Luminal nutrition and gut growth  

EDITOR,—We read with considerable interest the article by Jenkins and Thompson on the influence of enteral nutrition on the growth of the small intestine of the rat (J Anat 1971; 132: 167-74). We agree that the elucidation of the mechanisms by which the intestinal mucosa interacts with nutrients is a very important area of research. This may be of particular relevance in the context of the bacterial translocation hypothesis, and we are extremely interested in the element nutritional and hormonally mediated mechanisms. However, we know of little direct evidence to support their hypothesis. It has been proposed by Playford et al that the presence of undigested growth factors in the proximal intestinal lumen may cause the proximal mucosal hyperplasia seen after pancreaticobiliary diversion.9 This proposal cannot, though, explain why mucosal hyperplasia after pancreaticobiliary diversion is not observed, in many patients, even though their diet has been reduced by maintaining the animals on total parental nutrition.6 We agree that many issues remain to be resolved about the mechanisms that mediate the trophic effects of luminal nutrients and hope that our article will stimulate further interest in this important area.

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8 Pell JD, Johnson TT, Goodlad RA. The effects of, and interactions between fermentable dietary fibre and lipid in germ free and conventional mice. Gastroenterology (in press).

Reply

EDITOR,—We are interested to receive the comments of Dr Playford and colleagues and would like to respond to the points they make.

We accept that pancreaticobiliary diversion causes trophic changes in the proximal (enzyme free) section of intestine, as well as distal to the site of diversion. One explanation for this could be the reduced secretions, and also poorly digested fatty acids, stimulate release of a trophic peptide from the distal gut that causes mucosal hyperplasia proximally in the small intestine. Nevertheless, infusion of harvested pancreaticobiliary secretions into isolated intestinal loops shows that these secretions do themselves have a direct trophic action on the mucosa.1

We agree that the effects of a circulating hormone alone cannot account for the changes seen in jejunal transposition, where the transposed ileum undergoes hyperplasia and the transposed jejunal hypoplasia.2 The effects of luminal nutrients may, however, be relevant to some of these changes. The transplanted ileum will be exposed to an increased luminal load and the jejenum to a correspondingly reduced luminal load. These changes may make a more important contribution to jejunal growth than hormonally mediated effects.

Although the study quoted by Dr Playford and colleagues (their reference 6) shows that an elemental diet does not maintain mucosal growth compared with a standard laboratory diet (chow), it also shows that the elemental diet stimulates growth in the proximal small intestine compared with total parental nutrition. The contents of the elemental diet do themselves have a trophic effect on the small intestine. It is true that this trophic action may be less than that of the standard laboratory diet, but this may not be because the elemental diet contains pure amino acids while the laboratory diet contains whole protein. The diets differ in many other ways, not least of these being the absence of bacterial translocation in the elemental diet and the presence in the standard laboratory diet. Moreover, studies quoted in our article prove that amino acids do themselves have trophic effects on the small intestinal mucosa.4

The suggestion by Dr Playford and colleagues that a semilabile luminal trophic agent may be responsible for adaptive changes to diet is interesting and is not incompatible with the proposal that luminal nutrients also stimulate mucosal growth through gut hormones, and hormonally mediated mechanisms. However, we know of little direct evidence to support their hypothesis. It has been proposed by Playford et al that the presence of undigested growth factors in the proximal intestinal lumen may cause the proximal mucosal hyperplasia seen after pancreaticobiliary diversion.9 This proposal cannot, though, explain why mucosal hyperplasia after pancreaticobiliary diversion is not observed, in many patients, even though their diet has been reduced by maintaining the animals on total parental nutrition.6 We agree that many issues remain to be resolved about the mechanisms that mediate the trophic effects of luminal nutrients and hope that our article will stimulate further interest in this important area.

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Duodenogastric reflux and pancreatic growth

EDITOR,—In their interesting paper (Gut 1995; 36: 137-41), Dr Gasslander et al describe an increase in pancreatic wet weight and serum gastrin at two weeks and six weeks after split gastrojejunostomy in rats, an operation that produces complete duodenogastric reflux. They speculate that the trophic effect of duodenogastric reflux may contribute to the increased incidence of pancreatic cancer reported after gastric operations in humans. In our own study,1 which the authors do not cite, we showed an increase in pancreatic wet weight and serum gastrin six months after exactly the same operation in rats. Pursuing this hypothesis, we found that duodenogastric reflux promotes neoplasia—we further showed an appreciable increase in preneoplastic pancreatic lesions in rats receiving azaferine.