

HOW TO PREVENT CORONARY HEART DISEASE WITH DIETARY**Sharapova Nozima Erkinjonovna**

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Abstract. *Coronary heart disease (CHD) remains the leading cause of mortality in industrialized countries and is rapidly becoming a primary cause of death worldwide. Thus, identification of the dietary changes that most effectively prevent CHD is critical.*

Key words: *unsaturated, saturated, trans-fats, lipoprotein, low-density lipoprotein, carbohydrates, fatty acids.*

**КАК ПРЕДОТВРАТИТЬ ИШЕМИЧЕСКУЮ БОЛЕЗНЬ СЕРДЦА С ПОМОЩЬЮ
ДИЕТЫ**

Аннотация. *Ишемическая болезнь сердца (ИБС) остается основной причиной смертности в промышленно развитых странах и быстро становится основной причиной смерти во всем мире. Таким образом, определение изменений в питании, которые наиболее эффективно предотвращают ИБС, имеет решающее значение.*

Ключевые слова: *ненасыщенные, насыщенные, трансжиры, липопротеины, липопротеины низкой плотности, углеводы, жирные кислоты.*

Compelling evidence from metabolic studies, prospective cohort studies, and clinical trials in the past several decades indicates that at least 3 dietary strategies are effective in preventing CHD: substitute non hydrogenated unsaturated fats for saturated and *trans*-fats; increase consumption of omega-3 fatty acids from fish, fish oil supplements, or plant sources; and consume a diet high in fruits, vegetables, nuts, and whole grains and low in refined grain products.

However, simply lowering the percentage of energy from total fat in the diet is unlikely to improve lipid profile or reduce CHD incidence. Substantial evidence indicates that diets using non hydrogenated unsaturated fats as the predominant form of dietary fat, whole grains as the main form of carbohydrates, an abundance of fruits and vegetables, and adequate omega-3 fatty acids can offer significant protection against CHD. Such diets, together with regular physical activity, avoidance of smoking, and maintenance of a healthy body weight, may prevent the majority of cardiovascular disease in Western populations. Until recently, most epidemiologic and clinical investigations of diet and CHD have been dominated by the diet-heart hypothesis.

However, the original hypothesis was overly simplistic because the effects of diet on CHD can be mediated through multiple biological pathways other than serum total cholesterol or low-density lipoprotein cholesterol.

Numerous controlled feeding studies of the effects of different dietary fatty acids on serum cholesterol levels have been summarized in several meta-analyses from which predictive equations have been developed. All such analyses confirm early reports by Keys and Hegsted that saturated fatty acids increase and polyunsaturated fatty acids decrease total and LDL cholesterol. All 3 classes of fatty acids (saturated, monounsaturated, and polyunsaturated) elevate high-density lipoprotein cholesterol (HDL-C) when they replace carbohydrates in the diet, and this effect is slightly greater with saturated fatty acids. Also, triglyceride levels increase when dietary fatty acids are replaced by carbohydrates. Because replacement of saturated fat with carbohydrates proportionally reduces both LDL-C and HDL-C, and, thus, has little effect on the LDL-HDL ratio and increases triglycerides, this change in diet would be expected to have minimal benefit on CHD risk.

However, when monounsaturated or polyunsaturated fats replace saturated fat, LDL-C decreases and HDL-C changes only slightly. Moreover, substituting polyunsaturated fat for saturated fat may have beneficial effects on insulin sensitivity and type 2 diabetes. In numerous controlled metabolic studies, *trans*-fatty acids (found in stick margarine, vegetable shortenings, and commercial bakery and deep-fried foods) have been shown to raise LDL-C levels and lower HDL-C relative to *cis*-unsaturated fatty acids, and the increase in the ratio of total to HDL cholesterol for *trans*-fat is approximately twice that for saturated fat. *Trans*-fat increases plasma levels of lipoprotein and triglycerides and may reduce endothelial function by impairing flow-mediated dilation. In addition, *trans*-fatty acids adversely affect essential fatty acid metabolism and prostaglandin balance by inhibiting the enzyme delta-6 desaturase. Finally, high intake of *trans*-fat may promote insulin resistance and increase risk of type 2 diabetes. Only a handful of dietary trials with CHD end points have been conducted and most were among patients with CHD.

Two dietary approaches were tested in earlier trials; one replaced saturated fat with polyunsaturated fat, leaving total fat unchanged; the other lowered total fat. In all the high-polyunsaturated-fat trials, serum cholesterol was significantly reduced. In the Finnish Mental

Hospital Study, soft margarine replaced stick margarine, so the reduction in CHD was probably in part due to reduction in *trans*-fat intake. In the Minnesota Coronary Survey, cardiovascular events were not significantly reduced by a high-polyunsaturated-fat diet despite a decrease in serum cholesterol, but the mean duration of dietary intervention was only about 1 year.

