

Impact of Diet on Cardiovascular Diseases: Coronary Artery Disease

Part III: Micronutrients and Supplements

Abstract

Micronutrients are essential for the proper physiological functioning of the human body. They are only needed in trace amounts. However, low, or excess serum levels can result in wide-ranging undesirable health effects and increase mortality. Further, some micronutrients like arsenic are toxic if the body is exposed to them. The most common micronutrients involved with coronary artery disease include minerals like sodium, potassium, magnesium, calcium, and iron. Several other dietary supplements may also affect coronary artery disease. These include fish oil, curcumin, and probiotics. Micronutrients are often misunderstood in their role in preventing and reducing the impact of CAD and often end up being misused. In parts 1 and 2, the various macronutrients and their relationship with coronary artery disease were discussed. This section looks at several micronutrients that play a modulating role in atherosclerosis and coronary artery disease.

Keywords: coronary artery disease, sodium, potassium, iron, probiotics, vitamins

Introduction

Globally, cardiovascular diseases (CVDs) are the leading cause of morbidity and mortality in the world¹. Ischemic heart disease (IHD) is a major player in CVDs. Its global burden is on the rise. According to GBD 2019, there were 197 million prevalent cases of IHD and 9.14 million deaths due to it in 2019¹. The World Health Organization (WHO) estimates that three-quarters of deaths due to CVDs can be prevented by controlling lifestyle risk factors². One lifestyle, diet, plays an important role in decreasing IHD burden. A study estimated that individuals can reduce their coronary heart disease (CHD) risk by 30% by following a heart-healthy dietary pattern³. A prudent diet can therefore play an important role in the quest for clean coronary arteries. The effect of macronutrients on CHD has been discussed earlier in Part I and Part II. This third and last **narrative review** will discuss several micronutrients and supplements that also play a role.

Discussion

Micronutrients (vitamins and minerals) are an essential component of the diet and are necessary for normal cellular and molecular function⁴. While micronutrients are only needed in trace amounts, their deficiency or excess can result in wide-ranging undesirable health effects⁵⁻⁷. These include developmental, and mental impairment, and several physical diseases including atherosclerotic coronary artery disease (CAD)⁸.

Sodium: Sodium plays a major role in the etiology and pathogenesis of hypertension (HTN)⁹. This relationship is well-established¹⁰. HTN is a major risk factor for CAD¹¹. HTN promotes

atherosclerosis¹². The worldwide sodium intake ranges between 3.5–5.5 g per day (corresponding to 9–12 g of salt per day). The WHO recommends that sodium intake should be limited to approximately 2.0 g per day (equivalent to approximately 5.0 g of salt per day) in healthy individuals¹³. A further decrease to less than 2 g/day (American Heart Association (AHA) recommends a reduction to 1.5 g/day) leads to a more significant blood pressure (BP) lowering: (- 3.39 mmHg for systolic BP and - 1.54 mmHg for diastolic BP)¹⁴. This helps reduce atherosclerotic cardiovascular disease (ASCVD)¹⁵. High BP is dangerous. A 2-mmHg increase in BP increases mortality from CAD by 7 percent¹⁶. A heart-healthy diet reduces sodium intake¹⁷.

Potassium: A high intake of K is inversely related to CAD and CAD mortality. There is an inverse association between potassium and blood pressure¹⁸⁻²⁰. A diet rich in potassium is associated with a reduction in BP²¹. In a study supported in part by the National Heart, Lung, and Blood Institute and the National Institute of Diabetes, Digestive and Kidney Diseases, mice fed a low-potassium diet demonstrated increased vascular calcification and artery stiffness²². In another study, it was estimated that for every 1,000 mg per day increase in urinary potassium excretion (indicating higher potassium intake), the risk of CVD (including CAD) was 18 percent lower²³.

Magnesium: A systematic review and meta-analysis of prospective studies that comprised 313,041 individuals, found that higher dietary magnesium (Mg) intakes (up to approximately 250 mg/day) were associated with a significantly lower risk of IHD²⁴. One study reported that higher dietary Mg (per 200 mg/day increment) was associated with a 22% lower risk of IHD (Risk Ratio or RR=0.78)²⁵. Another study showed that higher levels of serum Mg (per 0.2 mmol/L increment) are associated with a 17% lower risk of IHD (RR=0.83) and 39% lower risk of fatal IHD (RR=0.61)²⁶. Several other studies have found a similar inverse association^{27,28}. Higher Mg is also noted to be potentially protective against sudden cardiac death²⁹. Mg can prevent or delay atherosclerotic plaques³⁰. Mg is also involved in the production of nitric oxide and prostaglandins, thus protecting the vascular endothelium in the coronary arteries³¹. Several epidemiological studies also indicate an inverse relationship between serum and dietary Mg and HTN³², type 2 diabetes mellitus (T2DM)³³, and metabolic syndrome³⁴ – all risk factors for CAD.

