

Impact of Nutrients and Food Components on Dyslipidemias: What Is the Evidence?^{1,2}

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ABSTRACT

Dyslipidemias have been shown to bear a close association with an increased risk of cardiovascular diseases, atherosclerosis in particular. As efforts are being made to find alternative therapies and ways to prevent disease, there is a corresponding rise in public interest in food and/or active food components that contribute to an improved lipid profile and, thus, to better health. Besides supplying the basic nutrients necessary for well-being, some foods add further physiologic benefits. In fact, specific foods and bioactive components could be beneficial in controlling dyslipidemias. From a review of the literature on foods and bioactive compounds, their recommended quantities, and expected effects, we found that the following nutrients and food components could positively impact the lipid profile: monounsaturated and polyunsaturated fatty acids, soluble fiber, vegetable proteins, phytosterols, and polyphenols. Therefore, incorporating these components into the regular diets of individuals is justified, because they contribute additional positive effects. This suggests that they also be recommended in clinical practice. Adv Nutr 2015;6:703–11.

Keywords: nutrients; food components; dyslipidemias, cardiovascular disease, antioxidants

Introduction

Cardiovascular diseases $(CVDs)^5$ rank among the highest causes of death globally. WHO estimates indicate that CVDs will account for \sim 20 million deaths in 2015 (1). Atherosclerosis has been identified as the basis for the development of these diseases. It commences as a vascular endothelium aggression and is precipitated by several risk factors, including dyslipidemias (2).

A rise in LDL cholesterol directly influences the development of atherosclerosis. Endothelial dysfunction results in an increase in the permeability of the intima to the plasma lipoproteins, encouraging their retention in the subendothelial space. The LDL cholesterol particles thus trapped are oxidized, producing an increase in concentrations of oxidized LDL cholesterol, which is proinflammatory and immunogenic. This mechanism is regarded as the beginning of early atherogenesis, showing that LDL cholesterol oxidation is an independent risk factor for CVD. Because oxidation takes place proportional to LDL cholesterol concentration in plasma, maintaining this concentration and inflammation under control is of paramount importance in the prevention of new cardiovascular events (3, 4).

Any reduction in the concentration of HDL cholesterol also substantially influences the prevalent atherogenic lipid profile. HDL cholesterol carries cholesterol in a pathway called reverse cholesterol transport and is responsible for removing oxidized LDL cholesterol by inhibiting monocyte attachment to the endothelium. It also stimulates the release of nitric oxide, which prevents vascular bed atherogenesis (5). A meta-analysis of prospective studies has revealed that every 1 mg/dL increase in HDL cholesterol may be associated with a reduction in the incidence of coronary heart disease (CHD) by 2–3% independently of other factors (6). Any increase in HDL cholesterol concentrations reduces the risk of CVD irrespective of a decrease in LDL cholesterol (4).

Diet has been recognized as playing a key role in the control of noncommunicable chronic diseases. Several studies have shown the effectiveness of certain foods in reducing the risk of these diseases. In this respect, specific foods and food components emerge as potential adjuvant strategies in the reduction of the risk of dyslipidemia. They are consumed as part of one's normal diet and exert beneficial

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 5 Abbreviations used: ALA, α -linolenic acid; CHD, coronary heart disease; CVD, cardiovascular disease.

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physiologic effects and/or reduce the risk of chronic diseases, in addition to their basic nutritional functions (7). Food components were revealed to act upon the different metabolic pathways that influence lipid disorders in humans. They are believed to have the potential to ultimately become prominent in the future treatment and primary prevention of dyslipidemias and thereby in the control of CVDs (8).

The objective of this study was to review the scientific literature dealing with the impact of foods or food components in dyslipidemias and check for evidence of their effect on disease control. We also wished to present studies with quantities of foods and food components for use in clinical practice. We performed a literature search of Lilacs, Scielo, PubMed, and Science Direct databases, specifically of the last 15 y of publications related to foods and dyslipidemia, using the keywords "foods," "food compounds," and "dyslipidemia."

