LETTERS TO THE EDITOR

Differential expression of cyclooxygenase 2 in human colorectal cancer

EDITOR,—We were puzzled by the recent paper by Dimberg and colleagues (Gut 1999;48:730–732) which reported that up-regulation of cyclooxygenase 2 (COX-2) protein expression was prominent in rectal adenocarcinomas compared with that in adenocarcinomas arising from the colon. “Low or undetectable levels of COX-2 protein expression” were demonstrated in 15 of 19 colonic adenocarcinomas located proximal to the rectum. Overall, upregulation of COX-2 protein expression was reported in only 56% of colorectal cancers.

Previous reports,1–3 which include one by the current authors on a not dissimilar case series,1 and two in the joint authorship of the accompanying commentary writer,2 have shown consistent upregulation of COX-2 expression in colonic and rectal adenocarcinomas (in 85–90% of cases) compared with matched normal colonic mucosa using different techniques, including northern blot analysis, RT-PCR, western blot analysis, and immunohistochemistry. Furthermore, four of these studies refer to the distribution of adenocarcinomas throughout the colon without showing evidence of differential COX-2 expression between rectal and more proximal tumours.4–5 In the one previous study which analysed COX-2 protein expression in human colorectal cancers by western blot analysis,6 immunoreactive COX-2 was detected in 76% of cases with a 10-fold increase in median tissue COX-2 concentration compared with normal colonic mucosa.

In our view, the authors should attempt to explain the discrepancy between their results and previously published data. It is interesting to note that, in the study of Kargman et al., five of six patients taking NSAIDs had low or undetectable COX-2 protein expression.1 Moreover, COX-2 has recently been shown to suppress induction of COX-2 mRNA and protein in interleukin-1β and phorbol ester stimulated human endothelial cells and fibroblasts.7 Do the authors have data on NSAID use in their cohort of patients prior to surgery?

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Reply

EDITOR,—We agree with Drs Hull and Langman that we found upregulation of COX-2 protein expression in a lower fraction of colorectal cancers (CRC) than previously reported. In part, this may simply be explained by the composition of different tumour types within CRC—that is, the number of colonic versus rectal tumours in our cohort comprises of different tumours. In the papers referred to it is difficult to assess the fraction of the different tumour types studied. The differences may also be dependent on the genetic basis for the CRCs studied, which we also have indicated but perhaps not emphasised sufficiently. CRCs with a defective mismatch repair capability, recognised by microsatellite instability (MSI), are accompanied by reduced COX-2 levels.1 At present, we do not know the fraction of MSI type tumours in our series and therefore cannot assess this possibility. An indirect estimate may be achieved by using the Mi m mouse model and human studies provide direct evidence that COX-2 expression may be related to loss of APC function.2 APC and β-catenin muta-


Proton pump inhibitors for Barrett’s oesophagus

EDITOR,—Recently, the authors of two leading articles, Triadafilopoulos (Gut 2000;46:144–46) and Shepherd (Gut 2000;46:147–49) referred to our paper.1 We would like to draw attention to the fact that the legend in tables 4 and 5 in our paper should be read as (cm.month), (squares.month), and (%.month) since the variable is the area under the curve (AUC), which is the product of length and surface


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time. The printed notation (with a slash) might suggest that the figures concern the change per month. In spite of our suggested change in the galley proof, this notation was maintained. Nevertheless, it does not change the purport of our conclusion, nor the discussion in both leading articles.

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1 Peters FT, Gansh E, Kupers EJ, et al. Endo-
scopic resection of Barrett’s oesophagus duodenal treatment: a randomised

MCP-3 in inflammatory bowel disease

Editor,—We read with interest the article by Wedemeyer and colleagues (Gut 1999; 44:629–35) on chemokines in inflammatory bowel disease.

Monocyte chemotactic protein 3 (MCP-3) expression in inflammatory bowel diseases is a very interesting observation and we agree with the authors that MCP-3 might play an important role in the pathophysiology of these diseases.

We have recently published an article on the C-X-C chemokines interleukin (IL)-8 and IP-10, and the C-C chemokines MCP-1 and MCP-3 in the mucosa of active ulcerative colitis. It concerns an immunohistochemical study in which we showed increased expression of these chemokines in the lamina propria of patients with ulcerative colitis compared with normal controls. Furthermore, we observed a significant difference in expression between active and moderate/severe ulcerative colitis based on the histological grading in MCP-1, MCP-3, and IL-8.