Two secondary prevention trials testing the approach of total fat reduction did not find a significant reduction in serum cholesterol or CHD events. Omega-3 fatty acids may reduce risk of CHD by preventing cardiac arrhythmia, lowering serum triglyceride levels, decreasing thrombotic tendency, and improving endothelial dysfunction. An inverse association between fish intake and coronary mortality was first reported in a Dutch population, and more than 15 prospective studies have followed. A systematic review of the 11 studies published before 2000 concluded that the inverse association was stronger for fatal CHD than for nonfatal myocardial infarction (MI), and the benefit was most evident in populations with higher-than-average risk of CHD. Since that review, 4 additional prospective cohort studies and 1 case-control study have provided further support for the protective effects of marine omega-3 fatty acids against CHD in diverse populations. Notably, 2 recent studies have shown that consuming 2 or more servings of fish per week was associated with 30% lower risk of CHD in women and that blood levels of eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) were strongly associated with decreased risk of sudden cardiac death in men. Revising dietary recommendations have emphasized high intake of complex carbohydrates, mainly starch, and avoidance of simple sugars.

However, many starchy foods, such as baked potatoes and white bread, are rapidly digested to glucose and produce even higher glycemic and insulinemic responses than sucrose (half glucose and half fructose). The glycemic index (GI) ranks foods based on rise in blood glucose (the incremental area under the curve for blood glucose levels) after ingestion compared with glucose or white bread, standardizing the carbohydrate content to 50 g. Foods with a low degree of starch gelatinization (more compact granules), such as spaghetti and oatmeal, and a high level of viscous soluble fiber, such as barley, oats, and rye, tend to have a slower rate of digestion and, thus, lower GI values. In several controlled clinical studies, feeding low-GI meals to diabetic patients led to significant improvement in glycemic control and lipid profile, but larger studies are needed. Much evidence suggests that adequate folate consumption is important for the prevention of CHD. Epidemiologic studies have found an inverse association between folate intake measured by dietary questionnaire or serum folate level and risk of CHD, which is likely to be mediated through homocysteine-lowering effects of folic acid.

Two randomized placebo-controlled trials evaluated effects of folic acid supplementation on the development and progression of atherosclerosis. Vermeulen et al found that supplementation with folic acid and vitamin B₆ for 2 years significantly decreased subclinical atherosclerosis indicated by abnormal exercise electrocardiography tests among siblings of patients with existing cardiovascular disease.

In the Swiss Heart Study, treatment with a combination of folic acid and vitamins B₆ and B₁₂ significantly decreased restenosis and revascularization after coronary angioplasty at 6 months and a combined cardiovascular end point at 11 months. Ongoing clinical trials should provide more definitive data on the role of folic acid supplementation in CHD prevention, but the interpretation of the findings from trials conducted in the United States could be complicated by the fortification of flour with folic acid. The relationship between consumption of specific foods or overall dietary patterns and risk of CHD has been examined in recent studies.

Such analyses are valuable in evaluating additional diet-heart hypotheses and in making practical dietary recommendations. For example, replacement of red meat with chicken and fish has been associated with reduced risk of CHD. An inverse association between nut consumption and risk of CHD has been seen consistently in prospective studies. Which further underscores the importance of distinguishing different types of fat. Although nuts are high in fat and, thus, routinely proscribed in dietary recommendations, the predominant types of fat in nuts are monounsaturated and polyunsaturated, which lower LDL-C level. Recently, several studies have reported the role of overall dietary patterns in predicting long-term risk of CHD. In these analyses, a "prudent" pattern characterized by higher intakes of fruits, vegetables, legumes, whole grains, poultry, and fish was associated with lower risk of CHD, whereas a "Western" pattern characterized by higher intakes of red and processed meats, sweets and desserts, potatoes, french fries, and refined grains was associated with a higher risk, independent of lifestyle factors.

Compelling evidence from metabolic studies, epidemiologic investigations, and clinical trials in the past several decades converges to indicate that at least 3 dietary strategies are effective in preventing CHD: substitute unsaturated fats (especially polyunsaturated fat) for saturated and *trans*-fats; increase consumption of omega-3 fatty acids from fish oil or plant sources; and consume a diet high in fruits, vegetables, nuts, and whole grains and low in refined grains. A combination of these approaches can confer greater benefits than a single approach.

However, simply lowering the percentage of energy from total fat in the diet is unlikely to improve lipid profile or reduce CHD incidence. Obesity is an important avenue by which diet can influence risk of CHD. However, the relationship between diet, especially dietary fat, and obesity remains controversial. Although reduction in percentage of calories from dietary fat intake is commonly recommended for weight loss, long-term clinical trials have provided no good evidence that reducing dietary fat per se can lead to weight loss. There is a growing consensus that excess calories, whether from carbohydrates or fat, will induce weight gain. A mildly hypocaloric moderate-fat diet, which allows for a great variety in choosing foods, can have better long-term compliance than a typical low-fat diet.

Small short-term studies have suggested roles of several diets in weight control, including a low-GI diet, a high-protein diet, and a diet high in dairy products, but larger and long-term studies are needed.

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