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Review Article

The impact of lifestyle modifications on disease progression in patients with lupus

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ABSTRACT

Systemic lupus erythematosus (SLE) is a chronic autoimmune disease with a complex pathogenesis influenced by genetic, environmental, and lifestyle factors. This narrative review explores the impact of lifestyle modifications on SLE progression, focusing on diet, physical activity, smoking, alcohol, caffeine, and ultraviolet radiation (UVR) exposure. Evidence highlights the benefits of n-3 polyunsaturated fatty acids (PUFAs), vitamin D optimization, and calorie restriction (CR) in reducing inflammation and disease activity, while excessive n-6 PUFA intake worsens outcomes. Regular physical exercise improves fatigue, cardiovascular fitness, and psychological well-being, whereas smoking and UVR exposure are consistently associated with heightened disease activity and organ damage. The effects of alcohol and caffeine are less clear, with moderate alcohol intake showing potential protective effects and caffeine yielding inconclusive results. This review underscores the importance of lifestyle modifications as integral to SLE management, complementing pharmacological therapies to improve quality of life and long-term outcomes.

Keywords: SLE, Lifestyle, Diet, Exercise

INTRODUCTION

Systemic lupus erythematosus (SLE) is a chronic autoimmune disease characterized by systemic inflammation and autoantibody production. It can affect multiple organs, including the skin, kidneys, joints, cardiovascular system, and central nervous system. SLE predominantly affects women, particularly those of reproductive age, with a female-to-male ratio of approximately 9:1. Prevalence varies globally, ranging from 20 to 70 cases per 100,000 individuals, with higher rates reported in populations of African, Hispanic, and Asian descent.[1] The heterogeneous presentation of SLE, along with its fluctuating disease course, poses challenges for diagnosis and management.[2]

The pathogenesis of SLE is complex, involving genetic, environmental, and immunological factors. Numerous genetic susceptibility loci have been identified, including variants in immune regulatory genes such as N-acetyltransferase 2 (NAT2) and signal transducers and activators of transcription 4 (STAT4). These genetic factors interact with environmental triggers, including infections, UVR, and lifestyle factors, to disrupt immune tolerance and initiate autoimmunity. Despite these insights, the exact mechanisms underlying SLE remain incompletely understood, emphasizing the need for further investigation into modifiable environmental and lifestyle factors that contribute to disease development and progression.[3]

Pharmacological therapies for SLE, such as corticosteroids, antimalarials, and immunosuppressive agents, have significantly improved survival and reduced disease activity. However, these treatments are associated with substantial side effects, including infections, hypertension, and osteoporosis. Additionally, the heterogeneity of SLE means that no single therapeutic approach is universally effective. This highlights the need for complementary strategies, including lifestyle modifications, to optimize disease management.[4]

Lifestyle factors, such as diet, cigarette smoking, alcohol consumption, caffeine intake, and UVR exposure, have garnered attention as modifiable contributors to SLE. Research suggests that these factors may influence disease susceptibility, activity, and severity by modulating immune responses, inflammation, and oxidative stress.[5] For instance, dietary components such as n-3 PUFAs and vitamin D have demonstrated immunomodulatory effects, while cigarette smoking and UVR exposure are associated with exacerbated disease activity and organ damage. Alcohol and caffeine consumption have shown mixed effects, with some studies indicating protective roles and others suggesting increased risk.[6]

Understanding how these lifestyle factors impact SLE at molecular and clinical levels is essential for developing comprehensive management strategies. Incorporating lifestyle interventions alongside pharmacological treatments could enhance disease outcomes, reduce complications, and improve quality of life for patients.[7] This review aims to provide a detailed analysis of the current evidence linking diet, smoking, alcohol, caffeine, and UVR exposure to SLE. By exploring these associations, the review seeks to inform future research and guide the development of tailored interventions for SLE patients.

