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Lupus – Prevention and Treatment with a Plant-Based Diet

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Abstract

Systemic lupus erythematosus (SLE) is a chronic multisystem autoimmune rheumatic disease in which disease flares are interspersed with episodes of remission. In contrast to organ-specific autoimmune diseases, SLE comprises a constellation of signs and symptoms that can affect multiple organ systems. A plant-based diet may have a beneficial effect on SLE patients, due not only to the direct action of nutrients on the immune system and inflammation, but also to an indirect effect on insulin resistance, obesity and associated co-morbidities. Accelerated atherosclerosis is a significant comorbidity and the leading cause of death for patients with SLE. Patients are also more likely to experience metabolic syndrome leading to type II diabetes, obesity, and chronic kidney disease. A plant-based diet is a safe and efficacious prophylaxis and treatment for all these comorbidities. Three small interventional studies with a plant-based diet showed a beneficial effect on patients' symptoms and therefore their quality of life. A plant-based diet has the advantage of having no adverse reactions or contraindications and can be an effective adjunct to standard treatments.

Keywords: Autoimmune; Fatigue; Inflammation; Joint pain; Lupus; Muscle pain; Plant-based diet; Rheumatic; Skin lesions; Systemic lupus erythematosus

Abbreviations: CVD: Cardiovascular disease; BMI: Body Mass Index; MI: Myocardial Infarction; SLE: Systemic Lupus Erythematosus; WFPB: Whole Food Plant-Based

Introduction

Systemic lupus erythematosus (SLE) is a chronic multisystem autoimmune rheumatic disease in which disease flares are interspersed with episodes of remission. In contrast to organ-specific autoimmune diseases, SLE comprises a constellation of signs and symptoms that can affect multiple organ systems [1]. Common symptoms include fatigue, joint and muscle pains and skin lesions, as well as more severe manifestations affecting the kidneys, central nervous system, heart and lungs. Symptoms can be heterogeneous, intermittent and range in severity [2], requiring the establishment of eleven criteria with four needed for the formal diagnosis of SLE [3,4]. SLE affects up to 322,000 adults in the United States [5]. It can begin at any age and affect both sexes, but more than 90% of new patients presenting with SLE are women in the childbearing years, most often at the end of the patient's second decade and beginning of the third decade of life [6,7].

Pathophysiology and Epidemiology

Genetic predisposition, environmental triggers, and the hormonal milieu, interplay in SLE disease development and activity [8]. The action of these pathogenic factors results in the generation of autoantibodies, immune complexes, autoreactive or inflammatory T cells and inflammatory cytokines that may initiate and amplify inflammation and damage to various organs, contributing to the clinical manifestations of SLE. It is characterized by a deposition of immune complexes, formed in large amounts as antinuclear antibodies bind to the abundant nuclear material in blood and tissues, along with disturbances in both innate and adaptive immunity manifest by disorders in cytokines, apoptotic cell clearance, B-cell immunity and T-cell signaling [9,10]. SLE Patients with active SLE exhibit high cytokine levels, including IFN-γ, TNF, IL-4, IL-6, IL-10, IL-12, IL-17 and IL-18 in serum and plasma; by contrast, IL-2 levels are lower in comparison with healthy controls. [11].

Comorbidities

Accelerated atherosclerosis is a significant co-morbidity and the leading cause of death for patients with SLE. [12] Patients suffering with SLE have approximately double the risk of atherosclerotic cardiovascular disease (CVD), stroke, heart failure and atrial fibrillation compared with the general population [13]. One study showed that more than half of the patients with SLE presented three or more risk factors for CVD (mostly obesity, hypertension and dyslipidemias) [14]. In another study, patients with SLE were identified to have a twofold to threefold higher risk of stroke and MI [15].

In patients with SLE, traditional risk factors for CVD (age, hypertension, diabetes mellitus, dyslipidemia, obesity, smoking, and positive family history) are more prominent than non-traditional factors and appear to have a major role in lupus-enhanced atherogenesis [16]. In particular hypertension and hypercholesterolemia have been shown to be independently associated with premature atherosclerosis in several SLE cohorts [17-19]. The prevalence of dyslipidemia, with elevations in total cholesterol (TC), low-density lipoprotein (LDL), triglyceride (TG), and apolipoprotein B (ApoB), are about 30% at the diagnosis of SLE rising to 60% after 3 years [20]. CV events occur both early and late during the disease course, with younger patients being at much higher risk than their age-matched counterparts [13].

