Lactulose has been shown to exert a number of effects on the intestinal microflora and to influence the immune response to enteric bacterial antigens in patients with inflammatory bowel disease.

**Editor,—**We read with interest the paper by Dr Orishi 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 may be a further indication of the involvement of endotoxin in the aetiology of this disease. We would like to report that a similar increase in systemic antibody concentrations was noted in our patients with ulcerative colitis.

There was 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 was 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 (107·5±20·5 median units) and ulcerative colitis (93·0±24·2) in comparison with healthy controls (61·3±15·7). We are unable, as yet, to explain these differences. The study by Dr Orishi and colleagues is of interest with regard to treatment for impaired gut barrier function. Lactulose has been shown to eliminate systemic endotoxins in a hapten induced rat model of colitis. 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 oral bacteria in normal rats. In the experimental model of colitis, it has generally been assumed that lactulose is fermented rapidly by colonic bacteria and that colonic absorption after oral administration would be minimal. Their study is also interesting with regard to the treatment of inflammatory bowel disease. 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 oral bacteria in normal rats. In the experimental model of colitis, it has generally been assumed that lactulose is fermented rapidly by colonic bacteria and that colonic absorption after oral administration would be minimal.

1 Rausch RL, Bernstein JJ. Echinococcus vogeli sp n. (Cestoda: Taeniidae) from the dusk dog Spotted Varetus (Lund.): Tropenmed Parasiat 1972; 23: 25–34.


**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 increased by lactulose treatment (p>0·05). We agree that an increase in systemic antibody concentrations may reflect a state of systemic endotoxinemia in patients with inflammatory bowel disease. 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 oral bacteria in normal rats. In the experimental model of colitis, it has generally been assumed that lactulose is fermented rapidly by colonic bacteria and that colonic absorption after oral administration would be minimal. Their study is also interesting with regard to treatment for impaired gut barrier function.

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