developing fatal colorectal cancer. Aspirin seems to have immunostimulatory and tumouricidal properties in the colon \(^2\) thus accounting for the protection. It is these properties that may also make low dose aspirin useful in the chemoprevention of colorectal cancer in high risk patients. This is supported by the data of Manzano et al. Pre-neoplastic ulcerative colitis is associated with \(T\) lymphocyte immunosuppression and this may permit immunologically unchallenged malignant degeneration in a chronically inflamed mucosa. Aspirin may therefore be useful in the chemoprevention of colorectal cancer by augmenting the immune system thus destroying early tumours. This principle also applies in the oesophagus as there are several similarities between the carcinogenesis of colorectal and oesophageal cancer. Chronic inflammation may be a pre-neoplastic lesion in both organs \(^4\) while Barrett's oesophagus is also associated with immunosuppression. \(^3\) NSAIDs may thus be useful in the chemoprevention of both colorectal and oesophageal cancer in patients with pre-neoplastic disease. I agree with Choi and Zelig that long-term studies of low dose NSAID prophylaxis are warranted in patients receiving surveillance.

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Acid and gastric metaplasia in the duodenum

EDITOR,—I am writing with reference to the letter to the editor (Savarino et al Gut 1994; 35: 1151) and the reply concerning the paper by Noach et al (Gut 1993; 34: 1510–4). In this paper they support the concept that gastric metaplasia of the duodenum is a response to acid hypersecretion and may be a defence mechanism. \(^1,2\) An alternative concept is that it is a pathological change resulting in a loss of mucosal resistance to acid and pepsin. The small study that we reported comparing light and electron microscopy appearances of the duodenal mucosa in healed duodenal ulcer patients after one year's maintenance treatment with either cimetidine or sucralfate showed little change in the presence of gastric metaplasia and of Helicobacter pylori in 64% of the cimetidine group, and an appreciable reduction or absence of both in 73% of the sucralfate group. \(^3\) If gastric metaplasia were a response to acid the reverse would be expected, with a reduction in the group receiving cimetidine. Over the next two years the relapse rates were 69% and 18% respectively. Gastric metaplasia of the duodenal mucosa is an almost inevitable accompanying duodenal ulceration. In the absence of gastric metaplasia \(H\) pylori cannot survive in the duodenum. Opportunistic colonisation of the metaplastic mucosa by \(H\) pylori may be an additional factor either in the initial formation of an ulcer or in delayed healing or relapse. It is of interest that Noach et al reported no reduction in the extent and prevalence of gastric metaplasia after 12 months of eradication of \(H\) pylori. This means that these patients could be at risk of recolonisation and possible recurrence of duodenal ulceration. This emphasises the need for a treatment that will restore normal duodenal mucosa. Treatment with long-term sucralfate is one possibility, but alternatively there may be dietary factors that can achieve the same effect and thereby restore normal mucosal resistance. 

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Pathogenesis of bacterial colitis

EDITOR,—The leading article on the pathophysiology of bacterial colitis was timely and informative (Gut 1994; 35: 872–4). Some aspects of this fascinating topic may, however, warrant additional discussion.

As the authors pointed out, the cytotoxic toxins of \(Shigella dysenteriae\) type 1 and \(Escherichia coli\) O157:H7 are structurally...