LITERATURE SEARCH

This narrative review is based on a comprehensive literature search conducted on 19 November 2024 in the Medline and Cochrane databases. Utilizing medical subject headings (MeSH) and relevant keywords, the search aimed to identify studies examining the impact of lifestyle modifications on disease progression in patients with SLE. To ensure thoroughness, a manual search was also conducted using Google Scholar, and the reference lists of identified papers were reviewed to locate additional relevant studies. The review focused on articles addressing the effects of diet, physical exercise, smoking, alcohol and caffeine consumption, and UVR exposure on SLE progression. No restrictions were applied regarding publication date, language, participant age, or type of publication, ensuring a broad and inclusive exploration of the available literature.

DISCUSSION

Diet and SLE

Dietary factors influence immune function and inflammation, playing a critical role in SLE pathogenesis and management. Key components of diet studied in SLE include PUFAs, CR, and vitamin D levels.[6]

The contrasting roles of n-3 and n-6 PUFAs are well-documented in SLE. Dietary intake of n-3 PUFAs, primarily derived from fish oil, has demonstrated significant anti-inflammatory effects. In murine models of SLE, n-3 PUFA supplementation reduced autoantibody production, proteinuria, and renal damage, key markers of disease progression.[8-10] Clinical trials in humans have corroborated these findings, showing improvements in endothelial function and reductions in systemic inflammation following supplementation with n-3 PUFA.[11] Moreover, a double blinded factorial trial reported a decline in revised systemic lupus activity measure (SLAM-R) scores among SLE patients receiving eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) compared to a placebo group.[12] According to another study, daily oral intake of moderate doses of EPA and DHA led to sustained remission of SLE.[13]

N-3 PUFA mitigates inflammatory and autoimmune responses through anti-inflammatory and immune-modulating mechanisms. It inhibits inflammatory cytokine production, lymphocyte proliferation, cytotoxic T cell and natural killer cell activities, macrophage cytotoxicity, neutrophil/monocyte chemotaxis, MHC Class II expression, and antigen presentation.[14,15] Experimental studies show that N-3 PUFA lowers plasma levels of cytokines such as IL-6 and tumor necrosis factor alpha (TNF-α). N-3 PUFA enhances antioxidant enzyme production and down-regulates $CD4^+$ T cell-related gene expression, including Cd80 and Il6. These effects collectively reduced inflammation, oxidative stress, and autoimmune activity in murine SLE models.[10,16,17]

In contrast, n-6 PUFAs, found in vegetable oils, have been associated with increased inflammatory responses. A study conducted in an animal SLE model, revealed that n-6 PUFA-rich diets exacerbated renal damage and autoantibody production, likely through upregulation of inflammatory gene expression in $CD4^+$ T cells.[10]

CR has shown promise in alleviating SLE manifestations. In animal models, CR reduced proteinuria, glomerulonephritis, and immune complex deposition, while extending lifespan. These benefits are mediated by suppression of pro-inflammatory cytokines, including interferon-alpha (IFN-α) and TNF-α, and enhanced antioxidant defences.[18]

CR also reduces circulating levels of leptin, a pro-inflammatory adipokine. In SLE models, hypoleptinemia induced by CR expanded regulatory T-cell populations and decreased the prevalence of Th17 cells, contributing to reduced disease severity.[19,20] Human studies further indicate that CR improves fatigue, a common and debilitating symptom in SLE patients, underscoring its potential as an adjunctive therapy.[21]

Vitamin D deficiency is prevalent in SLE patients and has been linked to increased disease activity and poor outcomes.[22-24] However, some studies have found no association between vitamin D deficiency and SLE pathogenesis.[25,26] Vitamin D modulates immune responses by inhibiting B-cell activation, reducing pro-inflammatory cytokine production, and promoting regulatory T-cell activity. Clinical studies have demonstrated that vitamin D supplementation improves immune homeostasis in SLE, reducing disease activity and inflammation.[27-29] However, inconsistencies in findings necessitate further investigation to clarify the relationship between vitamin D and SLE progression.

