

Salt intake and *Helicobacter pylori* infection

D. Gareth Beevers, Gregory Y.H. Lip and Andrew D. Blann

Background The chance finding of an association between *Helicobacter pylori* infection and hypertension and the known relationship between salt intake and blood pressure led us to speculate whether these two environmental factors might be related. A positive relationship between them might suggest that a high-salt diet could in some way facilitate gastric *H. pylori* infection.

Methods We have therefore conducted an ecological study of the national *H. pylori* infection rates in the EUROGAST study and national salt excretion levels with the INTERSALT project. Ten nations were included in both projects.

Results Statistically significant correlation between *H. pylori* infection rates and salt excretion were found in older men and women ($r = 0.728$ and $r = 0.827$, respectively) and in younger men ($r = 0.728$), but not younger women ($r = 0.519$).

Conclusions Our findings raise the possibility that salt

intake may in some way facilitate *H. pylori* infection. A dedicated population survey is now justifiable to investigate the role of salt intake in *H. pylori* infection with the measurement of all possible confounding variables including particularly socio-economic status *J Hypertens* 22:1475–1477 © 2004 Lippincott Williams & Wilkins.

Journal of Hypertension 2004, 22:1475–1477

Keywords: salt intake, *Helicobacter pylori* infection

University Department of Medicine, City Hospital, Birmingham, UK.

Correspondence and requests for reprints to Prof. D. G. Beevers, University of Birmingham, Department of Medicine, City Hospital, Birmingham B18 7QH, UK. Tel: +44 (0) 121 507 5086; fax: +44 (0) 121 554 4083; e-mail: gareth.beevers@swbh.nhs.uk

Received 15 March 2004 Revised 4 May 2004
Accepted 4 May 2004

See editorial commentary page 1459

Introduction

Both a high-salt diet and *Helicobacter pylori* infection are associated with hypertension [1–3]. In the case of salt, the association is almost certainly causal. The association of *H. pylori* infection with hypertension is less likely to be causal and may be explained by other factors including low socio-economic status and residential overcrowding. Furthermore there does not seem to be a plausible mechanism whereby *H. pylori* might raise blood pressure. Both environmental factors have, however, also been implicated in the development of stomach cancer. We hypothesized therefore that a high salt intake might in some way facilitate *H. pylori* infection. If this is so, then *H. pylori* infection and salt intake might be closely correlated. We therefore conducted an ecological survey to study the international league tables for *H. pylori* infection and for salt intake.

Methods

Data on the international league tables for *H. pylori* positivity were available from the EUROGAST project [4]. This was an investigation of 17 populations in 13 countries in Europe, the USA and Japan, where random samples of 150–200 men and women aged 25–34 years and 55–64 years were examined. Serum was collected for analysis of *H. pylori* IgG antibodies by an enzyme-linked immunoassay. All assays were performed in a

single laboratory. In all populations the prevalence of *H. pylori* positivity was higher in the older age groups but there was no difference between men and women.

The international data on dietary sodium consumption were obtained from the INTERSALT project [1]. This was a 52-centre study conducted in 32 nations of 24-h urine electrolyte excretion (an index of salt consumption) and blood pressure. Data were collected in 1986 and 1987 in age-stratified and sex-stratified random samples of 200 participants per centre. All urinary electrolyte assays were performed in a single centre laboratory. While there was no relationship between salt intake and age, there was a marked gender difference with women consuming less salt.

Ten nations were represented in both the EUROGAST and INTERSALT projects. These were Belgium, Denmark, West Germany, Iceland, Italy, Poland, Portugal, the United Kingdom, the United States and Japan. In both surveys where more than one population was investigated within an individual nation, the data from the populations were averaged. In order to minimize the possible confounding effects of differences in ethnic origin, the USA data from Hawaii and the two African-American centres in INTERSALT were not included. As no Northern Ireland centre participated in

EUROGAST, the INTERSALT data from Belfast were not included so the UK data are confined to England and Wales. Nine of the populations studied were almost exclusively of European origin participants. The population samples from Japan were included but all statistical analyses were performed with and without the Japanese data.

In view of the marked differences in salt intake between men and women, the two sexes were considered separately. Because of the gradients of *H. pylori* positivity with age, the analyses were performed in younger and older men and women separately (INTERsalt 20–29 years with EUROGAST 25–34 years, and INTERsalt 50–59 years with EUROGAST 55–64 years).

