*See corresponding article on page 1001.*

# Harbingers of coronary heart disease: dietary saturated fatty acids and cholesterol. Is chocolate benign because of its stearic acid content?[1,2]

*William E Connor*

Consumption of foods rich in saturated fatty acids and cholesterol has long been recognized as an important precursor for the development of coronary heart disease; saturated fatty acids and cholesterol each independently elevate cholesterol and LDL concentrations in the blood (1). However, it is now thought that some saturated fatty acids may not deserve this reputation. Likewise, dietary cholesterol has been held in less opprobrium in recent years. What are the facts? The saturated fatty acids lauric (12:0), myristic (14:0), and palmitic (16:0) acids definitely raise plasma cholesterol concentrations. The medium- and short-chain fatty acids with ≤10 carbons are handled by the body more like carbohydrates than fats and have no effects on plasma cholesterol concentrations. On the other hand, stearic acid—which has 18 carbons—has been considered benign or neutral in this regard, which has led the food industry to consider producing fats rich in stearic acid to provide the taste and flavor of fat in foods without the usual disadvantage of saturated fat. For chocolate lovers, considerable euphoria has resulted because the fat in chocolate is especially high in stearic acid (≈30% of fatty acids).

The reason dietary stearic acid is considered benign is based on its failure to elevate plasma cholesterol concentrations (2, 3). Stearic acid is well absorbed by the gut and is transported in chylomicrons and remnant particles before being picked up by the liver. Once there, an interesting paradox occurs in that excess stearic acid is simply converted to the 18-carbon monounsaturated oleic acid via a desaturase enzyme in the liver (4) and then recirculates in lipoprotein complexes as oleic acid, which is not hypercholesterolemic. Thus, conversion to oleic acid may explain why stearic acid does not elevate plasma cholesterol concentrations. In contrast, when palmitic acid reaches the liver after absorption, it simply recirculates as palmitic acid in lipoproteins. Thus, dietary palmitic acid, along with lauric and myristic acids, elevates plasma cholesterol and LDL concentrations by down-regulating the hepatic receptor for LDL (5). Dietary cholesterol raises plasma LDL concentrations by exactly the same mechanism.

Whether dietary stearic acid is benign or pathogenic for coronary heart disease has been greatly clarified by Hu et al (6) in their Nurses' Health Study of >80 000 women. On the basis of 14 y of follow-up data and analyses of dietary intakes, dietary stearic acid was shown to increase the risk of coronary heart disease more so than did palmitic, myristic, and lauric acids. As expected, medium- and short-chain fatty acids were not associated with a greater risk.

Proposed mechanisms for this positive relation between dietary stearic acid intake and risk of coronary heart disease must relate to factors other than plasma cholesterol and LDL concentrations. In other words, stearic acid must be pathogenic in its prehepatic phase, not after its conversion to oleic acid. One such mechanism is stearic acid's known depression of the protective lipoprotein, HDL (3). Other mechanisms include the activation of factor VII, increased lipoprotein(a) concentrations, and impairment of fibrinolysis (3, 7). Furthermore, the consumption of foods rich in stearic acid and other saturated fats result in postprandial lipemia (chylomicrons). Such lipemia may be associated with a tendency toward thrombosis. In addition, dietary stearic acid has been associated with the progression of coronary lesions (8). In that study, Watts et al suggested that stearic acid is thrombogenic via platelet activity and the activation of coagulation (8). This suggestion is supported by evidence that stearic acid causes thrombosis in experimental animals (9).

Foods high in saturated fat (and stearic acid), which may be associated with coronary disease in the Nurses' Health Study or in other studies, are listed in **Table 1**. Of interest is the relation between whole-milk consumption and deaths from coronary heart disease. Such deaths were 5 times more frequent in Finland, where whole-milk consumption was very high compared with that in France, where milk was used in small quantities (10).

Not listed among the pathogenic foods in the table are several foods with a very high cholesterol content, such as egg yolk and organ meats (including brain). As Hu et al (6) indicated, dietary cholesterol is likewise directly correlated with death from coronary heart disease. On the other hand, fish and chicken, in contrast with red meat and dairy products, were protective against coronary heart disease, as were polyunsaturated fats, monounsaturated fats, and fiber. Although the polyunsaturated fats were not broken down specifically into n−6 and n−3 fatty acids, n−3 polyunsaturated fatty acids from

[1] From the Oregon Health Sciences University, Division of Endocrinology, Diabetes and Clinical Nutrition L-465, Department of Medicine, Portland.

