Correspondence

Dietary salt intake and epidemiological studies

SIR,—We have read with interest the recent article by Sonnenberg[1] suggesting that dietary intake of salt is a significant factor in mortality from gastric ulcer. In this article 'statistically significant linear correlations' have been reported to exist between geographic variations in salt consumption and death rates from gastric ulcer.

Further to his request, statistics relating to food grade salt were supplied in 1985 by trade associations such as the Salt Institute and the European Committee for the Study of Salt. As far as the latter is concerned, it explained to Sonnenberg that food grade salt consumption refers to a trade concept (sales) and not to a diet one (ingestion). It is not reasonable to calculate the amount of salt which is ingested by the population, by a mere division of sales to that population. The approach to such a problem by interpopulation studies is one of the worst in epidemiology and can therefore only be taken as pure speculation.

Instead of taking precautions for a comprehensive statistical approach, Sonnenberg extrapolates in a too simple way from salt sales to gastric ulcer. He considers that the available figures are showing a correlation between salt intake and mortality from gastric ulcer. We do not intend to dispute on the possible role of sodium chloride in the aetiology of gastric ulcer. We feel, however, that the article by Sonnenberg does not provide any firm proof for such a role. Much bias could have produced a false picture. How accurate is reporting of death from gastric ulcer from region to region? Moreover, many factors other than salt consumption are well known to influence rates of death from gastric ulcer such as smoking, alcohol use, type of blood group etc. Were these factors with their regional variations accounted for? In addition, as gastric ulcer is fortunately not a uniformly fatal disease, using death rate instead of prevalence is unwise. What Sonnenberg may have been looking at could be in fact the care of gastric ulcer or its variation in severity from one region to another, with its attendant mortality changing in a similar fashion.

For the sake of clarification, we feel it is advisable to comment in more detail on the statistical aspects of salt intake. Information about the average salt intake is needed in certain epidemiological studies. This information relates to three main sources: (1) salt naturally occurring in food stuffs (2–3 g/day); (2) salt added in food processing (4–5 g/day); and (3) salt added during cooking or at table (3–4 g/day). Within the average daily intake, individual intakes of salt vary to a considerable extent. The numbers reproduced in the Table constitute an attempt at giving an idea of the average daily intake of salt within the enlarged European Economic Community (EEC).[2]

Table *Average daily intake of salt, 1984 (in g/day)*

Belgium	9·9
Denmark	10·7
France	8·0
Germany	8·4
Italy	7·6
Netherlands	8·5
Portugal	11·4
Spain	10·3
United Kingdom	9·8
EEC	8·2

The above figures were calculated in 1985 by taking into consideration the following parameters: (1) food grade salt has a variety of non-food uses which represent an average of 50% of this type of salt in Western Europe. The most important deviation is private deicing; (2) a significant amount of salt is delivered to food industries and used for the production of goods which are to be exported. Besides, a lot of salt is used locally for water softening or for deicing purposes; (3) a certain proportion of salt used in food processing is discarded before cooking or in the cooking water. Epidemiological data (3) suggest that with British cooking methods only 24% of the salt added is actually ingested.

These estimates match very well with figures from other sources. Investigations into the content of household salt in food commodities in the Netherlands undertaken on behalf of the Ministry of Welfare, Public Health, and Culture have shown that the average daily consumption is 8·4 g NaCl in that country. Daily salt intake in Britain averages 10·7 g for adult men and 8·0 g for women. Calculations by Bull and Buss for 1978 showed that the total amount of salt entering the home amounted to 9·72 g.[3,4]

Sodium excretion in 24 hour urine collections does not indicate the source of dietary salt but characterises an individual's sodium intake. There have been few efforts in epidemiological studies to correlate the individual sodium intake level based on food consumption data with reliable urine samples.[5] When available, the results widely confirm the calculation based on food consumption data. Thus, Cottet concludes that salt intake averages 6–7 g/day in France.[6]

Figures for salt intake based on urinary sodium excretion are slightly lower than the ECSS's estimates or are similar. Discrepancies may be the result of methodological differences. It is usually assumed that about 90–95% of the ingested sodium appears in

the urine. On the other hand, some adjustments for deviated or wasted salt are lacking accuracy. In certain countries, it is no longer accepted that approximately 30% is the contribution of sodium naturally present in food. Recent studies have shown that this figure is probably around 15%.