Calcium: High calcium levels are statistically associated with a higher incidence of CHD³⁵. Several studies report that calcium supplementation resulting in higher calcium levels increases the risk of cardiovascular events, coronary artery calcification, CAD, and myocardial infarction (MI)^{36,37}. A genetic predisposition to higher serum calcium levels is also associated with an increased risk of CHD³⁸. Possible mechanisms include effects on vascular calcification, vascular cells, blood coagulation, and altered gene expression³⁹. Calcium supplements should not be used to prevent CAD/CHD.

Iron: Serum iron levels are related to the severity of CAD⁴⁰. Many clinical studies suggest that the level of myocardial iron is a prognostic factor of heart failure following MI⁴¹. Sullivan et al. (1981) reported that depletion of body Fe stores reduced the risk of CAD⁴². A high dietary iron intake and high serum level of ferritin are positively linked with MI⁴³. Supplementation has also been associated with an increased risk of nonfatal MI or fatal CAD⁴⁴. Iron plays an important

role in the production of free radicals and peroxidation of lipids, leading to oxidative stress, which leads to an increase in atherosclerosis^{45,46}.

Selenium: Several cohort studies have demonstrated a link between lower blood selenium (Se) levels and the increased occurrence of HTN and CAD⁴⁷. The vascular benefits of Se are based on its antioxidative and detoxification effects⁴⁸.

Manganese: Manganese (Mn) is an element essential for health in trace amounts, but toxic at higher levels. In atherosclerotic subjects, the plasma levels of Mn are higher than in healthy individuals⁴⁹⁻⁵¹. The urine of CAD patients also show higher Mn concentrations than that of healthy controls⁵². In another study, Mn concentration did not appear to be significantly increased in patients with CAD⁵³. Its association with CAD is therefore unclear.

Lead: Chronic exposure to low lead (Pb) levels results in HTN that persists long after the cessation of Pb exposure⁵⁴. Studies on Pb levels and CAD are sparse in the literature. Several small studies have identified a positive association of Pb exposure with CAD^{55,56}. In a recent study, it was found that mean levels of serum Pb tended to be higher in CAD patients⁵⁷.

Vitamins: Previous experimental and epidemiologic evidence suggested that some antioxidant vitamins appear to be important in reducing the risk of CAD⁵⁸. However, most individual, and multivitamin supplements have shown no efficacy in reducing CVD⁵⁹⁻⁶¹. A study examined the efficacy of multiple vitamins and minerals on the secondary prevention of 1708 post-MI patients (age ≥ 50 years, ≥ 6 weeks after MI). In this study, consumption of high-dose multivitamins and minerals did not significantly reduce secondary cardiovascular events⁶².

Vitamin D: Cholecalciferol deficiency is associated with increased cardiovascular risk (including CAD), above and beyond established cardiovascular risk factors⁶³. Clinical studies have reported a clear association between low cholecalciferol levels and CAD⁶⁴⁻⁷⁰. A meta-analysis calculated an adjusted relative risk of 1.4 for CAD when comparing the lowest to the highest categories of vitamin D levels. Recent large-scale observational studies have recognized that this relationship between plasma levels of 25(OH)D and coronary atherosclerosis is inverse in character. Further, greater vitamin D deficiency/insufficiency is associated with a more severe and extensive CAD⁷¹. Several mechanisms may explain this link between cholecalciferol deficiency and CVDs. Adequate levels of vitamin D are associated with decreased risk of endothelial dysfunction, calcification, and stiffness in the arteries⁷². Low cholecalciferol levels modulate plasma renin activity, raise blood pressure, increase insulin resistance, and lead to hyperlipidemia⁷³⁻⁷⁷. Vitamin D may also have a direct effect on angiogenesis^{78,79}, and help in the development of coronary collateral circulation⁸⁰. However, the results obtained from a review of relevant randomized control trials did not clearly show cardiovascular improvements following cholecalciferol supplementation⁸¹.