Physical exercise and SLE

Physical activity is increasingly recognized as a valuable non-pharmacological intervention for managing SLE. In 2024, Blaess et al outlined evidence-based recommendations for physical activity in SLE, emphasizing its potential to improve quality of life and physical health outcomes. Their consensus highlighted the importance of medical evaluation before initiating exercise programs, allowing for the identification of contraindications and adaptation of regimens based on individual capacities, preferences, and comorbidities. Specific considerations, such as avoiding inflamed joints and using photoprotection or appropriate clothing for Raynaud's phenomenon, are essential for safety. For patients with conditions like osteonecrosis or Jaccoud's syndrome, tailored assessments are necessary prior to commencing activity. Structured exercise programs should include low-intensity warm-ups, cooling-down periods, and a combination of aerobic and resistance training exercises. Recommendations suggest 150–-300 minutes of moderate aerobic activity weekly, coupled with strengthening exercises on at least 2 days/week, with resistance training involving 1-3 sets of 812 repetitions.[30]

Physical activity yields significant benefits in SLE, both physically and mentally. Regular exercise is associated with reduced fatigue, less pain, better sleep, and improved psychological well-being, including reduced depressive symptoms.[31,32] A 12-week progressive aerobic exercise program has been shown to improve fatigue significantly in women with SLE.[33] Cardiovascular fitness and flexibility improvements may also mitigate the inflammatory effects of increased body mass and adiposity. Regular exercise, particularly when combining aerobic and resistance training, enhances muscle strength and cardiorespiratory fitness without exacerbating arterial stiffness or oxidative stress.[34] Furthermore, physically fit women with SLE exhibit favorable body composition parameters, including lower body mass index and waist-to-hip ratios, which correlate with reduced cardiovascular risk and improved health-related quality of life.[35]

The immunological effects of exercise in SLE are promising but warrant further exploration. Acute aerobic exercise has been shown to down-regulate inflammatory gene expression in leukocytes, with no evidence of inducing inflammation or disease flares.[36,37] These findings suggest a potential role for exercise in reducing systemic inflammation. Despite these benefits, some studies report limited impact of exercise on disease activity or fatigue, underscoring the variability of outcomes and the need for personalized approaches.[38,39] Intervention programs should prioritize patients with significant fatigue or greater organ damage, as these groups tend to exhibit the most pronounced reductions in physical activity levels. Overall, physical activity represents a crucial component of holistic SLE management, complementing pharmacological therapies to enhance physical health and quality of life.

Cigarette smoking

Cigarette smoking is linked to worsening clinical outcomes in SLE. It is associated with photosensitivity, active rash, higher SLE disease activity index (SLEDAI) scores, pleuritis, peritonitis, neuropsychiatric manifestations, thrombosis, cardiovascular and peripheral vascular diseases, and metabolic syndrome. Smoking also increases anti-phospholipid antibody production and reduces the effectiveness of SLE treatments.[40] A Chinese study found that smoking exacerbated SLE symptoms like photosensitivity, nephropathy, and proteinuria, though SLEDAI scores did not differ significantly between smokers and nonsmokers.[41]

Mechanistic studies suggest that smoking exacerbates SLE through oxidative stress and epigenetic modifications.[42,43] DNA methylation patterns altered by smoking influence the expression of immune-related genes, potentially contributing to autoimmunity. Notably, the cessation of smoking may reverse some epigenetic changes, emphasizing the importance of smoking cessation in SLE management.[44]

Alcohol consumption

The relationship between alcohol and SLE is complex. While early studies suggested no association,[45,46] recent

research indicates that moderate alcohol consumption may have a protective effect against SLE.[47,48] Although the mechanism by which alcohol consumption reduces SLE risk remains unclear, proposed theories highlight its anti-inflammatory and antioxidant effects. Moderate alcohol intake has been shown to suppress the NF-κB pathway, reducing pro-inflammatory cytokines such as TNF-α and IL-1β while increasing anti-inflammatory IL-10.[49] Compounds in wine, like glycerol and ascorbate, protect against oxidative DNA damage.[50] Epidemiological evidence suggests moderate alcohol consumption benefits inflammatory regulation compared to abstinence or heavy drinking, potentially lowering systemic inflammation and cardiovascular disease risk.[51]