Current Status of Knowledge MUFAs

MUFAs include FAs with only one double bond [e.g., oleic acid (18:1n–9), palmitoleic acid (16:1n–7), and elaidic acid (18:1n–9)]; they mainly occur in vegetable oils such as olive and canola. From analysis of randomized trials, the cardioprotective effect of MUFAs has been demonstrated in individuals having metabolic syndrome and CVD by the positive modulation of blood pressure, insulin sensitivity, and serum lipids (9).

There is convincing evidence that replacing carbohydrates with MUFAs increases HDL cholesterol concentrations and that replacing SFAs with MUFAs reduces LDL cholesterol and the total-to-HDL cholesterol ratio (10). Several researches have shown that replacement of MUFAs enhances a rise in the concentration of HDL cholesterol and decreases TGs; it also lowers the susceptibility of LDL cholesterol to oxidation (9, 11).

These lipids are also believed to decrease the HDL cholesterol–lowering effect commonly seen in diets containing low total fat and/or those rich in PUFAs (12). They cause postprandial lipemia because of a reduction in the formation of the remaining chylomicrons (13). Moreover, they are found to reduce platelet aggregation and increase fibrinolysis and coagulation time, reducing the prothrombotic state, which is the characteristic feature of CVD (14).

Limited research has been done on the effect of isolated MUFA consumption in humans, although the literature supports the potential benefits of increased MUFAs and reduced complex carbohydrates in modulating in vivo lipoprotein metabolism (15). The Joint FAO and WHO Expert Consultation on Fats and Fatty Acids in Human Nutrition recommended that MUFA intake be 15–20% of total energy, based on total fat intake (10).

PUFAs

PUFAs are unsaturated FAs with \geq 2 double bonds that enhance human health. For example, linoleic acid (18:2n–6) and α -linolenic acid (ALA) (18:3n–3) are considered to be essential, because they cannot be synthesized endogenously. Whereas linoleic acid is the precursor of arachidonic acid (20:4n–6), ALA is the precursor of EPA (20:5n–3) and DHA (22:6n–3). Vegetable oils such as sunflower and corn oil contain the ω -6 PUFA family. These are consumed more often than those in the ω -3 PUFA family, which occur in flaxseed and fish such as mackerel, herring, and salmon (13).

The action mechanisms of the ω -3 FAs involve the inhibition of the endogenous synthesis and esterification of cholesterol, an increase in cholesterol excretion in the bile, and an increase in bile salt synthesis. These mechanisms also inhibit the activity of glucose-6-phosphatase in the liver, which controls the release of hepatic glucose into the bloodstream. This effect probably accounts for the protective role of ω -3 FAs in the excessive uptake of glucose as evident in hyperlipidemic diets (2, 4). Other effects of ω -3 FA consumption include a drop in TGs in the muscle and fatty liver, confirming its role in those with hypocholesterolemia (14, 15).

EPA and DHA are beneficial in patients with hypertriglyceridemia, because they reduce plasma TGs by lowering VLDL synthesis in the liver (4) . The ω -3 FA family exerts a positive influence by altering plasma lipid concentrations and causing greater endothelial relaxation, both of which are essential for atherogenesis. Normally, ω -6 FA has proinflammatory and procoagulatory effects (10). The ω -3 FAs produce anti-inflammatory and antithrombotic effects by competing with the ω -6 cyclooxygenase enzymes and eicosanoid formation in the pathway, steadily decreasing the thrombotic inflammatory state. The FAO/WHO in Rome has established a recommended ω -6: ω -3 ratio of 5–10:1 (16).

An analysis of the effects of PUFA consumption revealed specific associations between cardiac and vascular hemodynamic improvement, with better TGs and endothelial function, and control of the possibility of thrombosis, inflammation, and arrhythmia (17). Experimental research confirmed the involvement of numerous relevant molecular mechanisms, including alterations in membrane structure; influence on ion channels, gene regulation, and eicosanoid synthesis; and the formation of new mediators of inflammation (2, 4, 17).

