Diet and drinking habits in alcoholic pancreatitis

SIR,—In the paper by Wilson *et al* (*Gut* 1985; 26: 882–87) they concluded that there was no significant difference between the dietary and drinking habits of patients with clinically evident alcoholic pancreatitis and patients with clinically evident alcoholic cirrhosis. The purpose of this paper was to determine 'whether increased intake of fat and protein or particular drinking habits are associated with the development of alcoholic pancreatitis'.

Durbec *et al*¹ have compared 440 European patients with chronic calcifying pancreatitis with two different populations: normal controls and patients with functional disturbances. The patients were selected to be questioned on their dietary habits before their first symptoms. The conclusion of this work was that the log of the relative risk of chronic calcifying pancreatitis (CCP) was linearly correlated with the average daily intake of alcohol and of protein and that the lowest risk was observed with average fat diet (80–110 g/d), a higher risk with low fat diets and the highest risk with high fat diet. These different factors were found to be additive on the log of the risk. The role of high fat, high protein diet has been confirmed in Italy, Brazil, Mexico and Germany².

The paper from Wilson *et al* suffers some critics: (1) To determine the possible nutritional factor of alcoholic chronic pancreatitis, it would have been necessary to compare patients with a normal population and not to a group of patients (alcoholic cirrhosis) who could have modified their dietary habits in the same way as patients with pancreatitis. (2) The number of patients is very low compared with all previous studies—30 patients with pancreatitis compared with 33 alcoholic cirrhosis. (3) Wilson *et al* found that patients with pancreatitis consumed 10% more fat and 17% more protein than cirrhotics. The fact that this difference was not significant was probably because of the small number of subjects, and although these differences were significant, when the entire groups were compared and age and sex were adjusted these differences were not seen. This has certainly reduced the number of patients.

Therefore the only conclusion which can be drawn from this paper is that the number of cases was insufficient, and that a control group was necessary.

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References

- 1 Durbec JP, Sarles H. Multicenter survey of the etiology of pancreatic diseases. *Digestion* 1978; 18: 337–50.
- 2 Sarles H. In: Glass J, Sherlock S, eds. *Progress in gastroenterology* vol. 4. New York: Grune and Stratton, 1984.

Reply

SIR,—Thank you for the opportunity to respond to Professor Sarles. He suggests the use of a normal population as controls for the index patients with alcoholic pancreatitis. The problem with this is that it involves the study of two variables—alcohol intake and the presence or absence of pancreatitis. To overcome this difficulty, we used controls who had alcoholic cirrhosis. Thus, all our patients were alcoholics of sufficient severity to develop clinically evident damage of either the pancreas or the liver.

We also agree that our numbers were small and that if we had enlarged the study, we may well have come up with statistically significant results. The differences in recorded food intake were quite small, however, and incidentally, comparable with those reported by Sarles and coworkers. As we stated in our paper, we found it difficult to envisage how eating 10% more protein and 17% more fat (whether statistically significant or not) would predispose to pancreatitis. Of course, this is a clinical 'hunch' which cannot be proven or disproven but a mere consideration of the huge variations in food intake of alcoholics with only the rare development of pancreatitis prompts us to hold this opinion.

Finally, we would like to point out that we made every effort to obtain premorbid dietary information. It is not clear whether similar efforts were made by Professor Sarles and his colleagues. Hyperphagia is often encountered in patients with chronic pancreatitis who suffer from malabsorption and such increased dietary intakes clearly result from the disease rather than cause it.

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