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РЕКОМЕНДАЦИИ ПО КОРРЕКЦИИ ПИТАНИЯ В ЛЕЧЕНИИ АТЕРОСКЛЕРОЗА

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Recommendations For Nutritional Correction in The Treatment of Atherosclerosis

Резюме

Коррекция питания является основой российских и зарубежных рекомендаций по лечению атеросклероза, стабильных и нестабильных форм ишемической болезни сердца. Благодаря правильно подобранному сбалансированному рациону возможно достижение целевых показателей липидного профиля. Поэтому назначение диеты является обязательным и самым первым компонентом в лечении любых форм атеросклероза. Анализ научной литературы с использованием библиографических баз NCBI, WoS, Scopus и РИНЦ показал, что наиболее приемлемы в профилактике и лечении атеросклероза вегетарианская и веганская диеты, достоверно снижающие риск развития и прогрессирования атеросклероза, осложнений и смертности. Это связано с тем, что растительные волокна препятствуют всасыванию холестерина и способствуют нормализации микрофлоры кишечника. Содержащиеся в овощах, фруктах, ягодах, чае и зерновых полифенолы препятствуют агрегации тромбоцитов и воспалительным процессам, способствуют улучшению состояния эндотелия и соотношения липопротеинов крови. Антиатерогенными свойствами обладают соевый белок, витамин D, омега-3-жирные кислоты и многие другие описанные в обзоре компоненты. Для всеядных людей необходимо ограничение атерогенных продуктов, богатых холестерином, железом, сахаром, кальцием и фосфатами. Важное значение имеет способ приготовления, поскольку при жарке как растительных, так и животных продуктов образуются атерогенные вещества, способствующие воспалительным процессам в стенках артерий и дислипидемии.

Ключевые слова: *антиатерогенная пища, атеросклероз, вегетарианство, диета, пищевые волокна, растительные продукты*

Конфликт интересов

Авторы заявляют, что данная работа, её тема, предмет и содержание не затрагивают конкурирующих интересов

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Abstract

Nutrition correction is the basis of Russian and foreign recommendations for the treatment of atherosclerosis, stable and unstable forms of ischemic heart disease. A properly selected balanced diet allows achieving target lipid profile indicators. Therefore, the appointment of a diet is a mandatory and the very first component in the treatment of any form of atherosclerosis. Analysis of scientific literature using the bibliographic databases NCBI, WoS, Scopus and RINTS showed that vegetarian and vegan diets are the most acceptable in the prevention and treatment of atherosclerosis, reliably reducing the risk of development and progression of atherosclerosis, complications and mortality. This is due to the fact that plant fibers prevent the absorption of cholesterol and help normalize the intestinal microflora. Polyphenols contained in vegetables, fruits, berries, tea and grains prevent platelet aggregation and inflammatory processes, help improve the condition of the endothelium and the ratio of blood lipoproteins. Soy protein, vitamin D, omega-3 fatty acids and many other components described in the review have anti-atherogenic properties. Omnivores need to limit atherogenic products rich in cholesterol, iron, sugar, calcium and phosphates. The cooking method is important, since frying both plant and animal products produces atherogenic substances that contribute to inflammatory processes in the arterial walls and dyslipidemia.

Key words: *antiatherogenic food, atherosclerosis, vegetarianism, diet, dietary fiber, plant products*

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AS — atherosclerosis, IHD — ischaemic heart disease, HDL — high-density lipoproteins, LDL — low-density lipoproteins, MFC — macrophage foam cell, PSFA — polyunsaturated fatty acids, NFA –unsaturated fatty acids, CVD — cardiovascular disease, IL — interleukin

Introduction

The main strategy to prevent atherosclerosis (AS) is promotion of healthy lifestyles (HLS), quitting bad habits, especially smoking [1]. An important parameter of HLS is the use of special diets, reducing the risk of AS-induced cardiovascular diseases (CVD) [2]. Dietary interventions are the foundation of the Russian and foreign recommendations on the management of AS, stable and unstable ischaemic heart disease. An optimally selected, balanced diet can help in achieving lipid profile targets. In this context, vegan and vegetarian diets are the most efficient. In 2017, a study in 3,696,778 subjects demonstrated reduced risk of AS in individuals who consumed a lot of fruit and vegetables [3]. Meta-analyses conducted in 2023 showed significantly reduced risk of CVD, IHD [4] and AS [4] in vegetarians. The long-term plant-based diet (3.7 years on the average) results not only in slower AS progression, but also firm myocardium reperfusion, as confirmed by PET CT results, and AS regression seen on angiocardiograms [6]. At the same time, unhealthy diets are a leading cause of early death in a majority of countries, mainly deaths of CVD [7]. A meta-analysis conducted in 2024 demonstrated statistically better arterial stiffness and reduced risk of AS in vegetarians vs. general population consuming meat as well [8].

