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Dietary salt and gastric ulcer

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SUMMARY Statistically significant linear correlations between geographic variations in salt consumption and mortality from gastric, but not duodenal ulcer, are reported. It is suggested that dietary consumption of salt is a risk factor in mortality from gastric ulcer.

Although it has been speculated that dietary factors play a role in the aetiology of gastric ulcer disease, firm evidence to this effect is lacking.[1] Japan is known to have a high incidence of gastric ulcer disease,[2 3] and a conspicuous fact about Japanese diet is its high content of salted food.[4] Furthermore, salt has been found to induce gastritis in animals.[5] In the present paper, the possibility was examined that geographic variations in mortality from gastric ulcer might be related to those of salt intake. Because mortality from cerebrovascular diseases is known to be related to hypertension and salt consumption, additional evidence for a possible link between salt consumption and gastric ulceration was sought by comparing the geographic and temporal variations of mortality from gastric ulcer and cerebrovascular diseases.

Methods

SALT CONSUMPTION

Statistics concerning the consumption of food grade salt from different countries were supplied by the Verein Deutsche Salzindustrie in Bonn, FRG, the Comité Européen d'Étude du Sel in Paris, France, and the Salt Institute in Alexandria, Virginia, USA. The figures shown in the Table for New Zealand, Canada, the United States, and Japan pertain to the period of 1978–80, while figures for the other countries relate to the period of 1971–1975. The average annual consumption of each country was divided by the average population for the same period to calculate the per capita consumption. Food grade salt makes up only a small fraction (5–8%) of total salt production,[6 7] and most of it is used in food processing. Much is lost during the process of food preparation, due to food degradation, or remains in the brine and does not enter the human body. Therefore, the per capita consumption calculated from the total consumption of table salt exceeds the actual dietary load by a factor of 2–3.

To validate the correlations obtained from the data of total national consumption of food grade salt, additional sources of salt consumption were sought. Despite its limitations, the 24 hour measurement of urinary sodium output over several days is presently considered to be the most reliable and reproducible criterion, but for political reasons it is not applicable to large populations. For the present analysis the results of different studies compiled by Joossens *et al* supplemented by two other reports have been used.[4 8 9]

The mortality from cerebrovascular diseases, gastric and duodenal ulcer was analysed for countries where either the consumption of food grade salt or the urinary excretion of sodium chloride was known (Table). The mortality data from these 19 countries were made available by the National Departments of Health or the National Statistical Offices. The terms 'stroke' or 'cerebrovascular diseases' were used for the sum of all deaths from cerebrovascular diseases grouped under the ICD codes 430–438 in the detailed list of the 8th revision. The average age-specific death rates of the period 1971–1975 were calculated for each country, and in case of New Zealand, Canada, United States, and Japan also the averages of 1978–80. The death rates were adjusted to the age distribution of the population of England and Wales in 1971 by the method of direct standardisation.[10]

For the analysis of the temporal changes of cerebrovascular diseases, gastric, and duodenal ulcer, the Vital Statistics of the United States from 1921 to 1980 were used.[11–13] All deaths resulting

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Table 1 *Salt intake and death rates from cerebrovascular diseases (CVD), gastric (GU) and duodenal ulcer (DU)*

Country		*Average population 1971–5 (,000)*	*CVD*	*GU*	*DU*	*Consump. of NaCl (10^3 tons)*	*Urinary NaCl (g/day)*
United Kingdom	(UK)	55120	168	3·5	1·0	300	—
Scotland	(SCO)	5212	212	3·0	5·0	—	16·0
England	(ENG)	48378	162	3·6	4·0	—	9·9
Finland	(FIN)	4629	165	3·6	1·5	—	12·2
Denmark	(DEN)	5016	103	4·8	2·6	62	—
Netherlands	(NET)	13435	119	3·4	2·0	89	—
Belgium	(BEL)	9756	164	4·7	0·8	127	12·8
France	(FRA)	51224	145	3·7	0·7	369	—
West Germany	(GER)	61791	178	4·9	2·2	347	12·2
Austria	(AUS)	7511	188	4·8	4·4	43	—
Switzerland	(SWI)	6299	121	3·7	2·7	60	—
Spain	(SPA)	34780	188	5·7	2·2	503	—
Portugal*	(POR)	9506	330	7·0	4·4	198	18·0
Greece	(GRE)	8931	161	3·1	1·7	45	—
Italy	(ITA)	54207	161	3·6	4·5	380	—
Australia	(AUL)	13137	188	3·2	3·1	—	10·2
New Zealand†	(NEW)	3128	140	2·6	3·4	30	10·6
Canada†	(CAN)	23697	89	1·6	1·8	106	—
USA†	(USA)	224403	89	1·2	1·2	965	9·7
Japan	(JAP)	115431	212	6·0	1·1	1431	18·0

Death rates given as annual average per 100 000 living population of the period 1971–1975.
*Death rates of gastric and duodenal ulcer refer to the average of 1966–1970.
†Salt consumption and death rates refer to the average of 1978–1980.

