Editorial

The Reluctance of Physicians to Admit That Chronic Disease May Be Due to Faulty Diet

A challenging problem for the nutritionist is the fact that physicians, who so often ascribe acute illness to a recent dietary mishap, have always hated to accept any theory ascribing chronic disease to bad food. Thus, the causation of scurvy was clearly and convincingly described by Bachstrom in 1743; lemon juice as a preventive and cure, with excellent resistance to storage, was recommended in Lind's treatise in 1754. A critical test was given by the British navy in 1795, the year of Lind's death, and the brilliant success led to the orders which abolished scurvy forever from British ships. Thanks to Lind's work, Nelson's sailors enjoyed unusual health during the decades when his "distant storm-tossed ships stood between Napoleon and the conquest of the world."

In 1908 Holst and Fröhlich found that the guinea pig, alone among domestic or laboratory animals, developed scurvy on diets like those of the sailors before 1800, and could be cured by lemon juice. This test animal later served to control the isolation and synthesis of ascorbic acid (vitamin C). But all this left the medical profession cold. For a decade after Holst's work, and a century and a quarter after the Lords of the Admiralty accepted Lind's theories, the *Encyclopedia Britannica* and leading medical texts disparaged the dietary theory and spoke favorably of "infection by an unknown microbe." In 1950 a text widely used by English-speaking students of medicine stated that "young men totally deprived of vitamin C, but leading an active life with outdoor exercise have not developed any symptoms or signs of scurvy over prolonged periods." Thus two centuries after Bachstrom's studies and twenty years after Szent-György identified the chemical nature of the missing substance in scorbutic diets a distinguished internist found the theory so distasteful that he allowed himself to forget that sailors who died of scurvy had been young men getting 12 to 20 hours daily of "active outdoor exercise" on the decks and in the rigging of their ships, and that lemon juice had promptly cured every symptom and sign.

The story of scurvy is now being reënacted in the history of atherosclerosis and the effect of excessive dietary cholesterol. Rupture of the left ventricle was first described by William Harvey in 1649, in the heart of a noble knight. By 1900 physicians recognized that coronary atherosclerosis and angina pectoris were "afflictions of the better classes, not of the working classes" (Osler). Indeed, atherosclerosis was never found in wild, domestic, or laboratory beasts, no matter how fat or how old they became. The lesions were far more severe in those people who regularly used eggs and milk products than in those who ate only cereals, vegetables, fruit, fish, and meat.

Windaus and his pupils found cholesterol abundant in the atherosclerotic plaques and in the diets of those who developed the lesions. They knew that all animals synthesized this substance, and that herbivors on cholesterol diets excreted large quantities of cholesterol in

bile and milk. The facts of comparative and of geographic pathology suggested to Anitschkow that dietary cholesterol might be the cause of atherosclerosis and in 1914 he reported that the rabbit, normally free of antheroma, developed severe lesions when fed cholesterol. His work was confirmed in Europe, and in America, where feeding cholesterol to chicks and hypothyroid dogs also was found to produce severe disease. No lesions occurred in control animals on similar regimes but receiving no cholesterol by mouth. In all three species, as in man, arterial hypertension accelerated the development of the lesions.

Antischkow made an inspired guess when he ignored endogenous cholesterol as a possible source of the lesions. In alloxan diabetes or following intravenous injections of detergents, such as Tween 80®, extremely high endogenous plasma cholesterol levels have been maintained in rabbits.[1] No atherosclerotic lesions developed, while controls with lower plasma cholesterol levels due to *fed* material showed widespread atherosclerosis. At the University of California, endogenous cholesterol was tagged by giving rabbits radioactive hydrogen as H_2^3O; in others the dietary cholesterol was tagged. This study[2] showed that "exogenous cholesterol forms the bulk of the cholesterol in atheromatous deposits found in cholesterol fed rabbits" although "cholesterol synthesis continues even in the face of massive prolonged cholesterol feeding."

No one has yet produced such lesions in mammals except by adding cholesterol to their diets, although innumerable attempts have been made by increasing endogenous production, by causing severe injury to arterial walls by direct trauma, by experimental hypertension with periarteritis, and by injuring arteries with diets low in choline, riboflavin, and other factors. Similarly, in man, atherosclerosis decreases in wartime with restriction of dietary cholesterol, although other dietary deficiencies, as well as work, worry, and the increase in the infectious diseases, would be expected to increase "degenerative" diseases during blockade or enemy occupation.

"It is a principle well established in medicine that agents harmful to laboratory animals must be assumed to be harmful to men until definitely proved otherwise" (Shields Warren). Most clinicians and science writers take an opposite view when discussing atherosclerosis. Their bias toward this problem is summed up in their usual statement: "The rabbit has no cholesterol in its normal diet and hence lacks ability to handle this substance." As a matter of fact, rabbits secrete milk twice as rich in cholesterol as human or bovine milk, and baby rabbits consume far more milk per kilo per day than human babies.[3] Cholesterolosis of the aorta is commonly found in human infants prior to weaning, and similar lesions develop in most suckling rabbits. After weaning, these plaques disappear quickly both from children and young rabbits. A final point of likeness between man and the rabbit is that a few individuals of both species show little rise in blood cholesterol and no atherosclerosis as a result of prolonged cholesterol excess in the diet. The mechanism of "resistance" is unknown. The percentage of "resistant" men, who show no evidence of coronary or aortic atherosclerosis by the age of 55, is less than 10 per cent according to necropsy findings reported from Minnesota.

It is now clear that endogenous cholesterol plays no part in atherosclerosis except when hens are kept under high estrogenic stimuli for periods much longer than the laying period of wild fowl, and perhaps in some human beings with hypercholesterolemia who continue to excrete abnormal amounts of cholesterol in the bile even on cholesterol-free diets. In most such cases, however, blood levels of cholesterol fall strikingly when the patient is placed on a low cholesterol diet, liberal in protein.

Since two centuries of practical and experimental study have failed to convince some physicians that diets of salt pork, beans, and flour may be inadequate for maintaining health, it probably will take a long time to convince the profession that diets rich in eggs, butter, and cream cause the disease which now kills nearly one out of every three physicians. The biologist knows that such diets are as alien to adult mammals as those which cause scurvy, beriberi, and pellagra.

The nutritionist may fail to correct this

type of abnormal diet, but industry will find it profitable to have those who use these foods add sitosterols or dihydrocholesterol[4] to the diet. These substances block cholesterol absorption, and prevent both hypercholesterolemia and atherosclerosis in the experimental animal.

Thus, by a slight increase in cost of his foods, man may learn to eat cholesterol and not absorb it.

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