LETTERS

Dietary fat and risk of coronary heart disease in men

Studies quoted showed opposite of what is claimed

EDITOR,—Alberto Ascherio and colleagues were unable to show a significant association between intake of saturated fatty acids (as percentage of energy) or of cholesterol and total myocardial infarction and coronary heart disease after appropriate adjustments.[1] With saturated fatty acids they found a positive trend for fatal coronary heart disease but an inverse trend for all cases of myocardial infarction (fatal plus non-fatal). Thus these data indicate a clear inverse relation between saturated fatty acids and non-fatal myocardial infarction. The authors fail to comment on this surprising divergence.

From the numerous studies in the literature they quote "the evidence" for a direct association between intake of saturated fatty acids and risk of coronary heart disease—namely, four international comparisons and four prospective studies. But in fact these studies tend to show the opposite. In Scrimshaw and Guzman's study animal fat was not related to the degree of atherosclerosis. The two papers on Japanese emigrants reported no data on the relation between saturated fatty acids and coronary heart disease, although another analysis found that the cultural upbringing of the emigrants was far more important than their diet.[2] In the seven countries study the association was seen in cross cultural analysis but not within the cohorts. In two of the four prospective studies, the Framingham study and the Ireland-Boston diet-heart study, the associations did not reach significance.

In Goldbourt *et al*'s study there was an inverse trend between intake of saturated fatty acids (in g/day) and coronary heart disease, and data on intake of saturated fat as a percentage of energy were not reported. This leaves just one prospective study in the literature that directly related intake of saturated fatty acids to risk of coronary heart disease.[3]

Thus Ascherio and colleagues' results are in good agreement with the vast majority of data in the literature in showing no risk of coronary heart disease related to consumption of saturated fatty acids. It is time to realise that the diet-heart hypothesis as it relates to intake of saturated fatty acids and cholesterol can be upheld only if most observations are ignored and quotation bias is used.[4]

Finally, Ascherio and colleagues do not present any data on oleic acid, nor do they give their reason for withholding these data. This is all the more surprising since one of the authors (Willett) has claimed health benefits from consumption of olive oil—so far without sufficient evidence.[5]

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1 Ascherio A, Rimm EB, Giovannucci EL, Spiegelmann D, Stampfer M, Willett WC. Dietary fat and risk of coronary heart disease in men: cohort follow up study in the United States. *BMJ* 1996;**313**:84-90. (13 July.)
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Study gives clear message about diet

EDITOR,—Alberto Ascherio and colleagues studied over 40 000 health professionals to examine the association between fat intake and the incidence of coronary heart disease in men of middle age and older.[1] Investigation into the influence of dietary fats on risk of heart disease has long been a contentious field, and the literature shows a quick response to the publication of new work. For example, after the publication of the paper by Law *et al* on cholesterol reduction and risk of ischaemic heart disease in 1994[2] the *BMJ* published 10 letters commenting on the study.

If discussion of the minutiae of study design, results, analysis, etc is put aside then the message from Ascherio and colleagues' study is clear and simple: all middle aged and older men who want to reduce their risk of death from coronary heart disease must lower their intake of saturated fat and cholesterol while increasing the amount of n-3 fatty acids and fibre in their diets. Interestingly, these conclusions are similar to the information coming from intervention studies of Mediterranean diets, which have concluded that to reduce the risk of heart disease we must eat less meat (a major dietary source of saturated fat and cholesterol) and more fruit, cereals, and vegetables (sources of fibre).[3] We could probably improve our risk profiles even further if we drank a glass of red wine with our meals.[4]

This excellent and easily available dietary treatment is unlikely to become common practice among the general public, because we now live in the age of the soft option. The west of Scotland coronary prevention study indicates that similar or perhaps even better results can be obtained by giving 3-hydroxy-3-methylglutaryl coenzyme A reductase inhibitors prophylactically.[5] The pharmaceutical industry would have us believe that these drugs are the only way to combat cardiovascular disease, although this is clearly not the case.

Although a lower incidence of heart disease in the population would, in the long term, result in reduced costs to health services, an important issue to consider is that prescribing these drugs takes responsibility for maintaining health away from the individual and lays it at the door of health professionals. Should already strained health services shoulder the burden of prescribing drugs prophylactically to those people who would rather take a pill than observe a healthier diet?

