## Reply

SIR,—Dr Mahida restates the observations made in his earlier paper[1] on IL-2R expression in inflammatory bowel disease. In this work the classification of the IL-2R+ cells in frozen sections and on cytospins of isolated cells as macrophages or lymphocytes was made on morphology, whereas in our paper we attempted to do it by surface marker expression in situ. No double staining with anti-CD3 or a pan-macrophage antibody was carried out in the study of Mahida *et al*,[1] either in sections or on cytospins, to determine the phenotype of the cells. They described the morphological appearance of the IL-2R+ cells and not the phenotype. We think it unhelpful to ascribe cell lineages based on morphological appearance in a frozen section, thus the need for studies in which lineages are based on the presence of specific cell markers – for example, CD3 for T cells. In addition, it is difficult to be sure that depletion of cell subpopulations does not occur when preparing isolated cells from inflamed human intestine,[2] so that studies on isolated cells need to be carefully interpreted. It is not surprising that some IL-2R+ cells which look like macrophages isolated from inflamed gut can phagocytose zymosan.[1]

In the study of Mahida *et al*,[1] no quantitation of IL-2R+ cells in frozen sections was carried out so it is impossible to evaluate the assertion that no differences existed between ulcerative colitis and Crohn's disease. Likewise the assertion that the CD25+ cells are generally aggregated in the lamina propria is not borne out by the published figures (Fig 1 in our paper and Fig 2 in Mahida *et al*[1]). Certainly the subepithelial macrophage aggregates are strongly IL-2R+ in inflammatory bowel disease as we clearly stated in our paper. Outside of these aggregates in Crohn's disease, however, most of the IL-2R+ cells were CD3+ and this was not the case in ulcerative colitis.

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## Gastric epithelial dysplasia

SIR,—We read with great interest the two papers on gastric epithelial dysplasia in *Gut*.[1,2] These are only the latest of a series, which testifies to the increasing interest in gastric precancerous conditions and lesions in the early diagnosis of cancer, but only partly shows a better understanding of problems relating to the diagnosis and interpretation of dysplastic changes in gastric mucosa. Most reports agree that severe, or high grade, dysplasia is the most important precursor of gastric cancer and strongly recommend gastrectomy, particularly in light of the high percentage of early gastric cancers which this approach enables us to diagnose.[1-4] None the less, others have suggested, in a further paper published in an authoritative journal,[5] that 'gastrectomy is not always the treatment of choice for severe dysplasia and patients must receive a conservative clinical treatment and have frequent endoscopies until the appearance of early carcinoma.' Moreover, various papers report that mild, or low grade, dysplasia progresses to moderate dysplasia in only 9% of cases,[3] is associated with or progresses to cancer in a small but significant percentage of cases,[4] is not distinguished from high grade dysplasia in terms of evaluation of results,[1] and is not even included among cancer precursor lesions.[5] This is probably confusing for those who are not directly concerned in the problem and discouraging for those who would like to find in published papers a rational approach to premalignant gastric lesions. We think that the reasons for these contrasting results are as follows:

(1) Gastric epithelial dysplasia is a rare diagnosis and in all the reports quoted (all of which appeared in authoritative journals) there were no more than 250 cases; only multicentre studies, such as those carried out by the British Society of Gastroenterology in which we also collaborated, are therefore likely to provide us with sufficient information.

(2) As Lansdown and coworkers correctly emphasise,[2] distinguishing dysplasia, particularly in its mild form, from atypical hyperplasia is not done easily or always reliably; we think that the concept of mild dysplasia is changing and though only five years ago we were confident in saying that mild dysplasia was not an indication for follow up,[6] we now consider follow up of these lesions, when correctly classified, to be mandatory.[4,7]

(3) The stomach is a relatively large organ and in the absence of a persistent focal lesion it is difficult to target biopsies and ensure that samples are obtained from the same site (which is why[5] regression of severe dysplastic lesions is reported so often).

(4) Few papers have been published with results from a truly prospective study, and retrospective investigations, particularly in this field, are burdened by the risk of bias.

Nevertheless, we think that a few clinical aspects are fairly well established. Firstly, severe, or high grade, dysplasia, whether associated with gastric ulcer, polyps, erosions, or any endoscopic change, is the most reliable indicator that cancer is present or will develop in a short time and that patients must therefore undergo surgery when feasible. We think that such a policy will save the patient and the doctor medical and legal problems.

Secondly, new prospective and multicentre studies focusing more on mild and moderate or low grade dysplasias are needed because we still do not know the relative risk of cancer for each type of lesion (though we have made an attempt in this direction),[7] whether it is justified to consider moderate dysplasia as a separate entity, or how to follow up such patients.

Finally, we agree that when expert advice is not available locally specimens suspected of dysplastic changes should be examined by expert pathologists, who should be entrusted with educating, with suitable tools, their colleagues in the field.

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## Peritoneal tuberculosis

SIR,—The important study by Manohar and his Durban colleagues[1] draws attention to peritoneal tuberculosis in underprivileged communities. Their findings are in keeping with the Cape Town experience over the past 28 years.[2-5] We question their statement that 'ascitic fluid analysis is not usually of specific diagnostic value.' While acid fast bacilli are rarely found in the small volumes examined, determination of adenosine deaminase in the ascitic fluid[6] allows the diagnosis of peritoneal tuberculosis with a sensitivity and specificity of the order of 100% and 96%, respectively.[5,7,8] Adenosine deaminase determination in the ascitic fluid may obviate the need for the more invasive peritoneoscopic examination.