In a quantitative analysis of the effect of fish consumption on the incidence of fatal CVDs in human studies (observational studies and randomized controlled trials), it was found that consuming even very small quantities of fish (1 serving/mo of 100 g or 0.14 g of ω -3 FAs) can reduce the risk of mortality from CVD by 17%; furthermore, every subsequent increase of 1 serving/wk leads to an even greater reduction of risk by 3.9% (18). Consuming small amounts of fish was also associated with a 27% risk reduction in nonfatal heart attacks; however, additional fish servings did not produce any further reduction. In a 2014 study in 157 healthy adults, Molinari et al. (19) showed that consuming 400 mg/d of ω -3 FA–enriched milk (150 mg EPA, 150 mg DHA, and 100 mg ALA) for 8 wk increased EPA and DHA blood concentrations without altering the lipid profile.

The vegetable source of ALA, which occurs in flaxseed, canola oil, and soybean oils, as well as in smaller quantities in dark-green leafy vegetables, has also revealed its protective properties (20). A study was conducted in Spain that involved 211 untreated individuals having primary dyslipidemia. After adjustment for confounders, ALA intake was found to be inversely related to the risk of atherosclerotic plaque in the carotid and femoral arteries of those individuals with high CVD risk. This implies one of the antiatherosclerotic effects of the vegetable source of ALA (21).

The hypotriglyceridemic effects of fish oils are well established, and studies have demonstrated a beneficial effect of dietary and supplemental ω -3 FAs, including both EPA plus DHA and ALA, on CHD (22). Replacing SFAs with PUFAs decreases LDL cholesterol concentration and the total-to-HDL cholesterol ratio. A similar but lesser effect is achieved by replacing these SFAs with MUFAs. According to the FAO, based on epidemiologic studies and randomized controlled trials of CHD events, the minimum recommended amount of total PUFA consumption for lowering LDL and total cholesterol concentrations, increasing HDL cholesterol concentrations, and decreasing the risk of CHD events is 6%, and the recommended range for PUFAs is 6–11% of total energy intake (10).

Soluble fiber

Soluble fiber is usually highly viscous and fermented by the intestinal microflora. It is mainly composed of pectin (found in fruits), gums (oats, barley, and legumes such as soybeans), and mucilage. Several studies conducted with oat bran, psyllium, guar gum, and pectin validate the hypocholesterolemic effect of soluble fiber. The results demonstrate that an intake of 5–15 g/d of soluble fiber reduces LDL cholesterol by \sim 15– 20% (23–25). In an experimental study done in rats fed a high-fat diet, it was observed that the consumption of oats reduced weight, body fat, blood glucose, FFAs, TGs, total cholesterol, and the LDL-to-HDL cholesterol ratio, and increased HDL cholesterol concentrations. A dose-dependent response further reduced lipid accumulation in the liver. Thus, oat consumption was shown to increase LDL cholesterol receptors, which in turn exerted a beneficial effect by reducing serum lipids. Therefore, the inclusion of oats in the diet has been suggested as an adjuvant therapy in metabolic disorders (26).

In an intervention study in 100 postmenopausal women, after 3 mo of consuming dehydrated apple (75 g, equivalent to 2 apples) total cholesterol concentration was found to have been reduced because of the anti-inflammatory effect and antioxidant properties of the bioactive components of apples, including soluble fiber, pectin, and phenolic compounds (27). Other benefits are associated with a reduction in dietary saturated fat and protein, accompanied by a high intake of antioxidant substances from a vegetable source (28).

