

*Clarification of the link between polyunsaturated fatty acids and Helicobacter pylori-associated duodenal ulcer disease: a dietary intervention study*

It would have been interesting if Duggan *et al.* (1997), in their report in your journal, had been able to take duodenal biopsies to compare the presence and extent of duodenal gastric metaplasia between the subjects on a high- and on a low-polyunsaturated-fat diet. It would be of interest also to know whether those subjects who had active duodenal ulcers showed any endoscopic improvement as a result of taking a high-polyunsaturated-fat diet.

Duodenal gastric metaplasia is always found in association with duodenal ulceration and is a necessary prerequisite for *Helicobacter pylori* colonization of the duodenum from the antrum. The geographical distribution of duodenal ulceration, with the presence of areas of high and low prevalence, has shown a strong relationship with staple diets (Tovey & Tunstall, 1975; Tovey, 1979, 1992, 1994; Tovey *et al.* 1989). In India our own studies and those of others showed a high prevalence of duodenal ulcer in all of the polished-rice-eating areas, especially in the South and in Bangladesh, and a low prevalence in the unrefined-wheat-eating areas of the North, in particular in the Punjab. The prevalence was also low in isolated areas where certain millets, certain pulses or ragi (*Eleusine coracana*) formed a large part of the staple diet.

Our experiments on animal peptic ulcer models show that the North Indian Punjabi diet is protective against ulceration, whereas the South Indian diet is ulcerogenic (Jayaraj *et al.* 1980, 1986, 1987). This protective activity is present in certain foodstuffs such as unrefined wheat, soya, ragi and some millets and lentils. The lentil Horse Gram (*Dolichos biflorus*) is markedly protective. This protective activity has been shown to lie in the lipid fraction of these foodstuffs. This fraction contains essential fatty acids such as linoleic acid, phospholipids and phytosterols, all of which have been shown experimentally to enhance mucosal cytoprotection (Tarnawski *et al.* 1987; Lichtenberger *et al.* 1990; Romero & Lichtenberger, 1990).

From available evidence, *Helicobacter* infection of the antrum seems to be equally prevalent in the high- and low-duodenal-ulcer areas of India and is not a factor in explaining the differences in prevalence. It is possible that the presence of protective factors in diet may explain these differences. The protective factors may enhance the resistance of the duodenal mucosa, enabling it to withstand acid and pepsin, and consequently the development of gastric

metaplasia. To account for changes in duodenal ulcer prevalence it may be that we ought to be looking for the effect of diet on duodenal gastric metaplasia and not at any effect on the extent of *Helicobacter pylori* infection.

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*Clarification of the link between polyunsaturated fatty acids and Helicobacter pylori-associated duodenal ulcer disease: a dietary intervention study – reply by Duggan & Spiller*

We agree that the epidemiological data linking diet and duodenal ulcer disease quoted by Tovey & Hobsley (1998) have yet to be made clear. Our experimental studies show that very substantial amounts of PUFA in the diet do not

significantly alter *Helicobacter pylori* infection in the antrum. As Tovey and Hobsley point out, this still leaves a possibility that these diets are protective because of their impact on duodenal metaplasia which we did not examine.

**Reference**

Tovey FI & Hobsley M (1998) Clarification of the link between polyunsaturated fatty acids and *Helicobacter pylori*-associated duodenal ulcer disease: a dietary intervention study (letter). *British Journal of Nutrition* **80**, 116.

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