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### Reply

SIR,—We acknowledge the pertinent points raised by Lingenfelser and Marks. When our paper was submitted we thought that the role of ascitic adenosine deaminase in the diagnosis of tuberculous peritonitis[1 2] needed further investigation. Subsequent evaluations of ascitic adenosine deaminase in tuberculous peritonitis[3 4] have shown high diagnostic sensitivities.

We therefore accept that this determination is an important investigatory option in the diagnosis of peritoneal tuberculosis. The role of peritoneoscopy therefore seems to be limited to equivocal adenosine deaminase results where there is high clinical suspicion of tuberculous peritonitis or cases where malignant ascites is suspected.

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### Zinc deficiency in cirrhosis

SIR,—We read with interest the report by Goode *et al* (*Gut* 1990; 31: 694–9) on zinc status and hepatic functional reserve in patients with liver disease. We have reported hepatic zinc, copper, and magnesium concentrations in childhood cirrhosis, indicating that the serum concentrations do not always reflect the tissue concentrations of these trace elements. We have already indicated that the tissue zinc concentration decreases not only in cirrhotic children but also in patients with idiopathic portal hypertension.[1]

We have also reported that zinc is decreased in leucocytes of patients with cirrhosis, which affects their chemotactic function.[2] This function would be corrected when leucocytes are incubated with zinc (0·05 ml neutrophil mixture and 1 μg zinc) or control serum samples. Bactericidal function was shown to be appreciably decreased, as was nitro blue tetrazolium reduction and stimulated hexose monophosphate shunt activity of these cells.[3]

We have also shown (unpublished results) that zinc absorption is not greatly depressed but urinary zinc excretion is increased in patients with cirrhosis, which seems to be the basic cause of their zinc depletion.

Therefore we are in favour of zinc supplementation in these patients as suggested previously.[3 4]

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# BOOK REVIEWS

**Morson and Dawson's gastrointestinal pathology.** By B C Morson, I M P Dawson, D W Day, J R Jass, A B Price, and G T Williams. (Pp 748; illustrated; £19.) Oxford: Blackwell Scientific, 1990.

The new edition of Morson and Dawson will undoubtedly prove to be one of the 'best sellers' in the fields of gastrointestinal pathology, gastroenterology, and endoscopy. The volume is larger than its predecessors and there are now six authors, although individual responsibility for chapters is not specified. Documentation of disease entities is comprehensive and informative and the text is consolidated with a wealth of references and many new larger illustrations which clarify interpretation. The sections on inflammatory bowel disease, neoplasia, and infective colitis are superb. Vascular disorders of the small and large intestine are described in two separate chapters. A new section covering pathology of the peritoneum has been introduced towards the end of the text and this should be useful.

Lynch syndromes do not appear in the index, although they are discussed with references in the text. The role of antigliadin, antiendomysial, and antireticulin antibodies in the diagnosis and control of coeliac disease has probably been too recent to be included in view of the publication time. With reference to gastric dysplasia, the statement that 'severe dysplasia on its own is not an indication for surgical intervention' does not accord with current gastroenterological opinion. The entity of staphylococcal enteritis is challenged on the basis that *Clostridium difficile* infection cannot be excluded in documented cases but the authors overlook the fact that *C difficile* is predominantly a pathogen of the large intestine whereas staphylococcal pseudomembranous colitis involves the small intestine with predisposing factors such as hypovolaemia. Occasional other controversial views may stimulate publications in the relevant areas. The presentation, style, and content of this new multiauthor book are greatly improved, and I have no hesitation in recommending that this edition should be on the shelves of every department of histopathology.

H THOMPSON

**Current hepatology** (volume 10). Edited by Gary Gitnick. (Pp 357; illustrated; £60.) Chicago: Year Book Medical Publishers, 1990.

This is the 10th volume in this annual series edited by Gary Gitnick. The series aims to provide the reader with an overview of the world's published reports of the previous year in each of the major areas of liver disease. The need for annual reviews can be gauged from the introductory comments in the chapter on cirrhosis and portal hypertension, in which the authors refer to 500 articles pertaining to their topic, of which 139 were selected for review.

The reviews are generally well balanced, with a good mixture of science and clinical studies. Of particular note is the chapter reviewing Japanese reports. The remarkably increased incidence of hepatocellular carcinoma in that country and the prevalence of cryptogenic cirrhosis, or cirrhosis due to non-A, non-B chronic hepatitis, is a particular stimulus to workers in that country. So much of their data and the important scientific advances which have been made by them are published only in the Japanese journals. Many new advances are based on molecular biology, and for those who, like the author, have a struggle to keep up with the rapidly advancing techniques, the chapter on the molecular biology of liver disease can be thoroughly recommended. In recent years there has been a resurgence in the anatomy and pathophysiology of the liver, and the chapter reviewing recent advances here again makes very worthwhile reading.

This is a volume to be recommended to both clinical and research hepatologists.

ROGER WILLIAMS

**Inflammatory bowel disease and coeliac disease in children.** Edited by F Madziselimovic, B Herzog, A Burgin-Wolff. (Pp 199; illustrated; price not given.) Dordrecht, Netherlands: Kluwer Academic Publishers, 1990.

This slim volume records the proceedings of the International Falk Symposium on Paediatric and Surgical Gastroenterology held in Basel in 1989. The theme of the meeting, however, was restricted to inflammatory diseases of the gut and particularly Crohn's disease, ulcerative colitis, and coeliac disease. The book is divided into two sections. Section I is almost entirely devoted to Crohn's disease, and to a lesser extent ulcerative colitis, and section II is concerned with coeliac disease.

Section I makes a good start with a very readable summary of current understanding of the immune function of the gut and its postnatal development. The last part of the first chapter comments on the functional interaction between the immune system and nervous and endocrine systems. Potentially the most interesting area, it is disappointing in its