However, alternative diets are required, because not all people can adhere to a strict vegan or vegetarian diet due to individual differences. Since AS pathogenesis is impacted not only by dyslipidaemia [9], which depends on genetic and epigenetic factors [10], but also inflammatory processes [11] and endothelial dysfunctions [12], a Mediterranean diet can be recommended in this case. This diet means limited consumption of sweets and meat, moderate consumption of wine, fermented milk products and seafood, frequent consumption of olive oil, nuts, seeds, grain, vegetables, fruit, and legumes. There are reports of antiatherogenic effects of the Mediterranean diet for inflammation and vascular endothelium dysfunction due to changes in expression of genes *TCF7L2, CETP, APOA2, IL-6, COX-2* involved in this process [13]. The arterial endothelial function (production of VCAM-1, ICAM-1, VEGF molecules) is also normalised with limited consumption of food (small servings) to lose weight [12]. This can be the reason why Okinawa residents, who consume fewer calories, live longer [14].

Antiatherogenic effects of vegan, vegetarian and Mediterranean diet can be a result of a well-balanced ratio of food components, because plant products contain significantly more carbohydrates, less fat and sodium as compared to animal products [15]. According to meta-analyses results, the lowest risk of AS-induced CVD mortality is observed when the carbohydrate ratio is 50 % to 55 % (vs. fats and proteins), whereas a high risk was identified when this ratio decreased or increased. Consumption of plant proteins and fats (vs. animal proteins and fats) reduced the risk of AS mortality and associated CVD [16]. It is also essential to replace products with a high glycaemic load with whole-grain products and cereal with a low glycaemic load; and to consume less salt, red and processed meats [17]. Therefore, according to clinical guidelines of the National Atherosclerosis Society (NAS), Russian Gerontologist and Geriatrician Association, Eurasian Therapeutic Association, Eurasian Cardiology Association, Russian Society of Cardiosomatic Rehabilitation and Secondary Prevention, Russian Scientific Medical Therapeutic Society, a diet involving consumption of a lot of fibre, wholegrain cereals, vegetables, fruit and fat-free dairy products has been proven to be efficient in CVD prevention [18].

Inflammation is a significant factor of AS pathogenesis [11]; therefore, in addition to reduced levels of cholesterol and LDL, it is essential to cut on products with proinflammatory characteristics. Plant products are optimal. A randomised clinical trial in patients with IHD, who were on a vegan diet for 8 weeks, showed significant reduction in high-sensitivity C-reactive protein levels by 32 % vs. the group who were on the diet developed by the American Heart Association [19]. However, even plant products contribute to AS development if cooked incorrectly, because frying with animal or vegetable oils is

