Digest

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TIPS TO AVOID COLORECTAL CANCER

(1) Don't forget to check your own stools

Population screening for faecal occult blood reduces mortality from colorectal cancer. The finding is so well established that further trials seem unnecessary. However, if you do find yourself recruited to such a study and are randomised to the screening arm, do get your stools tested as the study dictates. Two studies in this month's *Gut* show that colorectal cancer mortality increases in those who fail to follow screening protocols assiduously.

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(2) Watch what you drink

Alcohol is really noxious to much of the digestive tract, although some organs bear the brunt of the damage. The large bowel may seem to have escaped the worst of the harm, yet some studies do indicate a weak association of alcohol with colorectal cancer. Now from the homeland of the world's greatest wines



Alcohol and colorectal cancer (pp 38-42).

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Double immunofluorescence staining for COX-2 and CD3 in sections from the small intestinal mucosal lesion of a coeliac patient (pp 84–9).

comes the news that heavy drinkers who already have a polyp run an increased risk of developing high risk colorectal adenomas and also cancers. *Note . . .it seems that the risk reflects the amount not the quality of the alcohol imbibed.*

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A SUBJECT OF INCREASING RESISTANCE

The clinical use of IV albumin is not without controversy. One role for albumin in patients with cirrhosis and tense ascites is to prevent post-paracentesis fall in arterial blood volume. If albumin use does cause concerns, vasoconstrictors might be an alternative therapy to counter the fall in arteriolar resistance induced by drainage of the ascites. A pilot study suggests that terlipressin may indeed be as effective as albumin in maintaining blood volume in this situation.

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COX-2 IN COELIAC DISEASE—A BLISTERING START

Coeliac disease is an inflammatory condition; COX-2 is induced by inflammation. It may reasonably be asked whether inflammatory cells in the coeliac small intestine express COX-2. In all 15 coeliac biopsies, COX-2 positive T cells were clustered where the epithelium seemed to blister or became detached. After dietary treatment the number of COX-2 cells decreased. Phenomenon or epiphenomenon?

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HOLY GRAILS AND CROHN'S DISEASE

No single clinical observation, investigation or predictive model predicts relapse of Crohn's disease after surgery. Can immunological markers assessed at time of operation identify those who are likely to relapse? Interleukin-10 (IL-10) is a major anti-inflammatory cytokine. Levels of IL-10 in ileal mucosa were significantly lower in those patients with (endoscopic) recurrence within three months of surgery. How better to prevent recurrence continues to be one of the holy grails of Crohn's disease. **See page 25**

NOT HAVING A RELAXING TIME

Transient relaxations of the lower oesophageal sphincter are the major mechanism for acid reflux—occurring both in normal individuals and those with reflux symptoms. Treatment directed towards the physiological defect seems attractive although the history of motility altering therapy in the oesophagus has been mostly littered with ineffective and/or toxic agents.

The GABA receptor, baclofen, inhibits transient relaxations and has now been shown to inhibit reflux episodes in patients with reflux disease. Perhaps surprisingly to those of us familiar with the drug in neurological disease, side effects were minimal.

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