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Dietary interventions for dyslipidemia

Abstract

The prerequisite for any diet is that it is nutritionally adequate, that is, it contains sufficient amounts of macro and micronutrients, in order to meet the recommendations. To do so, it must include a wide variety of foods. Fruits, vegetables and legumes (beans, peas) are good sources of vitamin A, C, folic acid, fiber and minerals. Cereals and derivatives (rice, flours, breads, etc.), especially whole grains, contain B vitamins, complex carbohydrates, fiber, as well as a small amount of protein and iron. Chicken, fish, beef and eggs are good sources of protein, iron and B vitamins. However, these last two food groups are also rich in saturated fats and cholesterol and therefore should be used in small amounts or in a modified form. More specifically, the following modifications should be considered: Total fat - The recommendation that total dietary fat should not exceed 30% of total calories has two goals, to facilitate the reduction in saturated fat intake and to promote weight reduction when necessary. In this article the focus is mainly on diet interventions and lipoprotein metabolism as understood so far.

Keywords: saturated fat, cholesterol, atherosclerosis, diet, nutrition

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Abbreviations: HDL-c, high-density lipoprotein-cholesterol; LDL, low-density lipoprotein; LDL-c, low-density lipoprotein-cholesterol; VLDL, very low-density lipoprotein

Introduction

The first approach to be adopted in the treatment of hyperlipidemias is dietary intervention. To achieve this goal, patients should be informed of the reason for the intervention, its importance and how to proceed with it. Three dietary factors have adverse effects on lipoprotein metabolism: high intake of saturated fat, cholesterol, and excessive caloric intake, which leads to obesity.[1] Traditionally, fat has been considered as the most important dietary factor with respect to chronic diseases, especially coronary heart disease and certain types of cancer.[2] Although other components of the diet, such as unsaturated fatty acid, carbohydrates, protein, and alcohol can affect lipoprotein metabolism, they are not included in the category of cholesterol-raising nutrients.[1] The phase 1 diet promotes a reduction in intake of the foods higher in saturated fats and cholesterol, which can often lead to some changes in eating habits. The phase 2 diet requires even more careful attention, as it advocates a more drastic decrease in the intake of these nutrients, which can make it very difficult to adhere to the diet and the quality of the diet, if it is not well planned. Therefore, frequent consultations with the nutritionist are recommended, preferably specialized in the area of lipids, in order to obtain the desired dietary modifications. The prerequisite for any diet is that it is nutritionally adequate, that is, it contains sufficient amounts of macro and micronutrients, in order to meet the recommendations. To do so, it must include a wide variety of foods. Fruits, vegetables and legumes (beans, peas) are good sources of vitamin A, C, folic acid, fiber and minerals. Cereals and derivatives (rice, flours, breads, etc.), especially whole grains, contain B vitamins, complex carbohydrates, fiber, as well as a small amount of protein and iron. Chicken, fish, beef and eggs are good sources of protein, iron and B vitamins. Milk and its derivatives (except butter), in addition to being good sources of protein, are also excellent sources of calcium. However, these last two food groups are also rich in saturated fats and cholesterol and therefore should be used in small amounts or in a modified form.

More specifically, the following modifications should be considered:

Total fat: The recommendation that total dietary fat should not exceed 30% of total calories has two goals: to facilitate the reduction in saturated fat intake and to promote weight reduction when necessary.

Saturated fatty acids: It is well defined that saturated fatty acids promote an increase in plasma cholesterol levels.[3,4] However, this is only true for some fatty acids, such as palmitic acid and myristic acid, present mainly in milk fat and its derivatives, in the fat of most meats and in some foods of plant origin, such as coconut, coconut oil, cotton and palm oil. Stearic acid, also saturated, does not seem to cause an increase in cholesterolemia.[5,6] Although beef fat has reasonable amounts of stearic acid, cocoa butter has the highest amount of this fatty acid. As the main component of chocolate is cocoa butter, some studies have been carried out, showing that moderate amounts of chocolate do not promote cholesterol elevation,[7,8] however, one cannot forget its high total fat content, which should be considered for overweight patients.