Vegetable protein

The intake of vegetable protein rather than animal protein produces better results in lowering cholesterol concentration, as observed in experimental animals. The cholesterol-reducing effect of soy has also been shown in human beings (29). Apart from soybeans, beans also play a beneficial role in the control of dyslipidemia. Beans (Phaseolus vulgaris) satisfy the main dietary recommendations for well-being because of their high amounts of fiber, starch, and other complex carbohydrates and their low amounts of fats and sodium (30). The USDA Dietary Guidelines for Americans recommend an intake of 3 cups/wk (1 cup = \sim 260 g; 780 g/wk) of legumes (dried beans) (31). In a study in 62 obese individuals (men and women), bean extract intake caused a moderate drop in serum cholesterol concentrations over both short- and long-term use (3–12 mo, respectively), whereas HDL cholesterol increased by 10% in 12 mo (32). The cholesterol-lowering mechanisms of bean intake are yet to be fully understood, and although some hypotheses have been proposed, it has not yet been proven that only one factor is involved; however, there may be a combined effect from several components in legumes, such as protein, soluble fiber, saponins, steroids, polyphenols, and phytates, which lower cholesterol (33).

Vegetable protein–rich diets produce greater satiety or fullness and research has demonstrated their success in producing greater weight loss, lower plasma TGs, reduced blood pressure, and a spare lean-mass effect compared with high-carbohydrate diets; moreover, no negative effects were observed in bone mineral density, renal function, or weight loss (34). The results showed substantial weight loss and control of hypertension and atherosclerosis (35).

Phytosterols

Phytosterols occur solely in plants and exhibit structural functions similar to that of cholesterol in animal tissues. The main action mechanism of dyslipidemia is the lowering of intestinal cholesterol absorption. Phytosterols have been observed to displace cholesterol from intestinal dietary micelles by dynamic competition, with cholesterol being eliminated unabsorbed in the stool, thus reducing absorption and the circulating pool (36). The action mechanism involved is assumed to be that daily phytosterol doses ensure that phytosterol interacts with dietary cholesterol in the intestine by enabling the excretion of cholesterol and bile acids. In the last decade, >200 clinical trials and several meta-analysis have confirmed the efficacy, effectiveness, and safety of its use as an agent of cholesterol reduction $(36-38)$.

Some food sources, such as vegetable oils, cereals, and oilseeds, are beneficial. Whereas omnivorous and vegetarian diets provide \sim 250–500 mg/d of phytosterols, fortified foods or supplements are required at a dose of 2 g/d to reduce cholesterol concentrations. Phytosterols have been suggested as a way of reducing the statin doses required in the treatment of dyslipidemia in order to minimize the side effects of drug therapy; however, further studies are warranted to support this impact (36).

The effectiveness of phytosterols at dosages of up to 3 g/d in reducing LDL cholesterol was demonstrated in several different populations, including people with type 2 diabetes and those with metabolic syndrome and familial hypercholesterolemia. On average, it was possible to achieve a 10% decrease in LDL cholesterol concentrations with an intake of 2 g/d of phytosterols or phytostanols (38). Phytosterolenriched products are normally well tolerated, with no clinically significant side effects (36).

Polyphenols

Flavonoids are polyphenols and include a wide range of water-soluble compounds that produce the coloring in yellow and red flowers, fruit, and leaves. The flavonoids occurring in fruits and vegetables (including proanthocyanidins, anthocyanins, and anthocyanidins) are found in cocoa, cinnamon, and red grapes. Epidemiologic studies imply that the regular intake of flavonoid-rich foods is associated with a decreased risk of CVDs, probably resulting from their antioxidant activity and ability to inhibit the synthesis of endogenous cholesterol (39).

Experimentally, in hamsters fed a cholesterol-enriched diet, anthocyanins were shown to be able to reduce concentrations of atherogenic LDL cholesterol in the plasma and increase HDL cholesterol concentrations by exerting antiatherogenic effects (40). Similar results also were observed after 3 mo of anthocyanin supplementation (160 mg, 2 times/d) in a double-blind, randomized, placebo-controlled trial in 120 subjects with dyslipidemia (41).