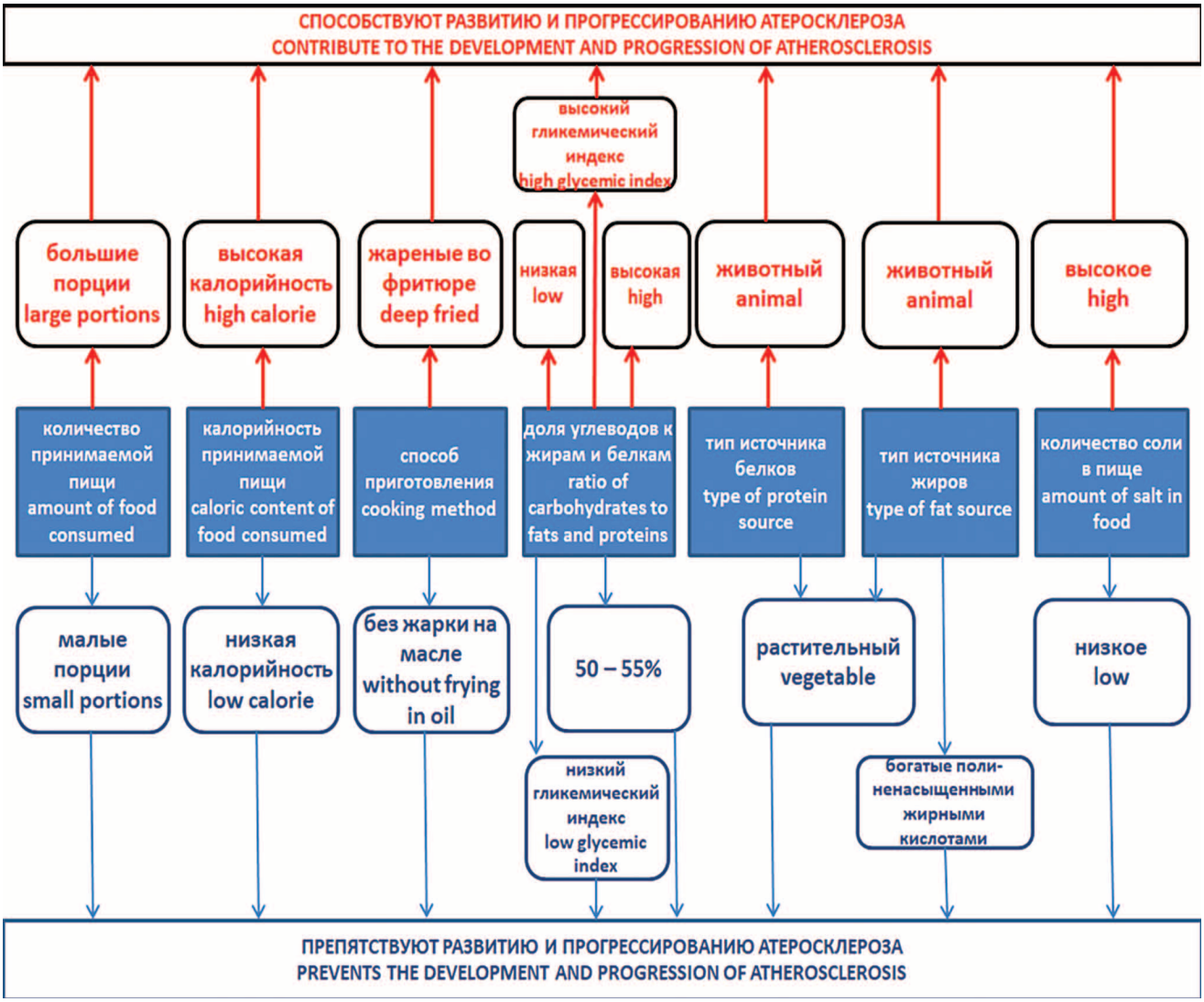

Figure 1. *Scheme of the effect of various components of the diet on the development of AS*

associated with production of oxidised food lipids, which cause vascular wall inflammation and atherogenesis [20]. Therefore, deep-fried potato and banana crisps, savoury snacks contribute to AS [21]. Thus, for AS prevention and management it is essential to consume low-calorie food, to ensure optimal carbohydrate composition and predominant content of plant protein and fat, as well as to limit consumption of salt, animal and deep-fried food (Fig. 1). It is important to consider in detail mechanisms of action of products possessing antiatherogenic effects, which can be recommended to patients with AS.

Effects of plant products on atherosclerosis

One of the mechanisms of favourable effects of vegan and vegetarian diets on AS [3, 4, 6, 8] is regulation of blood cholesterol levels. Up to 80 % of endogenous cholesterol is synthesised in the liver and is excreted with bile as bile acids. However, absorbed bile pool in the ileum contains 2/3 of biliary and 1/3 of endogenous cholesterol [22]. Effects of vegetarian and vegan diets on these processes facilitate reduced cholesterol levels and reduction in atherosclerotic load from atherogenic lipoproteins [5]. This is primarily due to the fact that plant products contain fibres, which are not digested by human gastric enzymes. These can be water-soluble and insoluble (coarse) dietary fibres. Water-soluble fibres contained in barley, oats, legumes, vegetables and fruit include beta-glucans, gums, pectin, slime, stable starches and fructans (inulin, fructo-oligosaccharides). They adsorb and bind cholesterol, reducing its absorption and facilitating its excretion with bile and faecal lipids [23].

Normally, 100 to 300 mg of cholesterol is evacuated by an adult with faeces daily [22]. Nuts, mill offals and wholegrain products contain insoluble dietary fibres (hemicellulose, lignin, cellulose), which reduce the transit time of cholesterol in intestines and its absorption. The recommended daily consumption of dietary fibres is 25 g for women and 38 g for men [23]. Although nuts contain a lot of fats, their consumption reduces total cholesterol and LDL levels, thus reducing the risk of AS [24].

In randomised controlled studies, daily consumption of 3 g of barley [25] and oat [26] beta-glucan, as well as food supplementing with oats fibres [27] facilitated reduction of blood cholesterol levels in subjects.

In addition to the effects of dietary fibres on intestinal cholesterol absorption, plant products contain various substances, possessing other mechanisms of antiatherogenic action. It has been demonstrated that higher consumption of tomatoes, tomato products and lycopene supplements has favourable effects on endothelial function and blood lipids [28]. Plant products contain various polyphenols, which protect against AS; this action is associated with antioxidative and anti-inflammatory action, modulation of lipid metabolism and intestinal microbiota function. Polyphenol-rich products are chocolate, coffee, tea, and fruit in any form [29]. Polyphenols (Fig. 2) include flavonoids (also subclasses of flavonols, flavones, isoflavones, flavanones, anthocyans), stilbenes (resveratrol and benzoic acid), phenolic acids (cinnamic acid), lignans (silimarin). Flavonols include kaempferol, quercetine, myricetin, which have antioxidant effects, stimulate endothelial NO-synthase, reduce VCAM-1 (vascular cell adhesion molecule-1) expression. They can be found in blueberries, merlot, kale, tea, broccoli, onions, and leek [30].