However, cohort studies have revealed that traditional atherosclerotic risk factors do not fully account for the CVD susceptibility in patients with SLE. It may also be due to pathophysiologic intermediates such as type I interferons and other inflammatory cytokines, oxidative stress, activated granulocytes and production of extracellular chromatin traps, antiphospholipid and other autoantibodies causing dysfunction of lipoproteins, altogether resulting in endothelial injury and pro-atherogenic dyslipidemia [13]. In SLE, autoantibodies and cytokines are able to modulate and decrease lipoprotein lipase activity, a key enzyme in lipid metabolism, producing the 'lupus pattern' of dyslipoproteinemia, characterized by elevated levels of VLDL (very low density LDL) and triglycerides and low HDL-cholesterol levels, which are directly correlated with SLE disease activity index (SLEDAI) scores [21]. These mechanisms may be further aggravated by chronic intake of prednisone, even at doses <7.5 mg/day [13].

Studies have reported a high prevalence of overweight and obese patients with SLE [22,23] In a representative sample of women with SLE, obesity (as defined by both FMI [fat mass index] and BMI) was independently associated with worse patient-reported outcomes including disease activity, depressive symptoms, and symptoms of pain and fatigue. Obesity may represent a modifiable target for improving outcomes among obese women with SLE [24].

With regards to diabetes mellitus risk, a study showed that patients with SLE are more likely to have metabolic syndrome [25]. Treatment with a daily glucocorticoid ≥10 mg prednisolone-equivalent dose was associated with increased risk of developing diabetes mellitus [26]. Interestingly, patients treated with hydroxychloroquine had their risk of type II diabetes attenuated [26].

Renal involvement is present in about 50% of patients with lupus, with a predilection for certain ethnic groups such as African Americans (70%). (27) Early detection and treatment are paramount since lupus nephritis is a major cause of morbidity and mortality in SLE and delayed diagnosis is a risk factor for end-stage renal disease. [28,29] Death in patients with SLE may be due to lupus activity (when vital organs or systems are involved), complications of treatment (particularly infections), or long-term sequelae (such as cardiovascular disease) [28].

Dietary Factors

A healthy diet may have a beneficial role in SLE patients, due not only to a direct action of nutrients on the immune system and inflammation, but also to an indirect effect on insulin resistance, obesity and associated co-morbidities [30,31]. Unhealthy dietary patterns may contribute to the development and course of SLE [32,33]. Specific dietary factors, including vitamins, mineral elements, fatty acids and polyphenols, play a major role in the modulation of immune responses, so an inadequate diet could constitute an important risk factor in SLE epidemiology [34-37].

Insufficient study has been done in this area, but in one study, women in the highest (versus lowest) AHEI-2010 (alternative healthy eating index) tertile of nut and legume intake had a 41% decreased risk of SLE [38]. Vitamin D deficiency may be a risk factor for SLE [39]. Vitamin D deficiency is commonly reported in SLE patients [40] and can contribute to the morbidity and mortality of these patients [41]. SLE may lead to lower vitamin D levels and vitamin D deficiency may have a causative role in SLE etiology and/or aggravation [42]. One study found that 67% of the SLE patients were vitamin D deficient, with mean levels significantly lower among African Americans (15.9 ng/ml) compared to Caucasians (31.3 ng/ml). Critically low vitamin D levels (<10 ng/ml) were found in a number of SLE patients [43].

Dietary Intervention studies

Interventional studies of SLE can be difficult to perform, given that SLE causes immunologically mediated injury in multiple body systems and symptoms can be heterogeneous, intermittent and range in severity [1]. However, one small study and two case studies have given us some information. In an interventional study, patients adhering to a WFPB (whole food plant-based) diet were more likely to experience benefits from dietary change, including a significant 26% decrease in SLE symptom severity, especially for SLE patients with initially severe symptoms [2].

