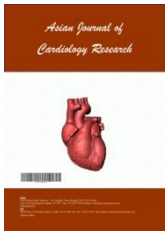

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Impact of Diet on Cardiovascular Diseases: Coronary Artery Disease Risk Factors

Shashi K. Agarwal [a*]

[a] *2227 US Highway 1, Suite 309, North Brunswick, NJ 08902, USA.*

Author's contribution

The sole author designed, analysed, interpreted and prepared the manuscript.

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ABSTRACT

Coronary artery disease is a leading cause of morbidity and mortality in the world. It is estimated that a large majority of coronary artery disease can be prevented by following healthy lifestyles. Healthy lifestyles are also linked to favorable changes in modifiable risk factors. These include hypertension, diabetes mellitus, dyslipidemia, and obesity. Since atherosclerosis starts in childhood, major lifestyle changes should be initiated at an early age. Modification of the diet is especially important at this age. Data accumulated from several meta-analyses indicate that a plant-based diet rich in fruits and vegetables, whole grains, legumes, nuts, and polyunsaturated fats are heart-healthy. On the other hand, diets rich in red meat, especially processed red meat, saturated fats, sodium, refined carbohydrates, and sugar-sweetened beverages are heart unhealthy. The role of the Mediterranean diet, DASH diet, and the vegetarian diet is also important in reducing the burden of coronary artery disease. This manuscript complements the three-part series on diet and coronary artery disease published earlier.

Keywords: Coronary artery disease; obesity; hypertension; dyslipidemia; diabetes mellitus; depression.

**Corresponding author: Email: usacardiologist@gmail.com;*

1. INTRODUCTION

Cardiovascular diseases (CVDs) are the major cause of suffering and death in the world [1]. Coronary artery disease (CAD or coronary heart disease: CHD) is the most common CVD [2]. It has a global prevalence of 2%–3% [3] and is the foremost single cause of loss of Disability Adjusted Life Years (DALYs) and mortality [4,5]. Much of this burden falls on low- and middle-income countries [6]. It is estimated that it affected 110 million people in 2015, was responsible for 164.0 million DALYs, and caused 8.92 million deaths [7]. Although CAD burden decreased in the high-income countries between 1990 and 2017, the rising world population and the increasing number of aged individuals, continue to increase the global crude numbers [8]. Low- and middle-income countries are expected to account for 82% of the future increase in CAD related mortality [9]. According to the World Heart Federation, the global cost of CVD in 2010 was approximately US$863 billion, which is expected to rise to more than US $1 trillion by 2030. In the low and middle-income countries, health expenditure (for CAD) is as high as 10% of the total healthcare expenditure [10].

More than 70% of at-risk individuals have multiple risk factors for IHD, and only 2%-7% of the general population have no risk factors [11]. These are mainly lifestyle factors, and it is postulated that with adequate changes in diet and lifestyle, about 80% of (premature) CVD mortality may be prevented [12]. Diet is one lifestyle that is a major driving force in the pathogenesis of atherosclerosis [13]. A healthy diet also significantly decreases the risk of CAD by attenuating several modifiable CAD-related risk factors.

2. DISCUSSION

Nutrition plays an increasing role in the primary and secondary prevention of several diseases, including CVDs [14]. The nutrition cardiovascular connection was first recognized in 1908, when a Russian scientist Alexander Ingatowski, demonstrated that high cholesterol intake caused the development of atherosclerosis in rabbits [15]. Atherosclerosis is the primary cause of coronary artery disease, and this can be attenuated [16] or even reversed with the intake of a healthy diet [17]. A healthy diet should not result in excess body weight (both BMI and WC should be normal) and should also be balanced, providing adequate macronutrients and micronutrients, with a good intake of water. Diets that are primarily plant-based, rich in whole grains, legumes, nuts, and fiber are heart-healthy. They have high amounts of polyunsaturated fats. These diets may include limited amounts of lean red meat and one egg a day. Seafood, low-fat dairy foods are safe. Coffee, tea, and chocolate intake are also beneficial. However, diets rich in red meat, especially processed red meat, saturated fats, trans fats, high levels of sodium and sugar, especially sugar-sweetened beverages and refined carbohydrates are harmful. Several common diets, such as the DASH diet, Mediterranean diet (MedD), and the vegetarian diet have CAD preventive effects. As mentioned in part II of the Diet and CVD: CAD series of articles, the MedD involves very low consumption of red meat (beef, pork, and lamb are reserved only for special occasions), very low or no consumption of processed meats, occasional intake of poultry, low or no consumption of butter, ice cream, or other whole-fat dairy products (only fermented dairy products, cheese, and yogurt, are consumed in moderate amounts). It includes an abundant consumption of olive oil, especially extra-virgin olive oil, together with high consumption of minimally processed, locally grown, fresh vegetables, fresh fruits, nuts, legumes, and cereals (mainly unrefined). An important source of protein is a moderate consumption of fish and shellfish. The diet also includes a moderate consumption of wine. DASH diet is rich in fruits, vegetables, whole grains, legumes, and nuts. It also includes low-fat dairy. seafood, skinless poultry, moderate intake of alcohol (for adults). It is low in red and processed meats, saturated fats, refined grains, sodium, and sugar-sweetened foods and beverages. Its focus is on reducing sodium intake for a beneficial effect on hypertension (HTN). The Western diet is a typical example of unhealthy food intake. It is higher in the intake of red meat, refined grains, processed meat, French fries, sweets and dessert, high-fat dairy products, and sugar-sweetened drinks. It is low in green leafy vegetables and fruits. Studies indicate that in contrast to the MedD and DASH diets, the Western diet significantly increases the LDL-C, TC, fasting TG, BP, BMI, and waist circumference. A heart-healthy diet also helps reduce several major modifiable risk factors for CAD.

2.1 Obesity

Obesity is an independent risk factor for CHD [18]. Weight gain increases the risk of CHD

events and CHD mortality [19]. A 10 kg rise in body weight increases the risk of CAD by 12% [20]. When the BMI exceeds 30 Kg/m^2, there is a 44% increase in risk for myocardial infarction21. Further, obese patients have more complex CHD disease and do not thrive well [22]. Following CABG, obese patients had a higher incidence of deep sternal wound infections [23]. Obese patients with CAD, also have higher mortality and unsatisfactory neurological recovery following a cardiogenic arrest recovery [24]. Adipocytokines from the fatty tissue in overweight/obese individuals induce insulin resistance, endothelial dysfunction, hypercoagulability, and systemic inflammation, promoting the atherosclerotic process [25]. Weight loss in obese CHD patients results in marked improvements in several CHD risk factors such as hypertension, CRP, lipids, insulin resistance, oxidative stress, and thrombogenicity [26]. This results in a lower incidence of CHD events, and reduced CHD mortality [27]. Dietary modification is central to the prevention and treatment of obesity [28,29]. One pound of weight is equal to 3500 Kcal [30]. Increasing calorie intake will therefore increase body weight, if not balanced by increased expenditure. Similarly, reducing caloric intake will induce weight loss. Many weight-loss diets work on this law of thermodynamics [31,32]. Restricting or increasing certain macro-ingredients in the diet also helps reduce weight through several complex mechanisms [33]. Some non-calorie and non-macronutrient restricted diets (such as the Mediterranean and vegetarian/vegan) are associated with a decreased risk of obesity [34,35]. Obesity paradox is an unusual phenomenon, where in an already developed disease, excess weight and obesity are associated with a favorable prognosis. The obesity paradox has also been noted in CAD [36]. Its discussion is beyond the scope of this manuscript.

3. HYPERTENSION

Hypertension is a major risk factor for CAD [37]. The risk for MI doubles when the systolic pressure is > 140 mm Hg when compared to that between 120 mm Hg and 129 mm Hg [38]. Similarly, compared to a diastolic pressure of <80 mm Hg, the risk ratio for CAD doubles if it increases above 94 mm Hg [39]. Hypertension is common in patients with CAD. In a large analysis of over 122,000 patients from 14 international randomized trials, the prevalence of hypertension in those with established CAD ranged from 30% to 70% [40]. Adverse clinical outcomes are higher in CAD patients if they have co-existing hypertension [41]. Studies have demonstrated that patients requiring more medications for blood pressure (BP) control or those with resistant or uncontrolled HTN have higher rates of adverse CAD outcomes, including all-cause death and nonfatal myocardial infarction [42]. It is estimated that the mortality rate of CHD is 2.3 times greater when hypertension is present [43]. Treatment of HTN results in a decrease in adverse cardiovascular outcomes (both with clinical and subclinical CAD). It is estimated that a modest short-term reduction in blood pressure confers a reduction in CAD events by about 16% [44]. Dietary modifications to reduce hypertension include decreased salt intake, weight loss, and moderation of alcohol consumption (among those who drink). The salutary effects of sodium restriction on BP are well established [45,46]. A meta-analysis (34 trials and 3230 participants) found that a sodium intake reduction of 4.4 g/day decreased BP by 5.4/2.8 mmHg in hypertensive individuals and 2.4/1.0 mmHg in normotensive individuals [47]. Besides its action in reducing HTN, sodium also directly damages target organs via multiple intricate pathways. In contrast to the Western diet, DASH [48], Mediterranean (MedD) [49], vegetarian [50], and Paleolithic [51] diets also help reduce BP. Hypertension exacerbates atherosclerosis by increasing sympathetic drive centrally via activation of the renin-angiotensin-aldosterone system. It increases oxidative stress, inflammatory cytokines, and endothelial and microvascular dysfunction [52]. It also causes a deficiency in vasodilators such as nitric oxide and prostacyclin. Hypertension may also increase afterload and result in left ventricular hypertrophy, which may compromise coronary blood circulation during diastole [53]. Hypertension is also frequently associated with insulin resistance and dyslipidemia, which are additional risk factors of atherosclerosis.

