

# Gut Digest

Robin Spiller, Editor

## OE SOPHAGEAL EPITHELIAL PROTECTIVE MECHANISMS AGAINST ACID REFLUX

The basis of the resistance of oesophageal squamous epithelium to acid injury is unknown. In this issue, Tanaka and colleagues investigated this, measuring oesophageal mucosal blood flow using a laser doppler probe while simultaneously imaging the oesophageal mucosa using a fluorescence microscope and a pH-sensitive fluorescent dye to assess the interstitial pH. Using these techniques, they were able to show that the oesophageal mucosa was able to exclude an acid solution of pH 1.5 without any change of the interstitial pH while exposing the serosal layer to a mildly acidic solution rapidly produced acidification of the interstitial space. However, the most striking effect was an increase in blood flow in the oesophageal mucosa which is likely to be protective.

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## NON-INVASIVE SCREENING TESTS FOR *H PYLORI* INFECTION IN CHILDREN

Since *Helicobacter* infection is acquired in early childhood, it is logical to detect and treat it then. However, at the age when infection occurs toddlers are easily traumatised by medical investigations. Even the urea breath test can be difficult for a child. A stool test would circumvent all these problems and might have considerable appeal both for research and clinical practice. Koletzko *et al* studied 302 symptomatic children, testing the stool using a new monoclonal enzyme immunoassay, which detects *H pylori* antigen. The test had high sensitivity and specificity, and in this group a positive predictive value of 98% with a negative predictive value of 99%. Interestingly, one of the false positive results, came from a child infected with *Campylobacter jejuni* suggesting there may be some cross reactivity between the two bacterial species. Further tests need to be done to make sure that this will not produce an unacceptable false positive rate in tropical countries.

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## AN ANIMAL MODEL OF DUODENAL ULCER—THE FULFILMENT OF KOCH'S POSTULATES

*Helicobacter pylori* infection causes gastric ulceration and gastric adenocarcinoma in the Mongolian gerbil model. The landmark paper by Ohkusa *et al* shows that it can also induce duodenal ulceration, finally fulfilling Koch's postulates for *H pylori* as a cause of DU. The report describes the artificial infection of gerbils with three separate strains of *H pylori*. All three strains induced gastric ulceration but two also induced consistent gastric metaplasia in the duodenum and duodenitis. Two gerbils developed superficial duodenal ulceration, and it is important that this occurred on a background of gastric metaplasia, as in humans. However, the DUs did have atypical features, for example they were always accompanied by gastric ulceration. Further work is needed to determine whether their pathophysiology is similar to chronic duodenal ulceration in humans, and if so, to develop this model to make it more consistent and reliable.

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## CAN PROBIOTICS ENHANCE MUCOSAL DEFENCE?

There is considerable interest, currently in the use of probiotics to treat a range of gastrointestinal diseases. The idea

that one might be able to enhance mucosal protection is an attractive one. By incubating a range of lactobacilli with cell lines expressing a membrane bound mucin MUC 3, Mack *et al* were able to show a specific upregulation by *Lactobacillus plantarum*, strain 999V. Co-culture of enteropathogenic *E coli* (EPEC) with *L plantarum* reduced the adherence of EPEC. They were also able to show an increased secretion of MUC3, which is normally retained, anchored in enterocyte membranes. The possibilities now arise of using probiotics to increase secretion of protective glycoproteins, providing protection or even treatment of infection without the adverse effect of antibiotics on the normal gut flora

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## NUTRITIONAL INFLUENCES ON SURVIVAL FROM COLORECTAL CANCER

There is strong epidemiological evidence that high energy intake and specifically high intakes of fat, red meat, and refined sugar all increase the risk of developing colorectal cancer. However, nutritional effects on subsequent progression of colorectal cancer remain to be determined. Dray *et al*'s study of 148 patients with colorectal cancer unsurprisingly showed that tumour stage was the most important factor in predicting survival. However, energy intake was also a highly significant predictor, with a risk of death at 5 years of a fifth for those with high intake of energy compared with those with low. This benefit was particularly seen in those with more advanced tumours (Dukes C and D) in whom it reduced the risk 16 fold. The authors speculate that high energy intake with ample body stores of nutrients may prevent the impairment of immunity associated with cancer cachexia. These studies would support the notion that maintaining adequate nutritional intake is an important part of cancer follow up care.

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## PREDICTING SURVIVAL AFTER TIPS

The paper by Angermayr *et al* in this issue address an interesting and important question: should we still rely on the "old" Child-Pugh score to predict survival in patients with cirrhosis or move to the more recently proposed index, the MELD score? It is perhaps surprising that the CPS has survived so long and is still the most frequently used prognostic index in chronic liver disease based on five variables, 3 laboratory (serum bilirubin, albumin, and prothrombin time) and 2 clinical (ascites and encephalopathy). The MELD (model for end stage liver disease) score is the latest attempt to refine the CP by removing the more subjective clinical variables and using an index constructed from just bilirubin, INR and creatinine. Initial studies suggested it might have major benefits in predicting survival after TIPS and in chronic liver diseases. The current study compared CPS with a modified MELD in a single centre in a large number of patients undergoing TIPS. Surprisingly the authors found no major benefit of MELD over traditional CPS. There are weaknesses with the study, notably its retrospective design, but the results are of clinical interest and suggest that the optimal prognostic index in chronic liver disease is still some way off. Since prioritisation for liver transplantation in the USA is now based on MELD scores this paper also has potential social and legal implications.

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