genesis of ulcerative colitis.1-3 It should not be noted that ulcerative colitis is a chronic debilitating illness characterised by episodic bloody diarrhoea; many patients suffer faecal incontinence, some require surgical intervention, and some are at increased risk of colon cancer. That such patients have a tendency to neuroticism and introversion is, perhaps, not surprising.

We would not wish to discourage the detailed psychological assessment of patients with ulcerative colitis, but this will only improve our understanding of disease pathogenesis if undertaken in the setting of appropriately controlled clinical trials. Further understanding of the pattern of bowel disease would be of untold benefit and at worst strengthen an already well-established bias.

S A RILEY
V MANI
M J GOODMAN
Hope Hospital, Leed Infirmary, and Burd General Hospital, Manchester


Enteroptapy associated with HIV

SIR,—We read with interest the study by Cummins et al (Gut 1990; 31: 317-21) on the enteroptathy associated with HIV infection.

There is a growing body of evidence for small bowel pathology in patients infected with the human immunodeficiency virus (HIV), and there is broad agreement that atrophy of villi is characteristic of such enteroptathy.1,2 Furthermore, there is evidence that jejunal mucosal pathology may be of importance in that reduced fat absorption correlates with the degree of villous atrophy.3

There are, however, conflicting reports on the changes in crypt cell proliferation in association with such villous atrophy. The paper by Cummins et al reports jejunal crypts of normal length, but with increased mitotic rate, in HIV infected patients. This is in general agreement with our own findings.4,5 As Cummins et al reiterate, we found a broad spread in crypt length in our patients, ranging from hypoplastic (in one patient with AIDS) through normality to hyperplastic. However, the mean jejunal crypt length in our total of 20 HIV infected patients suggested hyperplasia. Notably, we found a strong correlation between the degree of atrophy of villi and the degree of hyperplasia of crypts, and faced with these data it is difficult to avoid the conclusion that there may be a causal (rather than coincidental) relation between these two variables.

It is well recognised that enteric infection may induce hyperplastic villous atrophy, and no doubt the dynamics of HIV enteroptathy are complicated by opportunistic infections (even, possibly, with as yet unrecongnised pathogens).6 Ulrich et al have attempted to unravel the two, and have described an HIV specific hyperplastic enteroptathy, masked in some patients by the crypt hyperplasia induced by secondary pathogens. We quantified hyperplastic HIV enteroptathy also in the absence of indistinguishable opportunistic infections, but the paper by Cummins et al fail to exclude the effects of other pathogens.

Clearly, the mechanisms underlying the villous atrophy of HIV enteroptathy remain elusive and are likely to be multifactorial. Its clarification may have to await an in vivo or animal model of HIV infection, when complicating factors can be carefully controlled.

P A BATMAN
Department of Histopathology,
Bradford Royal Infirmary
M S KAPEMBA
E G GRIFFIN
Department of Communicable Diseases,
St George’s Hospital Medical School,
London


Reply

SIR,—We thank Bateman and colleagues, for commenting on our paper. Both ourselves and Ulrich et al have described villous atrophy and impaired crypt hyperplasia of the small intestine in HIV infection, particularly in patients with AIDS related complex or AIDS. Ulrich et al further found crypt hypoplasia in AIDS subjects who did not have enteric infections. Bateman et al have argued that the enteropathy of HIV infection is caused by hyperplasia, as they found a correlation of crypt length and villous atrophy, although they could show no difference in crypt length. We nevertheless argue that the enteropathy associated with HIV infection is a different pattern to that seen in other enteroptathies, as perhaps best exemplified by coeliac disease in which crypt hyperplasia is clearly discernible both by increased crypt length and increased mitotic count per crypt.

One explanation for these data could be that some crypt hyperplasia occurs in HIV infection that has not yet progressed to AIDS related complex or AIDS. A specially in response to enteric infections. As CD4 lymphocyte depletion occurs systemically and mucosally, the immune ‘drive’ for crypt proliferation is increasingly impaired. Longitudinal studies will help to confirm such an interpretation.

A G CUMMINS
J T LARRBOY
D J SHEARMAN
Queen Elizabeth Hospital,
Woodville South, South Australia 5011


NOTES

Falk Symposia, 1990

These will take place in Freiburg, West Germany as follows:
8-10 October Hepatic metabolism
11-13 October Bile acids as therapeutic agents
15-17 October Mechanisms of peptic ulcer healing
18-20 October Inflammatory bowel diseases

Further information: Falk Foundation eV, Leuzenweberstrasse 5, Postfach 6529, D-7800 Freiburg, West Germany.

Endoscopy workshop

The Chinese Society of Digestive Endoscopy, C.M.A. and the Hong Kong Society of Digestive Endoscopy will be holding the International Workshop and Symposium on Therapeutic Endoscopy and Gastroentrology on 9-12 October 1990 in Beijing, China.

Further details: Dr Joseph Leung, Department of Medicine, Prince of Wales Hospital, Shatin NT, Hong Kong; Tel: (852) 63631285; Fax: (852) 6350075.

Sir Francis Avery Jones BSG Research Award 1991

Applications are invited by the Education Committee of the British Society of Gastroenterology who will recommend to Council the recipient of the 1991 Award. Applications (15 copies) should include:
(1) a manuscript (2 A4 pages only) describing the work conducted;
(2) a bibliography of relevant personal publications;
(3) an outline of the proposed content of the lecture, including title;
(4) a written statement confirming that all or a substantial part of the work has been personally conducted in the United Kingdom or Eire.

The Award consists of a medal and a £100 prize. Entries must be 40 years or less on 31 December 1991 but need not be a member of the BSG. The recipient will be required to deliver a 40 minute lecture at the Spring Meeting of the Society in Manchester in 1991. Applications (15 copies) should be made to: The Honorary Secretary, BSG, 3 St Andrew’s Place, Regent’s Park, London NW1 4LB by 1 December 1990.