Onions are very rich in quercetine, a polyphenol compound; a clinical study in healthy males show that the daily consumption of quercetine normalises endothelial dysfunction compromised in AS patients [31]. Garlic contains flavonoids myricetin, isoquercitrin and isorhamnetin [32]. Regular consumption of garlic extract by AS patients for a year improved vascular elasticity and restored impaired microvascular perfusion [33]. In a randomised, double-blind, placebo controlled study in patients with a higher risk of CVD, garlic extract was statistically efficient in inhibition of calcification progression in coronary arteries and reduced production of proinflammatory cytokine IL-6 [34]. Flavones found in citrus rind (tangeritin, sinsensetin, nobiletin) inhibit phospholipases PLCγ2 and MAPK in platelets. Soya isoflavones (daidzen, glycitin, genistein) reduce the risk of IHD and myocardial infarction [30].

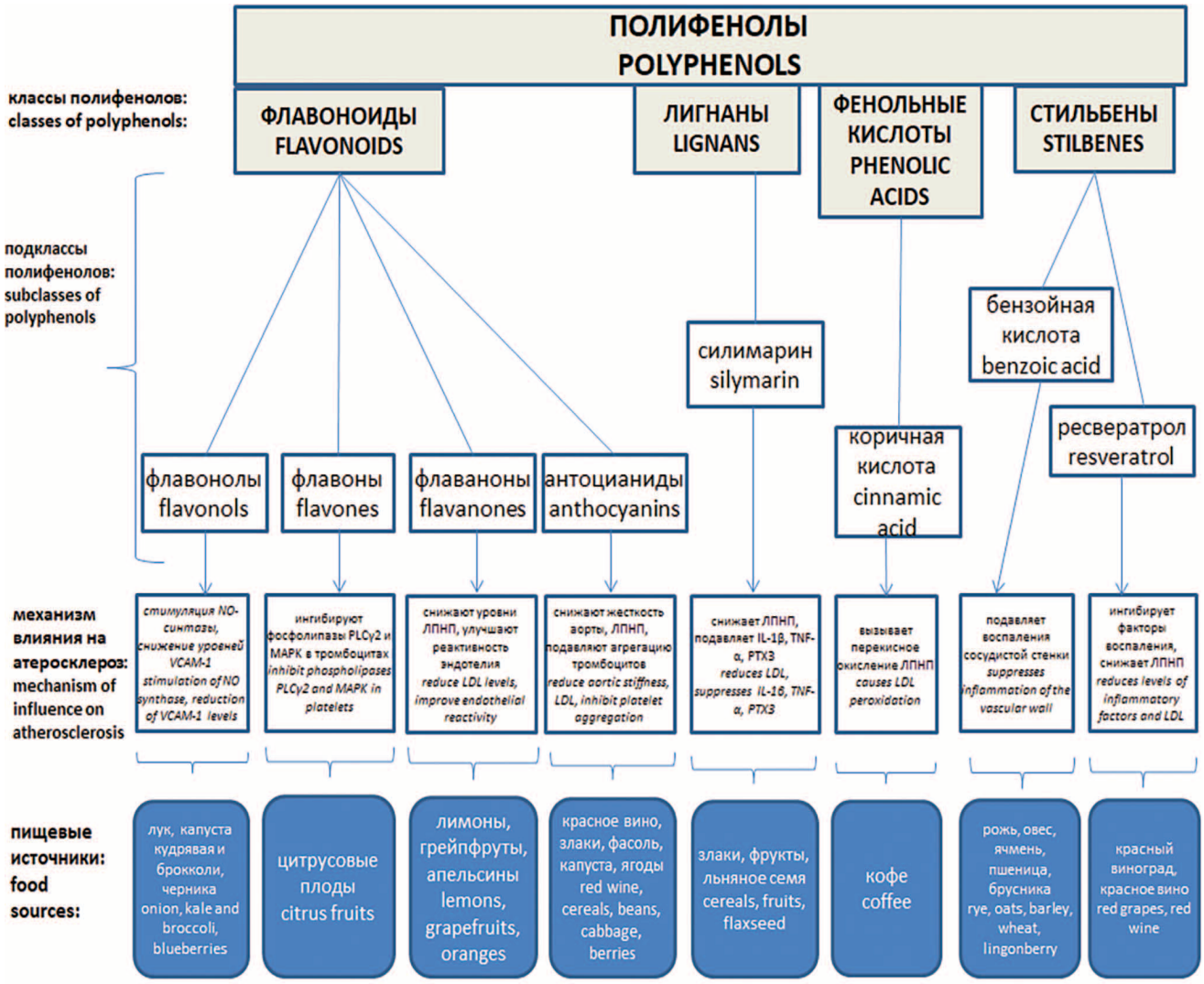

Figure 2. *Polyphenols contained in plant products and their effects on the development of atherosclerosis*