To reduce the intake of saturated fatty acids, especially those known to be hypercholesterolemic, one should use skim milk or yogurt, low-fat cheeses such as ricotta, cottage cheese and fresh Minas cheese with specification on the label of reduced amount of fat. In relation to beef, preference should be given to cuts with lower fat content. In addition, all apparent fat must be removed. White chicken meat and turkey meat, without skin, have lower total and saturated fat contents. However, the dark meat of these birds has values sometimes even higher than some cuts of beef. Lean pork loin, that is, with the apparent fat removed, contains an amount of total and saturated fat similar to that of some cuts of beef, and can therefore be used eventually. The best choice, however, is still fish meat, which has the lowest total and saturated fat contents.

Monounsaturated fatty acids: The main monounsaturated fatty acid present in foods is oleic acid, which appears to have a neutral or slightly beneficial effect on cholesterol levels.[9,10] This fatty acid is mainly present in olive, canola, rice, peanut, cashew, hazelnut and avocado oils. Monounsaturated fatty acids can also be produced from the hydrogenation of polyunsaturated oils in the production of margarines and hydrogenated vegetable fat; are the trans-isomeric forms. Recent data suggest that these fatty acids, in contrast to oleic acid, may promote elevations in cholesterol levels.[11,12] In the United States, the content of trans fatty acids ranges from 7 to 24% in

margarines, and hydrogenated vegetable fat contains 15% of these fatty acids.[13]

Thus, the controversy is created about what would be more appropriate for hypercholesterolemic patients: the use of butter or margarine? Butter contains about 50% saturated fat (mainly palmitic and myristic acid), 3% polyunsaturated and 23.4% monounsaturated, as well as containing 219 mg of cholesterol.[14] Margarines vary greatly in their composition, which depends on the types of oil used in their manufacture and the degree of hydrogenation. The creamier the margarine, the lower its degree of hydrogenation and therefore the lower the amount of saturated fat and probably the lower the content of trans-isomeric monounsaturated fatty acids. If we consider that the trans fatty acid content of margarines produced in Brazil is similar to those produced in the United States (7% to 24%), this value is far below the amount of saturated fat found in butter. In addition, margarines contain more polyunsaturated fatty acids and do not contain cholesterol. Therefore, it should be recommended to use creamy margarines, in limited quantity, so as not to increase the total fat content of the diet.

Polyunsaturated fatty acids: There are two categories of polyunsaturated fatty acids: omega-6, represented mainly by linoleic acid, and omega-3, whose plant representative is linolenic acid and the animal representative are eicosapentanoic acid (EPA) and docosahexaenoic acid (DHA), present in fish fat. The replacement of saturated fat in the diet by linoleic acid promotes a decrease in cholesterolemia.[3,4] Therefore, for many years diets with a high content of linoleic acid were recommended; however, little is known about the effects of consuming this fatty acid in large quantities over a long period of time. Therefore, the intake of linoleic acid should not exceed 7% of the total caloric value of the diet. Several vegetable oils are rich in linoleic acid, among them sunflower, corn and soybean oil. Although these oils are high in polyunsaturated fatty acids and low in saturated fatty acids, they should be consumed in moderation as they can also lead to weight gain due to the high energy content.

In recent years, there has been a growing interest in the possible benefits of omega-3 fatty acids on atherogenesis, particularly EPA and DHA, present in fish oils. Omega-3 fatty acids promote a decrease in plasma triglycerides when used in high doses,[15] however, other measures that are also effective, such as a decrease in the intake of simple sugars, alcohol and weight loss, are the first choice in the treatment of hypertriglyceridemias. There is no evidence that these fatty acids have any effect on cholesterolemia, and their role in reducing the risk of cardiovascular disease has not yet been established. In addition, little is known about the possible adverse effects of its consumption over a long period of time. Therefore, the use of fish oil capsules as a therapy in the treatment of hyperlipidemias is not recommended. Regarding the use of fish rich in omega-3 fatty acids, epidemiological studies suggest that frequent consumption of any type of fish, regardless of its fatty acid composition, is associated with a lower risk of cardiovascular disease.[16] Therefore, the increase in the consumption of fish instead of beef is a recommended measure in the treatment of hypercholesterolemias.