Quercetin is one of the major flavonols found in foods, and it is associated with the prevention of LDL cholesterol oxidation and atherosclerosis by exerting anti-inflammatory, antiproliferative, and antioxidative effects (41, 42). In a doubleblind crossover study in 49 healthy male subjects, quercetin intake (150 mg/d) was found to reduce postprandial TG concentrations and raise HDL cholesterol concentrations (43). In an experimental study with an mouse model of type 2 diabetes, quercetin supplementation (offered at 0.04% of the diet) improved hyperglycemia, hypertriglyceridemia, and antioxidant status, whereas quercetin offered at 0.08% also alleviated hypercholesterolemia and elevated HDL cholesterol in the mice. The authors suggest that quercetin may be useful in the management of diabetes and the prevention of diabetic complications (44). Drinking a functional beverage rich in fruit polyphenols (cranberry, apple, and blueberry) for 2 wk was found to reduce TG, total cholesterol, and hepatic cholesterol concentrations significantly in experimental spontaneously hypertensive rats fed an atherogenic diet (45).

Evidence in Food

Grapes

Grapes are a source of polyphenol antioxidants. A 6-wk randomized, double-blind, placebo-controlled study was done to evaluate the effects of the integral grape extract on the antioxidant status and lipid profile of 24 prehypertensive, overweight, and/or prediabetic subjects. The results showed an increase in HDL cholesterol, a reduction in the total-to-HDL cholesterol ratio, and improved antioxidant capacity (46).

Soybeans

Soybeans are a source of vegetable protein, isoflavone, phytoestrogen, and polyphenols that have an antioxidant having the capacity to inhibit LDL cholesterol oxidation. In a randomized, controlled, parallel-arm trial, soy was demonstrated to have a cholesterol-lowering effect in 65 men and women with moderate hypercholesterolemia in whom a reduction in total, LDL, and non-HDL cholesterol, as well as apoB, was observed (47). In a randomized crossover study in 24 subjects, the consumption of soy reduced lipid peroxidation in vivo and increased the resistance of LDL cholesterol to oxidation (48). In a meta-analysis of 8 randomized trials, Zhuo et al. (49) found that participants who consumed high concentrations of isoflavones had LDL cholesterol concentrations \sim 6 mg/dL lower than did participants who consumed the same amount of soy protein with low isoflavone concentrations ($P < 0.0001$). Similar effects were observed when patients who were normo- or hypercholesterolemic were analyzed separately. Because soy mostly has been associated with decreased serum cholesterol concentrations (50), the FDA also approved a daily intake of 25 g soy protein in a diet low in saturated fat and cholesterol as a means to reduce the risk of CVD. Soy has also been advocated in the treatment of hypercholesterolemia (51).

Cocoa

Polyphenols also occur in cocoa, from which they reduce the oxidation of LDL cholesterol, exert an anti-inflammatory effect, act as an antioxidant, moderate the immune response, enhance vascular function, and decrease platelet adhesion (52). These properties are beneficial in the control of pathologies such as CVDs, inflammatory processes, and even cancer (53). The antioxidants in cocoa polyphenols alter glycemic response, change lipid profile, and reduce platelet aggregation, inflammation, and blood pressure, and can moderate intestinal inflammation by cutting down neutrophil infiltration and producing proinflammatory enzymes and cytokines (54). An experimental study revealed that mice fed a fat-rich diet with a cocoa powder supplement showed less weight gain, inflammatory variables, insulin resistance, and fatty liver disease, as well as negatively regulated gene expression to adipose tissue (55). The role of cocoa in resistance to oxidative stress was also demonstrated by Khan et al. (56), who evaluated the effect of a 4 wk period of cocoa intake (40 g/d) in 42 individuals with CVD risk. They recorded a significant increase in HDL cholesterol and a reduction in LDL cholesterol oxidation. Another intervention study with 25 women and 25 men showed that consumption of 50 g/d dark chocolate enriched with flavonoids for 3 wk improved the lipoprotein profile, with greater beneficial effects seen in women than in men (57).

Mate

In South America, the commonly consumed infusion made from yerba mate (Ilex paraguariensis) was shown in an animal model to have excellent lipid-lowering effects by reducing lipid peroxidation, improving endothelial function, and modulating the genes involved in lipid oxidation and lipogenesis (58). In a study involving humans, yerba mate extract consumption by 60 overweight individuals for 6 wk resulted in a significant reduction in the percentage of body fat and adipose tissue, with no side effects (59).