4. DIABETES MELLITUS

CAD is the leading cause of morbidity in diabetic patients [54]. The atherosclerotic process occurs earlier and progresses at a greater rate and extent in the diabetic than in the non-diabetic population. Studies indicate that diabetic patients have more three-vessel disease and a more complicated coronary artery involvement than their nondiabetic counterparts [55,56]. They also have a higher CAD-related mortality [57]. Post-myocardial infarction, diabetic patients have a twofold increase in 30-day and five-year mortality

[58,59]. They also have a higher risk for complications post-intervention, including mortality, than nondiabetic subjects [60,61]. Diabetics also have a high prevalence (in 1 in five diabetics) of asymptomatic CAD [62]. Diabetes is now regarded as a CAD risk equivalent [63]. The role of diet in the pathogenesis of T2DM is well known [64]. Maintenance of ideal body weight is important to prevent or control diabetes mellitus [65]. Even a 5 to 7 percent weight loss markedly lowers the risk of progressing to diabetes in prediabetes [66]. Weight loss helps reduce DM by several mechanisms that include reducing ectopic fat [67], reducing glucotoxicity [68,69], and decreasing the demands on beta-cells [70]. The quality of diet is also extremely important. Indians found that T2DM was almost confined to the rich people who consumed oil, refined flour, and sugar in excessive amounts [71]. Diets that involve ingesting low amounts of saturated fat and refined carbohydrates, including sugar, are associated with a reduced incidence of T2DM [72]. On the other hand, meat-based diets are harmful. In one study, long-term adherence (over a 17-year interval) to a diet that included at least weekly meat intake was associated with a 74% increase (OR = 1.74) in odds of diabetes (compared to those adhering long term to a (no meat) vegetarian diet) [73]. It has been observed that vegetarian diets are inversely associated with the risk of developing T2DM [74]. The Adventist Society reported a 74% reduction in diabetes incidence from a vegetarian diet when compared to a meat-based diet in a 17-year follow-up [75]. Another study put this reduction at 35% [76]. In the Rotterdam study, T2DM risk was reduced by 18% with a vegetarian-based diet after a follow-up of four to seven years [77]. Studies show that lower glycemic index (GI) diets help improve insulin secretion, insulin resistance, body weight, composition, and HbA1c levels [78,79]. GI reflects the glycemic impact of the carbohydrates in a diet (irrespective of quantity) while glycemic load (GL) represents the overall glycemic impact considering both quantity and quality of carbohydrate. Both – high GI and high GL foods are associated with increased incidence of T2DM [80] and CAD [81]. Two common diets used to reduce CVDs, MedD [82] and DASH [83] are also helpful in reducing T2DM. The ATTICA study reported improved fasting glucose and improved insulin levels (15% reduction) with an adherence to the Mediterranean diet [84]. Intake of extra virgin olive oil, a major component of MedD, helps reduce both plasma glucose and HbA1c [85]. It is estimated that MedD reduces HbA1c by 0.32 to 0.53 percentage units [86]. Med D shows these benefits independent of weight loss. Putative mechanisms include improvements in insulin sensitivity via a reduction in inflammation87 and beneficial effects of fatty acids [88] and phenolic compounds on the beta-cell [89]. The DASH diet, commonly used to decrease sodium intake for the treatment of HTN, also helps against DM. In a systemic review and meta-analysis of five prospective cohort studies (n = 158,408) the consumption of a DASH dietary pattern reduced diabetes incidence by 18% (RR = 0.82) [90]. A vegetarian diet is also helpful [91]. There is, however, little scientific evidence regarding the effects of the Paleolithic diet on T2DM incidence [92].

5. DYSLIPIDEMIA

Lipoproteins are intricately involved in the development and progression of atherosclerosis [93,94]. Increased serum LDL-C is commonly pathogenic in CAD [95]. There is a linear relationship between the plasma concentration of LDL-C and coronary events [96]. The serum LDL-C level should be under 100 mg/dL in high-risk patients, and under 70 mg/dL in very high-risk patients97. There appears to be no threshold below which a further reduction in LDL-C leads to no further atherosclerotic vascular disease prevention [98,99]. Hence LDL-C may be safely lowered as much as possible [100]. Other lipid parameters often associated with an increased risk of CAD include high total cholesterol (TC), high triglycerides (TGs), a decreased level of HDL-C, high TG/HDL-cholesterol ratio, and high TC/HDL-cholesterol ratio [101,102]. Dietary intervention is often used as the initial treatment for dyslipidemia [103-105]. The Western diet is high in saturated fatty acids (SAFA). Replacing SAFA with unsaturated fatty acids in the diet (especially with polyunsaturated fatty acids (PUFA), lowers LDL-C without affecting HDL-C and TGs [106-108]. Replacing SAFA with carbohydrates does not improve the overall blood lipid profile. It lowers LDL-C but also lowers HDL-C and raises fasting TGs [109]. Supplemental intake of omega 3 PUFAs (EPA and DHA), from fish oil, helps lower TG concentrations [110]. Another component of the diet that significantly affects the lipid profile is soluble fiber (SF). It has been calculated that an intake of 4–10 g/day of SF results in a 5–10% reduction in LDL-C without substantially affecting HDL-C and TG concentrations [111]. Reducing SAFA and increasing SF intake can also be

achieved by following several special diets. The MED dietary pattern significantly lowers LDL-C by −0.07 mmol/L and TGs by −0.46 mmol/L [112] - this beneficial change was however not seen in another recent study [113]. MedD adherence also improves HDL-C levels [114]. The DASH diet is also good for dyslipidemia. Siervo et al. however, in a meta-analysis of 1917 participants observed a reduction in TC (-0.20 mmol/L) and LDL-C levels after the DASH intervention (−0.10 mmol/L) while there were no significant differences in HDL-c and triglyceride levels [115]. Similar results were obtained in a recently published controlled trial in 80 T2DM patients after 12 weeks following the DASH diet [116]. A 2019 umbrella review of systematic reviews and meta-analyses concluded that the DASH diet lowered the TC by −0.20 mmol/L and LDL-C by −0.10 mmol/L - without affecting HDL-C and TGs [117]. Two meta-analyses of randomized controlled trials (RCTs) found that vegetarian diets significantly lowered TC by −0.32 to −0.36 mmol/L and LDL-C by −0.32 to −0.34 mmol/L [118,119]. Vegetarian diets in these studies however also lowered HDL-C by −0.09 to −0.10 mmol/L while TGs were not significantly altered. A recent meta-analysis of RCTs in T2DM patients found that vegetarian diet patterns led to a reduction in LDL-C of −0.12 mmol/L with no significant effects on HDL-C and TGs [120]. In a recent meta-analysis of 36 RCTs, the substitution of red meat with plant-based protein foods showed a reduction in total cholesterol and LDL-C levels [121]. Ketogenic diets also appear to reduce TG levels (-0.18 mmol/L and increase HDL-C levels (+0.09 mmol/L) [122].

6. SLEEP DISTURBANCES

Abnormal sleep duration or disturbed sleep increases the risk of CAD. A sleep duration of ≤6 hours/night or ≥10 hours/night of sleep results in a higher prevalence of CHD, when compared to a reference group sleeping 7 to 9 hours [123]. In another study, the risk of CHD, at the end of a 10-year follow-up was 1.39-fold higher in women reporting <5 hours/night and 1.37-fold higher in those sleeping >9 hours compared to those sleeping 8 hours/night [124]. Similar data were reported in the Women's Health Initiative Study. In this study, compared to 7–8 hours of sleep per night, self-reported sleep of <5 hours or >10 hours, was associated with a 25% and a 43% respectively, raised risk for CHD [125]. Abnormal sleep duration also affects mortality. Cappuccio and group reported that fatal CHD was greater in those with a habitual sleep duration above or below 7 to 8 hours [126]. Several dietary components may affect sleep [127]. Tryptophan consumption improves sleep [128]. This amino acid is present in milk [129]. Conversely, the depletion of tryptophan has been shown to reduce sleep quality [130]. Tryptophan crosses the blood-brain barrier, where it is converted to serotonin, the precursor to the sleep-promoting hormone, melatonin [131]. Melatonin and serotonin intake in foods such as cherries is also associated with improvements in sleep duration and quality [132]. Sleep may also be disturbed by abnormal respiration, such as seen in obstructive sleep apnea (OSA) [133]. In a meta-analysis, it was noted that OSA resulted in an increased risk of incident clinically overt CAD in adults, especially in men [134,135]. Sleep apnea is estimated to confer a two-to threefold increase in poor cardiovascular outcomes and in all-cause mortality136. A Spanish study of 1,500 patients (10-year follow-up) demonstrated a reduction in coronary artery disease with CPAP treatment in these patients [137]. Obesity plays an important role in OSA, with the incidence of OSA in people with obesity being as high as 30% [138]. Weight loss in these patients, either by dietary intervention or by bariatric surgery leads to a reduction in apnea-hypopnea index (AHI) [139,140]. In one study, the benefits in AHI persisted over 4-years despite patients regaining 50% of the lost weight by the 4th year [141]. The mechanisms behind OSA leading to a higher incidence of CAD may be related to the intermittent hypoxia causing sympathetic nervous system overactivity and increased inflammation, oxidative stress, and endoplasmic reticulum stress [142].

