LETTERS TO THE EDITOR

Lymphocytic gastritis and coeliac disease

EDITOR,—We would like to comment on the paper by Miettinen *et al* on lymphocytic gastritis in patients with gastric lymphoma (*Gut* 1995; 37: 471–6). They found lymphocytic gastritis in about a third of patients with gastric lymphoma, and they looked for the relation between lymphocytic gastritis and *Helicobacter pylori* infection. They were surprised to find that lymphocytic gastritis occurred more frequently in patients without *H pylori* infection, and they were not able to explain the mechanism of the gastritis in patients with gastric lymphoma without *H pylori* infection. The authors, however, were aware that lymphocytic gastritis may occur in coeliac disease, but they did not discuss this possibility for their patients. We are convinced that coeliac disease may indeed explain the occurrence of lymphocytic gastritis in those patients with gastric lymphoma and without *H pylori* infection. In fact, as previously reported,¹ we have found the presence of lymphocytic gastritis in nine of 25 children with coeliac disease and in none of 36 children with *H pylori* infection. These nine coeliac patients represent all the cases of lymphocytic gastritis we found in 245 consecutive children who had upper gastrointestinal endoscopy. This strongly suggests that this peculiar form of chronic gastritis is, in children, almost exclusively related to gluten intolerance.

Taking into account that gastrointestinal lymphomata may be related to untreated coeliac disease,² there is one further reason to think that the patients reported by Miettinen *et al* with gastric lymphoma and without *H pylori* infection, could be affected by silent coeliac disease and could benefit from a gluten free diet.

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2 Holmes GKT, Prior P, Lane MR, Pope D, Allan RN. Malignancy in coeliac disease – effect of a gluten free diet. *Gut* 1989; 30: 333–8.

Reply

EDITOR,—We appreciate the interest Dr Maggiore and colleagues have shown in our article. We agree that lymphocytic gastritis may indeed be a manifestation of sensitivity to gluten.^{1 2}

We have evaluated all available duodenal biopsy specimens from our gastric lymphoma patients with lymphocytic gastritis to investigate possible association with coeliac disease (*Gut* 1995; 37: 471–6). Duodenal specimens were available in six cases among seven lymphoma patients with lymphocytic gastritis

(three, duodenal bulb; three, descending part of duodenum). No duodenal specimens had been taken in one *H pylori* negative patient. In five cases there was no villous atrophy; three of the patients were *H pylori* negative by histology, while two were positive. In one case a specimen from duodenal bulbous showed severe villous atrophy and an increase of intraepithelial lymphocytes. The gastric lymphoma in this patient was of B cell lineage (L26-positive and UCHL1 negative); no *H pylori* organisms were found in the gastric mucosa. No information about the use of gluten free diet is available.

Because of the retrospective nature of our study, we are not able to exclude duodenal villous abnormalities in all patients. In most of our patients, however, coeliac disease does not seem to explain the occurrence of lymphocytic gastritis.

The mechanisms of lymphocytic gastritis in most of our patients remain speculative. *H pylori* infection is closely associated with gastric lymphoma and has been suspected to be a cause of lymphocytic gastritis.^{3 4} As we have discussed in our paper, the absence of *H pylori* in some patients with lymphocytic gastritis and gastric lymphoma might be connected with atrophic changes in the body mucosa.

Most of the lymphomas complicating coeliac disease are located in the small intestine,^{5 6} and gastric lymphoma is rare.⁶ We agree with Maggiore *et al*, however, that the exclusion of coeliac disease is probably useful in patients with gastric lymphoma and lymphocytic gastritis.

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Concentrations of epidermal growth factor in human saliva and gastric juice

EDITOR,—We read with interest the article by Tunio and Hobsley (*Gut* 1995; 37: 335–9) examining the concentrations of EGF in gastric juice and saliva. We are somewhat surprised by their findings that the concentration of EGF in basal gastric juice exceeds that found in saliva. On the basis of these findings, they go on to conclude that much of the EGF in gastric juice is probably secreted by the stomach. Many groups have measured EGF concentrations in saliva and gastric juice using radioimmunoassay and agree with the salivary concentrations quoted in Tunio's and Hobsley's paper (about 3 ng/ml).^{1–3} However,

the concentrations of EGF found in gastric juice in this paper (about 4 ng/ml) are about 10 times higher than found by other groups,^{1–4} including the paper by Konturek quoted by the authors themselves.³ The major concern over the validity of this work is therefore not related to the salivary sample collection method, which is discussed in the paper, but over the concentration of EGF in basal gastric juice. Some explanation for this order of magnitude discrepancy between their results and other groups' findings needs to be given. This paper will therefore stand as a beacon to highlight the dangers of accepting 'established facts' such as the statement that 'it is well established that gastric juice EGF is mainly of salivary origin', which is often quoted in studies examining the importance of EGF; alternatively, it will crash on the rocks of methodological inaccuracy.

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4 Playford RJ, Marchbank T, Calam J, Hansen FH. EGF is digested to smaller, less active, forms in acidic gastric juice. *Gastroenterology* 1995; 108: 92–101.

Reply

EDITOR,—We thank Dr Playford, Professor Wright, and Dr Goodlad for their comments. The first of these is that they are surprised that the concentration of EGF in basal gastric juice in our study exceeded that in the contemporaneous saliva. However, we did not report that fact. Reference to our summary shows that 'There was no difference between basal salivary and gastric EGF concentrations ($p > 0.05$)'. We certainly therefore did not suggest that, to quote their letter, 'On the basis of these findings, they go on to conclude that much of the EGF in gastric juice is probably secreted by the stomach'. What we did conclude was that, after stimulation with histamine, the output of EGF was so greatly increased (compared with any changes in the saliva) that in those circumstances much of the EGF in the gastric juice was probably secreted in the stomach. The third comment is that, while our results for EGF concentration in resting saliva agree with those of other workers, our concentrations in the gastric juice were much larger than those workers (including themselves) have found. They conclude that our methods of measurement must have been in error, but fail to explain how the methodological rocks on which they believe our research vessel will founder were only present in the gastric and not the salivary samples.

There are considerable differences between the way we collected and stored the gastric juice samples and the methods used by other workers. The articles need to be read in detail to determine all of these, but we mention the