Flavanones eridicitol, hesperetin, naringenin in lemons, oranges and grapefruits reduce atherosclerotic plaque progression, reduce LDL and triglyceride levels, and improve microvascular endothelial reactivity. Anthocyans in blueberries, dewberries, strawberries, cherries, aubergines, opinions, radishes, cabbage, beans, grains, and red wine reduce aorta stiffness, LDL, triglyceride and total cholesterol levels, inhibit platelet aggregation. Resveratrol in grapes and wine inhibits production of nuclear factor NF-κβ and other inflammation factors, reduces LDL and thrombogenic PAI-1 (plasminogen activator inhibitor-1) levels, stimulates expression of *SIRT1* and production of anti-inflammatory adiponectin, and inhibits expression of proinflammatory factors eNOS, NFE2L2, ETI, IL-8 [30]. According to a meta-analysis conducted in 2024, daily consumption of 15 mg of resveratrol by individuals at a risk of IHD reduces TNF-α levels and improves endothelial function due to higher eNOS levels [35]. Benzoic acid in rye, barley, wheat and cowberry has anti-inflammatory and antibacterial effects. Cinnamic acid in coffee beans facilitates cross-oxidation of LDL. Silimarin in grains, fruit, holy thistle and flax seeds also reduces LDL cholesterol and total cholesterol levels, inflammatory biomarkers PTX3, TNF-α, IL-1β [30].

Citrus fruits are a source, that is very rich in flavonoids with anti-inflammatory and antioxidative effects (kaempferol, apigenin, luteolin, rutin, nobiletin, hesperetin, hesperidin, naringenin, naringin, quercetine). These flavonoids modulate adhesion molecules, including ICAM-1, VCAM-1, E-selectin, P-selectin. In preclinical AS models, administration of citrus flavonoids reduced levels of chronic inflammation TNF-α, NF-κB, interleukins, oxidative stress markers (glutathione, superoxide dismutase) [36]. Hesperidin in citrus fruit activates ABCA1 expression 1.8 times and boosts reverse cholesterol transport [37]. Citrus flavonoids naringenin, tangeritin, nobiletin and hesperetin are also potential therapeutic agents for the management of metabolic dysfunction. Epidemiological studies demonstrate positive correlation between consumption of large levels of flavonoids and reduced risk of IHD and stroke. Cell cultures and animal models confirmed lipid-lowering and anti-inflammatory effects of citrus flavonoids [38].

Naringenin and hesperetin in citrus fruit inhibit cholesterol synthesis as a result of 2.4-fold reduction in hydroxy-methyl-glutaryl-CoA-reductase production and can be an alternative to statins [37]. Plant products contain other promising substances, which can be used in production of new AS medications. For instance, curcuma is rich in 1,7-bis(4-hydroxy-3-methoxyphenyl)-1,6-heptadiene-3,5-dione, also known as curcumin, which reduces blood lipid levels and inhibits aortic AS [39]. In a randomised clinical trial, daily consumption of 500 mg of curcumin for 8 weeks contributed to statistically significant reduction in total cholesterol, triglycerides, LDL and TNF-α in patients who underwent elective coronary angioplasty [40]. Dietary supplements with capsaicinoid (found in pimento) reduce cholesterol and LDL levels without any effects on triglycerides HDL [41]. Antiatherosclerotic effects are observed in vegetable protein isolates due to their optimal amino acid composition and presence of residual bioactive plant substances. According to meta-analysis results, daily dietary supplementation with 25 g of soya protein reduces blood LDL levels in individuals with mild hypercholesterolaemia [42].