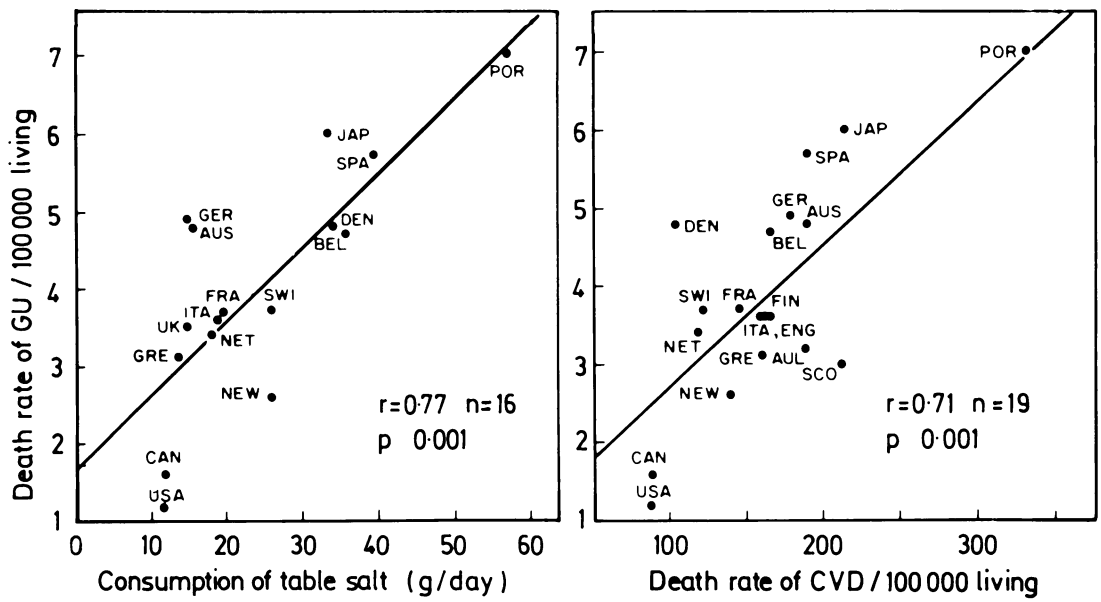

Fig. 1 *Correlations of death rates from gastric ulcer (GU) with average daily consumption of table salt and death rates from cerebrovascular diseases (CVD).*

from any type of cerebrovascular disease were grouped together. The age- and sex-specific numbers of deaths for each year were related to 100 000 male or female US residents of the same age groups and years. The rates for each year were adjusted to the age distribution of the American population of the census in 1980.

The correlations between the geographic and temporal changes in salt consumption and the death rates from cerebrovascular diseases, gastric and

duodenal ulcer were tested by least squares linear regression analyses.[14]

Results

Figure 1 shows a significant linear correlation between the consumption of food grade salt and mortality from gastric ulcer among different countries. The data in the Table show no such significant correlation between the mortality from duodenal ulcer and the consumption of food grade salt ($r=0{\cdot}27$, $n=16$, $p>0{\cdot}05$). Mortality from gastric ulcer, but not duodenal ulcer, is significantly correlated to mortality from cerebrovascular diseases (Fig. 1). A significant correlation is also found between the mortality of gastric ulcer and urinary excretion of sodium chloride from 10 countries (Fig. 2). Again, there is no significant correlation between duodenal ulcer and urinary sodium chloride ($r=0{\cdot}12$, $n=10$, $p>0{\cdot}05$). Calculation of the death rates either for all ages or for persons only between 45 and 75+ years of age leaves the results of the regression analyses unaffected. The correlations remain significant when tested for men and women separately.

In the United States, the female death rates from cerebrovascular diseases and gastric ulcer run parallel since 1921 ($r=0{\cdot}92$, $n=60$, $p<0{\cdot}001$). In men, there is a rise in the mortality from gastric ulcer until 1940; after the initial rise, the curves of gastric ulcer and cerebrovascular disease run parallel ($r=0{\cdot}75$, $n=60$, $p<0{\cdot}001$). All curves show a smooth peak between 1956 and 1965 (Fig. 3). The parallel time course does not concern the mortalities from duodenal ulcer or other benign gastrointestinal diseases. The same kind of similarity in the secular trends of cerebrovascular diseases and gastric ulcer can also be demonstrated for European countries and Japan.

