LETTERS TO THE EDITOR

Strongyloidiasis

EDITOR,—We were delighted to read Dr Grove’s leading article on the protein manifestations of strongyloidiasis (Gut 1994; 35: 437–40) and the case report by Dr Delarocque-Astagneau et al of a case presenting with biliary obstruction (Gut 1994; 35: 705–6), particularly with the reported increase in the incidence among migrant workers and holiday travellers to endemic areas. We recently submitted a case report of a West Indian man living in the United Kingdom since 1965 with disseminated strongyloidiasis who, similar to the patient in the case report, was also seropositive for human T lymphotropic virus (HTLV)-1. These cases highlight the well recognised association between HTLV-1 infection and disseminated strongyloidiasis, particularly in areas of high prevalence in the West Indies and southwest Japan.

Delarocque-Astagneau et al report a marked increase in infections with Strongyloides stercoralis in the United States. Although their cases were not in immigrants, it seems unusual that such an increase has not been reported in previously endemic areas. Of interest, they describe a patient with HTLV-1 infection and disseminated strongyloidiasis. This association was first described in Okinawa, Japan, and HTLV-1 infection in Okinawa, area of Japan. Cancer 1989; 64: 1290–5.

We agree with Dr DiMaggio and others that the development of pancreatic insufficiency in chronic renal failure is a matter of considerable clinical interest. The hypothesis that recurrent attacks of acute pancreatitis might partly explain the changes in uraemic pancreas is not as improbable as Dr DiMaggio suggests because the incidence of acute pancreatitis is significantly increased in patients with end stage renal disease. We agree with Dr DiMaggio that it would be very interesting to include an additional non-invasive control model to represent chronic pancreatitis in our study (compared with the animal changes associated with the caerulein model used). Unfortunately no such animal model exists.

The hypothesis proposed by Dr DiMaggio that a chronic increase of circulating CCK is responsible for the changes associated with uraemic pancreas is intriguing, and, although we have not measured CCK in our rat model, it is not excluded by our data. The same increase in

Reply

EDITOR,—Dr DiMaggio raises some important questions regarding our study and the participation of the exocrine pancreas in chronic renal disease. The data in the earlier report3 indeed show that the exocrine pancreas responds with increased digestive enzyme secretion (trypsin) to cholecystokinin (CCK) stimulation in vivo and this finding is in accordance with the increased secretory response (amylase and lipase), which we found in vitro at the time interval where renal insufficiency was most severe in our animals (Gut 1994; 35: 401–7). His work was meant to be referenced at a different place in our paper and we apologise for this error.

Dr DiMaggio further suggests that renal failure was only severe during the initial phase of our observation period, while, at the stage of developing glomerulosclerosis2 uremia was much milder. We agree that an animal model entailing total (in contrast with subtotal) nephrectomy combined with pancreatitis might be more adequate and that subtotal is simply insufficient. There is, however, extensive data on the recovery of the pancreas following nephrectomy and it is possible that the pancreas is functionally damaged but with normal morphology in chronic renal failure.

Dr DiMaggio reminds us that the pathophysiology as well as the sequelae of uraemic pancreopathy are still a matter of controversy and ongoing study.3 As indicated in the abstract of our report it was previously unclear whether uraemic pancreopathy should be regarded as a manifestation of chronic pancreatitis, arising from recurrent attacks of acute pancreatitis, or represents a distinct entity.4 To consider these questions we used a combination of case studies of pancreatitis and a model of pancreatitis that combines most of the biochemical and cell biological characteristics reported in other existing animal models of pancreatitis. We are now repeating the pancreas in renal failure experiment on the resemblance with any of the different features of pancreatitis we investigated. They did, however, resemble toxic damage of the pancreas and we therefore concluded that uraemic pancreopathy is distinct from pancreatitis and probably does not result from recurrent attacks of pancreatitis. The initial hypothesis that recurrent attacks of acute pancreatitis might partly explain the changes of the uraemic pancreas is not as improbable as Dr DiMaggio suggests because the incidence of acute pancreatitis is significantly increased in patients with end stage renal disease. We agree that it would be very interesting to include an additional non-invasive control model to represent chronic pancreatitis in our study (compared with the changes associated with the caerulein model used). Unfortunately no such animal model exists.

The hypothesis proposed by Dr DiMaggio that a chronic increase of circulating CCK is responsible for the changes associated with uraemic pancreas is intriguing, and, although we have not measured CCK in our rat model, it is not excluded by our data. The same increase in

Pancreas in end stage renal disease

EDITOR,—I read with interest the article by Lerch and colleagues (Gut 1994; 35: 401–7) describing the development of pancreatic insufficiency in experimental renal failure. I, however, was very surprised to see one of my papers1 given as a reference for statements in the introduction and discussion of their manuscript to support claims that exocrine pancreatic insufficiency occurs in renal failure. In contrast, we showed increased trypsin and normal lipase secretion in patients with renal failure undergoing dialysis.2 Confusion may have arisen because we reviewed the data of others published before our article and gave a number of references that report no normal or decreased pancreatic secretion in renal insufficiency.3–6

Of possible interest, we also found that patients with renal insufficiency had raised plasma concentrations of hormones that affect pancreatic secretion, which led us to hypothesis that the raised hormone concentrations may cause morphological changes of the pancreas.7 One of the important problems of the rat model of renal failure discussed by Lerch and colleagues is that severe renal insufficiency is only transient; serum creatinine decreased to almost normal values by 56 days. It is entirely possible that severe renal insufficiency is not present long enough to cause prolonged physiological changes such as constantly increased hormonal concentrations to cause morphological abnormalities.

Another possible problem with the study of Lerch et al was use of questionable controls; comparisons were made to caerulein pancreatitis which, according to the authors, causes acute pancreatitis whereas the aim of their study was to determine if renal insufficiency causes chronic pancreatitis.

These problems raise the question of whether the study of Lerch et al provides much useful information in resolving the question of pancreatic functional and morphological abnormalities in human renal failure.

P E DIMAGGIO

Mayo Foundation, Rochester, Minnesota 55905, USA