Cholesterol: Dietary cholesterol causes hypercholesterolemia and atherosclerosis in several laboratory animals. However, metabolic studies in humans, properly controlled, demonstrate that dietary cholesterol can cause non-uniform elevations in cholesterolemia.[4,17] It seems, however, that there are individual differences in the modification of cholesterolemia compared to diets high in cholesterol.[18] In addition, it is known that some types of saturated fat promote higher elevation of serum cholesterol levels than dietary cholesterol alone.[19] The interest in the atherogenicity of dietary cholesterol seems to be beyond the promotion of the elevation of serum cholesterol levels, since the absorbed dietary cholesterol enters the circulation with the chylomicrons, which are later degraded to the remaining chylomicrons, rich in cholesterol, which appear to be atherogenic.[1]

Cholesterol is found only in foods of animal origin. Particularly high in cholesterol are the egg yolk, the viscera, such as liver, heart, tongue, etc. and some crustaceans and molluscs, such as shrimp, lobster, oyster, etc. Beef, chicken and pork contain similar amounts of cholesterol, around 80 mg per 100 g. Dairy products also contain cholesterol, especially butter. To meet the recommendation of the phase 1 diet, that is, a cholesterol intake of less than 300 mg/day, foods higher in cholesterol should be avoided and the others should be used in moderate amounts. Preference should also be given to those containing the lowest saturated fat content.[20–22] There are many scientific papers that study the effects of different kinds of foods with lipid profile levels as, for example, to systematically evaluate the evidence regarding the effects of foods on Low-Density Lipoprotein-cholesterol (LDL-c) levels and to compare the findings with current guidelines.

Data synthesis: from origin through June 2019, we searched PubMed, Cochrane Database of Systematic Reviews, and Cochrane Central Register of Controlled Trials for guidelines, systematic reviews, and RCTs (for coffee intake only) of at least 13 days duration. Additionally, we searched Trip database for guidelines from 2009 through October 2019. Language was restricted to English. The strength of evidence was evaluated using The Grading of Recommendations Assessment, Development, and Evaluation (GRADE). A total of 37 guidelines, 108 systematic reviews, and 20 RCTs were included. With high evidence, foods high in unsaturated and low in saturated and trans fatty acids (e.g. rapeseed/canola oil), with added plant sterols/stanols, and high in soluble fiber (e.g. oats, barley, and psyllium) caused at least moderate (i.e. 0.20-0.40 mmol/L) reductions in LDL-c. Unfiltered coffee caused a moderate to large increase. Soy protein, tomatoes, flaxseeds, and almonds caused small reductions. With moderate evidence, avocados and turmeric caused moderate to large reductions. Pulses, hazelnuts, walnuts, high-fiber/wholegrain foods, and green tea caused small to moderate reductions, whereas sugar caused a small increase. Other identified foods were either neutral or had low or very low evidence regarding their effects.[23–27] Several foods distinctly modify LDL-c levels. The results may aid future guidelines and dietary advice for hypercholesterolemia.

Although not being in the scope of this review the more recent tracks of the matter are being derived towards gut microbiome and metabolome independently of energy intake, also micro and macronutrients with particular attention to Mediterranean diet.[20–22] Even though the experimental studies tend to focus on molecular biology and related techniques the complexity of the external facts and overall interactions linked to lipoproteins still remain very important issues as associated factors to the quality of the diets and dietary interventions aimed at lipid levels amelioration.[28,29]

