

many others right up to George Bernard Shaw the great critic⁹ may serve as examples. They despise doctors, convinced one is far better off without them. In a way, such vehement accusations nevertheless reflect the ubiquitous authority physicians enjoy. On the other hand, the fact that professional ignorance or carelessness will meet its immediate consequences, makes doctors a welcome target in society. So much a single physician may command in an individual person to person setting, so little the doctors, as a small stratum in society, must be feared on the whole. Mockery and derision can all too easily be poured over them.

In today's world, doctors all too wilfully accept a stance to which they aren't up to. Miracles cannot be performed, doctors cannot transcend the terrestrial sphere, they are no saints, they even have to make their living on the sickness of their fellow beings.

Let us be humble and keep a low profile, both in what we are and in what justifiedly may be expected from each and every one of us anytime. This is the only way to keep (justified?) criticism down to a minimum.

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Diet and coronary heart disease

Professor Yudkin's arguments (September 1992 *JRSM*, p 515) concerning the central role of sucrose in the aetiology of CHD are unconvincing, and some of his conclusions surprising. His assertion that, 'there is no substantial and convincing evidence that dietary fat or cholesterol is a cause of CHD', does not follow from the evidence presented in his article.

In fact, there is compelling scientific evidence to support the view that the serum total cholesterol concentration is the most important single factor in determining the risk of CHD, with the level of risk rising progressively with increasing levels of cholesterol¹⁻⁴. Moreover, primary prevention of coronary heart disease by reduction of cholesterol levels, has been conclusively demonstrated in a number of randomized, controlled clinical trials^{5,6}. Professor Yudkin rightly draws attention to the fact that in some of these studies, the reduction in cardiac mortality has been offset by an apparent increase in non cardiac deaths⁷. This observation clearly requires further investigation, and there is no doubt that the value of treating individuals with only moderate elevations in blood cholesterol, has to be set against the potential risk of non cardiac related mortality. However, this does not negate the value of treating severe hypercholesterolaemia where the potential risk of a cardiovascular event is very much greater.

Professor Yudkin also ignores an impressive and growing body of evidence to suggest that reducing cholesterol levels can lead to regression of coronary atheroma⁸. In the St Thomas' Atherosclerosis

Regression Study (STARS), dietary change alone retarded overall progression and increased overall regression of coronary heart disease, and in combination with cholestyramine produced a net increase in coronary lumen diameter⁹.

In summary; there is good evidence that hypercholesterolaemia is a major independent risk factor for CHD; that reducing cholesterol levels reduces risk and (in some studies), produces demonstrable regression of atheroma. I doubt that Professor Yudkin could offer such an impressive and consistent body of evidence in support of sucrose.

Since diet is the main environmental determinant of plasma lipid concentrations, it is reduction in saturated fat consumption not sucrose, which should continue to be the main dietary intervention for the prevention of CHD.

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Yudkin claims in his editorial (September 1992 *JRSM*, p 515) that there is stronger experimental and epidemiological evidence against dietary sucrose than fat as a causal factor of coronary heart disease (CHD). I should like to point out that in direct comparisons the evidence against lactose is stronger than that against sucrose.

In experimental hypercholesterolaemia and atherosclerosis, the addition of lactose to the diet produced significantly greater enhancements than did sucrose, in rabbits¹ and baboons². In controlled metabolic studies in young men, the average serum cholesterol rose from 185 mg% to 231 mg% on lactose and fell to 162 mg% after replacement by sucrose³; and the average plasma lipid profile was significantly affected adversely by 2 US quarts of skimmed milk (about 87 g of lactose, my estimate) daily compared with a control period in which the carbohydrate was balanced mainly by a sugar-containing drink⁴.

Correlation of per caput food supply⁵ for 1985 with age-standardized pooled male and female mortality