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

DU patients. This lag could be related to the time it takes to develop gastritis and subsequent GU. My paper deals with mortality from GU and stroke during 1971–1975.[13] The average daily intake of salt in 1984 is not very helpful in this regard and supplying data from 1951–1960 would have been far more revealing.

In their letter, Hanneman and Moinier concentrate on the correlation between GU mortality and consumption of food grade salt. They fail to account for the other arguments which have been raised in favour of salt being able to affect the gastric mucosa. At present this contention is based on four additional lines of evidence.

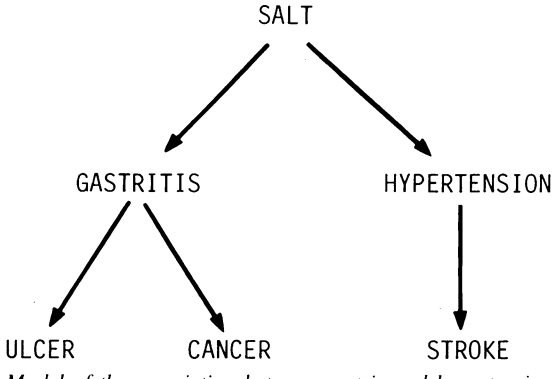
1 The occurrence of GU correlates with that of gastric cancer.[2 14] Countries with a high incidence of GU, such as Japan, Portugal, and Spain, have also a high incidence of gastric cancer. The geographic distribution of both diseases correlates with salt consumption as well as urinary salt excretion.[13 15–17]

2 Stroke represents a reliable epidemiologic marker for the occurrence of hypertension. Mortality from stroke correlates with mortality from GU and gastric cancer. This type of correlation concerns the geographic distribution of the three diseases,[13 18–20] their temporal behaviour,[13 20 21] and their occupational variation.[22 23]

3 It has been shown by multiple case control studies that salt consumption represents a risk factor for the development of gastric cancer.[16 24 25] At least one additional case control study has shown salt to be a risk factor also in GU.[26]

4 Gastric and hypertensive diseases are frequently diagnosed to coincide in the same subjects. Patients with GU or gastric cancer tend to have concomitant hypertensive diseases, such as ischaemic heart disease or cerebrovascular diseases, more often than people without gastric diseases.[27]

The hypothesis underlying the association of gastric and hypertensive diseases is shown in the figure. If Hanneman and Moinier wish to exonerate salt, they will need to supply alternative explanations not only for the relationship salt-GU, but also for all other relationships outlined in the figure – for example, salt-gastric cancer, stroke-GU, stroke-gastric cancer, etc.



Model of the association between gastric and hypertensive diseases.

AMNON SONNENBERG

VA Medical Center,
Gastroenterology Section,
5000 W National Avenue,
Milwaukee, WI 53295, USA

References

1 Sonnenberg A. Geographic and temporal variations in the occurrence of peptic ulcer disease. *Scand J Gastroenterol* 1985; **20:** suppl 110: 11–24.
2 Sonnenberg A, SenGupta A, Bauerfeind P. Epidemiology of peptic ulcer disease. In: Rees WDW, ed. *Advances in peptic ulcer pathogenesis.* Lancaster: MTP Press, 1988: 1–31.
3 Sonnenberg A. Geographic variation in the incidence of and mortality from inflammatory bowel disease. *Dis Col Rectum* 1986; **29:** 854–61.
4 Sonnenberg A, Møller H, Pace F. Birth-cohort analysis of peptic ulcer mortality in Europe. *J Chron Dis* 1985; **38:** 309–17.
5 McKeown T. *The role of medicine: dream, mirage or nemesis.* London: Nuffield Provincial Hospital Trust, 1976.
6 Ostensen H, Gudmunsen TE, Ostensen M, Burhol PG, Bonnevie O. Smoking, alcohol, coffee, and familial factors: any association with peptic ulcer disease? A clinically and radiologically prospective study. *Scand J Gastroenterol* 1985; **20:** 1227–35.
7 McIntosh JH, Byth K, Piper DW. Environmental factors in aetiology of chronic gastric ulcer: a case control study of exposure variables before the first symptoms. *Gut* 1985; **26:** 789–98.
8 Lee PN, Wilson MJ. *Tobacco consumption in various countries.* Research paper 6, 4th ed. London: Tobacco Research Council, 1975.
9 Sonnenberg A. Smoking and mortality from peptic ulcer in the United Kingdom. *Gut* 1986; **27:** 1369–72.
10 Sonnenberg A, Møller-Lissner SA, Vogel E, *et al.* Predictors of duodenal ulcer healing and relapse. *Gastroenterology* 1981; **81:** 1061–7.
11 McConnell RB. *The genetics of gastro-intestinal disorders.* London: Oxford University Press, 1966: 76–111.
12 Sonnenberg A, Møller H. Cohort and period effects in peptic ulcer mortality from Japan. *J Chron Dis* 1984; **37:** 699–704.
13 Sonnenberg A. Dietary salt and gastric ulcer. *Gut* 1986; **27:** 1138–42.
14 Segi M, Fujisaku S, Kurihara M. Mortality for gastric and duodenal ulcer in countries and its geographical correlation to mortality for gastric and intestinal cancer. *Schweiz Z Path Bakt* 1959; **22:** 777–84.
15 Joossens JV, Geboers J. Nutrition and gastric cancer. *Nutr Cancer* 1981; **2:** 250–61.