Wedemeyer and colleagues state in their discussion that MCP-1 is expressed in the epithelial cells and lamina propria whereas MCP-3 is almost exclusively produced by epithelial cells. However, in the results section and further in the discussion the authors mentioned spurious MCP-3 expression in the lamina propria of inflamed tissue. The photographs show only epithelial cells and it is not possible to see the staining pattern of the lamina propria.

We found MCP-3 expressing cells in the lamina propria which was significantly increased in active ulcerative colitis compared with both inactive ulcerative colitis and normal controls. Furthermore, MCP-3 expression in lamina propria was also enhanced in patients suffering from pouchitis compared with patients with a normal pouch (unpublished data).

In the study of Wedemeyer et al, unfortunately the data on MCP-3 expression in Crohn’s disease were not significant which might be because of the small number of patients examined. It would be interesting to further evaluate the role of chemokines in Crohn’s disease.

In conclusion, albeit with some minor differences, both studies have shown that MCP-3 plays an important role in ulcerative colitis.

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Reply

Editor,—We thank Dr Helwig and colleagues for their interest in our recent paper in which we showed enhanced expression of the C-C chemokine MCP-3 in inflammatory bowel disease mucosa. In the article by Ugucioni et al we noted their slightly different findings in terms of localisation of MCP-3 expression. Using different techniques (cystrostat and paraformaldehyde fixatives, different anti-MCP-3 antibodies) we found consistent expression of MCP-3 in the lamina propria of patients with ulcerative colitis compared with normal controls. Furthermore, MCP-3 expression in the lamina propria of inflamed tissue is not possible to see the staining pattern of the figures concern the change per month. In spite of our suggested change in the galley proof, this notation was maintained. Nevertheless, it does not change the purport of our conclusion, nor the discussion in both leading articles.

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Thalidomide treatment of oesophageal ulceration

EDITOR,—Iread with interest the case report concerning oesophageal ulceration in Crohn's disease. We wish to report the results of a survey to evaluate the status of this disease. A world wide survey to evaluate the status of this disease

Thalidomide treatment of oesophageal ulceration

EDITOR,—We read with interest the article by Maconi et al (Gut 1996:38:936) on the use of PAIR. We found it encouraging that other workers are interested in superior mesenteric artery Doppler ultrasound in Crohn's disease. Our group has been working on the subject for several years. We found it surprising that according to the literature, SMA flow does not correlate with disease activity. Firstly, disease activity needs to be defined. The Crohn's disease activity index does not correlate with disease activity in individual patients and the reference standard used by Maconi et al is probably not a reliable indicator for disease activity. Secondly, it is not correct in our view to correlate the resistant index in one article with mean velocity in another and flow volume in yet another, and make the statement "yielding conflicting results" on page 654. In our opinion only flow volume measurements can be used as a reliable indicator. The fact that Maconi et al did not find a correlation between SMA volume flow and disease activity is probably caused by their choice of reference standard, as pointed out by Kielidou and colleagues, and van Oostayen and colleagues.

Use of Doppler ultrasound in Crohn's disease

EDITOR,—We read with interest the article by Maconi et al (Gut 1996:38:936) on the use of PAIR. We found it encouraging that other workers are interested in superior mesenteric artery Doppler ultrasound in Crohn's disease. Our group has been working on the subject for several years. We found it surprising that according to the literature, SMA flow does not correlate with disease activity. Firstly, disease activity needs to be defined. The Crohn's disease activity index does not correlate with disease activity in individual patients and the reference standard used by Maconi et al is probably not a reliable indicator for disease activity. Secondly, it is not correct in our view to correlate the resistant index in one article with mean velocity in another and flow volume in yet another, and make the statement "yielding conflicting results" on page 654. In our opinion only flow volume measurements can be used as a reliable indicator. The fact that Maconi et al did not find a correlation between SMA volume flow and disease activity is probably caused by their choice of reference standard, as pointed out by Kielidou and colleagues, and van Oostayen and colleagues.

Percutaneous drainage of echnococcal cysts (PAIR—puncture, aspiration, injection, reaspiration): results of a worldwide survey for assessment of its safety and efficacy

EDITOR,—In 1996 a letter (Gut 1996:38:936) on the use of PAIR was published. We found it surprising that according to the literature, SMA flow does not correlate with disease activity. Firstly, disease activity needs to be defined. The Crohn's disease activity index does not correlate with disease activity in individual patients and the reference standard used by Maconi et al is probably not a reliable indicator for disease activity. Secondly, it is not correct in our view to correlate the resistant index in one article with mean velocity in another and flow volume in yet another, and make the statement "yielding conflicting results" on page 654. In our opinion only flow volume measurements can be used as a reliable indicator. The fact that Maconi et al did not find a correlation between SMA volume flow and disease activity is probably caused by their choice of reference standard, as pointed out by Kielidou and colleagues, and van Oostayen and colleagues.