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1 Ascherio A, Rimm EB, Giovannucci EL, Spiegelman D, Stampfer M, Willett WC. Dietary fat and risk of coronary heart disease in men: cohort follow up study in the United States. *BMJ* 1996;**313**:84-90. (13 July.)
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Population studied was peculiarly healthy and preoccupied with health

EDITOR,—According to Alberto Ascherio and colleagues, saturated fat may not be as dangerous with respect to coronary heart disease as commonly supposed, some of the risk being counteracted by consumption of fibre.[1] As might have been expected from the data in their table 1, consumption of saturated fat was positively correlated with cigarette smoking ($r = 0.981$) and mean body mass index ($r = 0.986$) and negatively correlated with exercise ($r = -0.983$), consumption of carotene ($r = -0.986$) and vitamin E ($r = -0.950$), and, curiously, alcohol consumption ($r = -0.984$).

Could it be that the population of male health professionals in the United States is peculiarly healthy and preoccupied with health? As has been noted elsewhere, this could mislead the unwary.[2] When figures from the study are

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When deciding which letters to publish we favour originality, assertions supported by data or by citation, and a clear prose style. Wit, passion, and personal experience also have their place.

Letters should have fewer than 400 words (please give a word count) and no more than five references (including one to the *BMJ* article to which they relate); references should be in the Vancouver style. We welcome pictures.

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compared with targets in the Health of the Nation[3] a smaller proportion of the fifth of the population in the study that had the highest saturated fat intake smoked cigarettes (14.4% *v* 20%, the British target for 2000) and this fifth of the population was the only one to derive more than 35% of total food energy from fat (the British target for 2005). Mean alcohol consumption was low in all the groups, at below 14 units a week, whereas the Health of the Nation's target for 2005 is to reduce the proportion of adult men drinking 21 units a week to 18% (taking 1 unit as 8.5 g alcohol[4]). Furthermore, only one group failed to achieve the British target of eating five servings of vegetables and fruit a day.

Regardless of the representativeness of the American health professionals, the reported mean serum cholesterol concentration of the groups was remarkably consistent at 5.2-5.3 mmol/l.

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Relative risks should not have been adjusted for body mass index

EDITOR,—In their prospective study of 43 757 health professionals in the United States, Alberto Ascherio and colleagues found that the multivariate relative risk of myocardial infarction was increased to 1.22 (95% confidence interval 0.96 to 1.56) and that of fatal coronary heart disease was increased to 2.21 (1.38 to 3.54) for men in the top versus the lowest fifth of saturated fat intake.[1] They conclude that these data do not support the strong association between intake of saturated fat and risk of coronary heart disease suggested by international comparisons.

The authors' conclusion is questionable because the relative risks were also adjusted for differences in body fatness (body mass index), which is generally believed to be promoted by a high fat diet and is a strong risk factor for coronary heart disease. By adjusting the relative risks for body fatness the authors eliminated the effect of a high fat diet on coronary heart disease exerted through increased body fatness. From the figures in table 1 of the authors' paper the correlation between saturated fat as a percentage of energy and body mass index is 0.97 ($P<0.002$) and that between total fat as a percentage of energy and body mass index is 0.99 ($P<0.001$). This strongly suggests that obesity was associated with a high dietary fat and saturated fat intake in this cohort. As some of the detrimental effect of a high fat diet is mediated through increased body fatness it seems more appropriate to present multivariate relative risks for coronary heart disease unadjusted for body mass index.

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Authors' reply

EDITOR,—Nicolai Worm notes that our data imply an inverse association between intake of saturated fat and risk of non-fatal myocardial infarction. Any factor that increases the case fatality rate of a disease without affecting its incidence, however, will be inversely associated with non-fatal occurrences of the disease. Such associations are of dubious interpretation. Also, we did not report results on intake of oleic acid because this is mainly derived from red meat and dairy fat (rather than olive oil, as in the Mediterranean diet) and is highly correlated with intake of total fat ($r = 0.96$) and saturated fat ($r = 0.73$). Attempts to adjust for these correlated nutrients cause wide confidence intervals, and results are not very informative. For example, the relative risk for men in the top fifth compared with those in the bottom fifth of oleic acid intake was 0.85 (95% confidence interval 0.53 to 1.44) after adjustment for total fat and polyunsaturated fat intakes.

