

THE ROLE OF DIETARY NUTRITION IN ATHEROSCLEROSIS

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Abstract

The significance of dietary nutrition in the prevention of cardiovascular diseases (CVD) has been well established [3,4]. Proper nutritional regimen and healthy lifestyle habits are among the key strategies for preventing complications associated with these conditions. Educating the population about principles of healthy living—including rational nutrition, physical activity, and smoking cessation—is crucial.

Introduction

Therapeutic diets serve as an adjunct to pharmacological treatment, exerting beneficial effects comparable to medications, and can mitigate some adverse drug reactions. In patients with cardiovascular diseases, prescribing disease-specific diets and engaging in physical exercise can help regulate blood lipid levels and reduce atherosclerotic stenosis of coronary arteries (based on coronary angiography results) (Basukova N.L., Zambrjiskiy O.N., Fomina A.I., Doroshevich V.I.). Dietary therapy is an integral component of comprehensive treatment. As a fundamental principle of dietetics, M.I. Pevzner emphasized that nutritional management is a core aspect of therapy and should be implemented alongside other therapeutic interventions. Without a therapeutic diet, effective treatment is unlikely.

A healthy diet plays a vital role in restoring health and preventing disease complications in patients with cardiovascular conditions. Therefore, a healthy diet for patients with cardiovascular diseases should be viewed not as a temporary measure, but as a lifelong, mandatory lifestyle modification. Rational nutrition helps regulate blood pressure and body weight, owing to its effects on lipid profiles and blood glucose levels, thereby significantly reducing the risk of thrombosis. Excessive salt intake, limited consumption of fruits and vegetables, and unregulated intake of animal fats contribute to overnutrition and weight gain. Moreover, ensuring that the diet is rich in essential nutrients and diverse in food types is equally important.

The impact of dietary factors on atherogenesis—either directly or through known mechanisms such as dyslipidemia, hypertension, or hyperglycemia—is well substantiated.

A diet rich in monounsaturated fats markedly enhances insulin sensitivity compared to diets high in saturated fats [7]. This process is especially evident during postprandial periods, characterized by a reduction in triglyceride levels. Furthermore, the intake of high doses of n-3 polyunsaturated fatty acids (PUFAs) results in decreased triglyceride levels. However, it is often impossible to reduce triglycerides solely through natural food sources. In such cases, pharmacological supplements or artificially enriched foods with n-3 PUFAs are prescribed [6,7].



In patients with fasting hypertriglyceridemia, dietary fat intake should be sharply reduced—by approximately 10% of the total daily caloric intake—to mitigate triglyceride levels. Fructose consumption, when maintained at habitual levels, can increase plasma triglycerides by 15–20%. The primary dietary source of fructose is sucrose (disaccharide), which contains glucose and fructose [1].

Weight loss enhances insulin sensitivity and leads to a decrease in triglyceride levels. Some studies report reductions of 20–30% in triglycerides following weight reduction.

Moderate alcohol consumption (10–30 grams of ethanol daily) has been shown in some studies to negatively affect triglyceride levels, leading to their increase [9]. While small amounts of alcohol can raise triglycerides in certain populations, overall, evidence suggests that alcohol may have adverse effects on lipid metabolism when consumed excessively.

High levels of saturated fatty acids in the diet contribute to an increase in low-density lipoprotein (LDL) cholesterol-rich lipoproteins and a rise in lipoproteins with lower density. Conversely, trans fats tend to decrease high-density lipoprotein (HDL) levels and increase low-density lipoproteins (LDL), thereby exacerbating atherogenic profiles. Replacing saturated fats with monounsaturated fatty acids has minimal or no significant impact on high-density lipoprotein levels, while omega-3 polyunsaturated fatty acids slightly decrease the levels of dense lipoproteins—yet they are known to increase HDL cholesterol by approximately 0.08–0.15 mmol/L (3.1–6 mg/dL) [5]. Cessation of smoking also results in an increase in HDL cholesterol levels [2,8].

In recent years, European cardiology and atherosclerosis societies have developed dietary guidelines aimed at reducing serum cholesterol levels and slowing atherosclerosis progression. These recommendations advocate for limiting all types of fats, especially during cooking—prohibiting the consumption of fats produced during frying. Poultry is recommended over red meat, and dairy products should be low-fat (e.g., skim kefir and low-fat cheese). Fish such as cod, hake, mackerel, sardines, and tunny are encouraged. A variety of vegetables, nuts, beans, and olives are recommended, along with whole grains, cornmeal bread, products made from pearl barley, and rice-based dishes. Additionally, baked goods from pearl barley and sugar-free fruit drinks are included in the diet. Fats like heavy cream, mayonnaise, and fatty oils are restricted.

Adherence to a cholesterol-lowering diet should be lifelong. Properly balanced daily nutrition not only improves mood but also serves as an effective measure to prevent additional health complications.

References

1. Bantle JP., Raatz SK., Thomas W., Georgopoulos A. Effects of dietary fructose on plasma lipids in healthy subjects. *Am J Clin Nutr.* 2000.- P.1128–1134.
2. D'Agostino RB Sr., Vasan RS., Pencina MJ., Wolf PA., Cobain M., Massaro JM., Kannel WB. General cardiovascular risk profile for use in primary care: the Framingham Heart Study. . *Circulation.* 2008. -P.743–753.
3. Grundy SM. Lipids, nutrition and coronary heart disease. *Atherosclerosis and coronary artery disease.* -1996.- Vol. 120.-P.132-134.
4. Hu F, Willett WC. Optimal diets for prevention of coronary heart disease. *JAMA.* -2002.-№. 288(2): -P.2569–2578.



5. Kraus WE., Houmard JA., Duscha BD., Knetzger KJ., Wharton MB., McCartney JS., Bales CW., Henes S., Samsa GP., Otvos JD., Kulkarni KR., Slentz CA. Effects of the amount and intensity of exercise on plasma lipoproteins. *N Engl J Med* . -2002. -P.1483–1492.
6. Liu S., Manson JE., Stampfer MJ., Holmes MD., Hu FB., Hankinson SE., Willett WC. Dietary glycemic load assessed by food-frequency questionnaire in relation to plasma high density-lipoprotein cholesterol and fasting plasma triacylglycerols in postmenopausal women. *Am J Clin Nutr*. -2001.-P. 560–566.
7. NCEP Expert Panel on detection, evaluation and treatment of high blood cholesterol in adults. Executive summary of the third report of the National Cholesterol Education Program (NCEP) Expert Panel on detection, evaluation and treatment of high blood cholesterol in adults (Adult Treatment Panel III). *JAMA*.- 2001.-№.285.-P.2486–2497.
8. Rabkin SW. Effect of cigarette smoking cessation on risk factors for coronary atherosclerosis. A control clinical trial. *Atherosclerosis* . 1984.-№. 53.-P. 173–184.
9. Rimm EB., Williams P., Fosher K., Criqui M., Stampfer MJ. Moderate alcohol intake and lower risk of coronary heart disease: meta-analysis of effects on lipids and haemostatic factors. *BMJ*.1999. -P.1523–1528.
10. Shaw K., Gennat H., O'Rourke P., Del Mar C. Exercise for overweight or obesity. *Cochrane Database Syst Rev*. -2006.-№.4 -P.3817.

