Editorial

Diet and Atherosclerosis: Significance of Exceptions That Test the Rule

In the enormous literature on diet and atherosclerosis pouring out in increasing volume each year, there are two main refrains. One ignores nearly every factor but diet, and skips lightly over national statistics which do not fit on the curve relating coronary deaths to per capita ingestion of animal fat. The other emphasizes every factor except diet and concludes "there is much more work to be done before any conclusion can be drawn as to the relation of diet to arterial disease." Of course there is always more work to be done in clarifying any biologic problem, and it is obviously most desirable that much more information be obtained on species and individual responses to emotional stress, physical activity and dietary factors in relation to atherogenesis, thrombogenesis, and of both of these to plasma lipids. One way to throw light on this crucial problem is to investigate the apparent exceptions to the rule that in large cultural groups the coronary death rate is related to the intake of saturated fats.

It is, of course, irrelevant that there is a relation between intakes of protein and saturated fat, or even between the latter and per capita purchases of television sets, automobiles, or mink. There is clear-cut experimental evidence, in many species of warm-blooded vertebrates, that raising the intake of saturated fats raises plasma lipids and evokes atherogenesis and thrombogenesis. There is no evidence that such lesions can be evoked by high intake of protein, nor by exposing animals to television, carrying them in automobiles, or setting the cages on second-hand furs. The fact that rich people have more severe atherosclerosis than the poor was what led Ignatovski to feed egg yolk and butterfat to rabbits, since the intake of these foods has always been high in the diet of the rich and low in the diet of the poor. The fact that coronary disease is correlated to various indices of per capita wealth is not new, and is relevant only when experiment shows a connection between wealth and factors important in atherogenesis. This has been shown for cholesterol and fat.

Although experiments on animals first proved the importance of dietary lipids in determining the levels of plasma cholesterol and rates of atherogenesis, studies of human populations have not always confirmed the relationship between diets rich in animal, egg and dairy fats and the coronary death rates and plasma lipid levels.[1,2] When one compares death rates from coronary disease with diets on a national scale, one finds that the death rates rise from 6.5 per 10,000 per year, at age fifty-five to sixty-five in Japanese men, with almost no eggs and no dairy products in their diets, to 82 per 10,000 per year, in the United States, with its very high intake of these foods.[2] These countries seem to fit nicely on a slope relating animal fat intake to coronary death rate. So do many other countries, as emphasized by Keys.[3]

However, Yudkin[1] and Yerushalmy and Hilleboe[2] point out the striking exceptions to this relationship, and see in these a complete refutation of the idea that diet, and notably animal fat, plays a great role in atherosclerosis. Obviously, much more needs to be known about the life-long levels of fat ingestion in countries with low coronary death rates, such as France (12 per 10,000), or Holland, Denmark and Norway (under 25 per 10,000) where the intake of animal fat is relatively high.[2] These coun-

tries, and England with a coronary death rate of 45 per 10,000, also consume more fish, which has protective unsaturated fats, but this alone scarcely can be credited with the difference in coronary death rates. It is worth noting that groups with low coronary death rates, but high rates for cerebrovascular deaths, have low fat but high salt intake, and much hypertension. This has been striking in Japanese and in African Bantus.

One place to look for a possible explanation of these exceptions to the rule is in the rates of atherogenesis and in diets during adolescence. In America, as Holman[4] in New Orleans and Lober[5] in Minnesota have shown, atherogenesis in white males is most rapid between the ages of ten and twenty-five years. American dietary surveys show an incredibly high intake of dairy and egg fat by white males during these years, but no corresponding increase in either dietary fat or in atherogenesis in white girls.[6] While dietary surveys and studies of arteries in European adolescents have not been published, it is apparent in discussing the problem with European parents and pathologists that it is in this age group that a great difference from the American population is seen. Boys rarely have eggs or cream for breakfast or lunch, they consume little ice cream and do not drink milk. Their load of school work and modes of travel to school force them to a life which favors high caloric expenditure with a diet actually lower in animal fat than the diets of their parents (who have much more substantial mid-day meals).

Another factor is that of thrombogenesis. This can best be determined by the incidence of postoperative thromboembolism and of pulmonary emboli in autopsy material in various countries. (In several hundred autopsies of Negroes over forty years of age in St. Louis, Mo., myocardial infarction was found in 18 per cent, thromboembolic lesions in 22 per cent; in a similar number, matched by age and sex, in Uganda, the incidence was: thromboembolic disease in 2 per cent, infarcts in 0.4 per cent.[7]) If, as the English believe, thrombotic disease is more severe and atherosclerosis about the same in populations with high levels of coronary deaths as compared to others with lower rates, the incidence of thromboembolic disease should also be higher. How the way of life affects this mechanism and why it should be only one-third as frequent in France as in England demands further inquiry. Perhaps differences in habits of drinking, in the relation of the largest meal to activity or to sleep and in the use of salt rather than skill in cooking, play a part in the different rates on the two sides of the Channel.

The Latin aphorism "Exceptio probat regulam" is distorted when "probat" is translated as "proves" and not as "probes." Study of the exceptions which test the rule that diets and atherosclerosis are related undoubtedly will cast new light on the causes of the high rate of coronary disease in the United States. Perhaps it will turn out that psychologic factors, rather than differences in diet and physical activity, make the difference between a rate of 12 per 10,000 per year in France and 82 in America. This would confirm the belief of Kipling that the French are "lifted over all By the light sane joy of life, the buckler of the Gaul." But the readers of Celine and Simenon, of Anouilh and Camus, and of Jean Genet and Giono, take it for granted that both rural and urban Frenchmen are profoundly disturbed and frustrated by life, and that the stresses which may alter plasma lipid and thrombosis are as numerous and effective in France as in Manhattan or London.

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