Total calories: Every dietary prescription has as one of the goals to get patients to achieve the desired weight. In the case of hyperlipidemias, this recommendation has a special significance, since obesity, in addition to being a risk factor for cardiovascular disease, is directly related to the elevation of cholesterol and plasma triglyceride levels. The reduction of body weight can be achieved through a diet nutritionally compatible with the needs of the patient, and also by increasing physical activity. Both weight reduction and increased physical activity are extremely important elements in the treatment of

hypercholesterolemia. These factors not only promote the reduction of cholesterol levels, but also provide other benefits, such as reduced triglyceride levels, increased high-density lipoprotein-cholesterol (HDL-cholesterol), reduced blood pressure and decreased risk of diabetes mellitus.[30] In the specific case of overweight patients, it is very common to detect elevated plasma triglycerides, and the intake of simple sugars and the consumption of alcoholic beverages should be reduced. Hyperglycidic diets stimulate the synthesis of very low-density lipoproteins (VLDL) and, therefore, often raise triglyceride levels and reduce serum HDL-cholesterol concentration.[1]

Fibers: Dietary fiber is a generic term that includes substances with characteristic chemical structure, physical properties, and physiological effect.[31] Dietary fibers are classified, according to their solubility in water, into soluble (pectins, gums, mucilages and some hemicelluloses) and insoluble (cellulose, lignin and remaining hemicelluloses).[32] Insoluble fibers accelerate intestinal transit, increase fecal cake, slow down the hydrolysis of starch and glucose absorption; soluble fibers, on the other hand, delay gastric emptying, intestinal transit, glucose absorption and reduce serum cholesterol.[32–34] Foods are composed of various types of fiber in varying amounts. Soluble fibers are found in greater quantities in oats, barley, legumes, apples and citrus fruits, while insoluble fibers are found in wheat bran, whole grains and cereals and vegetables.[32,35,36]

The recommended intake of total dietary fiber for adults is 20 to 30g/day, being around 25% (6g) of soluble fiber.[30] The best way to achieve this recommendation is to increase your intake of fiber-rich foods. The group of cereals and their derivatives and those of legumes represent good sources of dietary fiber, as well as fruits. A small apple, a medium orange and a medium banana have, respectively, 0.9, 1.4, and 0.7 of soluble fiber and 1.1, 0.7 and 0.4 of insoluble fiber.[37] The addition of 3g/day of soluble oat fiber to the diet reduces cholesterol and this reduction is greater when the intervention happens in individuals with higher cholesterol levels.[38] The increase in fiber consumption is only one of the components of the diet that affects plasma cholester levels.[39] Thus, the effects of a moderately restricted fat diet can be potentiated by the addition of soluble fiber.[40] It is estimated that every 1% decrease in serum cholesterol can promote a 2% reduction in heart disease.[38]

Alcohol: There is evidence that moderate alcohol consumption is related to lower mortality rates from coronary heart disease. This cardioprotective effect of alcohol is partially attributed to its ability to raise the concentration of HDL, a well-defined negative risk factor for coronary heart disease.[41] It has been shown that both HDL_2 and HDL_3 are positively related to alcohol consumption and inversely associated with rates of cardiovascular disease.[2]

There is controversy regarding the protective equivalence that all alcoholic beverages would exert on atherosclerotic disease. Rimm et al.[42] report a 26% reduction in the risk of cardiovascular disease in men who consume 5-30g of alcohol/day (350 ml of beer, 30 ml of hard liquor and 100 ml of wine), when compared with teetotalers, after controlled intakes of fat, cholesterol and fiber. However, the deleterious effects of alcohol should be considered. In individuals who are prone to dyslipidemia, high alcohol intake can cause elevated triglyceride levels by stimulating VLDL production by the liver. Alcohol can also lead to increased blood pressure and body weight[1] and is hepatotoxic. Therefore, its intake should be carefully evaluated.

With regard to wine, it has been shown that red has more phenolic components than white, and among these phenols the main difference is in the flavonoids, which are present in red wine in a concentration 20 times higher than that of white wine. Flavonoids include components such as quercetin, rutin, which have been shown to have potent biological effects in vitro, including inhibition of eicosanoid synthesis as well as cancer growth and development. It is estimated that for the average North American population, the addition of two glasses of red wine per day increases the flavonoid content of the diet by 40%.[41] Flavonoids encompass a class of natural pigments found with great frequency in nature, being components of a variety of fruits and vegetables, such as cherry, jambolan, grape, strawberry, blackberry, jabuticaba and eggplant.[43] However, the bioavailability of flavonoids in fruits and vegetables is not yet clearly established. They are present in complex polymeric and glycosidic forms, which cannot be easily degraded by digestive juices, and their insolubility can limit or even prevent their absorption. During the fermentation of wine, degradation to monomeric forms occurs; In addition, the alcohol content in wines maintains the stability of flavonoids for a very long period of time in the bottles and, most likely, also in the human intestine.[41]

