Letters to the Editors

Trends in fat consumption

I read the paper 'Trends in individual fat consumption in the UK 1900–1985' by A. M. Stephen and G. M. Sieber (1994) with interest. This is an important subject for anyone concerned with possible links between fat consumption and chronic disease, especially in view of the intense educative activity about dietary fats that is now being encouraged.

Try as I may, I cannot reconcile the conclusions of the authors with the data as actually presented. The first and most crucial point concerns the time at which the consumption of fat, expressed as a percentage of dietary energy, started to decline. The authors state variously that this 'began in the mid-1970s' (Results, p. 780) or 'mid to late 1970s' (Discussion, p. 783). Information on this point is given in three forms. The Tables give data in 10-year groupings, which gives only a rough indication of trends. Fig. 1 is better because all data points are plotted. (Incidentally, the legend to their figure and the Abstract state that data are taken from ninety-seven studies, Table 1 lists ninety-five studies, whereas Table 2 clearly shows that only eighty-seven were included. The points on Fig. 1 are difficult to distinguish in some places, but add up to something more like eighty-seven than ninety-seven.)

The spread of points in Fig. 1 for the 1970s and 1980s indicates that in any one year the range of values spanned about 7 or 8 percentage points (for example in 1980, highest 44%, lowest 37%). This suggests that the variability in results from different studies was too great to demonstrate that a 1·2% decline from 1979 to 1985 (Table 3, males) was significant. MAFF Food Survey data show that fat intakes as a percentage of energy have remained constant at about 42% from 1968 to 1992. Although the authors' decision to avoid household consumption data is understandable, the MAFF data, while perhaps not being an accurate reflection of individual intakes, should provide a reasonable view of trends with time. The principal visual indication of a downward trend after the mid-1970s is Fig. 2, the derived plot to which I will return later.

The second point concerns the authors' enthusiasm to show that regional trends in fat consumption may account for regional differences in coronary heart disease (CHD): something that has not been demonstrated so far in the UK. Data are cited in Table 5 and Fig. 3 as evidence that the trend in fat consumption in South East England is declining while that in Scotland is continuing to rise. The authors fall short of relating this directly to changes in CHD mortality in the two regions, but they imply this by the remark: 'With the UK studies, however, there were a large number from both Scotland and the South East, two areas with different mortality rates'. Table 5 shows Scottish fat intakes (% energy) in 1960–69, 1970–79 and 1980–85 as 40·0, 41·3 and 38·1 and in South East England as 38·8, 40·8 and 40·8 in the same periods. The graphical representation (Fig. 3) shows a quite different story, a continuing rise in Scotland and a marginal fall in South East England. While I am not in a position to dispute the authors' mathematical representation of their data with weighted quadratic regressions, I suggest that few reasonable people presented with the different forms of the data in this paper would accept this as compelling evidence for substantially different trends in the two regions.

From the public health standpoint, an important message the authors wish to give is that the decline in CHD mortality observed in the UK relates well to changes in fat consumption in South East England and in Scotland. One's interpretation clearly depends not only on when fat intake and CHD mortality are judged to have peaked, but also what

is considered to be an appropriate 'lag time' between the two. This last matter is highly controversial. Thus, Rose (1982) suggests an 'incubation period' of more than 10 years while Vartiainen and colleagues (1994) suggest 2 years or less. The authors did not cite a reference for their contention that CHD mortality in the UK began to fall in 1979 but my reading of the information published by The Office of Public Censuses and Surveys is that CHD mortality in Scotland started to decline in about 1972–73 in all age-groups. In England and Wales the matter is more complicated because disease mortality peaked at different times in different age groups but the general decline began somewhere between 1970 and 1975. Thus, during a period when CHD mortality first rose, peaked and then declined, fat intakes remained relatively constant. This does not seem compelling evidence for an important influence of fat consumption on CHD mortality.