[2] Address reprint requests to WE Connor, Oregon Health Sciences University, Division of Endocrinology, Diabetes and Clinical Nutrition L-465, Department of Medicine, 3181 SW Sam Jackson Park Road, Portland, OR 97213. E-mail: connorw@ohsu.edu.

*Am J Clin Nutr* 1999;70:951–2. Printed in USA. 

**TABLE 1**
Foods rich in stearic acid and other saturated fats

| |
|---|
| Red meat (beef, pork, or lamb) |
| High-fat dairy products (whole milk, cheese, butter, and ice cream) |
| Chocolate |
| Lard |
| Coconut oil |
| High-fat "fast foods" |
| Highly hydrogenated margarines and shortenings, which also contain *trans* fatty acids |

fish and fish oil are especially protective against coronary heart disease by a variety of mechanisms (11). These mechanisms include a reduction in plasma triacylglycerol concentrations, an inhibition of the growth of atherosclerotic plaques, and prevention of thrombosis.

Finally, there is documentation by the Harvard study that Americans are reducing their intakes of all saturated fats, including stearic acid; intakes decreased from 15.58% of total energy in 1980 to 10.63% in 1990 (6). These data fit nicely with other trends: less dietary cholesterol and saturated fat intakes, lower serum cholesterol concentrations, and fewer deaths from coronary heart disease. Altogether, these data indicate that favorable lifestyle changes will prevent coronary heart disease. Unfortunately for chocolate lovers, including myself, chocolate's high content of stearic acid puts it in the same category of risk of coronary disease as meat and butter, ie, pathogenic! Chocolate should be savored on special occasions only.

## REFERENCES


1. Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults. Summary of the second report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel II). JAMA 1993;269:3015–23.
2. Yu S, Derr J, Etherton TD, Kris-Etherton PM. Plasma cholesterol-predictive equations demonstrate that stearic acid is neutral and monounsaturated fatty acids are hypocholesterolemic. Am J Clin Nutr 1995;61:1129–39.
3. Aro A, Jauhiainen M, Partanen R, Salminen I, Mutanen M. Stearic acid, *trans* fatty acids, and dairy fat: effects on serum and lipoprotein lipids, apolipoproteins, lipoprotein(a), and lipid transfer proteins in healthy subjects. Am J Clin Nutr 1997;65:1419–26.
4. Lin DS, Connor WE, Spenler CW. Are dietary saturated, monounsaturated, and polyunsaturated fatty acids deposited to the same extent in adipose tissue of rabbits? Am J Clin Nutr 1993;58:174–9.
5. Spady DK, Dietschy JM. Interaction of dietary cholesterol and triglycerides in the regulation of hepatic low density lipoprotein transport in the hamster. J Clin Invest 1988;81:302–9.
6. Hu FB, Stampfer MJ, Manson JE, et al. Dietary saturated fats and their food sources in relation to the risk of coronary heart disease in women. Am J Clin Nutr 1999;70:1001–8.
7. Mitropoulos KA, Miller GJ, Martin JC, Reeves BEA, Cooper J. Dietary fat induces changes in factor VII coagulant activity through effects on plasma free stearic acid concentration. Arterioscler Thromb 1994;14:214–22.
8. Watts GF, Jackson P, Burke V, Lewis B. Dietary fatty acids and progression of coronary artery disease in men. Am J Clin Nutr 1996;64:202–9.
9. Connor WE, Hoak JC, Warner ED. Massive thrombosis produced by fatty acid infusion. J Clin Invest 1963;42:860–6.
10. Artaud-Wild SM, Connor SL, Sexton G, Connor WE. Differences in coronary mortality can be explained by differences in cholesterol and saturated fat intakes in forty countries but not in France and Finland. Circulation 1993;88:2771–9.
11. Connor SL, Connor WE. Are fish oils beneficial in the prevention and treatment of coronary artery disease? Am J Clin Nutr 1997;66(suppl):1020S–31S.