into dietary strategies for the management of CV risk factors within the context of autoimmune diseases. It is imperative to recognize that the atypical risk factors associated with autoimmune diseases are intrinsically linked to the pathology of the diseases themselves. To comprehensively apprehend the influence of diet on these non-traditional risk factors, it is essential to elucidate both their effect on the disease and the mechanisms through which they exert their influence. The relationship between dietary choices and autoimmune diseases is multifaceted, characterized by a dichotomy of effects. The Western dietary pattern, characterized by high levels of sodium, sugar, and heavily processed meats, has been demonstrated to exacerbate states of inflammation, thereby intensifying disease severity. The Mediterranean diet, characterized by its high content of unsaturated fatty acids, vitamins, and dietary fiber, promotes the consumption of minimally processed foods and tends to improve inflammatory responses and reduce the overall burden of disease [65]. Regarding the complex effects of diet on autoimmune diseases, we summarized several common mechanisms. Further molecular effects need to be explored.

We recommend several nutrients based on their anti-inflammatory and improving CV function effects. Unfortunately, there are few clinical studies on their cardiovascular effects in autoimmune systemic diseases, and this is one of the areas that needs more attention in the future.

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Declarations

Ethics approval and consent to participate Not applicable.

Conflict of interest The authors declare no conflict of interest.

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