Correspondence

Intestinal permeability

SIR,—Judging from recent review articles on the subject of intestinal permeability, it seems to have become the fashion to ignore completely the original contributions made by Dr Axon and successive coworkers in Leeds. I was, therefore, not too surprised to see the recent article by Ramage and colleagues (*Gut* 1988; **29:** 57–61) which covered very similar ground (albeit with different probe molecules) to a study on intestinal permeability changes in an experimental rat model reported some time ago.[1] After all, it is always important to see confirmatory studies and there were some interesting new aspects in the recent paper. I was surprised, however, to see that Ramage and colleagues (and presumably the referees of their paper) did not think it necessary to acknowledge the existence of such a closely related study, which was one of the first to describe the use of the Nippostrongylus infected rat as an experimental model for studying passive intestinal permeability, perhaps it appeared in too obscure a journal?

In their discussion, they hint that future experiments on animal models might yield further information on the relationship between structural damage and intestinal permeability. Perhaps if they were to scan other obscure journals,[2,3] they might find some help.

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References

1 Cobden I, Rothwell J, Axon ATR. Intestinal permeability in rats infected by *Nippostrongylus brasiliensis*. (*Gut* 1979; **20:** 716–21).
2 Cobden I, Rothwell J, Axon ATR. Passive permeability in experimental intestinal damage in rats. *Clin Sci* 1981; **90:** 115–8.
3 Sandhu JS, Frazer DR. Effect of dietary cereals on intestinal permeability in experimental enteropathy in rats. *Gut* 1983; **24:** 825–30.

Activity of phospholipase A2

SIR,—We read with interest the paper by Otamiri *et al* (*Gut* 1987; **28:** 1445-53) concerning the activity of phospholipase A2. The authors suggest that the increase in enzyme activity may be the result of a cellular influx of calcium ions or that the ischaemia may have inactivated an endogenous phospholipase A2 inhibitor.

It has been shown in a rat model[1] that platelet activation results in a profound increase in phospholipase A2 activity. Our work on experimental acute pancreatitis in the rat confirms this finding.[2] The authors make no reference to the administration of anticoagulants in their experimental protocol; we therefore suggest that platelet activation may have made a significant contribution to the measured phospholipase activity.

The differences between plasma and serum phospholipase A2 activity in the rat are not seen in man. This model therefore may be of limited relevance to the clinical problems associated with intestinal ischaemia.

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References

1 Etienne J, Gruber A, Polonowski J. Activite phospholipasique A2 du serum de rat – Association de deux proteines. *Biochim Biophys Acta* 1980; **619:** 693–8.
2 Bird NC, Peng SY, Goodman AJ, Johnson AG. Phospholipase A2 activity in taurocholate-induced acute pancreatitis in the rat model. *Int J Pancreatol* (in press).

Lymphokine activated killer cell activity in patients with GI cancer

SIR,—Several fundamental aspects of tumour immunology are important to the interpretation of the valuable article by J R T Monson *et al* (*Gut* 1987; **28:** 1420–5) on lymphokine activated killer (LAK) cell activity in patients with gastrointestinal carcinoma.

The study confined itself to peripheral blood (PB) lymphocytes, the relevance of which has been brought into serious doubt by the work of Vose and Moore[1] and Holmes,[2] indicating that PB lymphocyte activity and type bear little similarity to the immune-tumour relationship and micro-environment *in vivo*. It is also important to bear in mind that cytotoxic effector lymphocytes in tissues would appear to be different in number and type to those in peripheral blood.

Of the many theories thought to underlie down regulation of the immune system in carcinoma patients, that of longterm specific cellular immunosuppression, relating to cancer cell minor histocompatibility antigen expression has been shown to be the case *in vitro*.[3] This, in association with the idiotype network concept of immune function[4] may be an explanation for the inability of most patients