



Bimodal distribution of fasting gastric acidity in a rural African population

A M Sammon, M Mguni, L Mapele, K O Awotedu, J E Iputo

Setting. The people of Transkei eat a diet high in linoleic acid, the principal fatty acid in maize. The theory has been put forward that a diet high in linoleic acid and low in fat and riboflavin, such as the traditional diet in Transkei, results in overproduction of prostaglandin E₂ in the gastric mucosa, and that this overproduction in turn causes a suppression of gastric acid production.

Objective. To investigate the effect of diet on fasting gastric pH in a rural black African population.

Design. Fasting gastric acid samples were obtained by fine nasogastric tube aspiration from 150 volunteers at a rural health clinic. The pH of these samples was measured and a full dietary questionnaire was used. *Helicobacter pylori*

serology was done on a subgroup of 30 volunteers.

Results and conclusions. A bimodal pH distribution was found. Approximately half the population had a gastric pH within the range 1 - 4. Half had a pH of over 4. A high pH was significantly associated with consumption of maize ($p = 0.006$), and with consumption of both pumpkin and beans ($p = 0.006$).

A high proportion of this rural African population has a diet-associated abnormally high gastric pH. The pattern of upper gastrointestinal disease may be significantly affected by diet in this community and in others with a similar diet.

S Afr Med J 2003; 93: 786-788.

The rural people of Transkei, South Africa, eat a diet high in maize, pumpkin and beans, and low in other dietary constituents. In this community, with one of the highest incidences of oesophageal cancer in the world,¹ diet-associated high gastric pH has been postulated as a major factor.² The diet is based on maize, and a significant proportion of the population have little added fat or protein. This diet is therefore high in linoleic acid, low in other fatty acids, and low in riboflavin. It has been suggested² that these dietary elements promote high prostaglandin E₂ (PGE₂) production in the stomach; PGE₂ suppresses gastric acid production and raises the intragastric pH.

This study examined the relation between diet and gastric luminal pH in a rural Transkeian population.

Methods

Ethical permission for the study was obtained from the Research Committee of the University of Transkei. Volunteers were recruited from patients attending a rural health clinic and informed consent was obtained. Patients who were smokers, who had symptoms of upper gastrointestinal disease, or who were receiving non-steroidal anti-inflammatory drugs were

excluded from the study.

A food frequency questionnaire was used which included 23 items identified as the most frequently eaten foods in rural Transkei (Table I). These items fell into five broad categories: maize-based foods, animal products, fat and oil, fruit, and vegetables. Consumption of each food item was defined according to frequency of intake: 2 - 3 times per week, once a week, once a month, once a year, or never. *Amarewu* is a drink of fermented maize meal. *Holsum* is a proprietary solid cooking fat. Known as *umsobo* in Transkei, *Solanum nigrum* is used as a wild vegetable. Portion size was not estimated for each item.

Fasting gastric juice was obtained from each volunteer using a fine-bore nasogastric tube. The fluid obtained was snap frozen, transported in liquid nitrogen, and stored at -40°C. pH was measured by glass electrode. Serum for *Helicobacter pylori* antibodies were obtained by venepuncture, and the Novum *H. pylori* immunoglobulin A (IgA) enzyme-linked immunosorbent assay (ELISA) kit (Novum Diagnostica, Dietzenbach, Germany) was used. This kit is a microtitre enzyme immunoassay system that uses recombinant antigens for the detection of IgA class antibodies to *H. pylori* in human serum. The relationships of individual dietary components and pH were analysed by means of Spearman's rank correlation, using SPSS. The median score for frequency of consumption of each food group was calculated. This score was used to divide subjects into high and low consumption subgroups for correlation analysis. For example, subjects who consumed 10 or more fruit per month were compared with those who consumed less than 10 per month for correlation with pH.



Results (Table I)

One hundred and fifty volunteers took part in the study. Maize-based foods were the main source of calories for most of the volunteers. Soft maize porridge was consumed daily by 49% of volunteers, stiff maize porridge by 27%, and stamped maize by 37%. Bread was eaten daily by 45%, and beans by 29%. Some cow's milk was consumed daily by 45%, but no other animal fat or protein source was a usual part of the daily diet. Vegetables and fruit were eaten reasonably commonly, although only 18% ate cabbage daily, and 7% an apple daily. No individual food consumed had a significant association with high gastric pH. In the subgroup of 30 volunteers who had *H. pylori* serology carried out, 15 were positive. There was a statistically non-significant trend for serum positivity to be associated with a low pH. Age and gender showed no significant association with pH. pH showed a bimodal distribution, with peaks at pH 2 and 7 (Fig. 1).

High pH was significantly associated with high frequency of maize consumption, high frequency of pumpkin and bean consumption, and high frequency of maize, pumpkin and bean consumption, each at a significance of $p = 0.006$ (Table II).