Cadmium: The literature is extremely sparse on cadmium (Cd) and CAD. One study reported that serum Cd levels were significantly decreased in patients with CAD⁷⁰.

Zinc: Zinc (Zn) is a major component of numerous enzymes within the human body. It controls the functioning of metalloenzymes, transcription factors, angiotensin-converting enzymes, desaturases, superoxide dismutases, and many others⁸². Consequently, deficiency of Zn leads to

apoptosis, inflammation, and oxidative stress, all well known risk factors for CVDs⁸³. Perturbations in Zn homeostasis affect the vascular endothelium⁸⁴. Proatherogenic factors, released during Zn deficiency, increase the incidence of arrhythmias, strokes, CM, and many other CV system pathologies^{85,86}. There is an inverse relationship between serum Zn concentrations and the risk of CVDs in high-risk populations⁸⁷. Zn affects oxidative stress and reduces the oxidation of low-density lipoprotein (LDL-C), which has a protective effect against atherosclerosis⁸⁸. In previous studies, it has been shown that in patients with CAD, serum Zn levels are lower than in healthy individuals. Kazemi et al in 2007, found that serum Zn level was significantly lower in coronary artery patients⁸⁹. In a meta-analysis, Liu, et al., found that low Zn intake was associated with a higher prevalence of CAD. It was also associated with a higher incidence of MI⁹⁰. In a study of patients undergoing coronary angiography, low serum Zn levels predicted mortality⁹¹. Although some studies have presented contradicting results⁹², it appears that low serum Zn levels are detrimental for the heart. One study linked them with increased cardiovascular mortality⁹³. Intracellular Zn plays a critical role in the redox signaling pathway, whereby certain triggers such as ischemia and infarction lead to the release of zinc from proteins and cause myocardial damage. In such states, replenishing with Zn has been shown to improve cardiac function and prevent further damage⁹⁴.

Copper: Copper (Cu) seriously affects the cardiovascular system, especially in the pathogenesis of CAD⁹⁵. In acute coronary syndrome there is a positive link between serum Cu levels and elevated troponin T, troponin I and CK-MB values⁹⁶. A MI is associated with a slight increase in serum Cu and a significant increase in urine Cu levels⁹⁷. Shokrzadeh et al. measured the levels of Cu in patients with ischemic cardiomyopathy and found higher serum Cu levels in these patients when compared to healthy subjects⁹⁸. Lutfi et al. did not find any significant association between CAD and Cu levels in Sudanese patients⁹⁹. A recent Mendelian randomization study found an inverse association for genetically higher Cu levels with risk of CAD (Odds ratio = 0.92)¹⁰⁰. Despite these studies, it is commonly believed that Cu levels are higher in patients with CAD. The lack of Cu leads to vascular elastic tissue degeneration and vascular smooth muscle proliferation, migration, and degeneration. It also impacts hypercholesterolemia and glucose tolerance and can increase chronic inflammation and oxidative stress¹⁰¹.

Supplements and CAD

Probiotics: Probiotics may have a role in preventing or reducing atherosclerosis and beneficially affect CAD/CHD. Stepankova, et al, demonstrated that some intestinal bacteria prevent or slow down the progression of atherosclerotic lesions¹⁰². Several species, such as *Eubacteria*, *Anaeroplasm*, *Roseburia*, *Oscillospira*, and *Dehalobacteria* are effective in preventing atherosclerosis. In mice, a reduction in atherosclerotic plaque has been noted Lactobacillus rhamnosus GG supplementation despite being fed a high-fat diet for 12 weeks¹⁰³. Probiotics may also help reduce BP, HbA1c, fasting blood glucose, and insulin resistance^{104,105}. In a review of 26 clinical studies and two meta-analyses, Shimizu et al. found that LDL-C was reduced by 8.9–11.6% with *L. reuteri* supplementation¹⁰⁶. LDL-C was lowered by 5% by *E. faecium* supplementation in mostly normal individuals¹⁰⁶. Selective bacteria given as a probiotic supplement may be of value in reducing atherosclerosis.