In a case study of a 24-year-old female patient treated with a WFPB diet, her estimated glomerular filtration rate increased from 14 to 27 ml/min in 6 weeks, and it was determined that she no longer needed dialysis or a kidney transplant. Her energy and joint pain levels also significantly improved [44]. In another case study of a 41-year-old male patient treated with a WFPB diet, symptoms resolved and laboratory tests were normalized on the diet. However, this patient experienced challenges with adhering to the diet, and it was clear that whenever he deviated from it, symptoms reappeared, and his estimated glomerular filtration worsened [44].

Looking specifically at fatty acid intake in patients with stable disease, a diet rich in polyunsaturated fatty acids (PUFA) was found to have a positive impact on overall clinical status [37]. With

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a concentration of 70% omega-3 PUFA and rich in α-linolenic acid, daily intake of 30g of flaxseed oil was found to lower serum creatinine in SLE patients with renal dysfunction [45,46].

A study, monitoring the dietary impact of two isocaloric diets on pregnant patients with SLE and antiphospholipid syndrome, emphasized that the diet with higher level of PUFA reduced the risk of fetal loss and symptoms [47]. Increasing polyunsaturated fatty acids and phytoestrogens showed a decrease in proteinuria and glomerulonephritis in animal models of SLE [48].

Clinical Considerations

SLE is characterized by its clinical and pathogenic complexity, difficult diagnosis and the high number of complications that can affect the patients' quality of life. The high morbidity and mortality associated with patients with SLE may be related to late diagnosis, problems in access to care, less effective treatments, and poor adherence to therapeutic regimens [49]. SLE management remains complicated owing to the biological heterogeneity between patients and the lack of safe and specific targeted therapies. Thus, the search for new therapeutic targets and strategies that can act more selectively on certain routes or biological processes, and improve the course of disease or reverse the outbreak phase, without generating collateral damage to unaffected tissues and organs, is the pillar underlying current research in SLE [32].

Pharmacological treatment in SLE aims to prevent organ damage and achieve remission. The choice of treatment is dictated by the organ system/systems involved and the severity of involvement. It ranges from minimal treatment (NSAIDs, antimalarials) to intensive treatment (cytotoxic drugs, corticosteroids). Newer agents for treatment of SLE are the B-lymphocyte inhibitor belimumab, the interferon antagonist anifrolumab, and the calcineurin inhibitor voclosporin.

The autoimmunity and inflammatory processes of SLE are related to the presence of dyslipidemia, obesity, systemic arterial hypertension, and metabolic syndrome. It is of prime importance to treat these comorbidities in order to decrease cardiovascular risk [50]. A diet with moderate protein and energy content, but rich in vitamins, minerals, antioxidants and mono/polyunsaturated fatty acids, can provide a beneficial protective effect against tissue damage and suppression of inflammatory activity, in addition to helping the treatment of comorbidities [45].

For over 50 years, evidence from interventional studies has strongly indicated that a low-fat plant-based diet is both safe and efficacious in the treatment of coronary artery disease (CAD) [51-57]. In particular, epidemiological studies show a 40% risk reduction of ischemic heart disease [58] and a 50% risk reduction of coronary heart disease mortality [59] for those following a vegetarian diet. Plant-based diets are effective in treating hypercholesterolemia [60-62]. In particular, a plant-based diet has been shown to be as effective as Lovastatin in treating hypercholesterolemia [61]. Studies also show a large reduction in the frequency of angina pectoris episodes using a plant-based diet [52,63,64] and coronary artery stenosis can show modest but significant regression or can be halted [57].

Studies have shown that the vast majority of patients can maintain a plant-based diet for at least 4 years, and experience its resulting benefits during that time [52-54]. Mortality due to myocardial infarction can therefore be greatly reduced [51]. Those following a plant-based diet have a 78% reduction risk of type II diabetes mellitus, as well as a 56% reduced risk of metabolic syndrome [65]. Interventional studies using a plant-based diet show a reduction of HbA1C by as much as 2.4 percentage pts, which is more than is usually achieved with the leading medication, Metformin [66,67]. While dietary regimens for patients with SLE are mainly aimed at reducing CVD risk, recent evidence suggests that healthier dietary habits could also improve inflammatory markers and immune function, with possible benefit on many disease symptoms [33,45]. In particular a plant-based diet reduces the risk of a number of other pathologies such as type 2 diabetes, [65] stroke, [68] osteoarthritis [69], prostate and colon cancer [70,71], diverticular disease [72], ulcerative colitis [73], Crohn's disease [74], chronic kidney disease [75], Grave's disease, Hashimoto's thyroiditis [76] and rheumatoid arthritis [77] just to name a few.