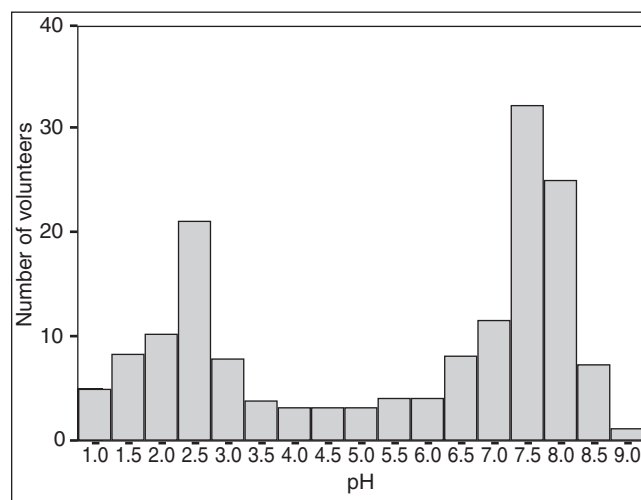


Fig 1. pH distribution in 150 rural volunteers.

Discussion

Quantity of each item consumed was not taken into account. The study assumed similar size food portions for all subjects.

Table I. Frequency of consumption of individual food items and relation to pH

Item	Consumed daily (%)	Consumed at least weekly (%)	Odds ratio for pH > 4 (food frequency above median)	95% confidence intervals
Apples	7	45	1.18	0.53, 2.61
Bananas	4	37	1.57	0.66, 3.72
Oranges	7	52	0.80	0.39, 1.63
Cabbage	18	89	0.73	0.36, 1.45
Spinach	6	51	1.04	0.51, 2.08
Fermented maize drink	18	51	1.14	0.59, 2.22
Soft maize porridge	49	69	1.05	0.54, 2.04
Stiff maize porridge	27	74	1.60	0.82, 3.15
Stamped maize	37	91	0.96	0.40, 2.28
Beef	4	31	0.84	0.33, 2.15
Chicken	10	63	1.16	0.59, 2.31
Mutton	3	36	0.79	0.33, 1.89
Eggs	5	54	1.22	0.60, 2.48
Milk	45	70	0.96	0.50, 1.86
Bread	36	87	1.38	0.66, 2.87
Beans	29	81	1.58	0.76, 3.29
Holsum	16	52	0.98	0.49, 1.92
Cooking oil	55	79	1.72	0.88, 3.36
Margarine	9	35	1.25	0.56, 2.77
Potatoes	21	77	0.74	0.38, 1.45
Pumpkin	5	46	1.93	0.89, 4.16
Rice	11	53	0.87	0.45, 1.69
Solanum nigrum	7	49	0.62	0.31, 1.22
Age 45+ yrs			0.65	0.31, 1.37
Female sex			1.10	0.45, 2.57
Positive serology for <i>H. pylori</i>			0.23	0.05, 1.18



Table II. Relation of food group consumption to pH

Food group	Odds ratio	95% confidence interval	Spearman's rank correlation with pH	p-value
Fruit	1.38	0.72, 2.67	0.14	0.08
Vegetables	1.06	0.55, 2.05	-0.01	0.90
Maize products	2.64	1.34, 5.20	0.19	0.02
Animal products	0.99	0.51, 1.90	-0.01	0.92
Pumpkin and beans	2.66	1.33, 5.30	0.22	0.006
Full traditional diet (maize, pumpkin, beans)	2.60	1.32, 5.13	0.22	0.006
Fats (Holsum, cooking oil, margarine)	1.02	0.53, 1.96	-0.04	0.63

The main findings of this study are a bimodal pH distribution and a strong association between the traditional dietary elements of maize, pumpkin and beans, and high gastric pH.

Less than half the population had a pH in the expected range of 1 - 4, with a greater number in the 4 - 8 range. The lower pH group equated with a relatively normal pH distribution.^{3,4} The other, bigger group whose pH peaked at 7, ate a diet high in maize, pumpkin and beans.

It has been postulated that a diet based predominantly on maize results in suppression of gastric acidity,² and the results of this study strongly support that theory. Linoleic acid is a precursor of PGE₂. In rats a long-term diet high in linoleic acid results in an increase in PGE₂ production in the stomach.⁵ PGE₂ production is also increased by a diet low in other fatty acids⁶ and riboflavin.⁷ PGE₂ is an inhibitor of gastric acid production.⁵

The scatter diagram of pH against maize, pumpkin and bean consumption (Fig. 2) indicates that there are other factors affecting pH, working either in parallel or synergistically with the dietary elements.

One such factor anticipated to be important is *H. pylori* infection. In this study it showed a non-significant association with a lower pH. This finding was unexpected. Current *H. pylori* infection increases PGE₂ production in the stomach,⁸ and so might be expected to be associated with a higher pH. However, polyunsaturated fatty acids including linoleic acid inhibit the growth of *H. pylori in vitro*,⁹ and therefore the possibility exists that the traditional diet reduces *H. pylori* prevalence.

