Editorial

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Assorted monounsaturated fatty acids promote healthy hearts[1,2]

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In the last half of this century, a variety of recommendations have been made concerning the optimal diet to lower cardiovascular disease (CVD) risk. The primary focus in the early years was on decreasing the intake of total fat, especially of saturated fat, in the diet. Hypotheses evolved from the epidemiologic studies of Keys (1), which correlated these fats with the risk of mortality from coronary heart disease (CHD). Equations were proposed that weighted saturated (SFA) and polyunsaturated (PUFA) fatty acid and cholesterol intakes to predict effects on circulating lipids and lipoproteins, and thereby on risk of CVD or CHD (2). Monounsaturated fatty acids (MUFAs) and stearate were considered neutral—without effect on serum lipid measures.

Effects of the concentration or composition of dietary fats and the intake of cholesterol on CHD development are the basis of the lipid hypothesis of atherosclerosis. Elevated blood concentrations of total cholesterol or LDL cholesterol increase the risk of CVD or CHD, whereas higher concentrations of HDL cholesterol decrease risk. Investigators proposed using the ratio of total to HDL cholesterol or of LDL to HDL cholesterol, to assess risk. Elevated concentrations of circulating triacylglycerols have also been considered independent risk factors for CVD (3). Thus, the composition of the diet relates not only to the concentrations of circulating lipids and lipoproteins but also to CVD incidence or prevalence in the population or in individuals. The size and density of LDL, influenced by genetics and diet, may play a role in an individual's risk of CHD (4). An inherited variant of LDL, lipoprotein(a), markedly enhances CVD risk, although we have little knowledge about the effect of diet (5).

The National Cholesterol Education Program (6) delineated the desirable concentrations of lipids and lipoproteins to minimize CVD, set guidelines for dietary and drug intervention, and recommended dietary interventions at 2 levels—the familiar Step I and Step II diets. The diets reduce total fat to $<30\%$ of energy, SFAs to $<10\%$ of energy, and cholesterol to <300 mg/d (Step I), or SFAs to $<7\%$ of energy and cholesterol to <200 mg/d (Step II). A lowering of LDL cholesterol by 1% reduces CVD risk by 1.5%. A 2.5% increase in risk occurs with each 1-mg decrease in HDL cholesterol, and risk increases by 25% for each 89-mg (1 mmol) increase in triacylglycerol concentration.

Newer proposals about dietary fats include the concept that MUFAs are not neutral but lower total and LDL cholesterol, similar to PUFAs, with the added benefit that replacing SFAs with MUFAs will not decrease HDL cholesterol. In this issue of the Journal, Kris-Etherton et al (7) propose that diets high in MUFAs from olive oil, peanut oil, or peanuts and peanut butter also lower concentrations of circulating triacylglycerols. This may enhance their salutary effect on CVD risk in contrast with either the Step II diet or the average American diet (AAD), which is high in SFAs (butter in this study). MUFAs may also reduce CVD risk with their antioxidant, antithrombotic, and antihypertensive properties. Measures of vascular and coagulation activity extend the original lipid hypothesis and are a current focus of atherogenesis research. Growth factors, cytokines and other inflammatory components, and genetic abnormalities that predispose individuals to CHD are new areas for examining potential effects of diet on biomarkers of CVD.

Are all MUFAs alike in their effects on circulating lipids and lipoproteins or on CVD risk? When the data in this study are used to calculate the effect of MUFAs on risk of CVD in comparison with the AAD (rather than comparing MUFAs with the Step II diet as Kris-Etherton et al did), olive oil had the greatest benefit, lowering CVD risk by 18%. Peanut oil and peanut products lowered CVD risk by 15%, and the Step II diet lowered the risk by 12%. Adverse effects of peanut oil on atherogenesis were reported in animal studies (8), with amelioration if the peanut oil fatty acids were randomly distributed on the triacylglycerol molecule. The position of the MUFA in the triacylglycerol may influence its atherogenic effects. The oils in the present study, olive and peanut, are similar in their fatty acid composition ($\approx$77% oleic acid), but differ in other constituents (antioxidants, plant sterols, tocopherols, tocotrienols, and other phytochemicals).