Significant weight loss can also be achieved with a plant-based diet [52]. Vegetarian diets generally are associated with lower BMI in observational and randomized controlled studies. A study of American vegans found that they had a mean BMI of 23.6 [78]. A European study found the average BMI of vegetarians and vegans to be 23.3 and 22.4 respectively for men and 22.8 and 21.8 for women [79]. A study of German vegans found an average BMI of 22.3 [80]. In contrast, the average BMI for American men over age 20 for the year 2015-2016 is 29.1. The average BMI for American women over age 20 for the year 2015-2016 is 29.6 [81]. Plant-based diets are no longer very unusual as they have become more popular in the general population in recent years. Given all of the benefits, the plant-based diet deserves a place among the physician's treatment options for SLE.

In a study looking at the motivation for patients with SLE making dietary changes, the main reasons given for patients making dietary changes were to become healthier overall (83%) or to improve lupus symptoms (77%). In contrast, common reasons reported for not changing eating habits were due to not having enough information (41%) [2].

In another study, patients reported that there was a lack of clinical counselling regarding diet, with only 24% stating their doctor had spoken to them about diet. Despite this, 100% of patients stated they would change their diet if they knew it would help their symptoms [82]. This suggests that specific dietary advice is likely to be welcomed by patients, when carefully explained. Since 90% of patients are female and of child-rearing

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age, it is important to note that well-planned plant-based diets have been confirmed as safe during pregnancy and lactation, as well as conferring additional health advantages, such as lower risk of excessive gestational weight gain and preeclampsia [83].

When treating comorbidities, it's important to titrate relevant medications as the effect of the plant-based diet become evident. Lab work should be done before starting treatment with a plant-based diet and then 6 to 8 weeks afterwards, since the therapeutic effects of a plant-based diet often take several weeks to become evident. Vitamin D deficiency can affect disease activity and disease damage in SLE patients [84]. In addition, patients with SLE may avoid the sun because of photosensitive rashes and potential for disease flare, [41] so adequate vitamin D supplementation is vital for them. Lab work for SLE patients should include testing for vitamin D deficiency [85].

Discussion

Treatment goals for patients with SLE include long term patient survival, prevention of organ damage, and optimization of health-related quality of life. Achieving these goals can be challenging for patients and physicians alike.

A plant-based diet can help prevent and treat comorbidities commonly associated with SLE. It is a safe and efficacious prophylaxis and treatment for hypercholesterolemia, atherosclerosis, essential hypertension and coronary artery disease. It is also a safe and efficacious treatment for chronic kidney disease, type 2 diabetes and obesity. The plant-based diet can reduce symptoms and increase quality of life of SLE patients. While medications are available to treat SLE, it's important to note that the plant-based diet has no adverse reactions or contraindications and can therefore act as a useful adjunct to standard treatments, both for SLE and its comorbidities. It can also be a valuable monotherapy between flare ups. Its safety profile can be especially valuable for pregnant patients or patients wishing to become pregnant. SLE patients often face large healthcare costs. A healthy plant-based diet costs no more than a standard diet. Given its efficacy, it can also decrease the cost of treating comorbidities.

Conflict of Interest

The authors state no conflicts of interest.