The estimates of salt intake have been exaggerated in the past. These inaccurately high averages have paved the way to various programmes and recommendations for reducing salt consumption. A recent report on Healthy Nutrition[7] was commissioned by the WHO from a small group of medical nutritionists and prepared in 1985–86. It has been suggested in this report nutrient goals be adopted and these goals be translated into dietary guidelines by European governments.

The goals for salt intake are as follows: (1) intermediate nutrient goals, for the general population, 7–8 g/day, and for the cardiovascular high risk group 5 g/day; and (2) the ultimate nutrient goal, 5 g/day for everybody. Are these medical experts not aware of the average salt intake in Western Europe – that is, 8 g/day? On what evidence is the recommendation based that dietary salt intake should be reduced in the near future to a 'safe' level for sodium sensitive hypertensive patients?

The desirability of preventing diseases by modifying dietary habits is a generous goal. But, in the case of salt, recent epidemiologic investigations have failed to show a clear cut role of sodium in the prevalence of hypertension. Moreover, the blood pressure response to heavy oral salt intakes (exceeding 1000 mmol Na/day) by normal healthy volunteers over several days has been shown to be quite variable: one third of subjects had a decrease, one third had no change and only one third had an increase in arterial blood pressure.[8] Objective findings on salt as a significant factor in mortality from gastric ulcer are still missing.

A statistically inconsistent and basically subjective approach does not seem very helpful. When Sonnenberg supposes that gastric ulcer used to be a rare disease in Europe before the onset of the 19th century, he should be more cautious regarding the incidence of 'the peculiar history of salt consumption'. It is not true to emphasise that a high taxation level has restricted salt consumption in the past centuries. Before 1789, French people were obliged to buy a minimum of about 3·5 kg/head/year, notwithstanding the heavy taxes (gabelle) which were levied. Moreover, this quantity corresponded to cooking salt only. Food preservation required extra quantities.

No doubt that freezing as a means of preserving food instead of salt has led to a steady decline in salt consumption over the years. But it is adventurous to allege that 'this reduction in dietary consumption of salt may form the basis for the parallel decline in mortality from gastric ulcer'. Salt for food preservation or fish cure was generally discarded. This evolution explains a progressive decline in the sales of food grade salt. But it has no incidence on salt ingestion and on the so called salt related diseases.

Not all food grade salt is eaten. Only a certain proportion is used for food preparation and a small quantity is effectively ingested. According to Van der Veer, a large scale study done in Hungary[9] showed the following average data per individual: sold 11·0 g/day; used 8·3 g/day; ingested 3·4 g/day.

Any policy of salt reduction would depend at present on a biased selection of statistics and result in dogmatic advice for which there is no scientific evidence.

Physicians of international reputation in the field of hypertension are concerned with the way in which the salt issue is currently being handled. In a letter to the editor of the *Lancet*, [10] some of them underlined, in 1984, that dietary salt has some positive value and that it was dangerous to extrapolate, from limited studies, that the present intake of salt had to be reduced on a life long dietary basis for the whole population. It is essential that epidemiological studies, more especially from a statistical point of view, be properly conducted.

R L HANNEMAN
President
B MOINIER
Secretary General

Salt Institute,
206 North Washington St,
Alexandria, Virginia 22314,
USA, and
Comité Européen d'Etude du Sel,
11 bis avenue Victor-Hugo,
75116, Paris,
France.

References

1 Sonnenberg A. Dietary salt and gastric ulcer. *Gut* 1986; **26:** 1138–42.
2 Moinier BM. *Food grade salt intake: facts and figures.* ECSS np, 1986.
3 James WPT, *et al.* The dominance of salt in manufactured food. *Lancet* 1987; i: 426–9.
4 Bull NL, Buss DH. Contribution of food to sodium intakes. *Proc Nutr Soc* 1980; quoted in (3).
5 Pietinen T, Tuomilehto J. *Estimating sodium intake in epidemiological studies in epidemiology of arterial blood pressure.* The Hague: Martinus Nijhoff Publishers, 1980: 29–44.
6 Cottet Jean. *Evolution de la consommation de sel en France.*
7 James WPT. *Healthy Nutrition: preventing nutrition*

related diseases in Europe. Copenhagen: WHO/Nutr Unit, 1986: 5a.
8 Luft FC, Rankin LI, Block R, *et al.* Cardiovascular and humoral responses to extremes of sodium intake in normal black and white men. *Circulation* 1979; **60:** 697–706.
9 Van der Veer O. *The human intake of salt*. Wageningen: University of Agr./Dep. of Human Nutr, 1985: 30.
10 Brown JJ, Lever AF, Robertson JIS, *et al.* Salt and hypertension. *Lancet* 1984; ii: 456.