Oils with MUFA contents similar to those of peanut oil and olive oil include soybean oil and cottonseed oil. In a recent study that focused on *trans* unsaturated fatty acids, which have adverse effects at high intakes, the investigators showed a 12% lowering of CVD risk in hyperlipidemic subjects with a MUFA-modified Step II diet (9). Refined rice bran oil, with a MUFA content similar to those of peanut and olive oil, has potent lipid-lowering and antiatherogenic effects, in part attributed to its unique plant sterol ferulate complex, oryzanol (10). Walnuts and walnut oil are lipid lowering. Macadamia nuts, uniquely rich in palmitoleic acid, have not been studied as yet. Peanuts are not true nuts, but are legumes; Kris-Etherton et al discuss the fact that the high protein content of peanuts may influence circulating lipids. Peanuts also uniquely contain 7% very-long-chain saturated fatty acids.

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Food companies and nutrition scientists have created foods containing phytochemicals that may lessen the risk of CVD: eg, grain products enriched with psyllium or other fibers and soft margarines enriched with the plant sterols sitosterol or sitostanol. The National Cholesterol Education Program diet of the next millennium will be more varied and permissive than earlier diets that limited fat intake. All fats, however (other than synthetic fats such as sucrose polyesters), are higher in energy than are proteins or carbohydrates. Relatively free use of MUFAs in a high-fat diet (≥35% of energy) may be optimal for people near their healthy body weight or body mass index in association with appropriate levels of physical activity.

The present study was done in a healthy population with normal circulating lipid concentrations, who had ingested the experimental diets for 24 d. Such a brief and strictly controlled study (all foods were provided and dietary compliance was closely monitored) does not extrapolate to hyperlipidemic subjects trying to deal with the lure of so-called healthy foods high in energy and sugars that may adversely affect metabolism and atherogenesis.

Finally, in trying to differentiate so-called good and bad fats, it is puzzling that SFAs, which are the hallmark of an atherogenic diet (especially butterfat or dairy products), have the greatest HDL-increasing and lipoprotein(a)-lowering effects, and thus might be considered good. This discussion omitted the dietary effects on apolipoprotein concentrations, which are perhaps more predictive of CVD risk than are lipoprotein lipid concentrations, and on the various heritable enzymes and transfer factors that may be modified by the diet. I look forward to the publication of studies that will address these issues and allow us multiple choices, tailored to our genes, that provide a palatable and varied heart-healthy diet.

REFERENCES

1. Keys A. Seven countries: a multivariate analysis of death and coronary heart disease. Cambridge, MA: Harvard University Press, 1980.
2. Hegsted DM, Ausman LM, Johnson JA, Dallal GE. Dietary fat and serum lipids: an evaluation of the experimental data. Am J Clin Nutr 1993;57:875–83.
3. Criqui MH. Triglycerides and coronary heart disease. In: Gotto AM Jr, Paoletti R, eds. Atheroscler Rev 1991;22:75–9.
4. Austin MA, Breslow JL, Hennekens CH, Buring JE, Willett WC, Krauss RM. Low-density lipoprotein subclass patterns and risk of myocardial infarction. JAMA 1988;260:917–21.
5. Dahlen GH, Guyton JR, Attar M, Farmer JA, Kautz, JA, Gotto AM Jr. Association of levels of lipoprotein Lp(a), plasma lipids, and other lipoproteins with coronary artery disease documented by angiography. Circulation 1986;74:758–65.
6. 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). Bethesda, MD: National Cholesterol Education Program, National Institutes of Health, National Heart, Lung and Blood Institute, 1993. (NIH publication no. 93–3095.)
7. Kris-Etherton PM, Pearson TA, Wan Y, et al. High–monounsaturated fatty acid diets lower both plasma cholesterol and triacylglycerol concentrations. Am J Clin Nutr 1999;70:1009–15.
8. Kritchevsky D, Tepper SA, Vesselinovitch D, Wissler RW. Cholesterol vehicle in experimental atherosclerosis. 13. Randomized peanut oil. Atherosclerosis 1973;17:225–33.
9. Lichtenstein AH, Ausman LM, Jalbert SM, Schaefer EJ. N Engl J Med 1999:340:1933–40.
10. Seetharamaiah GS, Chandrasekhra N. Studies on hypocholesterolemic activity of rice bran oil. Atherosclerosis 1989;78:218–23.