This study suggests a short-term dietary cause of raised pH; however, the possibility of a long-term achlorhydria must also be considered. Chronic *H. pylori* infection may result in atrophic gastritis with reduced acid output. Failure to demonstrate a positive association between *H. pylori* seropositivity and raised pH means that *H. pylori* is very unlikely to be the cause of the bimodal pH distribution seen.

A raised intragastric PGE₂, by relaxing the pylorus,^{10,11} may produce a chronic duodenogastric reflux which may give rise

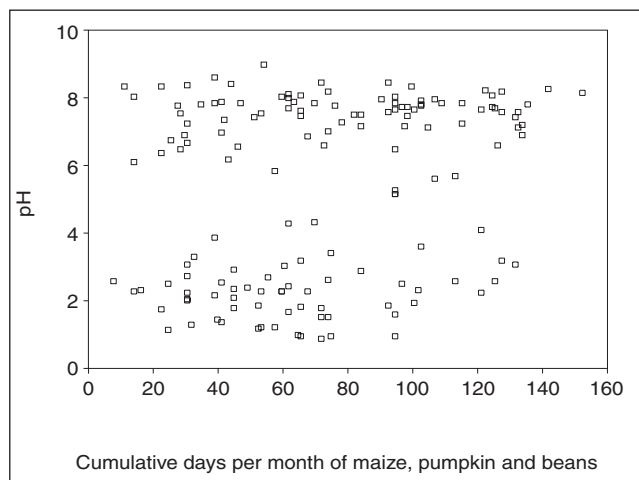


Fig 2. pH and traditional diet in Transkei.

to atrophic gastritis. It is probable that this mechanism has some effect on gastric pH in the population in addition to the short-term dietary effect.

It is possible that the traditional diet of maize, pumpkin and beans might be a spurious marker of some other aspect of Transkeian life which is the real cause of a raised gastric pH. It has been demonstrated in rats that dietary supplementation with linoleic acid results in a higher gastric pH.⁵ This effect, allied with the evidenced mechanism described, supports the existence of a direct link between a maize diet and raised gastric pH.

Help with funding came from the Minorities in Research Training programme of Howard University Cancer Centre, Southmead Hospital Research Fund, UK, and the Medical Research Council of South Africa. Statistical analysis was by Chris Foy, Research and Development Support Unit, Gloucestershire Royal Hospital, UK.

References

1. Doll R. The geographical distribution of cancer. *Br J Cancer* 1969; **23**: 1-8.
2. Sammon A, Alderson D. Diet, reflux and the development of squamous cell cancer of the oesophagus in Africa. *Br J Surg* 1998; **85**: 891-896.
3. Dressman JB, Berardi RR, Dermentzoglou LC, et al. Upper gastrointestinal (GI) pH in young, healthy men and women. *Pharm Res* 1990; **7**: 756-761.
4. Russell TL, Berardi RR, Barnett JL, et al. Upper gastrointestinal pH in seventy-nine healthy, elderly, North American men and women. *Pharm Res* 1993 **10**: 187-196.
5. Schepp W, Steffen B, Ruoff H-J, Classen M. Effect of dietary linoleic acid on acid secretion and cold-restraint ulcers in rats (Abstract). *Gastroenterology* 1986; **90**: 1617.
6. Raedersdorf D, Moser U. Influence of an increased intake of linoleic acid on the incorporation of dietary (n-3) fatty acids in phospholipids and on prostanoid synthesis in rat tissues. *Biochim Biophys Acta* 1992; **1165**: 194-200.
7. Pellicione NJ, Karmali R, Rivlin RS, Pinto J. Effects of riboflavin deficiency upon prostaglandin biosynthesis in rat kidney. *Prostaglandins Leukotrienes Med* 1985; **17**: 349-358.
8. Avunduk C, Suliman M, Gang D, Polakowski N, Eastwood GL. Gastroduodenal mucosal prostaglandin generation in patients with *Helicobacter pylori* before and after treatment with bismuth subsalicylate. *Dig Dis Sci* 1991 **36**: 431-434.
9. Thompson L, Cockayne A, Spiller RC. Inhibitory effect of polyunsaturated fatty acids on the growth of *Helicobacter pylori*: a possible explanation of the effect of diet on peptic ulceration. *Gut* 1994 **35**: 1557-1561.
10. Milenov K, Golenhofen K. Contractile responses of longitudinal and circular smooth muscle of the canine stomach to prostaglandins E and F2 alpha. *Prostaglandins Leukotrienes Med* 1982; **8**: 287-300.
11. Hausken T, Odegaard S, Berstad A. Antroduodenal motility studied by real-time ultrasonography. Effect of Enoprostil. *Gastroenterology* 1991; **100**: 59-63.

Accepted 9 July 2003.