

Gut

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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 of interest 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-labile, 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 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.

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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 uninfamed 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 β mucosal content in pouch biopsy specimens from pouchitis compared with specimens from un-

inflamed pouches, as in the case of ulcerative colitis.²

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NOTES

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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.

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Gut

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NB: Such conversion is the responsibility of the author.

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