Multivitamins: In 2011, one large multiethnic study on 182,099 participants showed no significant association between multivitamin intake for >10 years and CVD risk¹⁰⁷. Antioxidant vitamins like vitamin C, vitamin E and beta carotene have no clinical benefit in reducing CAD – and their use in high doses is often harmful¹⁰⁸. A study examined the efficacy of multiple vitamins and minerals on the secondary prevention of 1708 post-MI patients (age ≥ 50 years, ≥ 6 weeks after MI)¹⁰⁹. The consumption of a high-dose multivitamin and multimineral did not significantly reduce secondary cardiovascular events¹⁰⁹. No current results conclusively support the use of vitamin D supplementation as a strategy for CAD protection¹¹⁰. Minerals taken as a supplement also have not shown any CAD benefit¹¹¹. Folic acid ingestion may be helpful. One recent Mendelian randomization study found that high genetically predicted folate levels were associated with decreased risk of CAD¹¹². Daily supplementation with 0.5-5.0 mg of folic acid typically lowers plasma homocysteine levels by approximately 25%¹¹³. Hyper-homocysteinemia can cause endothelial injury, dysfunction of DNA, proliferation of smooth muscle cells, oxidative stress, decreased functioning of glutathione peroxidase, impaired nitric oxide synthase, and inflammation – all detrimental to the coronary arteries¹¹⁴. These data suggest that folic acid supplementation may reduce CAD¹¹⁵. However, supplementation for healthy people is not recommended by the AHA. Adequate intake of vitamins and minerals in their natural form (for example, in fruits and vegetables) is, however is CAD protective. Therefore, the AHA recommends the consumption of vegetables and fruits, especially green and yellow vegetables, but not antioxidant vitamin supplementation, to prevent atherosclerotic diseases such as CAD¹¹⁶.

CoQ10: Supplementation with CoQ10 (300 mg/day) increases antioxidant activity and lowers inflammation in patients who have CAD and are on statins therapy¹¹⁷. A meta-analysis (eight trials with 267 participants in the intervention group and 259 in the placebo group) showed that taking CoQ10 by CAD patients significantly decreased total cholesterol and increased HDL-C levels¹¹⁸. In patients who were post-MI reperfusion, CoQ10 decreased ventricular arrhythmias, improved LV function, and reduced total cardiac death^{119,120}. Another study, a double-blind placebo-controlled trial of 144 subjects with acute MI, also showed that CoQ10 was beneficial when given 120 mg per day within the first 3 days of an MI¹²¹. However, its use as a supplement, in the prevention and management of CHD remains unclear.

Fish Oils: Omega-3 polyunsaturated fatty acids (PUFAs) primarily eicosapentaenoic acid (EPA), and docosahexaenoic acid (DHA), are derived from fish and are available as fish oils¹²². Several primary and secondary prevention trials have shown that omega-3 supplementation reduce several cardiovascular endpoints, such as angina, fatal and non-fatal MI, and sudden cardiac death¹²³⁻¹²⁶. Alexander et al. found that there was a 14%-16% CHD risk reduction in high-risk populations with their intake. Higher intakes of EPA + DHA > 1 g per day resulted in an 18% reduction of any CHD event, including sudden cardiac death. This higher intake also reduced CHD by 25% in individuals with triglycerides (TG) over 150 mg/dl¹²³. They also help reduce stent restenosis¹²⁴, and CABG occlusion^{125,126}. They can be used concomitantly with statins, for further stabilizing and promoting coronary plaque regression¹²⁷. PUFAs improve the lipid profile - decrease very low-density lipoproteins and increase HDL-C¹²⁸. In a dose of 2 to 4 gm/day (EPA + DHA), they also help reduce TG levels¹²⁹. They retard atherosclerosis, plaque formation, coronary artery calcification, and promote atherosclerotic plaque stability^{130,131}. They also lower glucose, improve insulin resistance¹³²⁻¹³⁴ and reduce BP^{135,136}. Eating oily fish may be

better, as this also provides vitamin D, selenium, and other naturally occurring antioxidants which are not found in purified fish oil supplements. The AHA recommends consuming 1 serving of fatty fish or 1g of EPA/DHA-containing supplements twice a week for good cardiovascular health¹¹⁶.

Aspirin: Aspirin (ASA) is a cyclooxygenase-1 (COX-1) inhibitor. It binds irreversibly to platelets¹³⁷. Low dose use has been used for both primary and secondary prevention of ASCVD. Recent clinical trials demonstrate an increased risk of bleeding (gastrointestinal and cerebral) associated with aspirin use, which often outweighed cardiovascular risk reduction in primary prevention^{138,139}. The ACC/AHA guidelines recently recommended that aspirin be used for primary prevention only in patients 40-70 years of age who are at a high atherosclerotic vascular disease (ASCVD) risk and have a low bleeding risk, and who are unable to optimally control modifiable ASCVD risk factors¹⁴⁰. Low-dose aspirin should not be administered on a routine basis for primary prevention of ASCVD among adults younger than 70 years¹⁴¹. The recommended dose is 81 mg daily after a **PTCI or CABG**¹⁴². Low dose ASA is available over-the-counter without a prescription. Many individuals use low dose aspirin routinely to prevent CAD/CHD – in view of the increased bleeding noted, this use should be cleared by a physician.