7. SMOKING AND PHYSICAL INACTIVITY

Smoking and inadequate physical activity are both connected with unhealthy diets. Smokers eat poorly. Alkerwi et al. found an inverse relationship between the intensity of tobacco consumption and overall diet quality [143]. Smokers also tend to have less physical activity, drink more, and are less adherent to special diets [144,145]. People with healthier diets and less obesity are more likely to be more active [146]. The reverse is also relevant as more active people are less likely to be obese.

8. CHRONIC KIDNEY DISEASE

Chronic Kidney disease (CKD) patients have a five to tenfold higher risk for developing CAD [147]. Besides the traditional risk factors such as

hypertension, diabetes mellitus, obesity, etc. CKD patients are also exposed to many nontraditional, uremia-related risk factors, such as an abnormal calcium-phosphorus metabolism [148]. There is an abrupt decline in CAD risk after kidney transplant, despite years of exposure to traditional CAD risk factors [149]. In early CKD stages, the adoption of a healthy diet (primarily plant-based - a high intake of vegetables, fruits, nuts, whole grains, legumes, fish, fiber, and polyunsaturated fatty acids but low in saturated fatty acids and sodium.) slows glomerular filtration rate (GFR) decline and progression to end-stage renal disease (ESRD)150,151. Mortality is also reduced [152,153]. Salt restriction in CKD patients is beneficial irrespective of its antihypertensive effects [154]. Western diets include a substantial amount of meat and salt. Consumption of two or more servings per week of red meat was associated with more microalbuminuria and a faster decline in estimated GFR [155,156]. The consumption of two or more high-sugar content beverages has been reported to increase the risk of glomerular filtration impairment and proteinuria [157]. High consumption of sugary drinks/sodas leads to a faster decline in the GFR [158]. Fructose is especially harmful in CKD [159]. Brymora et al. demonstrated diminished inflammatory parameters, as well as lower fasting serum insulin levels and BP in patients with stage 2 and 3 CKD after they were switched to a low-fructose diet for 6 weeks [160]. A low protein diet offers a variety of clinical benefits in patients with renal insufficiency. A high protein diet may increase kidney damage [161]. Therefore, regular counseling with a nutritionist is required in these patients to ensure adequate protein and energy intake and to avoid or correct protein-energy-related wasting.

9. DEPRESSION

Several studies have shown a depression CAD link. In one meta-analysis of several prospective studies of individuals initially free of CHD, depression increased the risk of incident CHD by 30% [162]. The presence of depression in CHD patients also prognosticates poor outcomes, including a higher incidence of chest pain and shortness of breath, higher re-hospitalization rates, and a greater risk of non-fatal cardiovascular events [163,164]. Depression significantly affects functionality after an MI and worsens the QOL165. Patients with CHD and associated depression also have significantly increased mortality after an angiographically confirmed diagnosis [166-168], following an acute coronary syndrome [169] and following coronary artery bypass graft surgery [170]. Analyses of many trials indicate that prognosis improves in these patients when depression improves. Several observational and clinical studies have shown that diet plays an important role in the pathogenesis of depressive disorders. Plant-based diets, rich in olive oil, whole grain, fish, low-fat dairy, and low in animal foods lead to a decreased risk of depression [171,172]. A Western diet, rich in red and/or processed meat, saturated fats, refined grains, sweets, high-fat dairy products, and low in fruits and vegetables is associated with an increased risk of depression [173,174]. Obesity, often a consequence of poor diet, also increases the risk of depression [175]. On the other hand, individuals with depression are also more likely to consume poorer quality diets [176] and more likely to become obesity [177].

10. PSYCHOLOGICAL STRESS

Prospective studies show that chronic stress or long-term repetitive stress exposure (such as job stress, marital unhappiness, caregiving burden, or feeling of being treated unfairly) [178] is associated with a 40–50% increase in CAD [179,180]. Acute mental stress (for instance, anger, emotional turbulence) is more common and results in more cardiovascular events and more severe events, compared to chronic stress [181-183]. Stress impacts diet negatively. Stress decreases diet quality and contributes to consuming unhealthy foods such as fast foods, sweets, and other fat and energy-dense foods. The increased consumption of these hyper-palatable foods that are low nutrient-dense (e.g., butter, cream cheese, full-fat products), occurs in stressed individuals even when there is no hunger or bodily demand for food [184,185]. Further, stress lowers the intake of fruits, vegetables, fish, and unsaturated oils [186]. Poor diet quality is detrimental to the coronary vascular system. Stress can also affect the amount of food intake. Stress often results in over-eating [187] and binge-eating188. This leads to obesity, which as discussed above, is harmful to CAD [189].

11. CONCLUSION

Diet has direct effects on the coronary vasculature. However, there are several major modifiable risk factors for CAD that are also affected. These include obesity, hypertension,

diabetes mellitus, dyslipidemias, improper sleep, chronic kidney disease, and mood disorders. Even smoking and low physical activity relate to diet. Many of these relationships are bidirectional. Overall, a plant-based diet with small amounts of lean meat, the absence of trans fats, and low in sodium and added sugars is helpful. MedD, DASH, and vegetarian/vegan diets are also useful in combating these risk factors.

CONSENT AND ETHICAL APPROVAL

It is not applicable.

COMPETING INTERESTS

Author has declared that no competing interests exist.

REFERENCES

1. Available:https://www.who.int/news-room/fact-sheets/detail/the-top-10-causes-of-death. Accessed 12 Apr 2020.
2. Wirtz PH, von Kanel R. Psychological stress, inflammation, and coronary heart disease. Curr Cardiol Rep. 2017;19:111. DOI: 10.1007/s11886-017-0919-x.
3. Shahjehan RD, Bhutta BS. Coronary Artery Disease. [Updated 2021 Aug 7]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2021. Available: https://www.ncbi.nlm.nih.gov/books/NBK564304/.
4. Khan MA, Hashim MJ, Mustafa H, et al. Global Epidemiology of Ischemic Heart Disease: Results from the Global Burden of Disease Study. Cureus. 2020 Jul 23;12(7):e9349. DOI: 10.7759/cureus.9349.
5. Virani SS, Alonso A, Benjamin EJ, et al. American heart association council on epidemiology and prevention statistics committee and stroke statistics subcommittee. Heart Disease and Stroke Statistics-2020 Update: A Report From the American Heart Association. Circulation. 2020 Mar 3;141(9):e139-e596. DOI: 10.1161/CIR.0000000000000757.
6. Chow CK, Nguyen TN, Marschner S, et al. PURE Study. Availability and affordability of medicines and cardiovascular outcomes in 21 high-income, middle-income and low-income countries. BMJ Glob Health. 2020 Nov;5(11):e002640. DOI: 10.1136/bmjgh-2020-002640.
7. Zhang G, Yu C, Zhou M, Wang L, Zhang Y, Luo L. Burden of ischaemic heart disease and attributable risk factors in China from 1990 to 2015: findings from the global burden of disease 2015 study. BMC Cardiovasc Disord. 2018;18:18. DOI: 10.1186/s12872-018-0761-0.
8. Morovatdar N, Avan A, Azarpazhooh MR, et al. Secular trends of ischaemic heart disease, stroke, and dementia in high-income countries from 1990 to 2017: The Global Burden of Disease Study 2017. Neurol Sci. 2021 May 1. DOI: 10.1007/s10072-021-05259-2.
9. Available:https://www.who.int/cardiovascular_diseases/en/cvd_atlas_14_deathHD.pdf.
10. Gheorghe A, Griffiths U, Murphy A, Legido-Quigley H, Lamptey P, Perel P. The economic burden of cardiovascular disease and hypertension in low-and middle-income countries: A systematic review. BMC Public Health. 2018;18:975.
11. Sampasa-Kanyinga H, Lewis RF. Frequent use of social networking sites is associated with poor psychological functioning among children and adolescents. Cyberpsychology Behav Soc Netw. 2015;18:380–385.
12. Piepoli MF, Hoes AW, Agewall S, et al. Authors Task Force, M. 2016 European Guidelines on cardiovascular disease prevention in clinical practice: The Sixth Joint Task Force of the European Society of Cardiology and Other Societies on Cardiovascular Disease Prevention in Clinical Practice (constituted by representatives of 10 societies and by invited experts) Developed with the special contribution of the European Association for Cardiovascular Prevention & Rehabilitation (EACPR) Eur. Heart J. 2016;37:2315–2381.
13. Casas R, Estruch R, Sacanella E. Influence of bioactive nutrients on the atherosclerotic process: A review. Nutrients. 2018 Nov 2;10(11):1630. DOI: 10.3390/nu10111630.
14. Available:https://www.who.int/initiatives/behealthy/healthy-diet.
15. Konstantinov IE, Jankovic GM. Alexander I. Ignatowski: A pioneer in the study of atherosclerosis. Tex. Heart Inst. J. 2013;40:246–249.
16. Arnett DK, Blumenthal RS, Albert MA. 2019 ACC/AHA guideline on the primary

prevention of cardiovascular disease: a report of the American College of Cardiology/American heart association task force on clinical practice guidelines. J Am Coll Cardiol; 2019 Mar 7.
DOI: 10.1016/j.jacc.2019.03.010.