Results of randomised controlled studies show that replacement of saturated fat in food with linolic acid (omega-6, which is a main component of numerous vegetable oils, including sunflower, soya and corn oils) is efficient in lowering serum cholesterol levels, but does not reduce the risk of death of CVD [43]. Despite the beneficial effects of Mediterranean diet, rich in olive oil [13], literature analysis did not show any randomised clinical trials with statistically significant data on the impact of olive oil or oleic acid on AS development. This can be associated with the fact that, in addition to omega-6, virgin olive oil also contains oleuropein, hydroxytyrosol and tyrosol, which possess cardioprotective effects. There are reports on the inhibiting effect of oleuropein on collagen-induced platelet activation and IL-8 production. In experiments, administration of tyrosol at a dose of 4 mg/kg body weight in the presence of saturated fats and cholesterol reduced AS lesions as compared to animals on the same diet, but without tyrosol. Inhibition of collagen-induced platelet activation under the influence of hydroxytyrosol has been demonstrated. In men with elevated blood pressure, hydroxytyrosol normalised the blood lipid profile. Tyrosol and hydroxytyrosol inhibited phosphorylation of MAPK (mitogen-activated protein kinase, which boosts cell proliferation), production of reactive oxygen intermediates, and secretion of proinflammatory cytokines, including IL-8 [44]. However, a meta-analysis conducted in 2024 showed that consumption of omega-3 (the main component of flaxseed oil) reduces the all-cause mortality, including deaths caused by atherosclerotic cardiovascular diseases. An increase in omega-3 consumption by 1 g/day reduced the risk of death by 3.5 %. Consumption of omega-6 and total polysaturated fatty acids (including oleinic acid) did not demonstrate any significant correlation [45]. Another meta-analysis also confirmed that high doses of omega-3 significantly reduce AS progression [46]. In addition to plants, omega-3 is found in oily fish, consumption of which significantly reduces plasma triglyceride levels and increases the concentration of anti-atherogenic HDL [47]. Also, omega-3 consumption is associated with marked inhibition of platelet aggregation in patients with CVDs vs. healthy controls [48]. These effects were observed when sources of omega-3 were not heat treated (raw flaxseed oil), because aldehydes and epoxy fatty acids produced during frying neutralise favourable effects on AS [20].

Role of vitamins and intestine-active products in the development of atherosclerosis

Reduction in the risk of AS of peripheral arteries with consumption of vitamins A, B6, E, C and folic acid has been demonstrated [49]. It is essential to supplement food with products rich in vitamin B and not dietary supplements containing these vitamins, because a meta-analysis of randomised controlled studies did not show any proofs of the protective effect of vitamins B on AS progression [50]. Since the highest content of vitamins C and E, precursors of vitamin A and folic acid is found in plants, available data are another argument for the use of vegetarian and vegan diets in the prevention and management of AS. Randomised controlled studies using flow-mediated dilatation demonstrated that folic acid restores epithelium damaged by AS [51]. Beta-carotene, riboflavin and niacin affect AS pathogenesis by inflammation inhibition [11]. A meta-analysis showed that niacin also lowers LDL levels [52]. Although vitamin B supplements do not affect AS development, they do not induce disease. Therefore, vitamin supplements are not contraindicated in patients with AS and can be recommended in case of vitamin deficiency.

The main factors of AS pathogenesis are macrophage foam cells (MFC) in atherosclerotic plaques. MFC are dysfunctional for autophagy. Vitamin D3 and its receptor inhibit MFC production and induce autophagy, considerably restoring oxidative lipoprotein autophagy, boosting autophagy-mediated lipid destruction in macrophages, thus preventing macrophage conversion into MFC [53]. In comparative experiments in Yucatan micropigs, which were fed with cholesterol-rich feed for 48 weeks, vitamin D3 deficiency lowered plasma HDL levels and contributed to cholesterol accumulation and AS development. Animals, who received vitamin D3 at a dose of 1,000 IU/day and 3,000 IU/day, did not show such differences. On foam cells isolated from human monocytic macrophages, vitamin D3 contributed to polarisation to M2 macrophages, with reduced expression of pro-inflammatory TNF-α, IL-1β, IL-6, and improved cholesterol outflow [54].

In cell culture experiments, vitamin D3 prevented endothelial cell death by modulating apoptosis and autophagia due to inhibition of superoxide ion production, supporting mitochondrial function and cell viability, kinase activation and NO production induction [55]. In mice experiments, removal of vitamin D3 receptor accelerated AS development due to better adhesion, migration and transfer of MFC cholesterol to atherosclerotic plaques. MFC production was induced by interaction between vitamin D3 receptor with SERCA2b and activation of stress CaMKII-JNKp-PPARγ signalling, stimulation of CD36 and SR-A1 receptors [56]. According to a systemic review of clinical trial results, vitamin D3 deficiency is associated with asymptomatic AS [57]. Blood tests and ultrasonic Doppler vessel examination in Korean adults showed a negative association between vitamin D3 levels and carotid AS [58]. Clinical studies did not demonstrate adverse effects of high doses of vitamin D3 for AS development and progression. Even a single dose of 100,000 IU of vitamin D3 taken by female patients with AS did not affect endothelial function and vessel stiffness, inflammation and coagulation parameters [59]. Meta-analyses made it possible to reliably demonstrate that dietary supplementation with vitamin D3 improves endothelial function [60], significantly reduces the risk of intima-media complex thickening in the carotid [61] and the risk of CVD [62]. Table 1 shows specific food components and their effects on AS progression.