Cruciferae

The regular inclusion of these vegetables in the diet can reduce metabolic disorders related to adiposity (60). In particular, plants belonging to the family Brassicaceae (e.g., broccoli, cabbage, cauliflower, and Brussels sprouts) contain glucosinolates, polyphenols, carotenoids, and phytosterols, which are anti-inflammatory and antioxidant in nature. In an intervention study, 38 healthy volunteers (23 women and 15 men) were fed a serving of 300 g/d of black and red cabbage for a 2 wk period. They showed a significantly increased plasma concentration of carotenoids (lutein and b-carotene) and total antioxidant capacity. A reduction in blood glucose and an improved lipid profile were seen, along with a reduction in total and LDL cholesterol and LDL cholesterol oxidation (61).

Nuts

Nuts included in the diet can significantly improve CVD risk factors because of their content of MUFAs and PUFAs and the presence of high fiber and bioactive molecules such as tocopherols, polyphenols, and arginine (62). Hazelnut consumption (contributing to 18–20% of total daily energy intake) by 21 volunteers with hypercholesterolemia for 4 wk was shown to act beneficially on the lipid profile by reducing total and LDL cholesterol and TGs, increasing HDL cholesterol, and improving inflammatory markers (63). An experimental study in 40 hamsters fed a high-fat diet for 8 wk demonstrated that the consumption of a hazelnut skin extract reversed the increase in total and LDL cholesterol induced by the high-fat diet, decreased circulating concentrations of FFAs and TGs, and also increased the excretion of bile acids (64). There is increasing proof of the potential that nuts have in lowering LDL cholesterol and increasing HDL cholesterol, and the FDA advocates an intake of 42 g/d for cardiovascular health (62, 64).

Cranberries

Cranberries (Vaccinium macrocarpon), one of the main sources of polyphenols, have found widespread use for their antioxidant abilities. Cranberries have a variety of phytochemicals, including 3 classes of flavonoids (flavonols, anthocyanins, and proanthocyanidins), catechins, and phenolic acids, substances related to a wide range of biological effects, including acting as an antioxidant, modulating enzyme activity, and regulating genetic expression (65).

Their nutritional attributes make them one of the important dietary targets in the prevention of CVD by exerting a beneficial effect on the risk factors of CVD, which include dyslipidemia, oxidative stress, hypertension, inflammation, and endothelial dysfunction. After a chronic intervention with cranberries, clinical studies have shown remarkable improvement in the lipid profile, apoA-I, and oxidative stress, and decreased apoB, fasting plasma glucose, and C-reactive protein (66, 67). In an experimental study in animals that mimicked postmenopausal women, who show a natural susceptibility toward developing CVD, Yung et al. (68) found that an 8 wk intervention of cranberry juice induced a significant decrease in LDL cholesterol, TGs, total cholesterol, and non-HDL cholesterol. Many studies employing different models have attempted to evaluate the health benefits of cranberries, revealing their antioxidant capacity. This implies that cranberry polyphenols may act directly in eliminating free radicals from the body and/or influencing stronger expression and synthesis of the natural antioxidant enzymes (69–71). Cranberry consumption is also related to improved lipid profile and endothelial function; however, even in the literature, the amounts that must be consumed to achieve the benefits attributed to them are not mentioned.

Curcumin

The chief bioactive component of turmeric is curcumin. Its antioxidant and anti-inflammatory characteristics are said to be correlated with an improvement in CVD risk, a delay in oncogenesis, and having beneficial effects upon the modulation of various factors, including lipids and cholesterol. Curcumin as a supplement in an experimental model for atherosclerosis negated the ill effects of a high-fat diet on weight increase, the development of fatty liver, dyslipidemia, the expression of inflammatory cytokines, and atherosclerosis, with a dose-dependent response (72). However, in a recent systematic review and meta-analysis of randomized controlled trials, curcumin supplementation apparently did not affect total cholesterol, LDL cholesterol, TGs, and HDL cholesterol at all. Therefore, more controlled studies are necessary to confirm its properties in lipid modulation (73).

