LETTERS TO THE EDITOR

Evidence against an autoimmune aetiology for inflammatory bowel diseases

SIR,—The leading article that appeared in *Gut*[1] raised the question of whether inflammatory bowel diseases (IBD) are autoimmune disorders. Since IBD do not fulfil all criteria for classification as autoimmune disorders, analogies between the two groups of diseases may be of interest.

Circulating interferon (IFN) is commonly detected in patients with autoimmune disorders (the so called 'autoimmune-IFN') as well as in patients with AIDS, correlating with disease progression.[2-3] Although circulating IFN is not included in the list of criteria suggestive of autoimmune disorders, the presence of acid-labile IFN-α is believed to reflect continuing autoimmune reactions.[4-5]

We tested 51 sera from patients with either ulcerative colitis or Crohn's disease for the presence of IFN using a sensitive bioassay. For comparison, 41 sera of HIV infected patients were also tested. No IFN was detected in the serum samples from the IBD group while 10 sera from the HIV infected patients were positive for IFN with titres ranging from 5 to 200 IU/ml. This IFN was acid-lable, and characterised as α-type by sensitivity to neutralisation with specific antiserum. IBD sera were also tested for the presence of neutralising antibodies to IFN α or γ[6] and no IFN antibodies were found in IBD sera. Thus, although T cells and macrophages are activated in Crohn's disease and IFN-γ is actively released in the diseased gut,[7-9] no circulating autoimmune IFN can be detected in these patients. These observations provide new evidence against an autoimmune aetiology for IBD.

FRANCESCO PALLONE
Clinica Medica 2,
Università la Sapienza,
Viale de Policlinico 00161,
Rome, Italy
STEFANO FAIS
Cattedra di Gastroenterologia,
Clinica Medica II,
Policlinico Umberto I,
Rome, Italy
MARIA R CAPOBIANCHI
Instituto di Virologia,
Università di Roma,
La Sapienza,
Rome, Italy

Correspondence to: Professor F Pallone.

1 Snook J. Are the inflammatory bowel diseases autoimmune disorders? *Gut* 1990; **31**: 961–3.
2 Hooks JJ, Moutsopoulos HM, Geis SA, Stahl NI, Decker JL, Notkins AL. Immune interferon in the circulation of patients with autoimmune disease. *N Engl J Med* 1979; **301**: 5–8.
3 Eyster ME, Goedert JJ, Poon MC, Preble OT. Acid-labile alpha-interferon. A possible preclinical marker for the AIDS in hemophilia. *N Engl J Med* 1983; **309**: 583–6.
4 Preble OT, Black RJ, Friedman RM, Klippel JH, Vilek J. Systemic lupus erythematosus: presence in human serum of an unusual acid-labile leukocyte alpha interferon. *Science* 1982; **216**: 429–31.
5 Yee AMF, Buyon JP, Yip YK. Interferon-alpha associated with systemic lupus erythematosus is not intrinsically acid labile. *J Exp Med* 1989; **169**: 987–93.
6 Antonelli G, Currenti M, Turriziani O, Dianzani F. Neutralizing antibodies to interferon-alpha: relative frequency in patients treated with different interferon preparations. *J Infect Dis* 1991; **163**: 882–5.
7 Pallone F, Fais S, Squarcia O, Biancone L, Pozzilli P, Boirivant M. Activation of peripheral blood and intestinal lamina propria lymphocytes in Crohn's disease. In vivo state of activation and in vitro response to stimulation as defined by the expression of early activation antigens. *Gut* 1987; **28**: 745–53.
8 Mahida YR, Patel S, Wu K, Jewell DP. Interleukin 2 receptor expression by macrophages in inflammatory bowel disease. *Clin Exp Immunol* 1988; **74**: 382–6.
9 Fais S, Capobianchi MR, Pallone F, Di Marco P, Boirivant M, Dianzani F, *et al.* Spontaneous release of interferon-gamma by intestinal lamina propria lymphocytes in Crohn's disease. Kinetics of in vitro response to interferon-gamma inducers. *Gut* 1991; **32**: 403–7.

Macrophage subpopulations in pouchitis

SIR,—We read with great interest the article by De Silva *et al.* (*Gut* 1991; **32**: 1160–5) on lymphocytes and macrophages subpopulations in pelvic ileal pouches. We have recently performed a similar study characterising immunohistochemically macrophages subsets in ileal pouches with and without pouchitis. Pouch biopsy specimens were stained with monoclonal antibodies (RFD1-dendritic cells, RFD7-mature macrophages, RFD9-epithelioid cells, and tingible body macrophages) using the immunoperoxidase technique.

In agreement with the results of De Silva *et al*, we found a significantly higher proportion of RFD9+ cells in patients with pouchitis (n=10) than in uninflamed pouches (n=20) or normal ileum (n=10), while there were no differences between the three groups in the number of cells positive for the other macrophage markers. Since an increased presence of RFD9+ macrophages has been shown in inflammatory bowel disease, but not in infectious colitis,[1] we agree with the authors that the presence of this histochemical pattern in pouchitis may suggest pathogenetic mechanisms similar to those of original ulcerative colitis.

Macrophages play a major role in mediating and regulating inflammatory and immunological responses in gut mucosa through a number of specialised functions, including antigen presentation and secretion of mediators. The increased phenotypic heterogeneity of macrophages may be therefore caused by the persistent stimulation and activation of these cells in an inflamed mucosa.

This hypothesis is further supported by our recent observation (unpublished data) of a significantly higher interleukin-1 ß mucosal content in pouch biopsy specimens from pouchitis compared with specimens from uninflamed pouches, as in the case of ulcerative colitis.[2]

P GIONCHETTI
M CAMPIERI
A BELLUZZI
G M PAGANELLI
M TAMPIERI
E BERTINELLI
C BRIGNOLA
G POGGIOLI
M MIGLIOLI
G GOZZETTI
L BARBARA
Instituto di Clinica Medica e Gastroenterologia, and Istituto di Clinica Chirurgica II, Universita' di Bologna, Policlinico S Orsola, Via Massarenti 9, 40138 Bologna, Italy

1 Mahida YR, Patel S, Gionchetti P, Vaux D, Jewell DP. Macrophage subpopulations in the lamina propria of normal and inflamed colon and terminal ileum. *Gut* 1989; **30**: 826–34.
2 Gionchetti P, Campieri M, Belluzzi A, Ferretti M, Boni P, Brignola C, *et al.* Interleukin 1-ß (IL-1ß) in patients with ulcerative colitis (UC). *Gastroenterology* 1991; **100**: A582.

NOTES

Sir Francis Avery Jones BSG Research Award 1993

Applications are invited by the Education Committee of the British Society of Gastroenterology who will recommend to Council the recipient of the 1993 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. Entrants must be 40 years or less on 31 December 1993 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 1993. Applications **(15 copies)** should be made to: The Honorary Secretary, BSG, 3 St Andrew's Place, London NW1 4LB by 1 December 1992.

Dysphagia Research Society

The Inaugural Meeting of the Society will be held from 6–8 November 1992 in Milwaukee, Wisconsin. Further details and abstract forms from Dysphagia Research Society, Organising Office, c/o Reza Shaker MD, GI Section/111C VA Medical Center, 5000 W National Avenue, Milwaukee, Wisconsin, 53295 USA. Tel: 414 384 2000 extn 6943; fax: 414 384 8480.