It is worth noting that AS development is affected by gut microflora, which is impacted not only by fermented milk products, containing live bifidus bacteria and lactic bacteria, but also by plant food (Table 2). When consumed with moderation, skimmed and full-fat dairy products do not contribute to the risk of AS, while regular consumption of yoghurt and small amounts of cheese has antiatherogenic effect [17]. This is associated with probiotics blocking development of abnormal gut microflora, which causes inflammation (contributing to AS plaques), with cholesterol and lipid fermentation by microbiota and formation of short-chain fatty acids, which are harmless for vascular walls [63]. Bacteria metabolites, such as trimethylamine N-oxide and bile acids, have protective effects against AS [64]. Antiatherogenic effects of naringenin in citrus fruit are due to induction of bile acid synthesis by gut microflora [37]. Quercetine in onions improves enzyme activity of intestine microbiota and normalises blood lipid profile [65]. The effects of berries and phytochemicals are a result of their ability to modulate gut microbiota, reducing *Firmicutes/Bacteroidetes* ratio and activating beneficial bacteria, such as *Akkermansia muciniphila* [66].

Atherogenic food products

Intake of cholesterols with food has an important role to play in the development of AS [67]. Therefore, animal-derived food rich in cholesterol (especially red and white meat) contributes to higher LDL cholesterol levels [68], higher risk of AS and AS mortality [16]. The development of AS is also facilitated by products, which are associated with inflammatory processes (rich in vitamin B12, cholesterol, fat and carbohydrates, high-calorie products, saturated and trans fats), which increase IL-1β, IL-4, IL-6, IL-10, TNFα, and C-reactive protein levels, involved in disease pathogenesis [69].

Since AS is associated with ageing and telomere shortening [70], irrespective of demographics, lifestyle and consumption of other products and drinks,

Table 1. *Specific food components with anti-atherosclerotic effect and their mechanisms of action*

Name of the substance	Impact effect	Food sources	Author
Lycopene	normalizes endothelial function and blood lipid composition	tomatoes	[28]
Naringenin and Hesperetin	inhibit cholesterol synthesis by inhibiting hydroxymethylglutaryl-CoA reductase	citrus	[37]
Curcumin	reduces levels of total cholesterol, triglycerides, LDL, TNF-α	turmeric	[39, 40]
Capsaicinoids	reduce cholesterol and LDL levels	hot peppers	[41]
Soy protein	reduce LDL level	Soybeans	[42]
Omega-6 fatty acids	reduce cholesterol level	vegetable oils (corn, sunflower, soybean)	[43]
Oleuropein	inhibits IL-8 production and collagen-induced platelet activation	olive oil	[44]
Hydroxytyrosol and Tyrosol	normalize blood lipid profile, inhibit MAPK phosphorylation and secretion of proinflammatory cytokines	olive oil	[44]
Omega-3 fatty acids	reduce triglyceride levels and increase HDL, suppress platelet aggregation, slow atherosclerosis progression	flaxseed oil, fish	[45-48]
Folic acid	restores impaired endothelial function	whole grain bread, legumes, citrus fruits	[51]
Beta-carotene (provitamin A)	suppresses inflammation of the vascular wall	carrots, pumpkin, mushrooms	[22]
Niacin (vitamin PP)	suppresses inflammation of the vascular wall, reduces LDL levels	beans, tomatoes, buckwheat, peanuts, carrots	[22, 52]
Vitamin D3	inhibits the formation of MPC, induces autophagy and lipid breakdown, reduces the synthesis of IL-6, TNF-α, IL-1β in macrophages, prevents the death of endothelial cells and improves their function	mushrooms, fish, yeast, cheese, butter, egg yolk	[53-55, 60]

Table 2. *Foods that inhibit the development of atherosclerosis by affecting the intestines*

Name of the substance	Impact effect	Food sources	Author
Water-soluble dietary fiber (beta-glucans, gums, pectin, mucilage, resistant starches and fructans)	adsorb and bind cholesterol, reducing its absorption and increasing its excretion with bile and fecal lipids	barley, oats, legumes, vegetables, fruits	[23]
Insoluble dietary fiber (hemicellulose, lignin, cellulose)	reduce the transit time of cholesterol through the intestines, thus reducing its absorption	nuts, bran, whole grains	[23]
Lactic acid bacteria (probiotics)	prevent the development of infection that causes inflammation, participate in cholesterol metabolism in the intestine	cheese, yogurt, kefir, cottage cheese	[63]
Naringenin	induces the synthesis of bile acids by intestinal microflora	citrus	[37]
Quercetin	improves the enzymatic activity of intestinal microbiota	onion	[65]
Phytochemicals	activate beneficial bacteria in the gut, promoting cholesterol metabolism	berries	[66]