Antioxidants: The term "antioxidant nutrients" has been used to indicate the ability of some nutrients to prevent oxidative damage in cells. The current theory states that some clinical conditions, such as cardiovascular diseases, can be initiated and propagated by the action of free radicals (superoxide, peroxide or hydroxyl radicals).[44] The antioxidant behavior of vitamins C and E and beta-carotene has received much attention in reducing the risk of coronary artery disease. However, the incomplete stage of knowledge in this area should be emphasized.[45] Antioxidant micronutrients, according to the theory, would be able to neutralize free radicals, inhibit the oxidation of LDL, and thus potentially reduce the risk of coronary artery disease.[46] Some studies emphasize the role of oxidized LDL as an important factor, as it is more atherogenic.[47] Vitamins C, E and beta-carotene are mistakenly characterized only as antioxidants. They are reducing agents, being antioxidants in some circumstances (often in the physiological amounts found in food) and pro-oxidants (producing billions of free radicals in other circumstances (often in the pharmacological amounts found in supplements). In the form naturally present in food, these compounds are chemically balanced, that is, they are part of a mixture in which half of the reducing agents are in oxidized form and half in reduced form, but every supplement is chemically unbalanced.[48] The use of antioxidant vitamins should not be done indiscriminately.[47] The current stage of knowledge does not yet allow its use on a large scale. Several studies present conflicting results.[49–53] In addition, there is a warning about the risk of using supplements instead of lifestyle modification, through well-established preventive measures, such as the use of prudent diet, regular physical exercise and smoking cessation.[54]

Garlic and onion: Some effects on blood pressure, fibrinogen and fibrinolysis, platelet aggregation and plasma viscosity have been attributed to the intake of garlic and onion essential oils.[55,56] Studies using garlic-based products have shown a reduction in total cholesterol, LDL and plasma triglycerides and a reduction in the susceptibility to LDL oxidation.[55,57–59] Garlic's mechanisms of action appear to include effects on cholesterol synthesis, degradation and excretion. Large amounts of these oils (such as 50g) were required to have any effect. This is very difficult to achieve through dietary intake as the oils comprise only 0.06% - 0.1% of the weight of garlic and only 0.005% of the weight of onions. Despite possible beneficial effects, garlic has some negative characteristics. In some people, its use can cause anemia, weight loss, dermatitis and asthma, and may also lead to increased fragility of red blood cells.

Implementation and monitoring of dietary treatment: The combined effect of a diet rich in fruits, vegetables, complex carbohydrates, and low in animal fat and simple sugar, is probably

the appropriate way to reduce chronic diseases. As an alternative, the diets used in the Mediterranean and Asian regions have received considerable attention, since the population of these regions has a low occurrence of cardiovascular disease and longer life expectancy. Although non-dietary factors have probably contributed to longevity in these countries, it is believed that diet plays an important role. Their diets are characterized by the daily consumption of large amounts of foods of plant origin (fruits, vegetables, breads, other forms of cereals, potatoes, beans, nuts, etc.), with fresh fruits being the typical dessert and olive oil the main source of fat. Dairy products (mainly cheese and yogurt), fish, poultry and eggs are consumed weekly in small to moderate amounts. Red meat is little consumed and wine used daily along with meals. It is important to report the practice of regular physical activity, which also contributes to lower rates of cardiovascular disease.[60]

