The lipid hypothesis: orthodoxy by default?

John Rivers, a London-based nutritionist, looks at the debate over diet and coronary heart disease

DR George Mann is a university teacher, a physician, a nutritionist and a heretic. He is one of the small number of outspoken opponents of the theory that the key to the modern epidemic of coronary heart disease (CHD) is the consumption of too much fat which is too high in cholesterol and has too low a ratio of polyunsaturated to saturated fatty acids. That this opposition should justify the term 'heretic' is not a reflection of the unchallengeable nature of the evidence supporting the 'lipid hypothesis', but the extent to which official gatherings of experts in different countries have made it national policy.

CHD is a sudden event, but as it only occurs in arteries narrowed and made less resilient by atherosclerosis, its origin must be sought in the factors which lead to years of deposition of cholesterol-rich atheromatous plaques. Virchow's observation over a century ago that the cholesterol in these plaques is derived from serum cholesterol suggested high serum cholesterol was the primary cause. Ample epidemiological evidence backs this view. The disagreement starts when one seeks the cause of high serum cholesterol.

As over 90% of our body cholesterol is endogenously synthesised, Mann dismisses dietary cholesterol as an important determinant of serum cholesterol. Rather, he suggests, one should look for factors which alter rates of synthesis or excretion. In his view the latter is crucial, and he notes a reduction in cholesterol breakdown is known to result from exposure to high levels of atmospheric carbon monoxide—hence the importance attached to smoking and pollution—and from consumption of *trans* unsaturated fatty acids.

Powerful evidence can be adduced to support his view that present dietary hypotheses of hypercholesterolaemia are mistaken. For example, a recent study of 2,000 subjects in Michigan found no significant differences in intakes of energy, total fat, unsaturated fat, saturated fat, or cholesterol between the upper and lower tertiles for serum cholesterol. What ultimately matters, however, is not the effect of diet on serum cholesterol but its impact upon mortality from CHD. Reviewing the dietary modification trials that have been conducted to date, Mann concludes that differences in mortality in primary or secondary clinical trials have yet to be shown. Although mortality from CHD is reduced in some, a diagnosis of the cause of death is relatively soft data, and the total death rate has remained constant. Mann argues that if CHD mortality really fell in these trials, mortality from something else must have risen for the total deaths to have been constant. He points out that this often seems to be cancer.

The idea that polyunsaturated fats predispose towards cancer dates from the studies of Pearce and Dayton (*Lancet* i, 464–467, 1974). Many nutritionists regard the idea as unproven—at the least the evidence about it is conflicting. But it is Morton's Fork. If, like Heyden (in *The Role of Fats in Human Nutrition*, Academic Press, 1975), one dismisses the apparent rise in cancer deaths as due to poor design of the trials, then the trials are also too poor to provide evidence about deaths from CHD. The trouble, says Mann, is that the experiments are not scientific. Scientists should design experiments to disprove hypotheses—dietary trials are still being done to confirm them. Because the lipid hypothesis is still not proven after 27 years, he says, the time has come for a rethink.

More troubling than Mann's scientific objections to the

lipid hypothesis are the effects that widespread acceptance of it has had. It has become self-sustaining: heretical opponents find it difficult to obtain research funds, and the medical hypothesis is being increasingly used as an advertising ploy—a massive vegetable oil industry needs evidence for the benefits of polyunsaturated fats.

Even more important, though, we may be doing exactly the wrong thing. Pursuit of the lipid hypothesis does not mean just swapping polyunsaturates for saturates. Polyunsaturates are too unstable, so much of the extra polyunsaturated fatty acids are provided by the substitution of soft margarines and stabilised vegetable oils for butter and animal fats. The trouble is, both changes lead to a dramatic increase in consumption of *trans* fatty acids, compounds whose known hypercholesterolaemic effect (Vergossen and Gottenbos, *The Role of Fats in Human Nutrition*) outweighs the putative benefits of polyunsaturated fats.

Butter contains 8% of its fatty acids as *trans* isomers, the product of biohydrogenation in the rumen. Vegetable oils, which like soft margarines are usually partially hydrogenated, contain *trans* fatty acid levels of up to 40%. Manufacturers of polyunsaturated oils and margarines may proudly state that they are high in polyunsaturates, but they do not state the levels of *trans* fatty acids, nor do they feel obliged to point out the crucial modification that those fatty acids may have undergone.

Like many of the critics of the lipid hypothesis, Mann has sought supporting evidence for his view from studies on the Masai. These meat- and milk-eating pastoralists have low serum cholesterol in spite of a high consumption of animal products. Mann argues that this can be expected—they have low levels of environmental pollution and eat little *trans* fatty acid. Nevertheless, his studies on 50 aortae obtained post mortem show that they do get atheroma but its effects are minimised by the tendency of the coronary artery to widen with age. This Mann attributes to heavy exercise, pointing to epidemiological studies which show a protective effect of heavy exercise. The question obtrudes itself. Is a dietary solution being sought because it is more acceptable than exercise?

It was sad that, to hear Mann talk, I had to attend a press gathering organised by the Butter Information Council, a public relations body representing the dairy industries of Europe. One must regret that he was not asked to present his views to a scientific audience, although they do have available a good summary in his latest paper (*New England Journal of Medicine* 297, 644–650, 1977). Our dietetic fate is in danger of being decided by a public relations battle between margarine salesmen and cowherds.

In the face of this, however, we cannot seek reassurance from the deliberations of the expert committees on the topic. Two successive UK committees on heart disease, under the aegis of the Department of Health and Social Security (*Diet and Coronary Heart Disease*, HMSO, 1974), and the Royal College of Physicians (*J. Roy. Coll. Physicians*, 10, 213, 1976), have come to diametrically opposed conclusions about the value of the lipid hypothesis. The evidence available to both was substantially the same. The latest report, from a US Senate Committee under Senator George McGovern, has recommended among other things a reduction by 30% in dietary total fat intake accompanied by a 50% rise in the consumption of polyunsaturated fatty acids. Such sweeping changes are only likely to be achieved by a substantial shift towards the use of vegetable oils and, according to Mann, may cause more problems than they solve. Not all that he says is convincing, but it is a powerful criticism of a view that is in danger of becoming orthodoxy by default. □