References

1. Shaikh M, Jordan N, D'Cruz D (2017) Systemic lupus erythematosus. Clin Med (Lond) 17(1): 78-83.
2. Knippenberg A, Robinson GA, Wincup C, Ciurtin C, Jury EC, et al. (2022) Plant-based dietary changes may improve symptoms in patients with systemic lupus erythematosus. Lupus. 31(1): 65-76.
3. Tan E, Cohen A, Fries J, Masi A, McShane D, et al. (1982) The 1982 revised criteria for the classification of systemic lupus erythematosus. Arthritis Rheum 25(11): 1271-1277.
4. Noble PW, Bernatsky S, Clarke AE, Isenberg DA, Ramsey-Goldman R, et al. (2016) DNA-damaging autoantibodies and cancer: the lupus butterfly theory. Nat Rev Rheumatol 12(7): 429–434.
5. Helmick C, Felson D, Lawrence R, Gabriel S, Hirsch R, et al. (2008) National arthritis data workgroup. Estimates of the prevalence of arthritis and other rheumatic conditions in the United States. Part I. Arthritis Rheum 58(1): 15-25.
6. Mina R, Brunner H (2013) Update on differences between childhood-onset and adult-onset systemic lupus erythematosus. Arthritis Res Ther 15(4): 218.
7. Fanouriakis A, Tziolos N, Bertsias G, Boumpas DT (2021) Update on the diagnosis and management of systemic lupus erythematosus. Ann Rheum Dis 80(1): 14-25.
8. Fava A, Petri M (2019) Systemic lupus erythematosus: Diagnosis and clinical management. J Autoimmun 96: 1-13.
9. Crispin JC, Liossis SNC, Kis-Toth K, Lieberman LA, Kyttaris VC, et al. (2010) Pathogenesis of human systemic lupus erythematosus: recent advances. Trends Mol Med 16(2): 47-57.
10. Lisnevskaia L, Murphy G, Isenberg D (2014) Systemic lupus erythematosus. Lancet 384(9957): 1878-1888.
11. Chun HY, Chung JW, Kim HA, Yun JM, Jeon JY, et al. (2007) Cytokine IL-6 and IL-10 as biomarkers in systemic lupus erythematosus. J Clin Immunol 27(5): 461–466.
12. Appleton B, Major A (2021) The latest in systemic lupus erythematosus-accelerated atherosclerosis: related mechanisms inform assessment and therapy. Curr Opin Rheumatol 33(2): 211-218.
13. Kostopoulou M, Nikolopoulos D, Parodis I, Bertsias G (2020) Cardiovascular disease in systemic lupus erythematosus: Recent data on epidemiology, risk factors and prevention. Curr Vasc Pharmacol 18(6): 549-565.
14. Bruce I (2005) 'Not only...but also': factors that contribute to accelerated atherosclerosis and premature coronary heart disease in systemic lupus erythematosus. Rheumatol 44(12): 1492–1502.
15. Yazdany J, Pooley N, Langham J, Nicholson L, Langham S, et al. (2020) Systemic lupus erythematosus; stroke and myocardial infarction risk: a systematic review and meta-analysis. RMD Open 6(2): e001247.
16. Esdaile JM, Abrahamowicz M, Grodzicky T, Li Y, Panaritis C, et al. (2001) Traditional Framingham risk factors fail to fully account for accelerated atherosclerosis in systemic lupus erythematosus. Arthritis Rheum 44(10): 2331–2337.
17. Petri M, Perez-Gutthann S, Spence D, Hochberg MC (1992) Risk factors for coronary artery disease in patients with systemic lupus erythematosus. Am J Med 93(5): 513–519.
18. Rahman P, Aguero S, Gladman DD, Hallett D, Urowitz MB (2000) Vascular events in hypertensive patients with systemic lupus erythematosus. Lupus. 9(9): 672–675.
19. Bruce I, Urowitz M, Gladman D, Hallett D (1999) Natural history of hypercholesterolemia in systemic lupus erythematosus. J Rheumatol 26(10): 2137–2143.
20. Szabo M, Szodoray P, Kiss E (2017) Dyslipidemia in systemic lupus erythematosus. Immunol Res 65(2): 543-550.
21. Borba E, Carvalho J, Bonfa E (2006) Mechanisms of dyslipoproteinemias in systemic lupus erythematosus. Clin Dev Immunol 13(2-4): 203-208.
22. Meza-Meza MR, Vizmanos-Lamotte B, Munoz-Valle JF, Parra-Rojas I, Garaulet M, et al. (2019) Relationship of excess weight with clinical activity and dietary intake deficiencies in systemic lupus erythematosus patients. Nutrients 11(11): 2683.
23. Borges MC, dos Santos FdMM, Telles RW, Lanna CCD, Correia MITD. (2012) Nutritional status and food intake in patients with systemic lupus erythematosus. Nutrition 28(11–12): 1098–1103.