Garlic

The bioactive ingredients in garlic are enzymes (e.g., allinase) and sulfur-rich compounds such as the enzymatically produced alliin and its compounds (e.g., allicin, the active ingredient). Allicin concentrations in garlic vary based on the garlic processing method. Because allicin is an unstable compound, it gets quickly transformed into different chemicals. However, even in the absence of allicin, garlic still preserves it positive effects on CVDs. Clinically, garlic's benefits have been reported in the alleviation of several conditions, including hypertension, hypercholesterolemia, diabetes, and atherosclerosis. The possible antibacterial, antihypertensive, and antithrombotic abilities of garlic also render it an important antiatherogenic (74).

Onions

Onions rank high among the vegetables that are rich in flavonoids, predominantly containing quercetin. Their most substantial benefits related to CVDs involve lowering blood pressure and oxidized LDL cholesterol and acting as an inflammatory marker. However, the best effects of onions are noted when \sim 150 mg of quercetin is consumed, which corresponds to an intake of \sim 700 mg/d onion (75).

Cinnamon

The beneficial effects of cinnamon (Cinnamomum zeylanicum) in vitro and in animal models have been revealed in the alleviation of diabetes associated with weight loss and a decrease in fasting glucose concentrations and glycated hemoglobin (a substance contained in red blood cells that binds to glucose and provides a measure of average blood glucose, useful in the diagnosis of diabetes) (76). Cinnamon

TABLE 1 Possible involvement of nutrients and food components in the lipid profile and cardiovascular health as demonstrated in studies¹

Food/component	Recommended amount	Effect/allegation	Reference
ω -3 FAs	$2 - 4$ g/d	Supplementation with ω -3 FAs from	(11)
		seafood is recommended for	
		severe hypertriglyceridemia	
	\geq 2 Meals/wk including fish	↓ CVD risk	(11)
	$1 - 2$ g/d	J TGs 3-9%	(24)
		J Mortality 20-30%	
Phytosterols	2 q/d	I TC	(36)
	2 g/d	LLDL cholesterol 10-12%	(36)
	$1 - 2$ g/d	L LDL cholesterol 5-10%	(25)
	$1 - 3$ g/d	\perp TC 2-13%	(24)
	$1 - 3$ g/d	LLDL cholesterol 4-13%	(24)
	2 g/d	LLDL cholesterol 10-15%	(83)
	$3-4$ g/d	Amount that can be used as a	(83)
		supplement to treat patients	
		with hypolipidemia	
Phytosterols and phytostanols	\geq 1.3 g/d Phytosterols	↓ CVD risk	(23)
	\geq 3.4 g/d Phytostanols	J CVD risk	(23)
	3 g/d (for a minimum of 2-3 wk)	LLDL cholesterol 11.3%	(23)
Soy protein	$1.5 - 2$ g/d	J LDL cholesterol 10%	(84)
	2 g/d	↓ LDL cholesterol up to 10%	(38)
	30-50 g/d soy protein powder	\downarrow TC 2-10%	(24)
	(100-200 mg isoflavones)		
	30-50 g/d soy protein powder	LLDL cholesterol 3-11%	(24)
	(100-200 mg isoflavones)		
	25 g/d	LLDL cholesterol 4-6%	(25)
	25 g/d	L LDL cholesterol 6%	(83)
	50 g/d	L LDL cholesterol 3-9%	(85)
Soluble fiber	25 g/d	J CVD risk	(51)
	\geq 3 g/d β -glucan from oats and/or barley	J CHD risk	(23)
	\geq 7 g/d psyllium		
	$5 - 15$ g/d	\downarrow TC 2-12%	(24)
	$5 - 15$ g/d	J LDL cholesterol 5-20%	(24)
Oatmeal	$5 - 10$ g/d	\downarrow LDL cholesterol \sim 5%	(25)
	60 g/d	LLDL cholesterol 2-6%	(24)
Oat bran	60 g/d	\downarrow TC 4-6%	(24)
	$50 - 75$ g/d	J LDL cholesterol 5%	(25)
Psyllium	$2-3$ teaspoons (6-9 g)/d	J LDL cholesterol 5%	(25)
Dietary fiber	Effect for every 1 g consumed	↓ TC 1.75 mg/dL	(23)
	Effect for every 1 g consumed	↓ LDL cholesterol 2.2 mg/dL	(23)
	20-30 g/d and 5-10 g soluble fiber	\downarrow TC	(83)
Nuts	80 g/d almonds or 50 g/d nuts	J LDL cholesterol 5%	(24)
	(replacing 30% of the fat obtained		
	from monounsaturated fat)		
	42 g/d	L LDL cholesterol 5%	(25)
Green tea	1200 mL/d (for 3 mo)	J TC 2.3%	(24)
	1200 mL/d (for 3 mo)	LLDL cholesterol 10%	(24)
	1200 mL/d (for 3 mo)	\downarrow TGs 6%	(24)
	1200 mL/d (for 3 mo)	↑ HDL cholesterol 3.8%	(24)
Red wine	300 mL/d for men	↑ HDL cholesterol 13%	(24)
	200 mL/d for women		
Legumes	3 cups/wk (780 g/wk)	\downarrow TC	(30)
	3 cups/wk (780 g/wk)	J LDL cholesterol	(30)

