St. Mary's Hospital Medical School

(University of London)

London, W.2.

EPIDEMIOLOGY DEPARTMENT

01-723 1252 Ext. 135

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Dr. H. Blackburn,
Laboratory of Physiological Hygiene,
School of Public Health,
University of Minnesota,
Stadium Gate 27,
Minneapolis,
Minnesota 55455,
U.S.A.

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Dear Henry,

Thanks for your letter. I am sorry that my fellow countrymen have not been very helpful. You ask me to be devil's advocate: this is somewhat difficult, and I shall not put up a very good performance, since as you know I am a believer. In essence my own position is this: I think that a high fat diet (and consequent elevation of blood cholesterol level) is probably the one essential cause of a population having any serious incidence of CHD. Within our population or yours I think that differences in fat intake are only minor determinants of who gets the disease, but I am not sure how big 'minor' is. I believe that the evidence for causation is strong enough, and obtains sufficient support from trials of its reversibility, to justify advice to the public, the forcefulness of the advice matching the level of an individual's risk. However, none of this is what you actually wanted to know.

I think you are mistaken in speaking of a 'United Kingdom view', in two respects. The first is that our minds are not clear enough to qualify for such consistence. The second is that it is my impression that a Gallup poll of those doctors who have thought about the subject would now show a large majority believing that diet is probably a major causal factor. Gerry Shaper is chairman of a working party (of which I am also a member) of our Cardiac Society and the Royal College of Physicians, which will shortly give birth to a report on the prevention of CHD. This should not be quoted prior to publication but it will certainly express the view that high fat diet is an important cause of the disease, that it should be generally modified by the public, and that this can only be achieved by a regime which includes some substitution of SF by PF.

Nevertheless you are certainly correct in saying that British opinion has been slow to come to this point and is still much less committed to it than American opinion.

Now at last let me try to give at least a partial answer to your question as to why this should be.

1. Within any one population dietary fat has not been found to correlate either with risk of CHD or with cholesterol level. The arguments of Ancel and others that this is due to dietary homogeniety coupled with inaccurate assessment methods is unconvincing as it appears never to have been quantified by him. When Harold Kahn did quantify it on the Israeli Heart Study data, using pessimistic estimates of error in dietary assessment, he came up with the conclusion that in that population dietary fat variation accounted for substantially less than 20% of cholesterol variance.

Dr. H. Blackburn 11th November 1975

2. United Kingdom data on the predictive significance of serum cholesterol levels are regrettably scanty. Morris's small study was clearly positive. Archie Cochrane's data have not been published but are negative. Our Whitehall Study data are about to be published and show a very shallow gradient of risk. (Actually the gradient is similar to the Framingham findings over the same rather low part of the cholesterol range.) Thus, if in the United Kingdom we were able by dietary change to get average cholesterol levels down to, say, 200mg/dl, we should not expect by that alone to achieve a very large reduction in CHD incidence.

3. The experience of physicians here in controlling obesity by dietary advice has left them pessimistic about the effectiveness of dietary advice in general.

4. The Helsinki trial has been somewhat discredited (to a much exaggerated extent, I think) by statistical criticisms of its design, and the VA trial has similarly tended to be discounted because of the absence of any effect on total mortality. (I would personally add concern at the Framingham finding that serum cholesterol was a negative predictor of total mortality in most age and sex groups. In our Whitehall Study data it is a quite unimpressive predictor of total mortality, but since these facts are not generally known, they have not hitherto had any important influence.)

The above are scientific reasons, but they are probably not the ones that have been most effective. More important in practice are the following:-

(i) The tradition of British cardiologists has been physiological and haemodynamic in its orientation and they have been slow to look at the environment.

(ii) Local findings tend to carry more weight than those from foreign parts. British data have been scanty on cholesterol but more abundant on smoking and physical activity. The latter have therefore been more emphasised.

(iii) There have been many diversionary alarms on the dietary front, from sugar onwards. The British have perhaps a national tendency to pay little heed to 'the latest notion', and the dietary fat study has been presented as but one among many dietary theories.

Your question on ECG coding is fortunately simpler. We train our own people only, using the document which I produced for WHO (Euro) some years ago. We hope shortly to be able to change to your training manual when this is available. We provide a calibration service for other centres in our collaborative trial, and on an ad hoc basis for a few friends.

I have not heard anything recently concerning progress in Budapest. I enclose a copy of a letter on the subject which I sent earlier this year to George Lamm. I think it is important that Budapest should be calibrated against Minneapolis, which should unquestionably continue to be accepted as the ultimate reference centre.

Yours,

Geoffrey Rose

Encl.