Cholesterol and CHD

To the Editor:

I read with interest the International Lecture, “Effect of Cholesterol-Lowering Diet on Mortality from Coronary Heart Disease and Other Causes.” by Turpeinen (Circulation 59: 1, 1979). In his demonstration of international statistics, the author attempts to correlate consumption of saturated fats and mortality from coronary heart disease (CHD). His recommended changes in the quantity and quality of dietary fats are based on a mental hospital study in Finland, where total replacement of dairy fats by vegetable oils in the diet was followed by a substantial reduction in CHD mortality of men.

The validity and significance of this study have been doubted.1 Scrutiny of the figures in this article does not bear out the author’s contention that fats derived from dairy products are causally associated with CHD mortality. He has not explained why the 25-g daily fat consumption of United States men, aged 35 to 64 years, is associated with CHD mortality of approximately 400 per 100,000 whereas a similar population in France and Switzerland consumes close to 40 g of dairy fat, with a subsequent CHD mortality experience of less than 150 per 100,000, his regression line notwithstanding.

Following the jujitsu principle of using an opponent’s strength to prove your own point of view, I submit a different analysis of the data presented by Turpeinen. In 1971, I published a correlation of CHD mortalities based on the presence of biologically available xanthine oxidase in homogenized, pasteurized cow’s milk.2 The biological availability of xanthine oxidase is artificially created by the homogenization process, causing the enzyme to be entrapped in liposomes. These liposomes can be persorbed and transported via the white blood cells in humans. We have demonstrated that xanthine oxidase is targeted into the vessel wall and the myocardium, constituting an initiation of the atherosclerotic process. These observations are based not on epidemiological studies, but on reproducible experimental findings.3,4 Wherever milk is ultra-pasteurized, as in Switzerland, France and Japan, or customarily boiled before consumption, as in most Mediterranean countries, the xanthine oxidase is inactivated and the CHD mortality is low. Wherever milk is homogenized and, particularly, when consumed in large amounts in youth — Finland, United States, New Zealand, Australia, United Kingdom, and Canada — the CHD mortality rate is high.

Instead of changing the quantity and quality of dietary fats, we recommend only the removal of biologically available xanthine oxidase from milk. Homogenized cow’s milk is the only commonly consumed food which contains an enzyme entrapped in liposomes. A simple and easily performed technology should reduce the high CHD mortality of the United States and Finland to a rate closer to that of France, Switzerland and Japan. A routine test for antibodies against bovine milk xanthine oxidase in human blood serum could screen the population which has been exposed to this enzyme.

A treatment for the atherosclerotic lesion initiated by ectopically deposited xanthine oxidase is provided by pharmacological doses of folic acid (80 mg/day). This well-known xanthine oxidase inhibitor has been used successfully for the treatment of atherosclerotic manifestations in humans for the last 8½ years.

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References


The author replies:

To the Editor:

Dr. Oster points to certain inconsistencies in my diagram (fig. 4) showing the correlation between the consumption of dairy fats and mortality from coronary heart disease (CHD). It is true that France
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