The National Cholesterol Education Program (NCEP)[30] recommends as fundamental the institution of nutritional therapy in the treatment of hyperlipidemias. This modification in eating behavior should be permanent and accompanied by an increase in physical activity. Attention to nutrition should begin with the nutritional assessment that will guide the dietary treatment. For better adherence to the diet, it should be as close as possible to the usual, be nutritionally adequate and pleasant to the palate. In dietary counseling, the patient should receive instructions on proper selection, quantity, and preparation techniques of food. Although there is no need to use special foods, in some circumstances the use of modified, low-fat products can facilitate treatment adherence. The patient should be monitored periodically, and the follow-up of the diet evaluated through a dietary survey, which can be performed through 24-hour recall, frequency or record of food consumed. The support and encouragement of the patient by the health team and the family are important. The family should adopt new eating habits and patterns, since the phase 1 diet is recommended as an appropriate dietary pattern for the population over 2 years of age. Success in accepting and incorporating the diet depends on adequate encouragement, continued nutritional education, and long-term patient monitoring. The attitude of the health team professionals, their interest and involvement are determining factors for treatment adherence. The nutritionist has an important role in the team, since he represents the professional trained to prescribe the dietary intervention.

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Conflicts of interest

No conflict of interest.

References

1. Grundy SM, Ahrens EH Jr. The effects of unsaturated dietary fats on absorption, excretion, synthesis, and distribution of cholesterol in man. *J Clin Invest*. 1970;49(6):1135–1152.
2. Rimm EB, Ellison RC. Alcohol in the Mediterranean diet. *Am J Clin Nutr*. 1995;61(6 Suppl):1378S–1382S.
3. Keys A, Anderson JT, Grande F. Serum cholesterol response to changes in the diet: IV. Particular saturated fatty acids in the diet. *Metabolism*. 1965;14(7):776–787.
4. Hegsted DM, McGandy RB, Myers ML, et al. Quantitative effects of dietary fat on serum cholesterol in man. *Am J Clin Nutr*. 1965;17(5):281–295.
5. Mensink RP. Effects of the individual saturated fatty acids on serum lipids and lipoprotein concentrations. *Am J Clin Nutr*. 1993;57(5 Suppl):711S–714S.
6. Denke MA, Grundy SM. Effects of fats high in stearic acid on lipid and lipoprotein concentrations in men. *Am J Clin Nutr*. 1991;54(6):1036–1040.
7. Kris-Etherton PM, Mustad VA. Chocolate feeding studies: a novel approach for evaluating the plasma lipid effects of stearic acid. *Am J Clin Nutr*. 1994;60(6 Suppl):1029S–1036S.
8. Kris-Etherton PM, Derr JA, Mustad VA, et al. Effects of a milk chocolate bar per day substituted for a high-carbohydrate snack in young men on an NCEP/AHA Step 1 Diet. *Am J Clin Nutr*. 1994;60(6 Suppl):1037S–1042S.
9. Woollett LA, Dietschy JM. Effect of long-chain fatty acids on low-density-lipoprotein-cholesterol metabolism. *Am J Clin Nutr*. 1994;60(6 Suppl):991S–996S.
10. Katan MB, Zock PL, Mensink RP. Effects of fats and fatty acids on blood lipids in humans: an overview. *Am J Clin Nutr*. 1994;60(6 Suppl):1017S–1022S.
11. Mensink RP, Katan MB. Effect of dietary trans fatty acids on high-density and low-density lipoprotein cholesterol levels in healthy subjects. *N Engl J Med*. 1990;323(7):439–445.
12. Judd JT, Clevidence BA, Muesing RA, et al. Dietary trans fatty acids: effects on plasma lipids and lipoproteins of healthy men and women. *Am J Clin Nutr*. 1994;59(4):861–868.
13. Wahle KW, James WP. Isomeric fatty acids and human health. *Eur J Clin Nutr*. 1993;47(12):828–839.
14. *USDA - United States Department of Agriculture, Composition of Foods*. Handbooks number 8-5 (1979), 8-10 (1983), 8-13 (1986).
15. Phillipson BE, Rothrock DW, Connor WE, et al. Reduction of plasma lipids, lipoproteins, and apoproteins by dietary fish oils in patients with hypertriglyceridemia. *N Engl J Med*. 1985;312(19):1210–1216.
16. Kromhout D, Bosschieter EB, de Lezenne Coulander C. The inverse relation between fish consumption and 20-year mortality from coronary heart disease. *N Engl J Med*. 1985;312(19):1205–1209.
17. Grundy SM, Denke MA. Dietary influences on serum lipids and lipoproteins. *J Lipid Res*. 1990;31(7):1149–1172.
18. Beynen AC, Katan MB, Van Zutphen LFM. Hypo- and hyperresponders: individual differences in the response of serum cholesterol concentration to changes in diet. *Adv lipid res*. 1987;22:115–171.
19. Hegsted DM, Ausman LM, Johnson JA, et al. Dietary fat and serum lipids: an evaluation of the experimental data. *Am J Clin Nutr*. 1993;57(6):875–883.
20. Meslier V, Laiola M, Roager HM, et al. Mediterranean diet intervention in overweight and obese subjects lowers plasma cholesterol and causes changes in the gut microbiome and metabolome independently of energy intake. *Gut*. 2020;69(7):1258–1268.
21. Muralidharan J, Moreno-Indias I, Bulló M, et al. Effect on gut microbiota of a 1-y lifestyle intervention with Mediterranean diet compared with energy-reduced Mediterranean diet and physical activity promotion: PREDIMED-Plus Study. *Am J Clin Nutr*. 2021;114(3):1148–1158.
22. Moreno-Frías MLR, Solís-Ortiz S. Dietary intake of micro- and macronutrients is associated with deficits in executive functioning in young women with high adiposity. *Nutrition*. 2022;101:111691.
23. US Department of Health and Human Services and U.S. Department of Agriculture. *2015–2020 Dietary Guidelines for Americans*. 8th edn, 2015.

