Letters

Reply

EDITOR,—We are glad that Dr Jolobe agreed with the importance of considering coeliac disease in patients with iron deficiency anaemia, even when symptomsatology is atypical. The precaution of taking endoscopic biopsy specimens prevents oversight of the diagnosis. We also note his comment on the abnormal appearance of duodenal folds in coeliac disease. We indeed routinely comment on such abnormalities having previously published evidence supporting the data of Brocchi et al.

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The Nm23 gene and colorectal cancer

EDITOR,—In the leading article Molecular biology of colorectal neoplasia (Gut 1993; 34: 289-92), the authors quote a report suggesting that Nm23 allelic deletion may prove to be a useful prognostic indicator in colorectal cancer.1 Cohn et al found that 73% of 11 large bowel cancer patients whose tumours had lost the Nm23 allele subsequently developed metastatic liver disease.

We also investigated allele loss at the Nm23 locus in 80 colorectal carcinomas using the same methodology, but could not detect allelic deletion in any of the 34 samples that were informative for this genetic marker (unpublished data). Myerhoff and Markowitz, using a ribonuclease protection assay, could not detect a single mutation in the Nm23 gene in 26 metastatic colon cancers, 17 non-metastatic colon cancers, or 43 matched normal controls. In addition, in this study, Nm23 gene expression was raised almost as often in metastatic as in non-metastatic cancers.2 They conclude that metastasis suppression by the Nm23 gene is a tissue specific phenomenon that does not play an important part in colorectal carcinogenesis. This study in conjunction with our own findings fails to support a possible role for Nm23 inactivation as a predictor of distant metastasis in colorectal cancer.

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Plateau activating factor and Crohn’s disease

EDITOR,—We read with interest the work of Ferraris et al (Gut 1993; 34: 665-8) reporting the contribution of intestinal epithelial cells to the enhanced generation of plateau activating factor (PAF) in ulcerative colitis. They emphasise that while PAF contributes to the pathogenesis of inflammatory damages in ulcerative colitis, it has no implication in Crohn’s disease.

We are very surprised by this conclusion. Ferraris et al can only assume that, in their in vitro model, intestinal epithelial cells or cells of the lamina propria of Crohn’s disease patients cannot produce more PAF than those of healthy controls. These data do not argue against a role for PAF in Crohn’s disease. For instance, other authors have reported increased contents of PAF in ileal and colonic mucosa of Crohn’s disease patients compared with controls.3 Furthermore, other data have reported PAF in the stool of patients with Crohn’s disease,4 and the deconjugation of fecal PAF contents after clinical remission.5 At this time, it is still unclear if PAF plays an important part, or is only a non-specific marker of inflammation, in Crohn’s disease. The use of PAF receptor antagonists during inflammatory bowel disease may bring interesting data about the role of this mediator.

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REFERENCES


Reply

EDITOR,—We are familiar with the work of Denizot et al on platelet activating factor (PAF) in Crohn’s disease. We completely agree with them that the exact role of PAF in inflammatory bowel disease is still unclear and that the use of PAF receptor antagonists in clinical settings might help to clarify this issue. We expected that if PAF’s contribution to the pathogenesis of Crohn’s disease is significant, its generation by both epithelial and lamina propria mononuclear cells would be enhanced in a similar fashion as we have reported for ulcerative colitis. As this was not the case, we assumed and stated that PAF’s implication in Crohn’s disease is less pronounced than in ulcerative colitis. We were cautious not to state that PAF has no implication in the pathogenesis of Crohn’s disease.

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