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Duodenal ulcer and carbohydrate

SIR,—We have read with interest the results of the study from Nottingham by Katschinski *et al* (*Gut* 1990; **31**: 993–6) concerning the association between duodenal ulceration and fibre and refined carbohydrate intake. The findings suggested that relative risks were reduced by a high vegetable fibre and low refined sugar intake but not a high intake of cereal fibre.

We have continued to gather information about the geographical distribution of duodenal ulceration and the staple diets of high and low incidence areas, and we find no correlation between duodenal ulcer incidence and fibre intake alone. There are high incidence areas in Ethiopia, Rwanda, and Burundi and in sorghum eating areas of India where the fibre intake is high.

The overall picture suggests that areas where polished rice, yams, or cassava are the staple foods the duodenal ulcer incidence is high. Where unrefined wheat, soya, some pulses or millets, or certain green vegetables form a large part of the staple diet the incidence is low.[1,2]

Experimental work on several animal models of peptic ulceration shows that the food substances mentioned above from low incidence areas contain a protective fraction which is liposoluble. The fraction is present in wheat bran but to a less degree in wheat germ.[3] We think that it is a protective factor present in certain high fibre foods and not the fibre itself that protects against duodenal ulceration.

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Gastric acid and urinary acid excretion

SIR,—Johnson *et al* (*Gut* 1990; **31**: 826–6) could not find a significant rise of urinary pH two hours after the start of a standard meal in normal subjects (despite a reduction in urine acid output) nor in patients after vagotomy. They concluded that changes in the rate of urinary acid output after a meal could not be detected by measuring pH because of the presence of buffers in normal urine. Their findings may reflect the inferiority of a standard meal to pentagastrin for maximal stimulation of gastric acid secretion.

We measured urinary pH in 14 duodenal ulcer patients with no vagotomy (group A) and in 14 patients after vagotomy (group B), before and two hours after pentagastrin 6 μg/kg subcutaneously. Median pH of basal urine in group A was 4·9 (range 3·9–5·7). Two hours after the meal the corresponding values were 6·2 (5·2–7·0). In group B preprandial and postprandial urine pH was 5·3 (4·7–7·0) and 5·3 (4·5–7·0).

Thus the conclusion drawn by Johnson *et al* is correct for pentagastrin stimulation after vagotomy, but not for duodenal ulcer patients who have not had a vagotomy.

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Reply

SIR,—Thank you for the opportunity to reply to these comments. Like us, Dr Niv and colleagues were unable to show a true 'alkaline' tide in the urine after gastric stimulation because their pH values were all 7 or less. Nevertheless, they showed an alkalinisation of the urine, as measured by rising pH in duodenal ulcer patients after pentagastrin stimulation, and were unable to show this effect in duodenal ulcer patients after vagotomy.

It would be interesting to know whether these measurements were taken during a standard gastric secretory study, with aspiration of gastric contents. This manoeuvre will clearly render the body's acid-base balance more alkaline by the removal of the gastric acid secretion. Perhaps this explains why they were able to show a change in pH in non-vagotomised ulcer patients. We maintain, however, that pH is a much less sensitive indicator of the amount of acid excreted in the urine than is direct titration of the acid content of the urine.

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Lipid pattern and plasma insulin in diabetics with gall stones

SIR,—We read with great interest the paper by Laakso and colleagues (*Gut* 1990; **31**: 344–7) regarding the relation between serum lipids, plasma insulin, and gall stones in non-insulin dependent diabetic women. The authors suggest that diabetics with gall stone disease have higher fasting insulin concentrations and lower total and low density lipoprotein cholesterol than diabetics without gall stones.

In the introduction they state that no studies have been published comparing lipids and lipoproteins in diabetics with or without gall stones. Some years ago we reported different results on the relations between gall stones and serum lipids in non-insulin dependent diabetic patients. We studied total cholesterol, serum triglycerides, and apolipoproteins A I and B in 81 subjects with non-insulin dependent diabetes mellitus affected by gall stones and 305 diabetics without gall stone disease.[1] We documented increased concentrations of triglycerides and decreased values of apolipoproteins A I in diabetic women with gall stones compared with those without, while no difference was shown in men. Total cholesterol and apolipoprotein B concentrations did not differ between groups. The observation of high concentrations of triglycerides in gall stones has been reported by most authors; our experience is in agreement with published papers and conflicts with the data reported by Laakso. Our finding of such an association only in women agrees with the observation of more severe lipid alteration in women with diabetes[2] and of an association of gall stones, low concentrations of high density lipoprotein cholesterol, and coronary disease found only in women.[3]

The serum lipid pattern in our patients might be related to increased serum insulin concentrations, as suggested by Laakso *et al* in another paper.[4] In this regard, in a case-control study (34 patients with gall stones and non-insulin dependent diabetes mellitus *v* 30 controls without gall stones, comparable for sex, age, body mass index, and duration and metabolic control of diabetes) we also documented increased values of C peptide in subjects with gall stones compared with controls.[5]

Concerning the possible mechanism by which hyperinsulinaemia could enhance gall stone formation, Laakso *et al* report that high insulin concentrations could activate low density lipoprotein receptors with an increased plasma-bile clearance of low density lipoprotein cholesterol. It, however, has also been reported that insulin is able to enhance the activity of ß-hydroxy ß-methylglutaryl coenzyme A reductase[6] and to suppress 7 alpha-hydroxylase[7] with consequent increased cholesterol and decreased bile acid secretion in bile. According to this finding Bennion and Grundy[8] showed that insulin administration in non-insulin dependent diabetics could increase cholesterol saturation of bile. In a preliminary retrospective evaluation of 386 subjects with non-insulin dependent diabetes mellitus we showed a significantly higher frequency of gall stones in patients treated with insulin compared with those being managed by diet or oral hypoglycaemic agents.[9] This finding seems to support the hypothesis of an increased risk of gall stones in diabetics treated with insulin, but prospective investigations on this topic are necessary.

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