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24. Patterson SL, Schmajuk G, Jafri K, Yazdany J, Katz P (2019) Obesity is independently associated with worse patient-Reported outcomes in women with systemic lupus erythematosus. Arthritis Care & Research 71(1): 126-133.

25. Chung CP, Avalos I, Oeser A, Gebretsadik T, Shintani A, et al. (2007) High prevalence of the metabolic syndrome in patients with systemic lupus erythematosus: association with disease characteristics and cardiovascular risk factors. Ann Rheum Dis 66(2): 208–214.

26. Chen YM, Lin CH, Lan TH, Chen HH, Chang SN, et al. (2015) Hydroxychloroquine reduces risk of incident diabetes mellitus in lupus patients in a dose-dependent manner: a population-based cohort study. Rheumatology (Oxford) 54(7): 1244-1249.

27. Bastian H, Roseman J, Mcgwin G, Alarcon G, Friedman A, et al. (2002) Systemic lupus erythematosus in three ethnic groups. XII. Risk factors for lupus nephritis after diagnosis. Lupus 11(3): 152–160.

28. Bernatsky S, Boivin J, Joseph L, Manzi S, Ginzler E, et al. (2006) Mortality in systemic lupus erythematosus. Arthritis Rheum 54(8): 2550-2557.

29. Faurschou M, Starklint H, Halberg P, Jacobsen S (2006) Prognostic factors in lupus nephritis: Diagnostic and therapeutic delay increases the risk of terminal renal failure. J Rheumatol 33(8): 1563–1569.

30. Minihane AM, Vinoy S, Russell WR, Baka A, Roche HM, et al. (2015) Low-grade inflammation, diet composition and health: Current research evidence and its translation. Br J Nutr 114(7): 999–1012.

31. Qin B, Yang M, Fu H, Ma N, Wei T, et al. (2015) Body mass index and the risk of rheumatoid arthritis: A systematic review and dose-response meta-analysis. Arthritis Res Ther 17(1): 86.

32. Aparicio-Soto M, Sanchez-Hidalgo M, Alarcon-de-la-Lastra C (2017) An update on diet and nutritional factors in systemic Lupus erythematosus management. Nutr Res Rev 30(1): 118–137.

33. Constantin M, Nita IE, Olteanu R, Constantin T, Bucur S, et al. (2019) Significance and impact of dietary factors on systemic lupus erythematosus pathogenesis. Exp Ther Med 17(2): 1085–1090.

34. Selmi C (2010) Autoimmunity in 2009. Autoimmun Rev 9(12): 795–800.

35. Pan Y, Ke H, Yan Z, Geng Y, Asner N, et al. (2016) The Western-type diet induces anti-HMGB1 autoimmunity in Apoe-/- mice. Atherosclerosis 251: 31–38.

36. Mu Q, Zhang H, Luo X (2015) SLE: another autoimmune disorder influenced by microbes and diet? Front Immunol 6: 608.

37. Rodríguez Huerta M, Trujillo-Martín M, Rúa-Figueroa Í, Cuellar-Pompa L, Quirós-López R, et al. (2016) Healthy lifestyle habits for patients with systemic lupus erythematosus: a systemic review. Semin Arthritis Rheum 45(4): 463–470.

38. Barbhaiya M, Tedeschi S, Sparks JA, Leatherwood C, Karlson EW, et al. (2021) Association of dietary quality with risk of incident systemic lupus erythematosus in the nurses' health study and nurses' health study II. Arthritis Care Res 73(9): 1250-1258.

39. Yang C, Leung P, Adamopoulos I, Gershwin M (2013) The implication of vitamin D and autoimmunity: a comprehensive review. Clin Rev Allergy Immunol 45(2): 217–226.

40. Attar S, Siddiqui A (2013) Vitamin D deficiency in patients with systemic lupus erythematosus. Oman Med J 28(1): 42-47.

41. Kamen D (2010) Vitamin D in lupus: new kid on the block? Bull NYU Hosp Jt Dis 68(3): 218-222.

42. Pakpoor J, Pakpoor J (2013) Vitamin D deficiency and systemic lupus erythematosus: cause or consequence. Oman Med J 28(4): 295.