At the same time the WHO Informal Working Group on Echinococcosis launched a procedure to evaluate the status of this procedure. A number of centres around the world known to be active in this field were requested to complete forms for patients treated with PAIR: 765 abdominal cysts, mostly hepatic, treated with this technique were reported from various countries. We report the results of this survey (table 1).

| Total cases (cysts) | Follow up >5 y | Follow up <5 y | Major complications | Anaphylactic shock | Spillage | Minor complications | Fever (33), rash (14), pain (30), infection of cavity (11), nausea and vomiting (10), intracranial haemorrhage (3), hypotension (2) | Recurrences |
|---------------------|----------------|----------------|---------------------|-------------------|-------|---------------------|------------------|---------------------|---------------------|
|                     |                |                |                     |                   |       |                     |                   |                     |                     |
| 765                 | 77             | 690            | 4 (0.52%)           | 1 (0.13%)         |       | 4 (0.52%)           | 105 (13.7%)       | 2 (0.26%)           | 12 (1.57%)          |
|                     |                |                |                     |                   |       |                     |                   |                     |                     |

5 Cutler AE. Testing for H pylori in clinical practice. Am J Gastroenterol 1994;89:5-41S.
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BOOK REVIEWS


I should say immediately that this is an excellent book. For those interested in mucosal immunology, little more is necessary. It comprises an up to date and comprehensive series of 13 reviews by scientists who have made important contributions to the field. I am very pleased to have a copy; it will be extremely useful.

Clinical gastroenterologists spend a great deal of their time battling with mucosal T cells, yet because these cells are too small to be seen with an endoscope (in any case they would be obscured in exudate or by the epithelial cell layer) and are difficult to stain on formalin fixed histopathology sections, they are rarely observed. The weapons used against these adversaries are principally non-specific drugs which have, obviously, worked if the patient gets better.

Thus, although it is tempting to take Sherlock Holmes' attitude and, when told by Watson that the earth revolves around the sun, feel that the mind is an attic that when filled with details of astronomy (or mucosal immunology), will leave no space for the more useful minutiae of Egyptian tobacco (or the indications for the large circular metal stent). But Holmes liked to have a comprehensive grasp of the background of the case, and I believe that he would not have missed a chance to study this book had he been a contemporary gastroenterologist.

The language of the book may be a problem for the non-immunologist, particularly if one's medical school notes stop at the Bursa of Fabricius. This is certainly state of the art immunology, but is directed at clinicians as well as scientists. Therefore, if you want to know more about current developments in inflammatory bowel disease, coeliac disease, or HIV, or you are just curious about what some of those cells that you see in biopsy samples might be doing, I strongly recommend that you invest some money in a copy of this book and some effort in reading it. Furthermore, I suggest beginning with the chapter on "Mouse models of gut inflammation"—such models may not be identical to human inflammatory bowel disease, but at least they give us an opportunity to understand it.

And if you can’t remember what CD25 is? Get a copy of Immunobiology by Janeway and Travers (3rd Edition; Current Biology Ltd, 1997); this is another excellent book where no previous knowledge is assumed. There you are—two rave reviews—or three if you count A Study in Scarlet.

A J S MACPHERSON


This is a collection of work by 31 predominantly North American, European, and Japanese gastroenterologists, digestive surgeons,
and radiologists. The list of authors includes leading figures in the field of digestive endosonography, namely those who took part in the development of the first pieces of equipment and who described the basic principles of endoscopic ultrasound, and the new generation of practitioners responsible for the most recent developments in this area, particularly the introduction of the endoscopic ultrasound guided puncture. This collective work is complete and exhaustive, it is in large format and divided into seven sections, supplemented by a very detailed and helpful index.

The book is a popular work and the teaching material it contains is very practical, detailed, and useful for beginners. However, the book relies on the experiences of the expert authors, which I find to be of much less interest. Much of their experience is now out of date, and endoscopic images are grouped at the beginning of the book and reproduced in black and white in appropriate chapters.

