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.


Concentrations of epidermal growth factor in human saliva and gastric juice

EDITOR,—We read with interest the article by Tunio and Hobbsy (Gut 1995; 37: 335–9) containing 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. The authors state that 40% of EGF concentrations found in saliva are of gastric origin. In our laboratories, we have found saliva concentrations quoted in Tunio’s and Hobbsy’s paper (about 3 ng/ml). 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.1 The major concern over the validity of this work is therefore not related to the salivary sample collection, 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 with the in vitro methodology is 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 a gastric origin2, which is often quoted in studies extolling the importance of EGF; alternatively, it will crash on the rocks of methodological inaccuracy.

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Concentrations of epidermal growth factor in human saliva and gastric juice.

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