17. Esselstyn CB, Jr, Gendy G, Doyle J, et al. A way to reverse CAD? J Fam Pract. 2014;63:356–364.
18. Wilson PW, D'Agostino RB, Sullivan L, et al. Overweight and obesity as determinants of cardiovascular risk: the Framingham experience. Arch Intern Med. 2002;162:1867–72.
19. Yarnell JW, Patterson CC, Thomas HF, Sweetnam PM. Comparison of weight in middle age, weight at 18 years, and weight change between, in predicting subsequent 14 year mortality and coronary events: Caerphilly Prospective Study. J Epidemiol Community Health. 2000;54:344-8.
20. Din-Dzietham R, Liu Y, Bielo MV, Shamsa F. High blood pressure trends in children and adolescents in national surveys, 1963 to 2002. Circulation. 2007;116(13):1488–1496.
DOI: 10.1161/CIRCULATIONAHA.106.683243.
21. Yusuf S, Hawken S, Ounpuu S, et al. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): Case-control study. Lancet. 2004;364:937–952.
DOI: 10.1016/S0140-6736(04)17018-9.
22. Garcia-Labbé D, Ruka E, Bertrand OF, Voisine P, Costerousse O, Poirier P. Obesity and coronary artery disease: evaluation and treatment. Can J Cardiol. 2015 Feb;31(2):184-94.
23. Buschmann K, Wrobel J, Chaban R, et al. Body Mass Index (BMI) and Its Influence on the Cardiovascular and Operative Risk Profile in Coronary Artery Bypass Grafting Patients: Impact of Inflammation and Leptin. Oxid Med Cell Longev. 2020 Jun 23;2020:5724024.
DOI: 10.1155/2020/5724024.
24. Sung CW, Huang CH, Chen WJ, et al. Obese cardiogenic arrest survivors with significant coronary artery disease had worse in-hospital mortality and neurological outcomes. Sci Rep. 2020 Oct 29;10(1):18638.
DOI: 10.1038/s41598-020-75752-9.
25. Landecho MF, Tuero C, Valentí V, et al. Relevance of Leptin and Other Adipokines in Obesity-Associated Cardiovascular Risk. Nutrients. 2019 Nov 5;11(11):2664.
DOI: 10.3390/nu11112664.
26. Esposito K, Pontillo A, di Palo C, et al. Effect of weight loss and lifestyle changes on vascular inflammatory markers in obese women: A randomized trial. JAMA. 2003;289(14):1799–1804.
DOI: 10.1001/jama.289.14.1799.
27. Bozkurt B, Aguilar D, Deswal A. Contributory risk and management of comorbidities of hypertension, obesity, diabetes mellitus, hyperlipidemia, and metabolic syndrome in chronic heart failure: A scientific statement from the American Heart Association. Circulation. 2016;134:e535–e578.
DOI: 10.1161/CIR.0000000000000450.
28. Raynor HA, Champagne CM. Position of the Academy of Nutrition and Dietetics: interventions for the treatment of overweight and obesity in adults. J Acad Nutr Diet. 2016;116:129–47.
29. Available:https://www.nhlbi.nih.gov/health/educational/wecan/downloads/calreqtips.pdf.
30. Schwartz MW, Seeley RJ, Zeltser LM, et al. Obesity pathogenesis: An endocrine society scientific statement. Endocrine Reviews. 2017;38:1–30.
31. Parretti HM, Jebb SA, Johns DJ, et al. Clinical effectiveness of very-low-energy diets in the management of weight loss: a systematic review and meta-analysis of randomized controlled trials. Obes Rev. 2016;17:225–34.
DOI: 10.1111/obr.12366.
32. Turner LR, Harris MF, Mazza D. Obesity management in general practice: does current practice match guideline recommendations? Med J Aust. 2015;202:370–2.
DOI: 10.5694/mja14.00998.
33. Halton TL, Hu FB. The effects of high protein diets on thermogenesis, satiety and weight loss: A critical review. J Am Coll Nutr. 2004;23:373–85.
DOI: 10.1080/07315724.2004.10719381.
34. Huo R, Du T, Xu Y, et al. Effects of Mediterranean-style diet on glycemic control, weight loss and cardiovascular risk factors among type 2 diabetes individuals: A meta-analysis. Eur. J. Clin. Nutr. 2015;69:1200–1208.
DOI: 10.1038/ejcn.2014.243.
35. Tonstad S, Stewart K, Oda K, et al. Vegetarian diets and incidence of diabetes

in the Adventist Health Study-2. Nutr Metab Cardiovas. 2011;1–8.

36. Romero-Corral A, Montori VM, Somers VK, et al. Association of bodyweight with total mortality and with cardiovascular events in coronary artery disease: a systematic review of cohort studies. The Lancet. 2006;368(9536):666–678.
DOI: 10.1016/S0140-6736(06)69251-9.
37. Benjamin EJ, Virani SS, Callaway CW, et al. Heart disease and stroke statistics-2018 update: A report from the american heart association. Circulation. 2018;137(12):e492.
DOI: 10.1161/CIR.0000000000000558.
38. O'Donell CJ et al. Hypertension and borderlineisolated systolic hypertension increase risks of cardiovascular disease and mortality in male physicians Circulation. 1997;95:1132–1137.
39. MacMahon S et al. Blood pressure, stroke and coronary heart disease. Part 1. Prolonged differences in blood pressure: prospective observational studies corrected for the regression dilution bias Lancet. 1990;335:765–774.
40. Khot UN, Khot MB, Bajzer CT, et al. Prevalence of conventional risk factors in patients with coronary heart disease. JAMA. 2003;290(7):898–904.
41. Elgendy IY, Bavry AA, Gong Y, et al. Long-term mortality in hypertensive patients with coronary artery disease. Hypertension. 2016;68(5):1110–1114.
42. Smith SM, Gong Y, Handberg E, et al. Predictors and outcomes of resistant hypertension among patients with coronary artery disease and hypertension. J Hypertens. 2014;32(3):635.
43. Stamler J, Stamler R, Neaton JD. Blood pressure, systolic and diastolic, and cardiovascular risks. US population data Arch Int Med. 1993;153:598–615.
44. McInnes GT. Hypertension and coronary artery disease: cause and effect. J Hypertens Suppl. 1995 Aug;13(2):S49-56.
DOI: 10.1097/00004872-199508001-00008.
45. He FJ, MacGregor GA. Importance of salt in determining blood pressure in children: meta-analysis of controlled trials. Hypertension. 2006;48:861–869.
46. Mozaffarian D, Fahimi S, Singh GM, et al. Global Burden of Diseases Nutrition and Chronic Diseases Expert Group. Global sodium consumption and death from cardiovascular causes. N Engl J Med. 2014;371:624–634.
47. He FJ, Li J, MacGregor GA. Effect of longer term modest salt reduction on blood pressure: Cochrane systematic review and meta-analysis of randomised trials. BMJ. 2013;346:f1325.
DOI: 10.1136/bmj.f1325.
48. Conlin PR, Chow D, Miller ER, et al. The effect of dietary patterns on blood pressure control in hypertensive patients: results from the Dietary Approaches to Stop Hypertension (DASH) trial. Am J Hypertens. 2000;13:949–955.
49. De Pergola G, D'Alessandro A. Influence of Mediterranean Diet on Blood Pressure. Nutrients. 2018;10(11):1700.
Published 2018 Nov 7.
DOI:10.3390/nu10111700.
50. Yokoyama Y, Nishimura K, Barnard ND, et al. Vegetarian diets and blood pressure: A meta-analysis. JAMA Intern Med. 2014 Apr;174(4):577-87.
DOI: 10.1001/jamainternmed.2013.14547.
51. Boers I, Muskiet FA, Berkelaar E, et al. Favourable effects of consuming a Palaeolithic-type diet on characteristics of the metabolic syndrome: a randomized controlled pilot-study. Lipids Health Dis. 2014 Oct 11;13:160.
DOI: 10.1186/1476-511X-13-160.
52. DeLalio LJ, Sved AF, Stocker SD. Sympathetic Nervous System Contributions to Hypertension: Updates and Therapeutic Relevance. Can J Cardiol. 2020 May;36(5):712-720.
DOI: 10.1016/j.cjca.2020.03.003.
53. Frohlich ED. Hypertension, left ventricular hypertrophy, and coronary flow reserve. Advances in Experimental Medicine and Biology. 1997;432:253–262.
54. van Zuydam NR, Ladenvall C, Voight BF, et al. SUMMIT Steering Committee; CARDIOGRAMplusC4D Steering Committee*. Genetic Predisposition to Coronary Artery Disease in Type 2 Diabetes Mellitus. Circ Genom Precis Med. 2020 Dec;13(6):e002769.
DOI: 10.1161/CIRCGEN.119.002769.
55. Hardin N. The myocardial and vascular pathology of diabetic cardiomyopathy. Coron Art Dis. 1996;7:99-108.
56. Woodfield SL, Lundergan CF, Reiner JS, et al. Angiographic findings and outcome in diabetic patients treated with thrombolytic therapy for acute myocardial infarction: the

