LETTERS TO THE EDITOR

Intestinal permeability

EDITOR,—We read with interest the paper by Dr Oriishi and colleagues (*Gut* 1995; **36:** 891–6) investigating intestinal permeability and the immune response to enteric bacterial antigens in patients with inflammatory bowel disease. Their finding of an increased systemic concentration of antibodies to lipid A in patients with inflammatory bowel disease is in agreement with studies showing increased titres of antibodies to the endotoxin core,[1] to peptidoglycan-polysaccharide complexes,[2] and to a variety of enteric bacteria[3] in these diseases. These studies offer indirect evidence of mucosal barrier dysfunction in patients with inflammatory bowel disease, which supports the more direct evidence provided by the measurement of systemic endotoxin concentration or by bacterial culture studies.[1 4]

The study by Dr Oriishi and colleagues also provides confirmation that the pattern of antibody response to endotoxin antigens is different in patients with Crohn's disease from those with ulcerative colitis.[1] There is an increase in the systemic concentration of IgG to endotoxin core/lipid A in patients with Crohn's disease but not in those with ulcerative colitis. Systemic IgM concentration to endotoxin core/lipid A is not increased in either disease. With regard to IgA, we similarly found that the plasma concentration of IgA to the endotoxin core was increased (though not significantly) in patients with Crohn's disease (107·3±20·5 median units) and ulcerative colitis (93·0±24·2) in comparison with healthy controls (61·3±15·7).[5] We are unable, as yet, to explain these differences.

Their study is also interesting with regard to treatment for impaired gut barrier function. Lactulose has been shown to eliminate systemic endotoxaemia in a hapten induced rat model of colitis[6] and had been suggested as treatment for patients with inflammatory bowel disease.[6 7] The mechanism of the anti-endotoxin action of lactulose is not clear, as lactulose treatment did not have any significant effect on the faecal count of Gram negative bacteria or faecal endotoxin concentration in the experimental model of colitis.[6] It has generally been assumed that lactulose is fermented rapidly by colonic bacteria and that colonic absorption after oral administration would be minimal.[8] The study by Dr Oriishi and colleagues, however, suggests that there is significantly increased lactulose absorption in the presence of colonic inflammation. It is possible, therefore, that absorbed lactulose was effective as an antiendotoxin agent in experimental colitis by exerting a direct neutralising effect on systemically circulating endotoxin.[6 9]

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Reply

EDITOR,—We thank Mr Keith Gardiner and his colleagues for their comments.

We are aware of the anti-endotoxin action of lactulose. In our study, however, we used lactulose as a marker of intestinal permeability. Anti-lipid A antibody concentrations were not influenced by lactulose with an antiendotoxin action, because antilipid A antibody concentrations were measured just before lactulose administration.

Lactulose may be useful in both evaluation of disease activity and treatment in diseases with an increasing intestinal permeability and endotoxaemia, such as inflammatory bowel disease, alcoholism.[1] Particularly in the inactive phase of Crohn's disease, the continuous administration of lactulose may be interesting. We look forward to further study on this subject.

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Hydatid disease

EDITOR,—We read with interest the leading article by Dr D L Morris (*Gut* 1994; **35:** 1517–8). We agree with the points concerning treatment of the hydatid disease, however, we are in disagreement with Dr Morris' statement 'there are two forms of echinococcus that affect the liver of humans, *E granulosus* and *E multilocularis*'.

Rausch and Bernstein in 1972 described a new species of echinococcus named *E vogeli*.[1] Furthermore *E vogeli* was found to be the aetiological agent of the hydatid disease in several patients from Colombia, Venezuela, Equador, and Panama, most of them showing hepatic involvement by the disease.[2] More recently, we had the opportunity to study nine patients with hydatid disease, seven of them from the Brazilian Amazon region; eight of nine showed extensive involvement of the liver.[3] Another study of six additional patients from the state of Acre (Amazon region) showed severe involvement of the liver (Meneghelli *et al*, unpublished data). All of these patients showed pathological findings that allowed us to establish the diagnosis of echinococcosis caused by *E vogeli*. Subsequently new cases of this neotropical hydatidosis have been detected in Brazil by Ferreira *et al*[4] and by D'Alessandro *et al* (unpublished data). It seems that the disease has an extensive distribution in South America, mainly in the Amazon region. All patients we studied had extensive involvement of the liver making a surgical approach impossible. We found that albendazole is effective for the treatment of the disease.[5] Thus we consider that we have enough evidence to say that there are at least three species of echinococcus that affect the liver of humans: *E granulosus*, *E multilocularis*, and *E vogeli*. Moreover there is a possibility, although rare, that another species, *E oligarthrus*, may also cause hepatic disease in humans.

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Gastric mucus viscosity and *Helicobacter pylori*

EDITOR,—The article in *Gut* by Markesich *et al* reports that *H pylori* infection does not cause reduction in the viscosity of human gastric mucus gel (*Gut* 1995; **36:** 327–9). The findings contradict the results of earlier studies,[1–3] and misquote the source of materials with which the work was carried out.[1 2] Contrary to the authors' assumption, we never used commercially obtained mucin and the work included human gastric mucus, as well as its purified mucin.[4 5] Furthermore, our assays of *H pylori* enzymatic activities were carried out with enzyme enriched protein fraction under well controlled conditions. These studies showed that *H pylori* through its mucolytic enzyme actions is capable of exerting a detrimental effect on gastric mucus gel viscosity.[6]

The viscosity data presented by Markesich *et al* were obtained directly on gastric juice samples from patients with and without *H pylori* infection, and do not provide any information on the mucolytic enzyme activities of the bacterium, nor for that matter on