Reply

SIR,—There are three mistakes commonly made in trying to refute epidemiological findings.

1 Observations are rejected because they may be based on less than perfect statistics. Mortality data are claimed to represent severity of disease, success of treatment, diagnostic accuracy, and coding practices rather than frequency of occurrence. Similarly, it is argued that actual dietary intake of salt may comprise a too small fraction of total consumption of food grade salt for the latter to give a representative estimate of salt ingestion. It is assumed that inaccurate data have led to a linear regression that would not have been observed with more precise statistics. Because the true information – that is, the original signal, was overlaid by too much random changes – that is noise, a wrong message was created. This assumption, however, defies the laws of statistics. Noise destroys rather than creates messages. It cannot give rise to new significant correlations, but only blur or distort existing ones. If the underlying data are so poor as Hanneman and Moinier claim, better and more refined statistics can be expected to strengthen the association between gastric ulcer (GU) and salt.

2 A hypothesis cannot be refuted simply by hinting at other seemingly possible explanations. If other mechanisms are assumed to have confounded the original observation, these assumptions need to be confirmed and subjected to the same scrutiny as requested for the original hypothesis. (a) It is suggested that death rates do not represent prevalence data. No evidence is given for this contention. Actually, all epidemiological studies dealing with GU and duodenal ulcer (DU) suggest quite the opposite. Mortality and incidence data of peptic ulcer disease show the same epidemiological pattern regarding their age, sex, race, geographic, and temporal variation.[1,2] Even in rare diseases, such as inflammatory bowel diseases which occur 10 times less frequently than GU, a parallel behaviour of mortality and incidence is found.[3] (b) It is suggested that the geographic distribution of GU mortality represents differences in outcome of treatment rather than frequency of occurrence. It is somewhat difficult to imagine how United States physicians manage to be five or three times more successful in treating GU than physicians from Japan and the United Kingdom, respectively. Longterm trends of mortality from peptic ulcer and many other diseases have remained largely unaffected by medical advancement.[3–5] In comparison with the impact of hygiene, technologic innovation, and other environmental influences, the beneficial effect of new diagnostic or therapeutic procedures tends to be overestimated.[5] Considering the marked decline in mortality from gastric cancer, gastric ulcer, stroke, myocardial infarction, and other diseases related to hypertension, the refrigerator may have saved more lives than the *x*-ray tube. (c) If death rates represent varying severity of GU rather than prevalence or incidence, why does the geographic variation in severity of GU correlate with consumption of food grade salt? (d) Smoking appears to be a more important risk factor in DU than GU.[6,7] The geographic variation of smoking does not match that of GU or DU.[4,8,9] (e) Liver cirrhosis rather than alcohol consumption seems to be the relevant risk factor precipitating peptic ulceration. If anything, alcohol seems to increase rather than decrease mucosal defence.[6,7,10] No correlation is found between the geographic variation of liver cirrhosis and GU (unpublished observations). (f) Blood group O has been shown to increase the risk for DU by 1·4. No firm relationship between GU and any blood group has been established.[11]

One could try to concoct some sophisticated and involved hypothesis why GU correlates with salt, although salt may have nothing to do with GU. Unless some confirmation is given for the alternative hypothesis, however, it makes more sense to accept the present correlation at face value rather than disregard it for some unsubstantiated hypercritical attitude. In case of several competing explanations, the most simple and straightforward one seems the most probable. Here, it means that salt correlates with GU, because salt has really something to do with GU.

3 The most recent statistics do not necessarily represent the data best suited to answer epidemiological questions. The occurrence of GU has declined in all western countries. The decline was more marked in countries with a high incidence leading to an increasing similarity between different countries.[4,12] Similarly, salt consumption has declined in most countries, the decline being more marked in countries with an initially high consumption. As it is more difficult to show a correlation between two variables when both cover a narrow range, the most recent data may not allow the establishment of meaningful or significant correlation. On the average, GU patients tend to be 10–20 years older than