clusions 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 conclusion it is important that 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.

In response to his first point, he has correctly identified an error on our part. The units quoted throughout our paper for butyrate and glutamine metabolism should have been nmol substrate metabolised/h/mg protein. We apologise for the confusion that has resulted. The statistical analysis and conclusions for all the data remain as stated.

The critical difference between our study and that of Chapman *et al*¹ is that we have examined butyrate metabolism in the presence of glucose but not glutamine, whereas their study was performed in the presence of all three energy substrates. Our own studies and those of Roediger² (but not those of Chapman *et al*) have shown increased metabolism of glutamine in ulcerative colitis. It seems likely that this may be a reflection of hyperplasia, as rapidly dividing cells have increased metabolism of glutamine.³ In the absence of glutamine we have failed to find any defect in metabolism of butyrate, and suspect that the results of Chapman *et al* may reflect the predilection of the mucosa for glutamine at least as much as the reduced ability to metabolise butyrate. To some extent a similar conclusion can be reached from part of Roediger's original study. He showed that the rate of oxygen uptake in the presence of butyrate as the sole energy substrate was normal in quiescent ulcerative colitis and only reduced in acute colitis, and although production of carbon dioxide from butyrate was reduced in ulcerative colitis, the most

striking change in the contribution of different fuels to total oxygen consumption was the increased metabolism of glucose in quiescent colitis.

With regard to the appropriate choice of glutamine concentration there is no clear answer. As Chapman *et al* themselves point out there is likely to be little or no glutamine available from the colonic lumen because it is so readily utilised by colonic flora,¹ and peripheral blood concentrations of glutamine are less than 0.5 mM⁴, so culture in the presence of 5 mM glutamine cannot be considered physiological.

We were interested by the results from ileal mucosal biopsy specimens reported by Chapman *et al* in their abstract.⁵ We would suspect 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 glutamine metabolism). The coefficients of variation for glutamine 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 infection 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 found¹ a correlation between 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, 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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- 1 Edwards FC, Coghill NF. Aetiological factors in chronic atrophic gastritis. *BMJ* 1966; 2: 1409.
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Reply

EDITOR,—Based on their findings, Edwards and Coghill suggest that social factors implied in the spread of *H pylori* infection could explain the epidemiological pattern of atrophic gastritis.

Crowding and promiscuity are probably the ultimate risk factors of *H pylori* infection. These conditions are mediated by a low social economic class, which is a good risk marker but not a causal factor of infection. Our study showed that when they have been living in the same conditions for years, cohabiting children from different ethnic and social origins attain the same level of risk for *H pylori* infection. Furthermore, the high seroprevalence known in gastroendoscopists¹ shows that infection can be acquired even in hygienic conditions.

This is not in disagreement with the social distribution of atrophic gastritis. *H pylori*, which is generally acquired in childhood, induces a chronic inflammatory reaction for years, but epithelial changes of atrophy and metaplasia are not constant and could vary with the host and circumstances. Atrophic lesions are uncommon in infected children, and it has been shown in adults that the mucosal colonisation area of *H pylori* can be larger than the area of chronic active gastritis.² Moreover, an aggravation of mucosal lesions of *H pylori* associated gastritis can be found after absorption of non-steroidal anti-inflammatory drugs (NSAIDs).³

Atrophic gastritis found in adults might be a multifactorial condition resulting from *H pylori* infection (as a necessary but not sufficient factor) associated with consumption of gastric toxic agents, such as NSAIDs, alcohol, tobacco, spiced food, etc.