Reply

EDITOR,—We appreciate Ockenga *et al*'s comments on our study. Although we did not assess pancreatic function in our patients, we would like to discuss the hypothesis that exocrine pancreatic insufficiency may contribute to steatorrhoea in malnourished HIV infected patients. As pointed out by Ockenga and colleagues, HIV infected patients frequently have elevated pancreatic serum enzymes and this can be due to several causes.¹ Furthermore, a recent study has shown that pancreatic abnormalities are frequently found on ERCP of HIV infected patients with cholangitis.² However, these biochemical or morphological abnormalities do not necessarily imply that exocrine pancreatic insufficiency is present. Indeed, roughly 90% of the secretory capacity must be lost before fat malabsorption occurs.³ In addition, previous studies have shown that faecal fat concentration is higher in patients with pancreatic insufficiency than in those with gastrointestinal diseases.⁴ In the paper by Bo-Linn and colleagues, all patients with pancreatic steatorrhoea had faecal fat concentrations of more than 9.5%. In our study, the faecal fat concentration was significantly lower in HIV infected patients than in patients with small bowel disease or short bowel syndrome, suggesting higher intestinal secretion; a faecal fat concentration of more than 9.5% was found in only one of 79 patients. Intestinal involvement is sufficient to explain faecal weight and steatorrhoea in HIV infected patients. Yet, these data do not exclude that, in some patients, particularly the severely malnourished ones, some degree of exocrine pancreatic insufficiency may aggravate malabsorption.

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Treatment of *Helicobacter pylori* infection

EDITOR,—Apart from validating the reversibility of the pathogenic processes mediated by *Helicobacter pylori* in the causation of duodenal ulcer, the study by Lam *et al* (*Gut* 1997;41:43–8) also served as a reminder that, even in *H pylori* associated ulcer disease, other mechanisms have a reversible aetio-pathogenic role, exemplified by the spontaneous remission documented through the use of placebo. There is a danger of underestimating these unknown mechanisms if, as the authors unfortunately did, like is not compared with like, an example being the reporting of the 100% 12 week healing rate achieved through adherence to the antibiotic protocol as a comparison with the 53.1% healing rate for placebo on the intention to treat analysis in the Results section (page 45). Strictly speaking, the former should have been compared with patients on placebo in the protocol

group, yielding a comparison of 100% *v* 63.4%. Finally, the penultimate line in table 3, dealing with the healing rate for *H pylori* positive patients allocated to placebo, did not make sense. These are minor criticisms in an otherwise good paper.

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Reply

EDITOR,—We are pleased that Dr Jolobe hit the nail on its head by pointing out that placebo can heal duodenal ulcers. In fact, placebo has been shown to heal up to 70% of duodenal ulcers.¹ Factors other than eradication of *H pylori* are clearly operating in the pathophysiology of ulcer healing. Our study does help to reassure us that as long as we can get rid of *H pylori*, whether temporarily or permanently, duodenal ulcers heal.

Dr Jolobe's two criticisms are valid. The first one represents a slip of the pen when writing the text, as the comparisons were done head for head, tail for tail in table 2. The second is a slip in proof reading, as there should be an absolute number, 14, in front of the percentage 37.8 in table 3. Our apologies.

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One week triple therapy for *Helicobacter pylori*

EDITOR,—The paper by Misiewicz *et al* (*Gut* 1997;41:735–9) is an important contribution which demonstrates the usefulness of lansoprazole and clarithromycin combined with either amoxycillin or metronidazole in the treatment of *Helicobacter pylori* infection.

However, there are some questions which arise:

- (1) Only half the patients had duodenal ulcers. There is good evidence that the response rate to eradication therapy is inferior in other patients and it is hard to justify grouping ulcer and non-ulcer patients together in the study.
- (2) Why use lansoprazole 30 mg twice daily? Optimal acid reduction should be achievable with 30 mg daily, and doubling the dose merely reduces the cost effectiveness of this drug. The balance of evidence suggests that omeprazole 40 mg daily, lansoprazole 30 mg daily and pantoprazole 40 mg daily should be equivalent in these regimens.
- (3) It is amazing to read that the combination of lansoprazole or omeprazole with twice daily amoxycillin and metronidazole was judged to be "effective". The success rates achieved were 66 to 83% depending upon the method of analysis. These sorts of incomplete success rates rightly led to the abandoning of dual therapy with omeprazole and amoxycillin or clarithromycin, and are no better than

the old triple therapy with bismuth, tetracycline and metronidazole, which has now been superseded.

If success rates of 90% cannot be achieved there are better treatments available.

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Reply

EDITOR,—We thank Dr Bateson for his interesting comments. We should like to reply to each of his questions in turn.

- (1) The success of *H pylori* eradication therapy depends on the patients' compliance with treatment and the sensitivity of the bacterium to antimicrobials.¹ We are unaware of studies reporting significantly greater efficacy in patients with duodenal ulcer disease, compared with patients with non-ulcer dyspepsia.
- (2) When combined with clarithromycin and a nitroimidazole, we agree that at present there seems to be no therapeutic advantage in increasing the dose of lansoprazole above 30 mg,² omeprazole above 20 mg,³ or pantoprazole above 40 mg⁴ daily. However, no such comparative data exist for the combination of a proton pump inhibitor (PPI) with clarithromycin and amoxycillin. When we planned the study, the data available supported the twice daily use of a PPI with antimicrobials, and also the patients' compliance seems to be better.
- (3) A PPI together with amoxycillin and metronidazole was "judged to be effective" as this combination produced eradication of *H pylori* in 88 to 94% of patients with metronidazole sensitive strains, according to intention to treat analysis. In such circumstances, these regimens were as effective as the other treatment arms. However, these regimens are significantly less effective in the presence of metronidazole resistant strains, with eradication between 46 and 62%. As discussed in the paper, these regimens are not recommended where antimicrobial sensitivities of *H pylori* are unknown or in areas where the prevalence of such strains is at least 30%, where a PPI in combination with amoxycillin and clarithromycin should be the first choice of treatment.

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