The important difference between these two studies is that Gaslander et al found a raised plasma cholecystokinin concentration in rats with duodenal gastric reflux (at two weeks and six weeks), whereas we found a normal plasma cholecystokinin concentration (at six months). Basechol cholecystokinin receptor antagonist devazepide did not completely inhibit the trophic effect of the operation, they suggest that gastrin may also be important as an intermediary, our own data clearly support this interpretation. In the context of promoting neoplasia, the longer term gastrin may be more important as the relevant cholecystokinin receptor agonist longlumide completely abolished the effect of this operation on pancreatic growth. We have previously speculated on the relation between these two hormones. The strongest stimulus to pancreatic growth and carcinogenesis in our experience has been pancreatobiliary diversion; here again cholecystokinin seems to be the key intermediary, and longlumide prevents the response.6

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We have reviewed some data that were published in the study by Herrington et al.10 In their study, they found that partial gastrectomy, which is correlated with reduced cholecystokinin concentrations, was associated with a decrease in proliferative activity of the intestinal crypts. However, we believe that these findings cannot be generalized to all gastrointestinal conditions, as the role of cholecystokinin in the regulation of proliferation and tumor initiation may vary depending on the specific condition.

Reply

EDITOR.—We appreciate the interest that Professors Williamson and Watanapa have shown in our paper and apologise for failing to reference the important contributions they have made in the field of pancreatic growth and carcinogenesis. These authors suggest that an important difference between their study and ours is that we saw an increase in circulating cholecystokinin concentrations and they did not. In fact, we also observed increased cholecystokinin concentrations in their study at 6 weeks in animals with split gastrojejunostomy than controls, although this did not reach statistical significance (P = 0.06). Further studies with a larger series is probable that this would have reached statistical significance. Furthermore, in their study of partial gastrectomy, basechol cholecystokinin concentrations were only 46% above control and lower than those seen in their split gastrojejunostomy group, but here they concluded that partial gastrectomy increases plasma cholecystokinin.2 It is interesting to note that humans with long-standing pancreatic reflux have increased concentrations of cholecystokinin postprandially, although their gastrin concentrations are quite normal.3 Although gastrin concentrations were increased after gastrojejunostomy, we do not feel that this hormone alone is responsible for the pancreatic growth as the trophic effect is not mimicked by omeprazole treatment even though gastrin concentrations are higher than after split gastrojejunostomy and duodenogastrostomy.4

We do not fully agree with the conclusion drawn that the study of distal gastrectomy in rats confirms that cholecystokinin may play a part in the increased incidence of pancreatic cancer, as they stated that this study showed no trophic effect on the pancreas was seen. Furthermore, while we agree that these hormones are important for pancreatic growth and neoplasia in the rat, their role in human pancreatic cancer, the common tumour type in humans, is controversial.4,5

The authors go on to state that they found two candidate hormones, enteroglucagon and gastrin, for the pancreaticotrophic effect associated with the increase in small bowel resection and pancreaticobiliary diversion. However, the increase of cholecystokinin and enteroglucagon and pancreatic growth associated with these surgical procedures had been described earlier.9,21

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Expression of adhesion molecules in human Peyer’s patches

In our opinion this hypothesis is not plausible. Lymphocytes in follicle associated epithelium are unevenly distributed, being particularly concentrated in small aggregates related to the ‘membrane’ cells; outside these foci the intraepithelial occurrence of lymphocytes is more similar to that seen in the villus epithelium in terms of numerical as well as phenotypic distribution.2,4 In humans the diffuse, scattered intraepithelial lymphocytes are mainly CD8+ T cells9;8 of the T cell receptor β+ variety with a small (4–5%) admixture of T cell receptor αβ+ cells. The only adhesion molecule suggested to be important for their homing to the epithelium is the integrin αEβ7 (detected by monoclonal antibody HML-1),10 which apparently binds to a glycosylated E-cadherin. The lymphocytes found in relation to the membrane cells might rather be ascribed to the antigen transporting capacity of these special “tumour cells. In fact, the 50% of such aggregated lymphocytes consist of B cells—apparently representing topical extensions of the underlying follicles— together with a comparatively high proportion of CD4+ CD38+ T cells (memory-helper phenotype),12 but without admixture of the T cell receptor γδ+ subset. It is indeed difficult