16 Hirayama T. A study of epidemiology of stomach cancer, with special reference to the effect of the diet factor. *Bull Inst Publ Health* 1963; **12:** 85–96.
17 Sato T, Fukuyama T, Suzuki T, *et al.* Studies of the causation of gastric cancer. 2. The relation between gastric cancer mortality rate and salted food intake in several places in Japan. *Bull Inst Publ Health* 1959; **8:** 187–98.
18 Whelton PK, Goldblatt P. An investigation of the relationship between stomach cancer and cerebrovascular disease. *Am J Epidemiol* 1982; **115:** 418–27.
19 Joossens JV. Dietary salt restriction – The case in favour. In: Robertson JIS, Pickering GW, Caldwell ADS, eds. *The therapeutics of hypertension.* Royal Society of Medicine Series No 26. London: Academic Press 1980: 243–50.
20 Joossens JV. Stroke, stomach cancer and salt. In: Kesteloot H, Joossens JV, eds. *Epidemiology of arterial blood pressure.* The Hague: Martinus Nijhoff Publishers, 1980: 489–508.
21 Tuomilehto J, Geboers J, Joossens JV, Salonen JT, Tanskanen T. Trends in stomach cancer and stroke in Finland. Comparison to Northwest Europe and USA. *Stroke* 1984; **15:** 823–8.
22 Public Health Service, Guralnick L. *Mortality by occupation level and cause of death among men 20 to 64 years of age: United States, 1950.* US Department of Health, Education, and Welfare. *Vital Statistics Special Reports*, vol 53, no. 5. Washington DC: US Government Printing Office, 1963.
23 The Registrar General. *The Registrar General's Decennial Supplement England and Wales 1961. Occupational Mortality Tables.* London: Her Majesty's Stationery Office, 1971.
24 Haenszel W, Kurihara M, Segi M, Lee RKC. Stomach cancer among Japanese in Hawaii. *J Natl Cancer Inst* 1972; **49:** 969–88.
25 Howson CP, Hiyama T, Wynder EL. The decline in gastric cancer: epidemiology of an unplanned triumph. *Epidemiol Rev* 1986; **8:** 1–27.
26 Stemmermann G, Haenszel W, Locke F. Epidemiologic pathology of gastric ulcer and gastric carcinoma among Japanese in Hawaii. *J Natl Cancer Inst* 1977; **58:** 13–9.
27 Sonnenberg A. Gastric cancer, gastric ulcer, and hypertensive diseases – A common epidemiologic risk factor? [Abstract] *Gastroenterology* 1987; **92:** 1649.

Psychological factors in the irritable bowel syndrome

SIR,—We were sorry to read in the Progress Report by Creed and Guthrie,[1] the statement that we had used the Beck Inventory wrongly for screening depression in surgical outpatients, taking a cut off point for depression of 5 instead of 14.[2]

In fact we used a simplified form of the Beck Depression Index, for which the range of normality is 0–4. 5–7 indicating mild, 8–15 moderate and over 15 severe depression. Had Creed and Guthrie read our article more carefully, they would have found that we correctly used the criteria of Beck and Beck,[3] and our finding of a 50% incidence of depression in gastrointestinal outpatients, and 68% in the irritable bowel syndrome, remains valid. They would also have learnt that the majority of patients were medical, and not surgical, outpatients.

PAUL M SMITH AND JOHN S HARVEY

Llandough Hospital,
Penarth,
South Glamorgan CF6 1XX

References

1 Creed F, Guthrie E. Psychological factors in the irritable bowel syndrome. *Gut* 1987; **28:** 1307–18.
2 Rose JDR, Troughton AH, Harvey JS, Smith PM. Depression and functional bowel disorders in gastrointestinal outpatients. *Gut* 1986; **27:** 1025–8.
3 Beck AT, Beck RW. Screening depressed patients in family practice: a rapid technic. *Postgrad Med* 1972; **52:** 81–5.

Reply

SIR,—Smith and Harvey are right to draw attention to the fact that they had used a shortened form of the Di-Beck questionnaire so that our criticism of their specific cutoff point does not hold; but we would still reject their claim that they have accurately demonstrated rates of depression of 50% and 68% in gastrointestinal and IBS patients respectively.

In their paper Rose *et al*[1] aimed 'to establish the number of patients suffering from depression' among new referrals to a gastrointestinal clinic. There are several reasons why the shortened Di-Beck questionnaire did not allow them to do this accurately. First, it is not a diagnostic tool but a measure of severity of depression.[2] Second, any selfadministered questionnaire cannot be used as a substitute for clinical assessment and should therefore be validated by a use of standardised interview.[3] We are not aware of any such validation of the shortened Di-Beck against clinical interviews among gastrointestinal outpatients, but there is evidence that 14 of the 21 items of the original questionnaire discriminated poorly between depressed and non-depressed patients in a general medical unit[4] and many of these items have been included in the shortened form.

Selfadministered questionnaires tend to overestimate the prevalence of depression in general medical patients because somatic symptoms and social dysfunction score on the questionnaire even when these are not the result of depressive illness.[5] In the case of the shortened Di-Beck, a score of 7 could be achieved by the patient who reports 'my appetite is much worse now' (2 points), 'I get too tired to do anything' (3 points), and 'I have to push myself hard to do anything' (2 points). Such complaints could be attributable to physical illness, and are common