The authors lay great stress on comparisons with the USA, referring to that nation of 250 millions and some 3000 miles across as if it were one homogeneous entity. The decline in CHD mortality began in California in the late 1950s, fully 20 years before the decline in some other areas (Rosenman, 1993). I know of no reliable evidence that fat consumption in California bore the sort of relationship with CHD mortality implied by the authors of this paper. Indeed, the rises and falls of CHD mortality in many countries bear little relation to dietary fat intakes when viewed objectively (Gurr, 1992), and in Japan, where CHD mortality has been steadily falling from an already low base, fat intakes have been gradually rising (Okayama *et al.* 1993).

Public health education to prevent CHD, as envisaged, for example, by the Health of the Nation initiative, will involve enormous resources. Many people will be expecting it to deliver what it promises. It must, therefore be based on sound science, not on the sort of tenuous association implied by this publication which does no credit to the Journal.

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Trends in fat intake in the UK

The purpose of conducting this study (Stephen & Sieber, 1994) was to examine intake of fat by the UK population, rather than fat availability, as provided by food balance data, or fat purchased, as provided by the National Food Survey. The use of published information drawn from dietary assessments carried out over this century seemed to be the only way to determine the intake over this period since no regular dietary assessment has been conducted. Our previous assessment for the USA (Stephen & Wald, 1990) demonstrated that individual intake shows a quite different trend from food supply data

and indicates that availability does not reflect what people are eating. While purchases into the home are closer to consumption than food supply, they nevertheless do not measure the intake and are, like food supply, subject to the increasing amounts of wastage and spoilage which accompany greater prosperity, as has occurred in developed countries including the UK and USA over the last 20–30 years We are not alone in having concerns about assessing consumption from measures other than individual intake. A recent comparison of USDA data from food supply and individual surveys conducted at intervals shows quite different results (Crane *et al.* 1992). Trends derived from food supply or food purchases must therefore be viewed with caution, for any country.

Most of our paper is concerned with the actual trends in intake and how these appear for different age–sex groups and different regions of the country. We did not try to pick an exact year at which a downward trend occurred because the data are not strong enough for that. Indeed, because the data end at 1985, it may be easier to see a definite trend in another 10 years or so; again, the USA picture was clearer because changes began to occur in the 1960s. What is important, and undeniable, is that fat intake increased in the first half of the century and represented less than 35% energy until the late 1930s.

The individual studies were dealt with statistically using quadratic regressions, with each study weighted by the number of subjects on whom assessments were carried out. Hence studies on a large number of subjects have considerable influence on the trend line. In the comparison of Scotland and the South East there were nearly three times as many subjects in the 1970s for Scotland than the South East, and the data from the studies in this decade therefore have a greater influence on the trend, while for the South East there were many subjects studied in the 1960s which have a strong influence on the trend for that region. This is why the trend lives and the 10-year averages do not appear to give the same results. When beginning this work we had many discussions on the best way to 'weight' the data, given that the studies were done on different numbers of subjects, and the statistical advice given to me was that weighting by numbers in each study was entirely appropriate.

We did, in fact, analyse all our data, unweighted, weighted by the numbers in each study and weighted by the rank of the study size, rather than the subject numbers. In most cases the weighting made little difference to the conclusions. Even the unweighted line for Scotland is similar to the weighted ones because of high values in the late 1970s which countered those from the three studies published in the 1980s.

The apparent discrepancies with numbers in the Tables are easily explainable. Many studies on adults were done on both males and females. For investigations of trends in males only or females only the data were separated and the results for each sex used. For overall results as shown in Fig. 1, however, where each point represented one study, the results from the males and females were combined. Hence, as indicated on Table 1, the numbers do not add up. Of the ninety-seven studies, fourteen were only on males; hence twelve of those given for males were done on females as well and are also included in the thirty-five studies noted for females. The ninety-seven studies included others which did not separate between males and females. Some studies were done on children and adults, so again would be separated in Table 1 but considered as one study in Fig. 1. There are indeed ninety-seven points in Fig. 1, they are just difficult to count. There were, for example, seven studies from 1984.

In re-examining our raw data and published tables for this response, however, we did find an error in Table 2. There were, in fact, fourteen studies from the 1970s, not four, giving the total number of ninety-seven.