Few studies have explored the impact of alcohol consumption on disease activity and damage in SLE. A previous cross-sectional study of SLE patients found an independent association between alcohol abstinence and an increased risk of nephritis.[52] This supports earlier in vivo and in vitro research suggesting that alcohol may exert an anti-inflammatory effect, potentially reducing disease progression.[49,50] Similarly, a Korean study reported that there was no significant relation between alcohol consumption and disease activity measured by the SLEDAI-2K scoring system.[53] These findings imply that while alcohol may not exacerbate SLE manifestations, its overall effect on disease activity remains inconclusive and warrants further investigation, particularly regarding alcohol dose and type.

Caffeine-rich beverages

Caffeine, functioning as a non-specific phosphodiesterase inhibitor, appears to interact with various components of the immune system, affecting both innate and adaptive responses. Studies on caffeine consumption and SLE disease activity reported positive results. They suggested that moderate caffeine intake attenuated SLE activity, potentially influencing chronic damage, while lower caffeine intake was linked to more frequent major organ involvement, including renal and neuropsychiatric manifestations, as well as anti-dsDNA positivity. Further, coffee consumption was positively associated with clinical remission over a six-month period.[54,55] Nevertheless, gene-environment interactions may modulate these effects, as demonstrated by previous research linking NAT2 genotypes to varying SLE risks associated with caffeine consumption.[56]

UVR

Pro-inflammatory effects of UV-A include stimulating IL-1 and IL-6 and altering lymphocyte function. However, UV-A also exhibits anti-inflammatory properties, leading to its use in phototherapy. These effects include T and B cell apoptosis and the reduction of inflammatory cytokines like IL-4, IL-10, and interferon-γ. Phototherapy using UV-A, often combined with psoralens, modifies T-cell receptor specificity and lymphocyte function.[57,58] In SLE mice, UV-A improved survival and reduced anti-DNA antibodies, likely due to preserving Langerhans' cell function and decreasing B-cell activation.[59,60]

UV-B radiation has immunosuppressive effects, primarily through DNA damage, which induces antigen-specific immunotolerance by generating regulatory T cells. UV-B can also promote SLE-related mechanisms, such as redistributing nuclear antigens to the cell surface and producing novel autoantigens. Additionally, UV-B damages keratinocytes and other cells through ROS production and DNA damage, including strand breaks and pyrimidine dimer formation. UV-B may also inhibit DNA methylation, enhancing lymphocyte function and autoreactive T cell production, as demonstrated in SLE-prone mice.[61]

It has been shown that increased UV radiation exposure can exacerbate pre-existing skin conditions in SLE patients, often causing new lesions on sun-exposed areas, including macular, papular, bullous lesions, and erythema. UV exposure can also trigger systemic SLE flares, manifesting as weakness, fatigue, fevers, or joint pain. However, patient-reported photo-induced symptoms often do not align with physician assessments or laboratory markers of SLE disease activity. Sunlight exposure likely contributes to both the onset and exacerbation of SLE, particularly in individuals prone to severe sunburns with blistering or rashes following midday sun exposure.[62] Therefore, photoprotection through sunscreen use and clothing is a cornerstone of SLE management to mitigate UV-related disease activity.

CONCLUSION

Lifestyle modifications, including dietary adjustments, smoking cessation, moderated alcohol intake, and UV protection, are critical for managing SLE. Evidence supports the benefits of n-3 PUFA supplementation, CR, and vitamin D optimization in reducing disease activity. Smoking and UV exposure unequivocally worsen disease outcomes, while the roles of alcohol and caffeine remain less clear.

Future research should focus on elucidating the mechanisms underlying these associations and designing targeted interventions. Integrating lifestyle modifications with pharmacologic treatments may enhance outcomes and quality of life for SLE patients, offering a holistic approach to disease management.

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