In summary, this is a book of high quality work with some good illustrations. The division between the technical sections and those on anatomy is well balanced, which is original to this type of work and is very informative. A number of chapters are extremely useful, particularly those on the linear array echoendoscope and portal hypertension. Some areas covered have less impact, particularly those concerned with the authors’ different experiences of gastrointestinal and retroperitoneal pathology. Overall, the chapter on retroperitoneal endosonography is poorly covered; biliary echoendoscopy is not discussed at all. This significant gap is an invitation to other authors to publish a work dedicated to bilipancreatic echoendoscopy.

Useful supplement to the work of doctors van Dam and Sivak.

I. PALAZZO


It seems almost unimaginable to me that, somewhere out there, exists a clinical gastroenterologist who would not want to own this book. Maybe I was destined to be the curator of the book review section of Gut just so that a review copy of this majestic atlas might come across my desk. What little effort it is to find words of praise for this tour de force of gastrointestinal radiology.

In one of the most delightfully understated introductions of the century, Reddy Mac-Sween writes that “...this volume brings credit to radiology as a discipline”. Oh yes indeed, and so very much more! Dr Vallance and selected colleagues have produced a book in which every single illustration (and there are many hundreds) is crystal clear. There are many radiological texts that are comprehensive, and there is a lesser number in which the pictures are clearly reproduced. There are few books indeed in which every picture credibly reveals the pathology in a totally convincing manner. I do not believe there is single illustration in this book that is not of a high order, and this applies equally to plain radiographs, barium studies, ultrasound, CT, MRI, angiography, or EUS.

Despite its visual excellence, there are idiosyncrasies. Quite what CT and MRI scans of parotid tumours are doing in a book of GI radiology quite escapes this reviewer. Less satisfactory still are some of the mini essays introducing each system. I suspect most readers will not be particularly enlightened by the two page essays that introduce each organ—too brief to say any more than most clinicians must surely know already. For example, who would learn much from: “Ileostomy enema. The distal small bowel may be examined satisfactorily in patients with an ileostomy by retrograde infusion of barium with or without air, introduced by Foley catheter.”

The essays are weak, but the legends and the figures are of exceptional quality. A well constructed legend obviates the need for arrows, or other marks, on the radiograph. In this atlas, arrows do appear from time to time, but they are not intrusive. I suggest that this atlas might very well be added to the extremely short list of books that every gastroenterologist should own.

IAN FORGACS

NOTES

Sir Frances Avery Jones British Society of Gastroenterology Research Award 2001

Applications are invited by the Education Committee of the British Society of Gastroenterology who will recommend to Council the recipient of the 2001 Award. Applications (TEN COPIES) should include:

• A manuscript (2 A4 pages ONLY) describing the work conducted
• A bibliography of relevant personal publications
• An outline of the proposed content of the lecture, including title
• A written statement confirming that all or a substantial part of the work has been personally conducted in the UK or Eire.

An applicant need not be a member of the Society. The recipient will be required to deliver a 20 minute lecture at the Annual meeting of the Society in Glasgow in March 2001. Applications (TEN COPIES) should be made to the Endoscopy Section Secretary, British Society of Gastroenterology, 3 St Andrews Place, London NW1 4LB by 1 December 2000.

CORRECTIONS

An error occurred in figure 1 in the paper by Fisher et al (Gut 2000;46:534–539). Levels of protein C, protein S, antithrombin and factor VII were (infold too high throughout the manuscript.

In the Methods section, normal ranges for protein C, protein S, antithrombin and factor VII should have read 66–122 U/dl, 68–146 U/dl, 75–140 U/dl and 50–150 U/dl. Similar corrections should apply throughout the Results section and in the legend to figure 1. This was an editorial error for which Gut apologises.

An error occurred in figure 1 by Jeppesen and Mortensen (Gut 2000;46:701–706). The correct figure is published below. The correct figure appears on the Gut website (www.gut.jnl.com) and thus diverges from the print version of the May issue. We apologise for any confusion this error may have caused.

Figure 1 48 hour balance studies defining intestinal failure. Absorption of net weight and energy in relation to the basal metabolic rate (BMR) calculated by the Harris-Benedict equations in 44 patients managing without parenteral support (non-HPN patients, open circles) and in 45 patients depending on home parenteral nutrition (black triangles). The 5% confidence limits of the non-HPN patients, defining intestinal failure, are given by the lines. Energy absorption/BMR was 84% and wet weight absorption 1.41 kg/day.

British Society of Gastroenterology Hopkins Endoscopy Prize 2001

Applications are invited by the Endoscopy Committee of the British Society of Gastroenterology who will recommend to the Council the recipient of the 2001 Award. Applications (TEN COPIES) should include:

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