43. Kamen D, Cooper G, Bouali H, Shaftman S, Hollis B, et al. (2006) Vitamin D deficiency in systemic lupus erythematosus. Autoimmun Rev 5(2): 114-117.

44. Goldner B (2019) Six week raw vegan nutrition protocol rapidly reverses lupus nephritis: A case series. Int J Dis Rev Prev 1(1).

45. Klack K, Bonfa E, Borba Neto E (2012) Diet and nutritional aspects in systemic lupus erythematosus. Rev Bras Reumatol 52(3): 384-408.

46. Petri M (2001) Diet and systemic lupus erythematosus: From mouse and monkey to woman? Lupus 10(11): 775–777.

47. Reifen R, Amital H, Blank M, Sklan D, Berkovich Z, et al. (2000) Linseed oil suppresses the anti-beta-2-glycoprotein-I in experimental antiphospholipid syndrome. J Autoimmun 15(3): 381–385.

48. Vieira S, Pagovich O, Kriegel M (2014) Diet, microbiota and autoimmune diseases. Lupus 23(6): 518-526.

49. Petri M, Orbai A, Alarcon G, Gordon C, Merrill J, et al. (2012) Derivation and validation of the Systemic Lupus International Collaborating Clinics classification criteria for systemic lupus erythematosus. Arthritis Rheum 64(8): 2677-2686.

50. Giannelou M, Mavragani C (2017) Cardiovascular disease in systemic lupus erythematosus: A comprehensive update. J Autoimmun 82: 1–12.

51. Morrison LM (1960) Diet in Coronary Atherosclerosis. JAMA 173: 884-888.

52. Ornish D, Scherwitz L, Billings J, Brown S, Gould K, et al. (1998) Intensive lifestyle changes for reversal of coronary heart disease. JAMA 280(23): 2001-2007.

53. Esselstyn CJ, Ellis S, Medendorp S, Crowe T (1995) A strategy to arrest and reverse coronary artery disease: a 5-year longitudinal study of a single physician's practice. J Fam Pract 41(6): 560-568.

54. Esselstyn CJ, Gendy G, Doyle J, Golubic M, Roizen M (2014) A way to reverse CAD? J Fam Pract 63(7): 356-364b.

55. Gupta S, Sawhney R, Rai L, Chavan V, Dani S, et al. (2011) Regression of coronary atherosclerosis through healthy lifestyle in coronary artery disease patients--Mount Abu Open Heart Trial. Indian Heart J 63(5): 461-469.

56. Arntzenius A, Kromhout D, Barth J, Reiber J, Bruschke A, et al. (1985) Diet, lipoproteins, and the progression of coronary atherosclerosis -The Leiden Intervention Trial. New Eng J Med 312(13): 805-811.

57. Rose S, Strombom A (2018) A comprehensive review of the prevention and treatment of heart disease with a plant-based diet. J Cardiol & Cardiovas Ther 12(5): 555847.

58. Kwok C, Umar S, Myint P, Mamas M, Loke Y (2014) Vegetarian diet, Seventh Day Adventists and risk of cardiovascular mortality: a systematic review and meta-analysis. Int J Cardiol 176(3): 680-686.

59. Phillips R, Lemon F, Beeson W, Kuzma J (1978) coronary heart disease mortality among Seventh Day Adventists with differing dietary habits: a preliminary report. Am J Clin Nutr 31(10 Suppl): S191-S198.

60. Najjar R, Moore C, Montgomery B (2018) A defined, plant-based diet utilized in an outpatient cardiovascular clinic effectively treats hypercholesterolemia and hypertension and reduces medications. Clin Cardiol 41(3): 307-313.

61. Jenkins D, Kendall C, Marchie A, Faulkner D, Wong J, et al. (2005) Direct comparison of a dietary portfolio of cholesterol-lowering foods with a statin in hypercholesterolemic participants. Am J Clin Nutr 81(2): 380-387.

62. Macknin M, Kong T, Weier A, Worley S, Tang A, et al. (2015) Plant-based, no-added-fat or American Heart Association Diets: impact on cardiovascular risk in obese children with hypercholesterolemia and their parents. J Pediatrics 166(4): 53-959.