¹ CHD, coronary heart disease; CVD, cardiovascular disease; TC, total cholesterol; \downarrow , reduction; \uparrow , increase.

reduces LDL cholesterol and increases HDL cholesterol. Although the positive influences of cinnamon are claimed in most animal models, clinically proven trials remain scarce and contradictory (77).

Other foods

We noted that there are other food matrices containing compounds with proven health benefits. In a study in rats fed a high-fat diet, the consumption of tomato juice and tomato products (sources of lycopene) reduced hallmarks of steatosis, TGs, and VLDLs, and increased lipid metabolism by inducing an overexpression of genes involved in more efficient FA oxidation (78). In humans, a randomized, single-blinded, controlled clinical trial demonstrated that raw tomato consumption produced a favorable effect on HDL cholesterol concentrations in overweight women (79). Moreover, recent studies suggest an important role of citrus flavonoids (including naringenin, hesperidin, nobiletin, and tangeretin) in the treatment of dyslipidemia, insulin resistance, hepatic steatosis, obesity, and atherosclerosis (80, 81). Human intervention studies with the use of chokeberries, cranberries, blueberries, and strawberries (fresh, as a juice, or freeze-dried) or purified anthocyanin extracts have demonstrated significant improvements in LDL cholesterol oxidation, lipid peroxidation, total plasma antioxidant capacity, dyslipidemia, and glucose metabolism (82). As natural sources of antioxidants, the regular consumption of these foods can exert positive effects, especially in improving biomarkers of oxidative stress and acting against dyslipidemia and other diseases.

Some nutrients and food components containing potential benefits for the control of dyslipidemia, the amount used, and the effects observed based on scientific evidence are listed in Table 1.

Conclusions

With respect to the link between health and food, several pieces of scientific evidence are available on the beneficial effects of foods and their bioactive components in the regulation of the lipid profile, and thereby in the prevention and control of dyslipidemia. In general, the following have been found to exert a positive impact on human health: PU-FAs and MUFAs in appropriate proportions, soluble fiber (oats and psyllium, in particular), phytosterols, soy protein, oilseeds, and nuts. Importance should also be given to a diet that includes plenty of fruits and vegetables and antioxidant sources, because they play a role in protection against LDL cholesterol oxidation. The combined effect of the inclusion of certain nutrients and food components and their additional effect on dyslipidemia justifies their daily recommendation in clinical practice.

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