24. Mach F, Baigent C, Catapano AL, et al. 2019 ESC/EAS Guidelines for the management of dyslipidaemias: lipid modification to reduce cardiovascular risk. *Eur Heart J*. 2020;41(1):111–188.

25. Arnett DK, Blumenthal RS, Albert MA, et al. 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. *Circulation*. 2019;140(11):e596–e646.

26. Mozaffarian D, Ludwig DS. Dietary guidelines in the 21st century - a time for food. *JAMA*. 2010;304(6):681–682.

27. Reynolds A, Mann J, Cummings J, et al. Carbohydrate quality and human health: a series of systematic reviews and meta-analyses. *Lancet*. 2019;393(10170):434–445.

28. Brito L, Sahade V, Weber B, et al. Factors associated with diet quality among Brazilian individuals with cardiovascular diseases. *J Hum Nutr Diet*. 2023.

29. Kirkpatrick CF, Sikand G, Petersen KS, et al. Nutrition interventions for adults with dyslipidemia: a clinical perspective from the National Lipid Association. *J Clin Lipidol*. 2023: S1933-2874(23)00185-X.

30. National Cholesterol Education Program. Second Report of the Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel II). *Circulation*. 1994;89(3):1333–1445.

31. Kritchevsky D. Dietary fibre and lipid metabolism. *Int J Obes*. 1987;11 Suppl 1:33–43.

32. Gorman MA, Bowman C. Position of The American Dietetic Association: health implications of dietary fiber. *J Am Diet Assoc*. 1993;93(12):1446–1447.

33. Kohn IJ, Ribeiro LG. The role of cholesterol in atherosclerosis and its potential management by dietary fiber. *Arq Bras Cardiol*. 1991;56(3):173–184.

34. Glore SR, Van Treeck D, Knehans AW, et al. Soluble fiber and serum lipids: a literature review. *J Am Diet Assoc*. 1994;94(4):425–436.

35. Anderson JW, Bridges SR. Dietary fiber content of selected foods. *Am J Clin Nutr*. 1988;47(3):440–447.

36. Cavalcanti MLF. Fibras alimentares. *Rev nutr PUCCAMP*. 1989;2(1):88–97.

37. McCance and Widdowson's. The Composition of Foods. In: Holland B et al. editors. 5th edn. Cambridge; Southgate. The Royal Society of Chemistry; 1991. p. 462.

38. Ripsin CM, Keenan JM, Jacobs DR Jr, et al. Oat products and lipid lowering. A meta-analysis. *JAMA*. 1992;267(24):3317–3325.