Table 3. *Food products that promote the development of atherosclerosis*

Food products	Impact effect	Author
Meat, cheese, egg yolk, lard, offal	exogenous cholesterol sources increase LDL levels	[67, 68]
Processed meat	telomere shortening, vascular endothelial aging	[71]
Meat products with phosphates	cause the development of coronary atherosclerosis	[72, 73]
Iron-rich foods (heme meats)	contribute to the progression of atherosclerosis	[74]
Rich in vitamin B12, cholesterol, fats and carbohydrates, high-calorie foods	induce the synthesis of IL-1β, IL-4, IL-6, IL-10, tumor necrosis factor alpha and C-reactive protein, involved in the pathogenesis of AS, promoting inflammation of the vascular wall	[69]
Fried foods in vegetable oil	form high concentrations of genotoxic and cytotoxic lipid oxidation products (epoxy fatty acids and aldehydes)	[20]
Potato chips	increase concentrations of oxidized LDL, IL-6, C-reactive protein	[75]
Products stored in plastic packaging containing phthalates	disrupt lipid metabolism, accelerate the development of atherosclerosis, thicken the intima media	[77]
Products with calcium additives	increase the risk of coronary heart disease	[78]
Drinks with sucrose	increase the risk of coronary heart disease in men	[79]

consumption of processed meat causes telomeres to shorten and facilitates the development of AS [71]. High concentrations of serum phosphate increase the risk of asymptomatic coronary AS and CVD death by 44 % [72]. Since, during processing, phosphates are actively added to raw meat (to improve the water-retaining properties and protein solubility) and meat products, their intake contributes to the development of AS [73]. Experiments in ApoE-/- mice demonstrated the role of iron overload in AS aggravation, as well as better vessel condition with low intakes of iron and chelated iron therapy. Therefore, it is recommended to limit consumption of products rich in iron in patients with AS (red heme meat) [74].

Vegetable fats can also have atherogenic potential when used for frying. When used for frying, cooking oils, which are rich in polyunsaturated fatty acids, produce highly concentrated genotoxic and cytotoxic products of lipid oxidation (including epoxy fatty acids and aldehydes) as a result of oxygen recirculating peroxide outbursts. These toxins are absorbed by intestine and contribute to inflammatory processes in vascular walls, inducing AS [20]. Therefore, in order to prevent AS, vegetarians and vegans are recommended to limit frying using vegetable oils. Daily consumption of potato chips by healthy volunteers for 4 weeks showed significant increase in levels of oxidated LDL, IL-6, C-reactive protein, and blood acrilamide levels. These changes were risk factors for the development of AS [75]. At the same time, elderly individuals with asymptomatic IHD, who ate jacket potato and mashed potato, did not show any effects for the disease progression and coronary artery calcification [76].

Food packaging and plasticisers in PVC materials contain phthalates. As a result of daily use of plastics contacting with food, phthalates inter the body; they interfere with lipid metabolism and speed up AS development. There is positive correlation between the use of phthalates and thickened intima-media complex [77]. According to several meta-analyses, dietary calcium supplements are associated with a higher risk of IHD, whereas consumption of food rich in natural calcium does not contribute to the development of coronary artery AS. Natural means calcium in consumed food (living plant and animal organisms) in the form of various salts as part of cytoplasm and extracellular fluid. Thus, any calcium salts (mainly calcium carbonate and citrate) added artificially are unnatural and contribute to AS [78]. Prospective clinical trials show increased risk of IHD in males who drink drinks sweetened with sugar [79]. Table 3 shows food products and their effects, which should be avoided or limited in order to prevent AS.

Conclusion

The fundamental element of the Russian and foreign guidelines for the prevention and management of atherosclerosis is a balanced diet, which can help in achieving the target blood cholesterol and LDL levels. The literature analysis demonstrated that the optimal diets are

vegetarian, vegan and Mediterranean diets. The effect is a result of antiatherosclerotic action of dietary fibres in plant products, which speed up bowel movements, adsorb and bind cholesterol, thus improving its removal from the body. Also, omega-6 fatty acids, soya protein, curcumin, and capsaicinoids lower blood LDL levels. There are reports of anti-inflammatory effects of beta-carotene, vitamins PP and D3, probiotics in plant products. Licopene, folic acid, vitamin D3 normalise function of vascular endothelium. There are reports on various effects of polyphenols in plant products on pathogenesis of atherosclerosis, as well as the ability of hesperetin and naringenin in citrus fruit to inhibit cholesterol synthesis in the liver due to inhibition of hydroxy-methyl-glutaryl-CoA-reductase. An important method of atherosclerosis prevention and management is avoidance of atherogenic food, frying with vegetable oils (contain aldehydes and epoxy fatty acids), products rich in cholesterol (meat, cheese, egg yolk, by-products), phosphates, iron, saturated and trans fats, with calcium supplements, which are stored in phthalate-containing packaging, sweet drinks. It is also essential to reduce consumption of high-calorie food, limit intake of carbohydrates with a high glycaemic index, crisps, and deep-fried products.

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