Food labelling can be of great value to those with a degree of sensitivity which makes it necessary for them to avoid particular substances, and further studies are certainly needed to identify the nature of the reactions which occur. There is no justification, however, for the alarmist advice which has led vulnerable people with vague symptoms to adopt inadequate, restricted diets which are themselves a cause of disease. It is a matter for concern that parents who are worried about behaviour disorders in their children or what they consider to be hyperactivity can sometimes be led to impose an obsessional dietary regimen on an already disturbed child. While increased parental attention may be beneficial in such cases, food prohibitions on the basis of an unconfirmed diagnosis cannot be justified.

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Diet and coronary heart disease: why blame fat?

The article by Gorringe[1] reminded us that coronary heart disease (CHD) is one of the diseases associated with affluence, and that it is more prevalent in populations with a high standard of living than in those with a low standard. This is reflected in the higher prevalence of CHD in countries where cigarette smoking is high and where many people have motor cars, radios and television.

Gorringe properly points out that such statistical evidence from populations does not prove cause, but it is not only in populations that they occur. Individuals that smoke have a higher risk of developing CHD than do non-smokers. And people with cars, radios and televisions are likely to be less active physically than those who do not, and physical activity is now also accepted as a risk in developing CHD.

As regards diet, differences between wealthy and poor populations are so numerous that it has become necessary to try and isolate those items and processes that are harmful; the current view is that it is dietary fat, especially the saturated fats, that are the dietary cause of coronary heart disease.

A more careful examination of the diets is needed in order to ensure that we have isolated the true positive items.

There are two characteristics of coronary heart disease that have to be considered in seeking a dietary cause of the disease. These are: (1) the abnormalities in the blood and (2) the clinical relationship between coronary heart disease and other diseases.

It is common to point to the raised blood concentrations of cholesterol as being the underlying cause of CHD. Other blood abnormalities include an increased concentration of glucose, triglyceride, uric acid and insulin; there is also a decreased concentration of high density lipoprotein[2,3]. Other changes are a reduction in glucose tolerance, an increase in insulin resistance,

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and a series of changes in the behaviour of blood platelets[4,5].

The evidence adduced led to the suggestion that reducing the fat content of the diet reduced the chances of developing heart disease. And so long as high blood cholesterol was in itself perceived as the cause of occlusive vascular disease, in the coronary circulation or in the peripheral circulation, it was thought necessary also to reduce the intake of cholesterol-containing foods. Since eggs have a relatively high concentration of cholesterol, people were often recommended to limit their intake of eggs.

The second piece of evidence that is cited is that several trials, involving tens of thousands of middle-aged men in which cholesterol concentrations in blood were reduced by diet and sometimes by drugs as well, have shown a reduction in the number of people dying from coronary diseases.

But what is frequently ignored is the fact that total mortality in such groups of people was not reduced[6]. That is, there was, unexpectedly, an increase in deaths due to a variety of other causes, notably accidents, violence, cancer and strokes. Another way of stating these findings is to say that, if you wish to increase the number of people dying from accidents, violence, cancer or strokes, then give them a diet low in cholesterol and fat, and perhaps give them also a cholesterol-lowering drug. At the same time, you will save the lives of a similar number of people from dying of coronary disease. Except for the fact that the proportion of people in each category is not very large, one could almost say, with 'Punch', 'you pays your money and you takes your choice'.

We have to conclude that there is no substantial and convincing evidence that dietary fat or cholesterol is a cause of CHD. However, this conclusion is not the same as saying that we must abandon altogether the view that diet has nothing to do with causing the disease. There is indeed a dietary item other than fat for which there is now overwhelming evidence of its involvement in producing the disease. This item is sucrose ('sugar').

In the first place, epidemiological evidence, which cannot of itself be conclusive, is nevertheless stronger for sucrose than it is for fats[3]. But it is the experimental evidence that is very strong, and answers all the reservations we have in relation to dietary fat.

In about 30% of men, an experimental increase in dietary sucrose produced all the changes that are seen in CHD and peripheral vascular disease[7,8]. These include increases in the blood concentration of cholesterol and other lipids, increases in uric acid and insulin, a fall in high density lipoprotein cholesterol and in glucose tolerance, and the production of insulin resistance. In addition, there are changes in the platelet behaviour that are characteristic of CHD. These abnormalities can also be shown in the sucrose-sensitive men who are taking the average quantity of dietary sucrose, and are reversed when the sucrose intake is reduced[9].

The results of these experiments led to the hypothesis that the underlying cause of CHD and of peripheral vascular disease is a raised concentration of blood insulin, and insulin resistance. Insulin resistance is also a feature of hypertension, diabetes, obesity and gout, all of which are frequently associated with CHD and PVD.

A third piece of evidence is that, in the sucrose-sensitive men, reducing the chances of developing CHD, by increasing physical activity, reduces insulin concentration in the blood.

In patients with PVD, there is a higher concentration of insulin and platelet adhesiveness than there is in control patients. Quantitatively, these are both related to their habitual sugar intake[8].

It is difficult to avoid the conclusion that sucrose can produce CHD in a significant proportion of men. And the considerable rise in CHD mortality in affluent countries during the past 50 years or so, may well be due to the considerable increase in sucrose consumption of those countries during that period.

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