J Munby and D F Weetman have calculated correlations between mean saturated fat intake and the means of other variables for the fifths of the population shown in our table 1. These calculations, however, greatly overestimate the correlations between these variables within the cohort. Also, the authors seem to suggest that saturated fat intake may be more strongly associated with risk of myocardial infarction in populations with a higher prevalence of smoking, a higher alcohol intake, and lower consumption of fruits and vegetables. Statistical power to detect these interactions was limited even in our large study, and therefore we cannot exclude this possibility. Although our population was decidedly more health conscious than the average British or American population, this provided the opportunity to evaluate the potential effects of diets with reduced levels of saturated fat.

Finally, Arne Astrup has also incorrectly calculated correlations between mean values; the actual correlation between saturated fat intake and body mass index was 0.09, and thus confounding by body mass index was slight. We certainly agree that maintaining a lean body contributes considerably to the prevention of coronary heart disease, but the role of dietary fat in causing obesity is minor.[1 2]

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Healthy sexual lifestyle should be emphasised when negative results of HIV tests are given

EDITOR,—Riva Miller and Marc Lipman welcome the Department of Health's recent guidelines for pre-test discussion on HIV antibody testing.[1] I am concerned, however, that there are two serious omissions from these guidelines, which may have an effect on the public health.

Firstly, no reference is made to offering testing for other sexually transmitted diseases. People who request testing for HIV antibody have a high incidence of sexually transmitted diseases, many of which are symptomless and are detected only by routine screening.

Secondly, the guidelines give no advice about counselling after the test for those in whom the result is negative. Most people who have an HIV test are given a negative result. This is an ideal opportunity to reinforce issues of general health promotion and of sexual health promotion in particular. Such an opportunity to emphasise the importance of a healthy sexual lifestyle should not be minimised when a negative result is given. Experience in genitourinary medicine clinics (where most HIV tests have been carried out) suggests that patients are particularly receptive to this advice at this time, when the anxieties about the HIV test have diminished.

Skill is readily available in genitourinary medicine clinics for both counselling before and after HIV tests and screening for sexually transmitted diseases.

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1 Miller R, Lipman M. HIV pre-test discussion. *BMJ* 1996;**313**:130. (20 July.)

Evidence for the risk of calcium channel blockers in hypertension was selective

EDITOR,—We have five comments on the Fortnightly review by D G Beevers and P Sleight.[1]

Firstly, Beevers and Sleight say that results from a Chinese study may not be readily applicable to a Western population and that a longer acting formulation of nifedipine was used.[2] It could be argued that if a beneficial effect was demonstrable in a population with low incidence of an event then it would be similar, if not more beneficial, in a population with a higher incidence of that event. Beevers and Sleight also did not attribute the better outcome in the nifedipine group to the use of a longer acting preparation, despite acknowledging the lack of information on these formulations. Thus, two potentially positive features of the study were presented as negative attributes.

Secondly, the authors cite highly selected reports to suggest an increase in mortality from events related to ischaemic heart disease in patients treated with nifedipine.[3 4] These studies have been criticised [5 6] for the small number of patients studied with limited statistical power of the data and for the confounding effects of the increased prevalence of pre-existing ischaemic heart disease (between 46% and 78% *v* 32% in the β blocker group) and diabetes in the group treated with nifedipine.[3] Moreover, 515 patients were treated with β blockers, 74 with nifedipine, and 77 with verapamil. Thus, the case mix compared in each treatment arm was not sufficiently matched, introducing the likelihood of confounding errors.

Thirdly, their statement "short acting calcium channel blockers like nifedipine" ignores the widely available longer acting formulations.

Fourthly, the reflex catecholamine surge is a physiological response to hypotension including that induced by all vasodilators without a negative chronotropic effect. Not only is this effect not group related it would be expected to be less obvious in the longer acting formulations.

Finally, two possibly advantageous properties of calcium channels—antiplatelet and antispasm effects—were judged as side effects whereas in some clinical situations they may be considered beneficial.