GUSTO-I experience. J Am Coll Cardiol. 1996;28:1661-1669.
57. Pan W, Lu H, Lian B, Liao P, Guo L, Zhang M. Prognostic value of HbA1c for in-hospital and short-term mortality in patients with acute coronary syndrome: A systematic review and meta-analysis. Cardiovasc Diabetol. 2019;18(1):169. DOI: 10.1186/s12933-019-0970-6.
58. Miettinen H, Lehto S, Salomaa V, et al. Impact of diabetes on mortality after the first myocardial infarction. The FINMONICA Myocardial Infarction Register Study Group. Diabetes Care. 1998; 21:69–75.
59. Herlitz J, Karlson BW, Lindqvist J, Sjolin M. Rate and mode of death during five years of follow-up among patients with acute chest pain with and without a history of diabetes mellitus. Diabet Med. 1998;15:308–14.
60. Herlitz J, Wognsen GB, Emanuelsson H, et al. Mortality and morbidity in diabetic and nondiabetic patients during a 2-year period after coronary artery bypass grafting. Diabetes Care. 1996;19:698-703.; Seven-year outcome in the Bypass Angioplasty Revascularization Investigation (BARI) by treatment and diabetic status. J Am Coll Cardiol. 1000;35:1112–9.
61. Aronson D, Bloomgarden Z, Rayfield EJ. Potential mechanisms promoting restenosis in diabetic patients. J Am Coll Cardiol. 1996;27:528-535.
62. Wackers FJ, Young LH, Inzucchi SE, et al. Detection of silent myocardial ischemia in asymptomatic diabetic subjects: the DIAD study. Diabetes Care. 2004;27:1954–61. DOI: 10.2337/diacare.27.8.1954.
63. Haffner SM, Lehto S, Ronnemaa T, Pyorala K, Laakso M. Mortality from coronary heart disease in subjects with type 2 diabetes and in nondiabetic subjects with and without prior myocardial infarction. N Engl J Med. 1998;339:229–234. PMID: 9673301. DOI: 10.1056/NEJM199807233390404.
64. Naito R, Miyauchi K. Coronary Artery Disease and Type 2 Diabetes Mellitus. Int Heart J. 2017 Aug 3;58(4):475-480. DOI: 10.1536/ihj.17-191.
65. Guess ND. Dietary interventions for the prevention of type 2 diabetes in high-risk groups: Current state of evidence and future research needs. Nutrients. 2018;10:1245. DOI: 10.3390/nu10091245.
66. Available:https://www.hopkinsmedicine.org/news/media/releases/people_with_pre_diabetes_who_drop_substantial_weight_may_ward_off_type_2_diabetes. Accessed November 29, 2021.
67. Lim EL, Hollingsworth KG, Aribisala BS, et al. Reversal of type 2 diabetes: Normalisation of beta cell function in association with decreased pancreas and liver triacylglycerol. Diabetologia. 2011;54:2506–2514. DOI: 10.1007/s00125-011-2204-7.
68. Robertson RP, Harmon J, Tran PO, Tanaka Y, Takahashi H. Glucose toxicity in beta-cells: Type 2 diabetes, good radicals gone bad, and the glutathione connection. Diabetes. 2003;52:581–587. DOI: 10.2337/diabetes.52.3.581.
69. Eizirik DL, Korbutt GS, Hellerström C. Prolonged exposure of human pancreatic islets to high glucose concentrations in vitro impairs the beta-cell function. J. Clin. Investig. 1992;90:1263–1268. DOI: 10.1172/JCI115989.
70. Brown RJ, Rother KI. Effects of beta-cell rest on beta-cell function: A review of clinical and preclinical data. Pediatr. Diabetes. 2008;9:14–22. DOI: 10.1111/j.1399-5448.2007.00272.x.
71. Seidell JC. Dietary fat and obesity: An epidemiologic perspective. Am J Clin Nutr. 1998;67(3 Suppl):546S–50.
72. Schwingshackl L, Hoffmann G, Lampousi AM, et al. Food groups and risk of type 2 diabetes mellitus: a systematic review and meta-analysis of prospective studies. Eur J Epidemiol. 2017 May;32(5):363-375. DOI: 10.1007/s10654-017-0246-y.
73. Vang A, Singh PN, Lee JW, Haddad EH, Brinegar CH. Meats, processed meats, obesity, weight gain and occurrence of diabetes among adults: Findings from adventist health studies. Ann. Nutr. Metab. 2008;52:9604. DOI: 10.1159/000121365.
74. Olfert MD, Wattick RA. Vegetarian diets and the risk of diabetes. Curr. Diabetes Rep. 2018;18:101. DOI: 10.1007/s11892-018-1070-9.
75. Vang A, Singh PN, Lee JW, Haddad EH, Brinegar CH. Meats, processed meats, obesity, weight gain and occurrence of diabetes among adults: Findings from adventist health studies. Ann. Nutr. Metab. 2008;52:9604. DOI: 10.1159/000121365

76. Chiu THT, Pan WH, Lin MN, Lin CL. Vegetarian diet, change in dietary patterns, and diabetes risk: A prospective study. Nutr. Diabetes. 2018;8:12.
DOI: 10.1038/s41387-018-0022-4
77. Chen Z, Zuurmond MG, Van der Schaft N, et al. Plant versus animal based diets and insulin resistance, prediabetes and type 2 diabetes: The Rotterdam Study. Eur. J. Epidemiol. 2018; 33:883–893.
DOI: 10.1007/s10654-018-0414-8.
78. Brand-Miller J, Hayne S, Petocz P, Colagiuri S. Low-glycemic index diets in the management of diabetes: A meta-analysis of randomized controlled trials. Diabetes Care. 2003;26:2261–2267
DOI: 10.2337/diacare.26.8.2261.
79. Martín-Peláez S, Fito M, Castaner O. Mediterranean Diet Effects on Type 2 Diabetes Prevention, Disease Progression, and Related Mechanisms. A Review. Nutrients. 2020 Jul 27;12(8):2236.
DOI: 10.3390/nu12082236.
80. Livesey G, Taylor R, Livesey HF, et al. Dietary Glycemic Index and Load and the Risk of Type 2 Diabetes: Assessment of Causal Relations. Nutrients. 2019 Jun 25;11(6):1436.
DOI: 10.3390/nu11061436.
81. Livesey G, Livesey H. Coronary heart disease and dietary carbohydrate, glycemic index and glycemic load: Dose-response meta-analyses of prospective cohort studies. Mayo Clin. Proc. Innov. Qual. Outcomes. 2019;3:52–69.
DOI: 10.1016/j.mayocpiqo.2018.12.007.
82. Mazzocchi A, Leone L, Agostoni C, Pali-Schöll I. The Secrets of the Mediterranean Diet. Does [Only] Olive Oil Matter? Nutrients. 2019;11(12):2941.
Published 2019 Dec 3.
DOI: 10.3390/nu11122941.
83. Challa HJ, Ameer MA, Uppaluri KR. DASH diet to stop hypertension. 2021 May 19. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2021 Jan.
84. Panagiotakos DB, Tzima N, Pitsavos C, et al. The association between adherence to the Mediterraneandiet and fasting indices of glucose homoeostasis: The Attica study. J. Am. Coll. Nutr. 2007;26:32–38.
85. Santangelo C, Filesi VR, Scazzocchio B, et al. Consumption of extra-virgin olive oil rich in phenolic compounds improves metabolic control in patients with type 2 diabetes mellitus: A possible involvement of reduced levels of circulating visfatin. J. Endocrinol. Investig. 2016;39:1295–1301.
86. Martín-Peláez S, Fito M, Castaner O. Mediterranean diet effects on type 2 diabetes prevention, disease progression, and related mechanisms. A review. Nutrients. 2020;12(8):2236.
Published 2020 Jul 27
DOI: 10.3390/nu12082236.
87. de Bock M, Derraik JG, Brennan CM, et al. Olive (*Olea europaea* L.) leaf polyphenols improve insulin sensitivity in middle-aged overweight men: A randomized, placebo-controlled, crossover trial. PLoS ONE. 2013;8:e57622.
DOI: 10.1371/journal.pone.0057622.
88. Maedler K, Oberholzer J, Bucher P, Spinas GA, Donath MY. Monounsaturated fatty acids prevent the deleterious effects of palmitate and high glucose on human pancreatic beta-cell turnover and function. Diabetes. 2003;52:726–733.
DOI: 10.2337/diabetes.52.3.726.
89. Maiorino MI, Bellastella G, Petrizzo M, Scappaticcio L, Giugliano D, Esposito K. Anti-inflammatory effect of mediterranean diet in type 2 diabetes is durable: 8-year follow-up of a controlled trial. Diabetes Care. 2016;39:e44–e45.
DOI: 10.2337/dc15-2356.
90. Jannasch F, Kroger J, Schulze MB. Dietary patterns and type 2 diabetes: A systematic literature review and meta-analysis of prospective studies. J. Nutr. 2017;147:1174–1182.
DOI: 10.3945/jn.116.242552.
91. Olfert MD, Wattick RA. Vegetarian diets and the risk of diabetes. Curr Diab Rep. 2018 Sep 18;18(11):101.
DOI: 10.1007/s11892-018-1070-9.
92. Masharani U, Sherchan P, Schloetter M, et al. Metabolic and physiologic effects from consuming a hunter-gatherer [Paleolithic]-type diet in type 2 diabetes. Eur. J. Clin. Nutr. 2015;69:944–948.
DOI: 10.1038/ejcn.2015.39.
93. Ahotupa M. Oxidized lipoprotein lipids and atherosclerosis. Free Radic Res. 2017;51:439–447.
DOI: 10.1080/10715762.2017.1319944.
94. Sanin V, Pfetsch V, Koenig W. Dyslipidemias and cardiovascular prevention: Tailoring treatment according to lipid phenotype. Curr. Cardiol. Rep. 2017;19(7):61.
Available:http://dx.doi.org/10.1007/s11886-017-0869-3.