Discussion

The present analysis shows that there is a significant linear correlation between salt consumption or

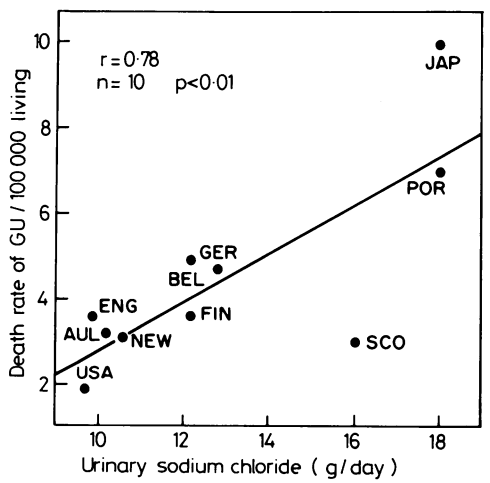

Fig. 2 *Correlation of death rates from gastric ulcer (GU) with urinary excretion of sodium chloride. Death rates correspond to the averages of 1971–75.*

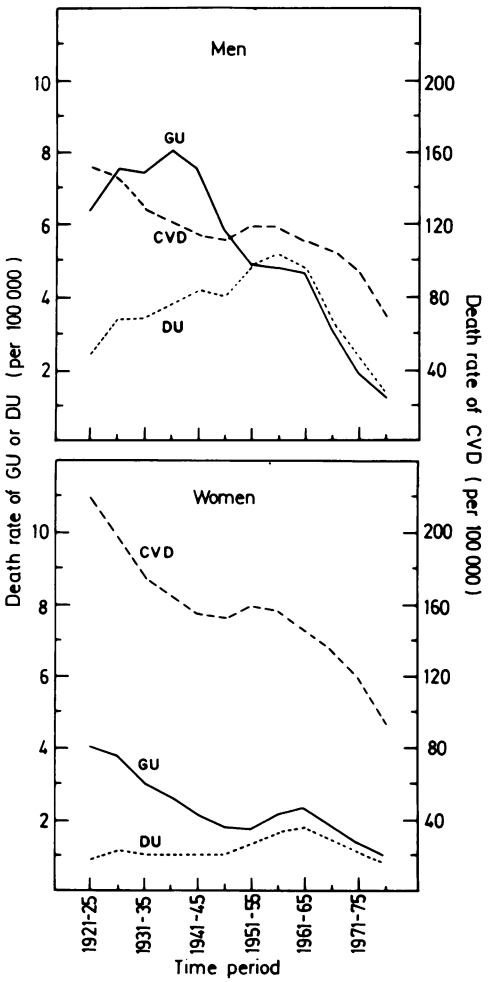

Fig. 3 *Comparison of temporal changes in death rates of gastric ulcer (GU), duodenal ulcer (DU), and cerebrovascular disease (CVD) in the United States from 1921 to 1980. Each point is the average of a five-year period.*

urinary sodium chloride excretion and death rates from gastric ulcer in several countries. A validation of this observation is obtained by a significant linear correlation between mortality from gastric ulcer and cerebrovascular diseases. There are additional data suggesting that the occurrence of gastric ulcer may be linked to the amount of dietary salt consumption. One piece of direct evidence is provided by a study by Stemmerman *et al* who analysed the risk of gastric ulcer among Japanese from Hawaii in a case control study.[15] The authors found a strong association between salt intake, gastric ulcer, and gastric metaplasia in 133 patients with gastric ulcer as compared with 244 controls. Also, salt was shown to induce gastritis of experimental animals.[5] The epidemiologic relationship between mortality from gastric cancer and cerebrovascular diseases gives further evidence to the contention that salt consumption can affect the gastric mucosa.[16 17] The geographic and temporal variations in the amount of urinary salt excretion are significantly correlated to those of mortality from gastric cancer,[8 16 18] and within countries like Japan and England, regions with a high mortality from gastric cancer also show conspicuously high mortality from cerebrovascular diseases.[5 18] During the last 20–30 years mortality from both diseases has declined in a strikingly parallel manner in most countries.[8 16 19]

The presently described relationship between the occurrence of gastric ulcer and salt consumption may be important in our understanding of the aetiology of gastric ulcer disease. The geographic distribution of gastric ulcer between Western countries and India and Africa,[20 21] for instance, might be related to different grades of salt consumption. The marked temporal variations displayed by the incidence of gastric ulcer in Europe could have been caused by the peculiar history of salt consumption. Gastric ulcer used to be a rare disease in Europe before the onset of the 19th century.[22] The incidence of gastric ulcer rose steadily during the 19th century and reached a peak in the generation born at the turn of the 20th century.[23 24] During the past decades the frequency of gastric ulcer has again declined.[24–26] This rise and fall of gastric ulcer is paralleled by a rise and fall in the dietary salt consumption. In Europe, salt was a precious and highly taxed commodity throughout the Middle Ages and the Renaissance.[7 27] Only after the French revolution were the high state taxes on salt abolished or markedly reduced, and only since then did salt become available to all sections of the population. Since the First World War, cooling as a means of preserving food has begun to replace salting, and this has led to a steady decline in salt consumption over the years.[18] This reduction in dietary consumption of salt may form the basis for the parallel decline in mortality from gastric ulcer and cerebrovascular diseases as shown in Figure 3.

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