conclusions can be drawn from specimens taken from only six patients of whom two had ileostomies. The results show a coefficient of variation of about 50% and the risks of a type 2 statistical error must be very high. In contrast to Finnie et al suggest an explanation as to why they found such a low protein weight to wet weight ratio. Until they do so it is very difficult to interpret their data.

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Reply

EDITOR,—We thank Mr Chapman for his comments regarding our paper and are grateful to the editor for the opportunity to respond to the points raised.

The finding is that butyrate metabolism in ileal mucosa is impaired in all specimens and that the results show that there is no clear answer. As Chapman et al themselves point out there is likely to be little or no butyrate available from the colonic lumen because it is readily utilised by colonic flora, and peripheral blood concentrations of butyrate are less than 0.5 mM, so culture in the presence of 5 mM butyrate cannot be considered physiological.

We were interested in the results from ileal mucosal biopsy specimens reported by Chapman et al in their abstract. We would expect that the reduced metabolism of butyrate that they have shown in the terminal ileum of patients undergoing colectomy, presumably for severe colitis, might be to some extent a reflection of the severe illness combined with backwash ileitis. For this reason we chose to study ileal biopsy specimens obtained at colonoscopy from patients in clinical remission. The figures for coefficients of variation we published were for all specimens, ileal and colonic included (20% for butyrate metabolism and 23% for glucose metabolism). The coefficients of variation for glucose metabolism in ileal biopsy specimens was 20% and for butyrate 18%, not 50% as stated by Chapman et al. There was, if anything, a very slight trend towards increased butyrate metabolism in ulcerative colitis in our study. We have subsequently increased the numbers of ileal biopsy specimens studied to 18 in all and the conclusion remains the same.

There clearly are some interesting differences in mucosal metabolism in ulcerative colitis but our view is that most of these differences are likely to reflect changes occurring secondary to hyperplasia.

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Risk factors for Helicobacter pylori

EDITOR,—The findings of the EUROGAST Study Group on risk factors for H pylori infection (Gut 1993; 34: 1672-6) interested us because of the relation between this organism and gastritis. The principal finding of the group was a positive correlation between infection and low educational standard. They suggested this showed that social class was a relevant factor. In 1966 we found a correlation among atrophic gastritis on gastric biopsy and social class in a series of 221 patients suffering from non-ulcer dyspepsia. The prevalence of gastritis increased with descending social class, and increased with age in all classes, becoming roughly equal in each class at 50 years and over.

Unlike the EUROGAST study our data suggested a positive correlation also between atrophic gastritis and excessive cigarette smoking and excessive alcohol consumption, and drinking hot tea.

The study by Vincent et al (Gut 1994; 35: 313) showed a high prevalence of H pylori in children cared for in a medical centre for mentally retarded children. The authors thought that factors related to close contact with other children were probably the cause of the high prevalence.

We were puzzled by our findings nearly 30 years ago. It is of interest that Bateson2 in his review of H pylori infection mentions its higher prevalence, and at an earlier age, in the developing world. Perhaps, if there is a relation between atrophic gastritis (and H pylori infection) and social factors it is mediated through interacting elements of living conditions probably associated with low grade education: financial hardship, restricted accommodation with people in close proximity and possibly sharing food and cooking utensils, poor hygiene, and low social class.

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