Results

Pooling the data from EUROGAST and INTERSALT, statistically significant correlations were found between 24-h urine sodium excretion and *H. pylori* positivity in men in both age bands and in older women (younger men, $r = 0.728$, $P = 0.017$; older men, $r = 0.728$, $P = 0.017$; younger women, $r = 0.519$, $P = 0.124$; older women, $r = 0.827$, $P = 0.003$) (Fig. 1). Weaker inverse correlations were found between urine potassium excretion and *H. pylori* positivity and between urine nitrate excretion and *H. pylori*. All correlations that

were significant remained so when the data from Japan (the only non-European-origin population) were excluded from the analyses.

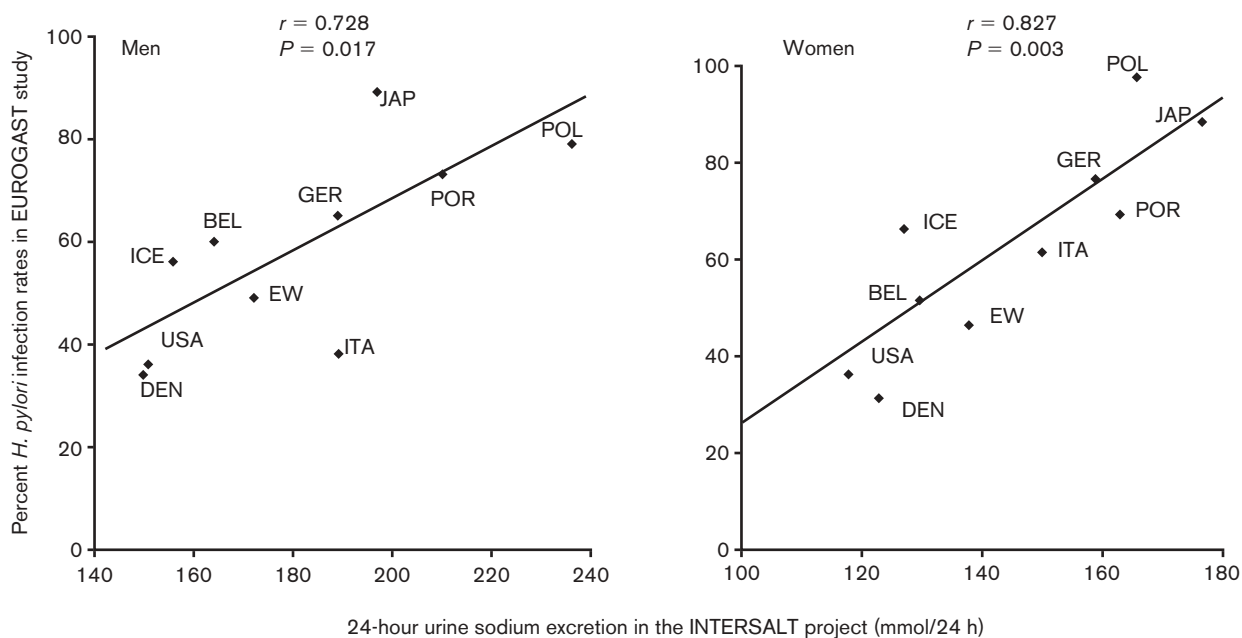
Discussion

Great care must be taken when interpreting these data. The EUROGAST and INTERSALT surveys were done at different times, in different populations and with different criteria for population sampling. We have attempted to minimize these possible sources of error by stratifying for age and gender and, in the case of the USA, by including only predominantly European origin populations.

Using Medline searches of the world literature we were unable to find any papers investigating the relationship between salt intake or excretion *H. pylori* HP infection or a pooling of the EUROGAST and INTERSALT projects. However, we were able to identify a reference to an association in a Ph.D. thesis by Dr P. Packer submitted in 1989, 4 years before the publication of the EUROGAST results. This thesis was unfortunately not available to us but is mentioned in the paper by Joosens *et al.* published in 1996 [5].

Despite many possible sources of error there were remarkably close correlations between sodium intake/excretion and *H. pylori* infection, with high coefficients

Fig. 1



Correlations between 24-h urine sodium excretion in the INTERSALT project and percent *Helicobacter pylori* infection rates in the EUROGAST project in older men and women in the 10 nations included in both studies. DEN, Denmark; BEL, Belgium; ICE, Iceland; ITA, Italy; GER, West Germany; POL, Poland; POR, Portugal; JAP, Japan; EW, England and Wales; USA, United States of America.

of correlation. The force of the association is closer than is usually found in epidemiological research.