95. Schulz R, Schlüter KD. PCSK9 targets important for lipid metabolism. Clin. Res. Cardiol. Suppl. 2017;12:2–11.
Available:http://dx.doi.org/10.1007/s11789-017-0085-0.

96. van de Borne P. Recent progresses in the treatment of dyslipidemia. Rev. Med. Brux. 2017;38(4):357–360.

97. Dyrbuś K, Osadnik T, Desperak P, Desperak A, Gąsior M, Banach M. Evaluation of dyslipidaemia and the impact of hypolipidemic therapy on prognosis in high and very high risk patients through the Hyperlipidaemia Therapy in tERtiary Cardiological cEnTer (TERCET) Registry. Pharmacol. Res. 2018;132:204–210.

98. Baigent C, Keech A, Kearney PM, et al. Efficacy and safety of cholesterol-lowering treatment: prospective meta-analysis of data from 90,056 participants in 14 randomised trials of statins. Lancet. 2005;366:1267–1278.
DOI: 10.1016/S0140-6736(05)67394-1.

99. Cholesterol Treatment Trialists Collaborators. Kearney PM, Blackwell L, et al. Efficacy of cholesterol-lowering therapy in 18,686 people with diabetes in 14 randomised trials of statins: a meta-analysis. Lancet. 2008;371:117–125.
DOI: 10.1016/S0140-6736(08)60104-X.

100. Descamps OS, Verhaegen A, Demeure F, et al. Evolving concepts on the management of dyslipidaemia. Acta Clin Belg. 2020 Feb;75(1):80-90.
DOI: 10.1080/17843286.2019.1702823.

101. Castelli WP, Anderson K, Wilson PW, et al. Lipids and risk of coronary heart disease. The Framingham Study. Ann Epidemiol. 1992;2:23–28.
DOI: 10.1016/1047-2797(92)90033-M.

102. Orozco-Beltran D, Gil-Guillen VF, Redon J, et al. Lipid profile, cardiovascular disease and mortality in a Mediterranean high-risk population: The ESCARVAL-RISK study. PLoS One. 2017;12(10):e0186196.

103. Rudel LL, Parks JS, Sawyer JK. Compared with dietary monounsaturated and saturated fat, polyunsaturated fat protects African green monkeys from coronary artery atherosclerosis. Arterioscler Thromb Vasc Biol. 1995;15:2101–10.

104. Bantle JP, Wylie-Rosett J, Albright AL, et al. Nutrition recommendations and interventions for diabetes--2006: A position statement of the american diabetes association. Diabetes Care. 2006;29: 2140–57.

105. Li YH, Ueng KC, Jeng JS, et al. Taiwan lipid guidelines for high risk patients. J. Formos. Med. Assoc. 2017;116(4):217–248.
Available:http://dx.doi.org/10.1016/j.jfma.2016.11.013

106. Mensink RP. Effects of saturated fatty acids on serum lipids and lipoproteins: A systematic review and regression analysis. world health organization; Geneva, Switzerland; 2016.

107. Schwingshackl L, Bogensberger B, Benčič A, Knüppel S, Boeing H, Hoffmann G. Effects of oils and solid fats on blood lipids: A systematic review and network meta-analysis. J. Lipid Res. 2018;59:1771–1782.
DOI: 10.1194/jlr.P085522.

108. The Scientific Advisory Committee on Nutrition (SACN) Report on Saturated Fats and Health; 2019.
[(Accessed on 31 August 2020)];
Available:https://www.gov.uk/government/publications/saturated-fats-and-health-sacn-report.

109. Mensink RP. Effects of saturated fatty acids on serum lipids and lipoproteins: A systematic review and regression analysis. World Health Organization; Geneva, Switzerland: 2016. The Scientific Advisory Committee on Nutrition (SACN) Report on Saturated Fats and Health; 2019.
[(Accessed on 31 August 2020)]
Available:https://www.gov.uk/government/publications/saturated-fats-and-health-sacn-report.

110. Zock PL, Blom WAM, Nettleton JA, Hornstra G. Progressing insights into the role of dietary fats in the prevention of cardiovascular disease. Curr. Cardiol. Rep. 2016;18:111.
DOI: 10.1007/s11886-016-0793-y.

111. Trautwein EA, McKay S. The role of specific components of a plant-based diet in management of dyslipidemia and the impact on cardiovascular risk. Nutrients. 2020 Sep 1;12(9):2671.
DOI: 10.3390/nu12092671.

112. Kahleova H, Salas-Salvadó J, Rahelić D, et al. Dietary patterns and cardiometabolic outcomes in diabetes: A summary of systematic reviews and meta-analyses. Nutrients. 2019;11:2209.

DOI: 10.3390/nu11092209.

113. Rees K, Takeda A, Martin N, et al. Mediterranean-style diet for the primary and secondary prevention of cardiovascular disease. Cochrane Database Syst. Rev. 2019;3.
DOI: 10.1002/14651858.CD009825.pub3

114. Kesse-Guyot E, Ahluwalia N, Lassale C, Hercberg S, Fezeu L, Lairon D. Adherence to Mediterranean diet reduces the risk of metabolic syndrome: A 6-year prospective study. Nutr. Metab. Cardiovasc. Dis. 2013;23:677–683.
DOI: 10.1016/j.numecd.2012.02.005

115. Siervo M, Lara J, Chowdhury S, Ashor A, Oggioni C, Mathers JC. Effects of the dietary approach to stop hypertension (DASH) diet on cardiovascular risk factors: A systematic review and meta-analysis. Br. J. Nutr. 2015;113:1–15.
DOI: 10.1017/S0007114514003341.

116. Hashemi R, Mehdizadeh Khalifani A, Rahimlou M, Manafi M. Comparison of the effect of dietary approaches to stop hypertension diet and american diabetes association nutrition guidelines on lipid profiles in patients with type 2 diabetes: A comparative clinical trial. Nutr. Diet. 2020;77:204–211.
DOI: 10.1111/1747-0080.12543.

117. Chiavaroli L, Viguiliouk E, Nishi SK, et al. DASH dietary pattern and cardiometabolic outcomes: An umbrella review of systematic reviews and meta-analyses. Nutrients. 2019;11:338.
DOI: 10.3390/nu11020338.

118. Wang F, Zheng J, Yang B, Jiang J, Fu Y, Li D. Effects of vegetarian diets on blood lipids: A systematic review and meta-analysis of randomized controlled trials. J. Am. Heart Assoc. 2015;4:e002408.
DOI: 10.1161/JAHA.115.002408.

119. Yokoyama Y, Levin SM, Barnard ND. Association between plant-based diets and plasma lipids: A systematic review and meta-analysis. Nutr. Rev. 2017;75:683–698.
DOI: 10.1093/nutrit/nux030.