39. Dietary fiber and health. Council on Scientific Affairs. *JAMA*. 1989;262(4):542–546.

40. Williams CL. Importance of dietary fiber in childhood. *J Am Diet Assoc*. 1995;95(10):1140–1146.

41. Goldberg DM. Does wine work? *Clin Chem*. 1995;41(1):14–16.

42. Rimm EB, Giovannucci EL, Willett WC, et al. Prospective study of alcohol consumption and risk of coronary disease in men. *Lancet*. 1991;338(8765):464–468.

43. Bobbio FO, Bobbio FO. Pigmentos naturais. In: Bobbio FO, Bobbio FO (Org.). Introdução à. Química de Alimentos. 2nd edn. São Paulo: Varela; 1992. cap. 6, p. 191–223.

44. Ruxton C. Antioxidant nutrients-do they have a protective role? *Food Chem Toxicol*. 1994;32(10):995–996.

45. McCornick DB. Supplemental antioxidants: No proven panacea. In: Wardlaw GM, Insel PM. Perspectives in Nutrition. 2nd edn. St. Louis: Mosby; 1993. p. 365.

46. Steinberg D, Parthasarathy S, Carew TE, et al. Beyond cholesterol. Modifications of low-density lipoprotein that increase its atherogenicity. *N Engl J Med*. 1989;320(14):915–924.

47. Hoffman RM, Garewal HS. Antioxidants and the prevention of coronary heart disease. *Arch Intern Med*. 1995;155(3):241–246.

48. Herbert V. The antioxidant supplement myth. *Am J Clin Nutr*. 1994;60(2):157–158.

49. Street DA, Comstock GW, Salkeld RM. A population-based case-control study of the association of serum antioxidants and myocardial-infarction. *Am J Epidemiol*. 1991;134(7):719–720.

50. Stampfer MJ, Hennekens CH, Manson JE, et al. Vitamin E consumption and the risk of coronary disease in women. *N Engl J Med*. 1993;328(20):1444–1449.

51. Salonen JT, Salonen R, Seppänen K, et al. Relationship of serum selenium and antioxidants to plasma lipoproteins, platelet aggregability and prevalent ischaemic heart disease in Eastern Finnish men. *Atherosclerosis*. 1988;70(1-2):155–160.

52. Riemersma RA, Oliver M, Elton RA, et al. Plasma antioxidants and coronary heart disease: vitamins C and E, and selenium. *Eur J Clin Nutr*. 1990;44(2):143–150.

53. Alpha-Tocopherol, Beta Carotene Cancer Prevention Study Group. The effect of vitamin E and beta carotene on the incidence of lung cancer and other cancers in male smokers. *N Engl J Med*. 1994;330(15):1029–1035.

54. Steinberg D. Antioxidant vitamins and coronary heart disease. *N Engl J Med*. 1993;328(20):1487–1489.

55. Harenberg J, Giese C, Zimmermann R. Effect of dried garlic on blood coagulation, fibrinolysis, platelet aggregation and serum cholesterol levels in patients with hyperlipoproteinemia. *Atherosclerosis*. 1988;74(3):247–249.

56. Mayeux PR, Agrawal KC, Tou JS, et al. The pharmacological effects of allicin, a constituent of garlic oil. *Agents Actions*. 1988;25(1-2):182–190.

57. Bordia A. Effect of garlic on blood lipids in patients with coronary heart disease. *Am J Clin Nutr*. 1981;34(10):2100–2103.

58. Lau BHS, Lam F, Wang-Cheng R. Effect of an odor-modified garlic preparation on blood lipids. *Nutr Res*. 1987;7(2):139–149.

59. Phelps S, Harris WS. Garlic supplementation and lipoprotein oxidation susceptibility. *Lipids*. 1993;28(5):475–477.

60. Willett WC, Sacks F, Trichopoulou A, et al. Mediterranean diet pyramid: a cultural model for healthy eating. *Am J Clin Nutr*. 1995;61(6 Suppl):1402S–1406S.