It is not within the scope of this study to speculate on the mechanisms of the association between salt intake and *H. pylori* infection. A high-salt diet might irritate the gastric mucosa and allow *H. pylori* to enter the body [6]. The reverse mechanism is implausible; that *H. pylori* infection somehow causes people to eat more salt and less potassium-rich fruit and vegetables.

Another possibility is that a high salt intake and *H. pylori* infection are simply markers of low socio-economic status and thus are not causally related. People of low socio-economic status tend to have higher blood pressure and body weight, possibly related to a high dietary intake of convenience foods, packet and canned foods and salted snacks. Thus salt intake is higher in poorer people. Similarly, *H. pylori* infection is commoner in people with low socio-economic status, assessed by duration of education. There is also a slight trend for *H. pylori* infection to be commoner in people with a high body mass index and in older people [7]. However, the inclusion of data from Japan, which now has a prosperous economy and low unemployment rates, makes this explanation less likely as salt intake and *H. pylori* infection rates are both high in that country. Within Japan, an association has been reported between the intake of salty foods and the risk of *H. pylori* infection [8].

This ecological study was an exercise in hypothesis generation and does not have the capacity to adjust for many confounding variables. In view of our findings, however, further research needs to be conducted along epidemiological lines. Within a single well-defined population, *H. pylori* infection rates should be examined, together with the measurement of 24-h urine electrolyte excretion and a detailed structured salt intake questionnaire. All possible confounding variables should be measured, including socio-economic status, duration of education and occupation, blood pressure and body mass index. Patients undergoing *H. pylori* testing or endoscopy for peptic symptoms would be unsuitable for such a study as they may have modified their diet in response to their symptoms. Furthermore, many antacids contain significant amounts of sodium.

There are important public health implications from our findings. If a high salt intake actually encourages or permits *H. pylori* infection, then a reduction in salt intake might reduce infection rates and lead to stomach cancer prevention. Whereas *H. pylori* eradication requires mass screening and costly antibiotic therapy, a modest reduction of salt intake of whole populations, achieved by cutting the salt content of processed foods, might, at no expense, achieve as much or even more

improvement in the nations health, with added benefits of hypertension and stroke prevention.

Conflict of interest

No funds were required for this study. Prof. Beevers is an advisor for Consensus Action on Salt and Health (CASH) and was a local principal investigator in the INTERSALT project.

Acknowledgement

The authors are grateful to Prof. I. J. Perry (Cork) and Prof. K. K. Cheng (Birmingham) for technical and scientific advice. They also thank Ms Ruby Stone for secretarial assistance.

References

- 1 INTERSALT Cooperative Research Group. Intersalt: an international study of electrolyte excretion and blood pressure. Results for 24 hour urinary sodium and potassium excretion. *Br Med J* 1998; **297**:319–328.
- 2 Barnes RJ, Uff JS, Dent JC, Gear MWL, Wilkinson SP. Long-term follow up of patients with gastritis associated with *Helicobacter pylori* infection. *Br J Gen Pract* 1991; **41**:286–288.
- 3 Lip GYH, Wise R, Beevers DG. Association of *Helicobacter pylori* infection with coronary heart disease. *Br Med J* 1996; **312**:250–251.
- 4 The EUROGAST Study group. An international association between *Helicobacter pylori* infection and gastric cancer. *Lancet* 1993; **341**: 1359–1362.
- 5 Joossens JV, Hill MJ, Elliott P, Stamler J, Lesaffre E, Dyer A, et al., on behalf of European Cancer Prevention (ECP) and the INTERSALT cooperative Research Group. Dietary salt, nitrate and stomach cancer mortality in 24 countries. *Int J Epidemiol* 1996; **25**:494–504.
- 6 De Koster E, Buset M, Fernandez E, Deltenre M. *Helicobacter pylori*: link with gastric cancer. *Eur J Cancer Prevent* 1994; **3**:247–257.
- 7 The EUROGAST study Group. Epidemiology of, and risk factors for, *Helicobacter pylori* infection amongst 3194 asymptomatic subjects in 17 populations. *Gut* 1993; **34**:1672–1676
- 8 Tsugane S, Tei Y, Takahashi T, Watanabe S, Sugano K. Salty food intake and the risk of *Helicobacter pylori* infection. *Jpn J Cancer Res* 1994; **85**:474–478.