120. Kahleova H, Salas-Salvadó J, Rahelić D, Kendall CW, Rembert E, Sievenpiper JL. Dietary patterns and cardiometabolic outcomes in diabetes: A summary of systematic reviews and meta-analyses. Nutrients. 2019;11:2209.
DOI: 10.3390/nu11092209.

121. Guasch-Ferré M, Satija A, Blondin SA, et al. Meta-analysis of randomized controlled trials of red meat consumption in comparison with various comparison diets on cardiovascular risk factors. Circulation. 2019;139:1828–1845.
DOI:10.1161/CIRCULATIONAHA.118.035225

122. Bueno NB, de Melo IS, de Oliveira SL, da Rocha Ataide T. Very-low-carbohydrate ketogenic diet v. low-fat diet for long-term weight loss: A meta-analysis of randomised controlled trials. Br. J. Nutr. 2013;110:1178–1187.
DOI: 10.1017/S0007114513000548.

123. Liu Y, Wheaton AG, Chapman DP, et al. Sleep duration and chronic diseases among US adults age 45 years and older: Evidence from the 2010 Behavioral Risk Factor Surveillance System. Sleep. 2013;36(10):1421–27.

124. Shankar A, Koh WP, Yuan JM, et al. Sleep duration and coronary heart disease mortality among Chinese adults in Singapore: a population-based cohort study. Am J Epidemiol. 2008;168(12): 1367–73.

125. Sands-Lincoln M, Loucks EB, Lu B, et al. Sleep duration, insomnia, and coronary heart disease among postmenopausal women in the Women's Health Initiative. J Womens Health. 2013;22(6):477–86.

126. Cappuccio FP, Cooper D, D'Elia L, et al. Sleep duration predicts cardiovascular outcomes: A systematic review and meta-analysis of prospective studies. Eur Heart J. 2011;32(12):1484–92.

127. Dashti HS, Scheer FA, Jacques PF, Lamon-Fava S, Ordovas JM. Short sleep duration and dietary intake: Epidemiologic evidence, mechanisms, and health implications. Adv. Nutr. 2015;6:648–659.
DOI: 10.3945/an.115.008623

128. Bravo R, Matito S, Cubero J, et al. Tryptophan-enriched cereal intake improves nocturnal sleep, melatonin, serotonin, and total antioxidant capacity levels and mood in elderly humans. J. Am. Aging Assoc. 2013;35:1277–1285.
DOI: 10.1007/s11357-012-9419-5.

129. Layman DK, Lönnerdal B, Fernstrom JD. Applications for α-lactalbumin in human nutrition. Nutr. Rev. 2018;76:444–460.

DOI: 10.1093/nutrit/nuy004.

130. Bhatti T, Gillin JC, Seifritz E, et al. Effects of a tryptophan-free amino acid drink challenge on normal human sleep electroencephalogram and mood. Biol. Psychiatry. 1998;43:52–59.
DOI: 10.1016/S0006-3223(97)80252-1.

131. Duan KM, Ma JH, Wang SY, Huang ZD, Zhou YY, Yu HY. The role of tryptophan metabolism in postpartum depression. Metab. Brain Dis. 2018;33:647–660.
DOI: 10.1007/s11011-017-0178-y.

132. González-Gómez D, Lozano M, Fernández-León M, Ayuso M, Bernalte M, Rodríguez A. Detection and quantification of melatonin and serotonin in eight Sweet Cherry cultivars (*Prunus avium* L.) Eur. Food Res. Technol. 2009;229:223–229.

133. Young T, Palta M, Dempsey J, Skatrud J, Weber S, Badr S. The occurrence of sleep-disordered breathing among middle-aged adults. N Engl J Med. 1993;328:1230–1235.
DOI: 10.1056/NEJM199304293281704.

134. Loke YK, Brown JW, Kwok CS, et al. Association of obstructive sleep apnea with risk of serious cardiovascular events: A systematic review and meta-analysis. Circ Cardiovasc Qual Outcomes. 2012;5:720-8.
DOI: 10.1161/CIRCOUTCOMES.111.964783.

135. Dong JY, Zhang YH, Qin LQ. Obstructive sleep apnea and cardiovascular risk: Meta-analysis of prospective cohort studies. Atherosclerosis 2013;229:489-95.
DOI: 10.1016/j.atherosclerosis.2013.04.026.

136. Yeboah J, Redline S, Johnson C, et al. Association between sleep apnea, snoring, incident cardiovascular events and all-cause mortality in an adult population:. MESA Atherosclerosis 2011;219(2):963–968.

137. Marin JM, Carrizo SJ, Vicente E, Agusti AGN. Long-term cardiovascular outcomes in men with obstructive sleep apnoea-hypopnoea with or without treatment with continuous positive airway pressure: an observational study. Lancet. 2005;365(9464):1046–1053.

138. Xu X, Xu J. Effects of different obesity-related adipokines on the occurrence of obstructive sleep apnea. Endocr J. 2020 May 28;67(5):485-500.
DOI: 10.1507/endocrj.EJ20-0036.

139. Araghi MH, Chen YF, Jagielski A, et al. Effectiveness of lifestyle interventions on obstructive sleep apnea (OSA): Systematic review and meta-analysis. Sleep. 2013;36(10):1553–1562, 1562a–1562e.

140. Greenburg DL, Lettieri CJ, Eliasson AH. Effects of surgical weight loss on measures of obstructive sleep apnea: A meta-analysis. American Journal of Medicine. 2009;122(6):535–542.

141. Kuna ST, Reboussin DM, Borradaile, et al. Sleep AHEAD Research Group of the Look AHEAD Research Group. Long-term effect of weight loss on obstructive sleep apnea severity in obese patients with type 2 diabetes. Sleep. 2013;36(5):641–649A.

142. Arnaud C, Bochaton T, Pépin JL, Belaidi E. Obstructive sleep apnoea and cardiovascular consequences: Pathophysiological mechanisms. Arch Cardiovasc Dis. 2020 May;113(5):350-358.
DOI: 10.1016/j.acvd.2020.01.003.

143. Alkerwi A. Baydarlioglu B. Sauvageot N. et al. Smoking status is inversely associated with overall diet quality: Findings from the ORISCAV-LUX study. Clin Nutr. 2017;36:1275-1282.

144. Albert NM, Butler R, Sorrell J. Factors related to healthy diet and physical activity in hospital-based clinical nurses. OJIN: The Online Journal of Issues in Nursing Vol. 19, No. 3, Manuscript 5); 2014.

145. Gangadi M, Kalpourtzi N, Gavana M, et al. Prevalence of tobacco smoking and association with other unhealthy lifestyle risk factors in the general population of Greece: Results from the EMENO study. Tob Prev Cessat. 2021 Sep 11;7:61.
DOI: 10.18332/tpc/140242.

146. Pietiläinen KH, Kaprio J, Borg P, et al. Physical inactivity, and obesity: a vicious circle. Obesity (Silver Spring). 2008;16(2):409-414.
DOI: 10.1038/oby.2007.72

147. Fujii H, Kono K, Nishi S. Characteristics of coronary artery disease in chronic kidney disease. Clin Exp Nephrol. 2019 Jun;23(6):725-732.
DOI: 10.1007/s10157-019-01718-5
Epub 2019 Mar 4
PMID: 30830548
PMCID: PMC6511359.

148. Sarnak MJ, Amann K, Bangalore S, et al. Conference Participants. Chronic Kidney Disease and Coronary Artery Disease: JACC State-of-the-Art Review. J Am Coll Cardiol. 2019 Oct 8;74(14):1823-1838.
DOI: 10.1016/j.jacc.2019.08.1017.

149. Edmonston DL, Pun PH. Coronary artery disease in chronic kidney disease: Highlights from a Kidney Disease: Improving Global Outcomes (KDIGO) Controversies Conference. Kidney Int. 2020 Apr;97(4):642-644.
DOI: 10.1016/j.kint.2019.12.010.

150. Stevens PE, Levin A. Kidney disease: Improving global outcomes 2012 and clinical practice guideline. Ann. Intern. Med. 2013;158:825–830.
DOI: 10.7326/0003-4819-158-11-201306040-00007.

151. Kelly JT, Palmer SC, Wai SN, et al. Healthy dietary patterns and risk of mortality and ESRD in CKD: A meta-analysis of cohort studies. Clin. J. Am. Soc. Nephrol. 2017;12:272–279.
DOI: 10.2215/CJN.06190616.

152. Slinin Y, Guo H, Gilbertson DT, Mau LW, Ensrud K, Collins AJ, Ishani A. Prehemodialysis care by dietitians and first-year mortality after initiation of hemodialysis. Am. J. Kidney Dis. 2011;58:583–590.
DOI: 10.1053/j.ajkd.2011.03.032.

153. Ricardo AC, Madero M, Yang W, et al. Adherence to a Healthy lifestyle and all-cause mortality in CKD. Clin. J. Am. Soc. Nephrol. 2013;8:602–609.
DOI: 10.2215/CJN.00600112.