One of the major criticisms of our paper is the relationship to mortality from coronary heart disease. Mortality data are expressed for entire countries, even if they are large and diverse. Hence our US data, which included most states in the United States, could be

related to mortality for the entire country. It may well be that the trend in fat intake was different for California than elsewhere, but there are insufficient data to examine that. However, both the fat trend data and the mortality data used include California, as well as all other states. As regards trends in the UK, examination of mortality in Scotland shows little change during the 1970s for males or females aged 35–74 years, with a similar lack of change in England and Wales until a consistent downward trend after 1979 (Marmot, 1984). We cannot subdivide our data any further by age and have therefore used mortality for age-groups combined.

If there is, as we suggest, a downward trend in fat consumption in the UK from the mid to late 1970s, this will become clearer as later data are obtained. We will continue to collect this information and update the trends in a few years. Our opinion is that trends in fat derived in the past from food balance figures have been misleading and we sought to provide as accurate a picture as possible. It is clear that the trend in fat intake in the UK is very different from the US, as are the mortality data. Associations of this kind provide support for other types of evidence which show the effect of fat on risk factors for coronary heart disease. Many are convinced of a relationship between the two. However, it appears that in Britain eating habits are not the only characteristics which are resistant to change.

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Bioavailability of nutrients
Conceptual aspects of definition and problems of determination

In nutritional sciences there is some confusion about the term bioavailability. Sometimes it is used as a measure of a nutrient's property which allows the nutrient to be utilized by the organism. This is meant by the expression 'a nutrient has a certain bioavailability'. Using the term in this manner, one should realize that the physical and chemical properties of nutrients are only one factor of the pathway of many biochemical reactions and physico-chemical processes from enzymic digestion in the gut to metabolic utilization. These processes are dependent on each other and controlled by complex feedback and hormonal mechanisms. They also vary with individual requirements, age, sex etc. At any point of this pathway bioavailability may be affected, enhanced or reduced. So bioavailability is not merely a measure for a single nutrient property; it also refers to the ability of man and animals to make a nutrient available. Hence the definition of bioavailability should comprise more than just a single nutrient property; a more comprehensive definition is required.

Sometimes the term bioavailability has also been used for gastro-intestinal digestion or even *in vitro* digestion and intestinal absorption. This, too, is incorrect. Digestion and absorption are only steps on the pathway of processes by which nutrients are made

available. So it is obvious that by measuring only one step of the whole pathway, e.g. intestinal digestion, no reliable information on bioavailability is obtained. Even measuring absorption, by determining the increase in a nutrient's serum concentration, often does not yield reliable information. Metabolism of a nutrient before reaching the central blood circulation, homoeostatic regulation of the serum level, and elimination interfere with the nutrient's increasing serum level after absorption. So the increase in the nutrient's serum concentration need not reflect equivalently the amount of a nutrient absorbed. Therefore, bioavailability should not be confounded with digestion or absorption.

Now, what does bioavailability in fact mean?

To my mind, bioavailability is the measure of the ability of man and animals, or the effectivity, by which nutrients, in a given chemical form, are liberated from food in the presence of certain food components. Bioavailability moreover includes intestinal absorption and transport of nutrients to organs and cells, where they finally fulfil their physiological function.

As the definition of bioavailability is closely linked to its determination, the question arises of how bioavailability is measured. The most reliable way to do this is by measuring the nutrient's physiological effects at some endpoints of the pathway by which a nutrient is made available in relation to the nutrient's intake. This may be done in terms of the nutrient's metabolic rate, its concentration in target organs, cells, characteristic body fluids, or body pools or by determining the activity of enzymes involving special nutrients. So, when studying bioavailability it is advisable to measure not only one but several characteristic bio-markers of the nutrient's bioavailability. The researchers' dilemma is, however, that bio-markers of bioavailability which are easy to determine are frequently not very reliable. So in future bioavailability research it is of primary importance to select more, reliable and easily determinable bio-markers for each nutrient.

It has been shown that there are different aspects of the term bioavailability which are often mixed up and which cause confusion. When studying bioavailability of nutrients we should always realize what bioavailability really means, and whether the variables we measure in fact allow a reliable determination of bioavailability.

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