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63. Frattaroli J, Weidner G, Merritt-Worden T, Frenda S, Ornish S (2008) Angina pectoris and atherosclerotic risk factors in the multisite cardiac lifestyle intervention program. Am J Cardiol 101(7): 911-918.

64. Ellis F, Sanders T (1977) Angina and the Vegan Diet. Am Heart J 93(6): 803–805.

65. Strombom A, Rose S (2017) The prevention and treatment of Type II Diabetes Mellitus with a plant-based diet. Endocrin Metab Int J 5(5): 00138.

66. Barnard N, Cohen J, Jenkins D, Turner-McGrievy G, Gloede L, et al. (2006) A low-fat vegan diet improves glycemic control and cardiovascular risk factors in a randomized clinical trial in individuals with type 2 diabetes. Diabetes Care 29(8): 1777-1783.

67. Johansen K (1999) Efficacy of metformin in the treatment of NIDDM. Meta-analysis. Diabetes Care 22(1): 33-37.

68. Rose S, Strombom A (2020) Preventing stroke with a plant-based diet. Open Access J Neurol Neurosurg 14(2): 555882.

69. Rose S, Strombom A (2019) Osteoarthritis prevention and treatment with a plant-based diet. Ortho & Rheum Open Access J 15(3): 555914.

70. Rose S, Strombom A (2018) A plant-based diet prevents and treats prostate cancer. Canc Therapy & Oncol Int J 11(3): 555813.

71. Rose S, Strombom A (2019) Colorectal cancer prevention with a plant-based diet. Canc Therapy & Oncol Int J 15(2): 555906.

72. Rose S, Strombom A (2019) Diverticular Disease risk reduced with a plant-based diet. Adv Res Gastroenterol Hepatol 14(2): 555884.

73. Rose S, Strombom A (2020) Ulcerative Colitis – prevention and treatment with a plant-based diet. Adv Res Gastroentero Hepatol 15(2): 555908.

74. Rose S, Strombom A (2018) Crohn's disease prevention and treatment with a plant-based diet. Adv Res Gastroentero Hepatol 9(1): 555753.

75. Rose S, Strombom A (2019) A plant-based diet prevents and treats chronic kidney disease. JOJ Uro & Nephron 6(3): 555687.

76. Rose S, Strombom A (2020) Preventing thyroid diseases with a plant-based diet, while ensuring adequate iodine status. Glob J Oto 21(4): 556069.

77. Rose S, Strombom A (2018) Rheumatoid arthritis - prevention and treatment with a plant-based diet. Orth & Rheum Open Access J 13(1): 555852.

78. Tonstad S, Butler T, Yan R, Fraser G (2009) Type of vegetarian diet, body weight, and prevalence of type 2 diabetes. Diabetes Care 32(5): 791–796.

79. Bradbury K, Crowe F, Appleby P, Schmidt J, Travis R, et al. (2014) Serum concentrations of cholesterol, apolipoprotein A-I, and apolipoprotein B in a total of 1 694 meat-eaters, fish-eaters, vegetarians, and vegans. Eur J Clin Nutr 68(2): 178-183.

80. Waldmann A, Koschizke J, Leitzmann C, Hahn A (2005) German vegan study: diet, life-style factors, and cardiovascular risk profile. Ann Nutr Metab 49(6): 366-372.

81. Fryar C, Kruszon-Moran D, Gu Q, Ogden C (2018) Mean body weight, height, waist circumference, and body nass index among adults: United States, 1999-2000 through 2015-2016. Natl Health Stat Report 122: 1-16.

82. Robinson GA, Mcdonnell T, Wincup C, Martin-Gutierrez L, Wilton J, et al. (2019) Diet and lupus: what do the patients think? Lupus 28(6): 755-763.

83. Rose S, Strombom A (2020) Managing the nutritional requirements of vegetarian and vegan mothers during pregnancy. J Gynecol Women's Health 18(2): 555985.

84. Sakthiswary R, Raymond A (2013) The clinical significance of vitamin D in systemic lupus erythematosus: a systematic review. PLoS One 8(1): e55275.

85. Rose S, Strombom A (2019) Ensuring adequate vitamin D status for patients on a plant-based diet. Ortho & Rheum Open Access J 15(3): 555913.

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