LETTERS TO THE EDITOR

Activated eosinophils in coeliac disease

SIR,—We read with interest the paper by Gallagher *et al* (*Gut* 1989; **30**: 1568–73) on the complement activation within the coeliac small intestine. Although we agree with a potential role of antibody, T cell, complement and mast cell responses in the pathogenesis of coeliac disease, we think that another component of the inflammatory reaction – that is, the eosinophils, might also be involved. Morphometric studies have shown that eosinophil populations are markedly expanded in the coeliac mucosa and that they return towards normal levels during gluten restriction.[1-3] More recently, an enhanced local release of eosinophil granule components with cytotoxic properties, such as eosinophilic cationic protein, has been shown in the jejunal tissue from patients with coeliac disease.[4] We would like to report here on further evidence, based on ultrastructural data, of eosinophil activation in the mucosa of three patients with active coeliac disease.

Three patients (two men and one women, age 50, 45, and 16 years) with coeliac disease were studied. At the time of the study they had active coeliac disease – that is, symptoms and subtotal villous atrophy of the jejunal mucosa. They since have had a symptomatic and histologic response to gluten restriction. Perendoscopic jejunal biopsies were obtained from macroscopically flattened jejunal mucosa and processed for standard and ultrastructural study as previously described.[5] Jejunal biopsy specimens revealed subtotal villous atrophy of the mucosa with crypt hyperplasia. The prominent cellular infiltrate of the lamina propria was dominated by lymphocytes and eosinophils. At least 50% of eosinophils were degranulated or lysed and had an altered structure. The typical normal structure of eosinophil granules with a crystalloid electron dense core and an electron radiolucent matrix[5] was dramatically modified according to two patterns of activation: in the epithelium (Fig 1), the eosinophil granules were grouped and their core density was inverted; in the lamina propria a more brutal type of activation could be recognised with eosinophils being in degranulation lysis (Fig 2).

Thus alterations of the structure of eosinophils, which have been previously associated with evidence of activation,[5] were present in the mucosa of those three patients with coeliac disease. This reinforces the possibility of eosinophil involvement in intestinal lesions. Activated eosinophils could release highly cationic cytotoxic proteins – for example, eosinophilic cationic protein and major basic protein which could have a role in cell damage in coeliac disease.[4] Further studies are needed to determine the relative contribution of inflammatory components in the pathogenesis of coeliac disease.

J F COLOMBEL
A JANIN
G TORPIER
Clinique des Maladies de l'Appareil Digestif, Hôpital Claude Huriez et Service d'Anatomie et Cytologie Pathologique, Hôpital Calmette, CHU Lille; Unité mixte INSERM U 167-CNRS 624, Institut Pasteur, Lille, France

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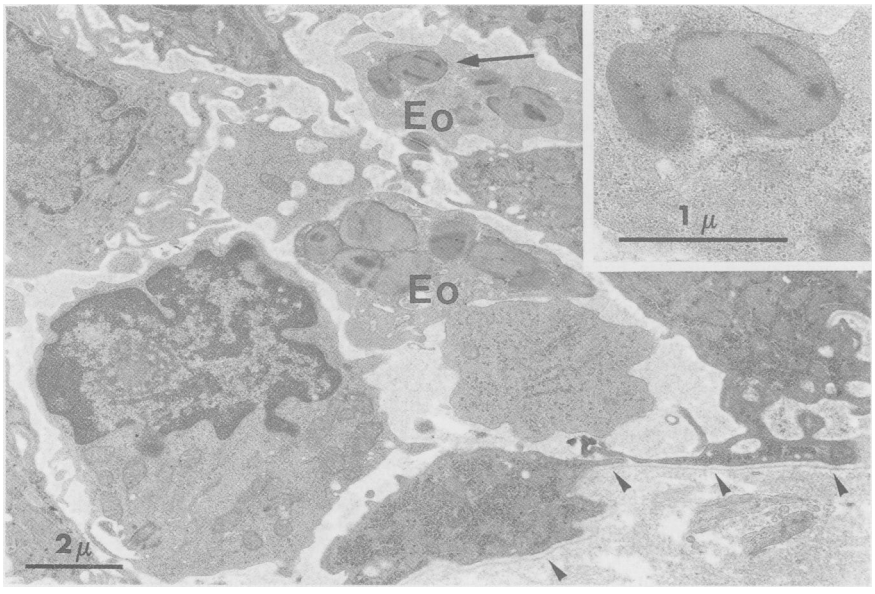

Figure 1: Coeliac disease. Intraepithelial eosinophils (Eo) (arrow heads on basement membrane). Eosinophil granules are grouped and their core have an inverted density (insert).

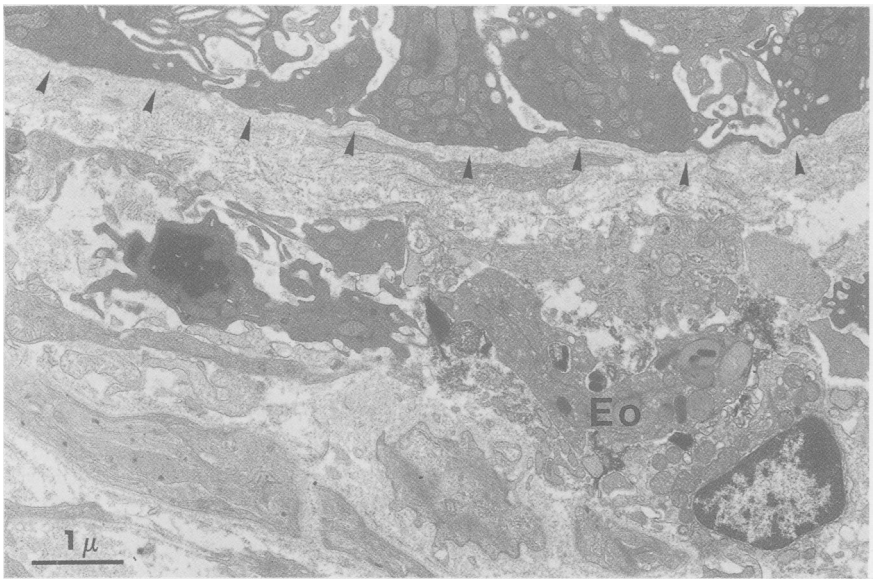

Figure 2: Coeliac disease. Eosinophil (Eo) in degranulation lysis in the lamina propria (arrow heads on the basement membrane).