in group c (6) ranitidine, 150 mg bid, for 12 months. Yates' correction was used to compare the percentage of patients with irritable bowel syndrome, showing normal morphology were used as controls. Although our experience has been reported in a previous study this was not cited by Vecchi et al. If in our study we have investigated the lectin binding sites in duodenoejejunal mucosa from 119 coeliac children (67 with subtotal villous atrophy and 52 with partial villous atrophy after gluten free diet for 12 months) (age range 1-12 years); moreover, 16 biopsies obtained from infants with short stature and normal intestinal mucosa and nine specimens obtained from infants suffering from posterioritis syndrome were also utilised as normal and pathological controls.

It would be of interest to know the age of patients studied in the paper by Vecchi et al., in addition no information about the blood groups of patients is available in this study.

Comparisons can be made with our results obtained by Vecchi et al with ours. Our findings show WGA and DBA in normal mucosa are different from those of Vecchi et al., in which a constant positivity at the tip of villi and in goblet cells; the latter findings were similar to our pathological control mucosa. Moreover, Vecchi et al considered 10 normal patients suffering from chronic diarrhoea with a final diagnosis of irritable bowel syndrome; these patients were indeed comparable with our pathological controls. Therefore we contend that only patients without gastrointestinal symptoms and histologically normal intestinal mucosa must be chosen as normal controls, especially in studies assessing lectin binding sites in pathological tissues.

In pathological specimens, there is striking disagreement about the distribution of some lectins; in our study, coeliac mucosa was evident in our coeliac mucosa and pathological controls, whereas no reactivity was encountered in normal control mucosa. On the contrary, Vecchi et al reported no differences in the WGA pattern in pathological and 'normal control' mucosa. As regards DBA distribution, Vecchi et al observed an evident decrease of goblet cells reactivity in mucosa with subtotal villous atrophy in comparison with controls and they suggested an imbalance in goblet cell number in the same way. However, in our study we have documented the appearance of goblet cells reactive in coeliac patients and pathological control mucosa. The DBA reactivity is considered as a specific marker of human colonic mucous goblet cells, as also outlined by Vecchi et al; it is noteworthy that colonic mucous goblet cells contain acidic sulphated mucosubstances, which are absent in normal duodenojejunal mucosa. In coeliac mucosa, however, we have previously shown with high iron diamine – alcin blue pH 2.5 (HID-AB method), the presence of weak and strong acidic sulphomucins. This fact may be related to the high reactivity found in the coeliac mucosa, suggesting the appearance of new specific lectin binding sites. Additional evidence on the appearance of sulphated glycoproteins was the finding of the PNA expression in our coeliac mucosa, which cannot be compared with normal and pathological control mucosa. The latter datum is another divergent point with negative findings by Vecchi et al in their control and pathological specimens.

Finally, Vecchi et al suggest that further studies about the lectin binding pattern in the jejunum of patients with GSE on a gluten free diet would be useful. In the aforementioned paper, we have extensively studied the lectin binding pattern in 52 treated coeliac patients on a gluten free diet for at least 12 months; in particular, data obtained from patients with coeliac disease and PNA obtained in these patients were similar to those found in untreated coeliac children. The hypothesis of a primary defect of the glycoconjugates metabolism in coeliac disease remains to be investigated in more extensive studies.


Reply

Sir,—We appreciate the comments of Barresi and colleagues who point out some very important matters of discussion on the use of lectin histochemistry. We feel, however, that the two studies cannot be considered fully comparable because of clinical and methodological differences.

First, while Barresi et al report the results obtained in a pediatric population, our series consisted of adult patients only. Although it is not known if age may induce changes in the lectin binding pattern in human subjects, this seems to be the case in animals. Indeed, the lectin binding pattern observed by Barresi et al differs not only from what we have observed but also from what has been observed by other authors.

In fact, as also mentioned by our colleagues, DBA has been shown to react with at least some of normal jejunal specimens studied by other authors. Furthermore, both epithelial and goblet cells is a common finding in normal jejunal mucosa, described by other authors and is one of the most consistent findings in our experience. Staining with DBA has been shown to occur in normal specimens by other authors but no evidence of staining was observed by us in either normal or pathological jejunal mucosa.

If these differences are indeed due to the fact that the only normal controls studied so far are those reported by Barresi et al, as the authors suggest, is also not known. Their statement is based on the absence of any gastrointestinal symptom in their children of short stature. Stating that normal controls, including adults, are both suggestive of coeliac disease. Thus, we believe that the presence of normal jejunal mucosa architecture, together with the negativity of other functional and serological evaluations (which were performed in our patients) should be considered as sufficient evidence to rule out the disease in our patients with diarrhoea as well as in their children with short stature.

Lectin histochemistry is a useful and interesting tool for the study of tissue glycoconjugates. One should keep in mind, however, the fact that the use of different fixatives and of different times the processing techniques may affect the presence and availability for staining.