Psyllium: Psyllium husk, derived from the seeds of *Plantago ovata*, has a 70 to 30 ratio of soluble/ insoluble fiber. Psyllium consumption reduces several risk factors for CAD. It helps reduce BP. A meta-analysis of 11 trials with 592 participants revealed a significant reduction of 2.04 mmHg in systolic blood pressure with its use¹⁴³. There is an improvement in hypercholesterolemia^{144,145}, HbA1c, and fasting blood glucose¹⁴⁶. It slows gastric emptying and helps control body weight¹⁴⁷. In an umbrella review, it was noted that consuming the highest amounts of dietary fiber intake can significantly reduce the incidence and mortality from CVD¹⁴⁸. Lim and Lee showed that psyllium prevented MI in rats¹⁴⁹.

Garlic: Garlic supplementation with aged garlic extract reduces systolic BP by 7-16 mm Hg and diastolic BP by 5-9 mm Hg. Total cholesterol is reduced by 7.4-29.8 mg/dL¹⁵⁰. However, supplementation benefits in CAD are still unconfirmed.

Chlorella: Chlorella products contain numerous nutrients and vitamins, including vitamin D and B12. A meta-analysis of 19 randomized controlled trials including 797 subjects showed that Chlorella supplementation improves TC, LDL-C and HDL-C levels, BP, fasting blood glucose, and body mass index¹⁵¹. Chlorella has multiple nutrients and antioxidant compounds that cause these beneficial effects¹⁵².

Curcumin: Curcumin comes from turmeric. It has anti-inflammation properties and improves lipid levels¹⁵³. It increases LDL-C size – smaller LDL-C particles are easier to oxidize¹⁵⁴. LDL-C oxidation leads to atherosclerosis. Wongcharoen et al. reported that post **coronary artery bypass grafting (CABG)**, curcumin in a dose of 4g per day given 3 days before and 5 days after the procedure, reduced inflammation, and reduced MI post CABG from 30% to 13%¹⁵⁵.

L-carnitine: L-carnitine (LC) is a derivative of amino acids lysine and methionine. It plays a role in energy metabolism and mitochondrial protection¹⁵⁶. It facilitates the transport of long-chain fatty acids into the mitochondrial matrix. This is associated with reduced oxidative stress,

inflammation, and necrosis of cardiac myocytes^{157,158}. Clinical studies show that LC supplementation (1000 mg/d) increases HDL-C and Apo-A1 levels with a slight decrease in TG levels¹⁵⁹. In one systemic review and meta-analysis (13 controlled trials with 3629 participants), supplementation with LC reduced angina by 40% and ventricular arrhythmias by 65% following an acute MI compared with placebo¹⁶⁰. However, there is not enough data to currently recommend its use in patients with CHD.

Vitamin K: Vitamin K is an essential bioactive compound required for optimal body function. Vitamin K has two main forms, namely, phyloquinone (K1) and menaquinones (K2). K1 is found primarily in green, leafy, and cruciferous vegetables. Vitamin K2 is found in some dairy products, pork, poultry, and fermented foods. Vitamin K2 is considered more important for vascular system health than vitamin K1. Vitamin K2 reduced incident CHD in a population of 4807 participants by 57%, when the upper versus lower tertile were compared¹⁶¹. Several studies have also documented the benefits of vitamin K1. A 16% reduction in CHD was noted in an NHS cohort of 72,874 female nurses, from lowest to highest quintile of vitamin K1 intake¹⁶². A similar reduction in CHD (13%-16%) was noted in a study of 40,087 men¹⁶³. In the Multi-Ethnic Study of Atherosclerosis study, there was an inverse association between vitamin K1 dietary intake and coronary artery calcification, especially in those on antihypertensive drugs¹⁶⁴. More studies are needed to determine the efficacy and proper supplementation dose. Both K1 and K2 are safely tolerated.