154. Cianciaruso B, Bellizzi V, Minutolo R, et al. Salt intake and renal outcome in patients with progressive renal disease. Miner Electrolyte Metab. 1998;24(4):296-301.
DOI: 10.1159/000057385. PMID: 9554571.

155. Lin J, Hu FB, Curhan GC. Associations of diet with albuminuria and kidney function decline. Clin. J. Am. Soc. Nephrol. 2010;5:836–843.
DOI: 10.2215/CJN.08001109

156. Lin J, Fung TT, Hu FB, Curhan GC. Association of dietary patterns with albuminuria and kidney function decline in older white women: A subgroup analysis from the Nurses' Health Study. Am. J. Kidney Dis. 2011;57:245–254.
DOI: 10.1053/j.ajkd.2010.09.027.

157. Shoham DA, Durazo-Arvizu R, Kramer H, Luke A, Vupputuri S, Kshirsagar A, Cooper RS. Sugary soda consumption and albuminuria: Results from the national health and nutrition examination survey, 1999–2004. PLoS ONE. 2008;3: e3431.
DOI: 10.1371/journal.pone.0003431.

158. Bomback AS, Katz R, He K, Shoham DA, Burke GL, Klemmer PJ. Sugar-sweetened beverage consumption and the progression of chronic kidney disease in the Multi-Ethnic Study of Atherosclerosis (MESA) Am. J. Clin. Nutr. 2009;90:1172–1178.
DOI: 10.3945/ajcn.2009.28111.

159. Gersch MS, Mu W, Cirillo P, et al. Fructose, but not dextrose, accelerates the progression of chronic kidney disease. Am. J. Physiol. Ren. Physiol. 2007;293:F1256–F1261.
DOI: 10.1152/ajprenal.00181.2007

160. Brymora A, Flisinski M, Johnson RJ, Goszka G, Stefańska A, Manitius J. Low-fructose diet lowers blood pressure and inflammation in patients with chronic kidney disease. Nephrol. Dial. Transplant. 2012;27:608–612.
DOI: 10.1093/ndt/gfr223.

161. Ko GJ, Obi Y, Tortorici AR, Kalantar-Zadeh K. Dietary protein intake and chronic kidney disease. Curr Opin Clin Nutr Metab Care. 2017 Jan;20(1):77-85.
DOI: 10.1097/MCO.0000000000000342.

162. Gan Y, Gong Y, Tong X, et al. Depression and the risk of coronary heart disease: A meta-analysis of prospective cohort studies. BMC Psychiatry. 2014;14:371.

163. Nicholson A, Kuper H, Hemingway H. Depression as an aetiologic and prognostic factor in coronary heart disease: a meta-analysis of 6362 events among 146,538 participants in 54 observational studies. Eur Heart J. 2006;27:2763–74.

164. Hagström E, Norlund F, Stebbins A, Armstrong P, Chiswell K, Granger C. Psychosocial stress and major cardiovascular events in patients with stable coronary heart disease. J. Intern. Med. 2018;283(1):83–92.

165. Lane D, Carroll D, Ring C, Beevers DG, Lip GY. Effects of depression and anxiety on mortality and quality-of-life 4 months after myocardial infarction. J Psychosom Res. 2000;49:229–238.

166. Barth J, Schumacher M, Herrmann-Lingen C. Depression as a risk factor for mortality in patients with coronary heart disease: a meta-analysis. Psychosom Med. 2004;66(6):802–13.
DOI: 10.1097/01.psy.0000146332.53619.b2.

167. May HT, Horne BD, Knight S, et al. The association of depression at any time to the risk of death following coronary artery disease diagnosis. ur Heart J. 2017;3(4):296-302.

168. Pelletier R, Bacon SL, Arsenault A, Dupuis J, Laurin C, Blais L, Lavoie KL. Relative associations between depression and anxiety on adverse cardiovascular events: does a history of coronary artery disease matter? A prospective observational study. BMJ Open. 2015 Dec 15;5(12):e006582.
DOI: 10.1136/bmjopen-2014-006582.

169. Meijer A, Conradi HJ, Bos EH, Thombs BD, van Melle JP, de Jonge P. Prognostic association of depression following myocardial infarction with mortality and cardiovascular events: A meta-analysis of 25 years of research. Gen Hosp Psychiatry. 2011;33:203–16.

170. Geulayov G, Novikov I, Dankner D, Dankner R. Symptoms of depression and anxiety and 11-year all-cause mortality in men and women undergoing coronary artery bypass graft (CABG) surgery. J Psychosom Res. 2018;106–114.

171. Jacka FN, Pasco JA, Mykletun A, et al. Association of western and traditional diets with depression and anxiety in women. The Amer J Psychiat. 2010;167(3):305–311.
DOI: 10.1176/appi.ajp.2009.09060881.

172. Jacka FN, Mykletun A, Berk M, Bjelland I, Tell GS. The association between habitual diet quality and the common mental disorders in community-dwelling adults: The hordaland health study. Psychosom Med. 2011;73(6):483–490.
DOI: 10.1097/PSY.0b013e318222831a.

173. Li Y, Lv MR, Wei YJ, Sun L, Zhang JX, Zhang HG, Li B. Dietary patterns and depression risk: A meta-analysis. Psychiatry Res. 2017 Jul;253:373-382.
DOI: 10.1016/j.psychres.2017.04.020.

174. Jacka FN, Pasco JA, Mykletun A, et al. Association of western and traditional diets with depression and anxiety in women. The Amer J Psychiat. 2010;167(3):305–311.
DOI: 10.1176/appi.ajp.2009.09060881.

175. Luppino FS, de Wit LM, Bouvy PF, et al. Overweight, obesity, and depression: a systematic review and meta-analysis of longitudinal studies. Arch Gen Psychiatry. 2010 Mar;67(3):220-9.
DOI: 10.1001/archgenpsychiatry.2010.2.

176. Kilian R, Becker T, Kruger K, Schmid S, Frasch K. Health behaviour in psychiatric in-patients compared with a German general population sample. Acta Psychiatrica Scand. 2006;114(4):242–248.
DOI: 10.1111/j.1600-0447.2006.00850.x.

177. Luppino FS, de Wit LM, Bouvy PF, et al. Overweight, obesity, and depression: A systematic review and meta-analysis of longitudinal studies. Arch Gen Psychiatry. 2010 Mar;67(3):220-9.
DOI: 10.1001/archgenpsychiatry.2010.2.

178. Dimsdale JE. Psychological stress and cardiovascular disease. J Am Coll Cardiol. 2008;51:1237–1246.

179. Steptoe A, Kivimäki M. Stress and cardiovascular disease. Nat Rev Cardiol. 2012;9:360–370. Stress has been associated with an increase in a prothrombotic state (Dimsdale JE. Psychological stress and cardiovascular disease. J Am Coll Cardiol. 2008;51:1237–46.

180. Schwartz BG, French WJ, Mayeda GS, et al. Emotional stressors trigger cardiovascular events. Int J Clin Pract. 2012;66:631–9.

181. Mostofsky E, Penner EA, Mittleman MA. Outbursts of anger as a trigger of acute cardiovascular events: A systematic review and meta-analysis. Eur Heart J. 2014;35:1404–1410.

182. Meisel SR, Dayan KI, Pauzner H, Chetboun I, Arbel Y, David D, Kutz I.. Effect of Iraqi missile war on incidence of acute myocardial infarction and sudden death in Israeli civilians. Lancet 1991;338:660–661.

183. Leor J, Poole WK, Kloner RA. Sudden cardiac death triggered by an earthquake. N Engl J Med 1996;334:413–419.

184. Oliver G, Wardle J, Gibson EL. Stress and food choice: a laboratory study. Psychosom Med. 2000;62(6):853–65.

185. Zellner DA, Loaiza S, Gonzalez Z, Pita J, Morales J, Pecora D, et al. Food selection changes under stress. Physiol Behav. 2006;87(4):789–93.
186. Khaled K, Tsofliou F, Hundley V. et al. Perceived stress and diet quality in women of reproductive age: a systematic review and meta-analysis. Nutr J. 2020;19(92). Available:https://doi.org/10.1186/s12937-020-00609-w.
187. Williamson DF, Thompson TJ, Anda RF, Dietz WH, Felitti V. Body weight and obesity in adulthood and self-reported abuse in childhood. Int. J. Obes. Relat. Metab. Disord. 2002;26:1075–1082. DOI: 10.1038/sj.ijo.0802038.
188. Fowler N, Vo PT, Sisk CL, Klump KL. Stress as a potential moderator of ovarian hormone influences on binge eating in women. F1000Research. 2019;8. DOI: 10.12688/f1000research.16895.1.
189. Valassi E, Schacchi M, Cavagnini F. Neuroendocrine control of food intake. Nutr. Metab. Cardiovasc. Dis. 2007;18: 158–168. DOI: 10.1016/j.numecd.2007.06.004.

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