In summary, numerous nutritional supplements have demonstrated improvement in surrogate endpoints (BP, lipids, glucose, carotid IMT, coronary calcification, etc.). However, there are limited data that dietary supplements reduce hard CV endpoints related to CHD and MI. Supplements can have unpleasant and often dangerous side effects. They can also interact with prescribed medication. Consuming too much vitamin D or calcium increases CAD. Supplements that are over-the-counter are not regulated and may have a poor quality. Instead of relying on supplements, a healthy diet provides a full complement of micronutrients needed for healthy coronary arteries.

Diet and other diseases/lifestyle factors

Besides obesity¹⁶⁵, type 2 diabetes mellitus¹⁶⁶, dyslipidemia¹⁶⁷, and HTN¹⁶⁸, a prudent diet also beneficially impacts other risk factors for CAD/CHD. These include depression¹⁶⁹, improper sleep¹⁷⁰, chronic kidney disease¹⁷¹, and smoking¹⁷².

Conclusion

Coronary artery disease is a major cause of morbidity and mortality around the world. Although abstinence from tobacco smoking and physical exercise plays an important role in reducing this burden, the part played by diet is extremely significant. The calories in the diet should not exceed the caloric expenditure, to avoid becoming overweight or obese. Ideally, the BMI should be normal and there should be no visceral obesity. Even a weight loss of 5–10% of body weight if overweight/obese reduces blood pressure and induces beneficial metabolic changes. The diet should also be balanced. Most professional nutrition associations recommend that a normal diet comprise of macronutrients in the following percentages: 45%-65% carbohydrates; 10%-30%

from proteins and 20%-30% from fats. An adequate intake of water (approximately 6 glasses per day) and micronutrients is also required. The ingredients in this balanced diet also matter. Ideally, the diet should be rich in vegetables (dark green, red, yellow, orange), fruits (preferably whole fruits), legumes (beans and peas), unsalted nuts (almonds, peanuts, pecans, pistachios, hazelnuts), whole grains, and fiber. The plant-based diet should provide an intake of viscous fiber of 5–10 g/day and plant sterols/stanols of 2 g/day. Meat should be eaten infrequently, with red meat restricted to lean cuts, and avoiding processed red meat. One egg a day appears to be safe in non-diabetics (and maybe even diabetics). Seafood intake, especially oily fish (DHA and EPA) is encouraged. Fat-free or low-fat dairy, including milk, yogurt, cheese are not restricted. Dietary saturated fat (fatty cuts of lamb, pork, beef, poultry with skin, beef fat, lard, bacon, sausage, hotdogs. whole milk & whole milk products: butter, ghee, cheese, cream, ice cream, yogurt made from whole milk; palm oil, palm kernel oil, coconut oil, and coconut cream) should be replaced with monounsaturated fatty acids and polyunsaturated fatty acids. Extra virgin olive oil and canola oil are good sources of monounsaturated fats. Plant-based omega-6 polyunsaturated fats are present in corn, safflower, sunflower, soybean oils, and sunflower seeds. Plant-based omega-3 polyunsaturated fats are available in flaxseed oil, canola oil, soybean oil, English walnuts, edamame, hemp seeds, chia seeds, flaxseeds, and fenugreek seeds. Overall, saturated fat intake should be reduced to 7% of total energy and dietary cholesterol to <200 mg/day. Trans fats (often labeled as partially hydrogenated fats) should be avoided (baked goods: pastries, cakes, donuts, cookies, fried foods: French fries, fried chicken, onion rings, and deep-fried snacks cooked in re-used oil, stick margarine, shortening; butter, meat, cheese, and dairy products). Refined carbohydrates and sugar-sweetened beverages should be avoided. Added sugars should be reduced to <10% of the total energy intake. Sodium intake should be reduced to 2300 mg per day (ideally to 1500 mg/day). Alcohol should be imbibed in low to moderate amounts. Filtered coffee, green tea, and dark chocolate intake are also beneficial. Supplementation with potassium, magnesium, iron, calcium, and vitamins is not recommended unless there is a deficiency. The role of probiotics is still unclear. Several common diets, such as the DASH diet, Mediterranean diet, and the vegetarian diet have CAD preventive effects. Plant-based diets are the only dietary pattern to have shown regression of atherosclerosis. A prudent diet will also help mitigate systemic atherosclerosis and its common manifestations - stroke, peripheral artery disease, and vasculogenic erectile dysfunction.

Ethical Approval:

As per international standard or university standard ethical approval has been collected and preserved by the authors.

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