LETTERS TO THE EDITOR

Osteopenia in adult coeliac disease

EDITOR,—We wish to comment on the paper by Valdimarsson *et al* (*Gut* 1996; 38: 322–7) regarding osteopenia in adult coeliac disease. Their results agree with ours^{1,2} with regard to the frequency, the severity of the derangement of bone and mineral metabolism, and the effect of gluten free diet. There are, however, some significant discrepancies between their results and ours. The first and most evident discrepancy concerns coeliac patients without malabsorption symptoms who currently represent the majority of all diagnosed adult patients.³ In this group they did not find a lower degree of osteopenia compared with those complaining of diarrhoea or weight loss. We have now examined 57 consecutive untreated coeliac patients (median age 30 years, range 17–68), of whom 23 presented the classic symptoms of malabsorption and 34 were subclinical or had been identified during the serological screening of coeliac relatives, patients with iron deficiency anaemia, recurrent aphthous stomatitis or alopecia areata. Unlike Valdimarsson, subclinical patients showed a significantly higher age adjusted bone mineral density ($p < 0.0005$) with respect to the coeliac patients with the classic disease, both at lumbar (-1.21 (0.97) v -2.32 (1.0) and femoral level (-1.31 (0.96) v -2.14 (0.92)). Moreover, the changes of the biochemical indices of bone metabolism and remodelling (strangely enough, these results were not provided by Valdimarsson) were significantly lower in our patients without diarrhoea or weight loss. The distinction between patients with and without malabsorption was made according to the same criteria adopted by Valdimarsson – that is, the presence or absence of diarrhoea or weight loss. It is in fact difficult to explain his results. Although there is no doubt that osteopenia may also be present in patients without diarrhoea, it is difficult to believe that it could be present to the same extent as in patients with a significant degree of malabsorption. This is confirmed by the fact that their seven patients with dermatitis herpetiformis, a condition characterised by a less extensive enteropathy and consequently by mild or no symptoms of malabsorption, had normal bone mass. Mazure *et al*,⁴ who divided their patients in a similar way, obtained results that were the same as ours.

Another important discrepancy concerns the relation between bone mass and age at diagnosis, which was lower in our patients (median 30 years, range 17–68) than in the study by Valdimarsson (median 53, range 17–79), but this merely accentuates the extent of this discrepancy. Valdimarsson, in fact, concludes that osteopenia represents a late complication of coeliac disease, occurring only after the third decade. This is not so in our experience nor in that of authors who have demonstrated osteopenia also in coeliac children.^{5,6} Our results show that 85% of patients diagnosed in their third decade fail to reach a normal peak bone mass and that other parameters such as the degree of physical activity and exposure to sunlight, the possible presence of amenorrhoea and, above all,

nutritional status and the presence of malabsorption symptoms are more important than age in conditioning the presence of osteopenia.

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- 6 Mora S, Weber G, Barera G, Bellini A, Pasolini D, Prinster C, *et al*. Effect of gluten-free diet on bone mineral content in growing patients with coeliac disease. *Am J Clin Nutr* 1993; 57: 224–8.

Reply

EDITOR,—Di Stefano *et al* are correct to note that our untreated patients with coeliac disease who did not have symptoms of malabsorption (diarrhoea or weight loss) had osteopenia of the same degree as patients with these symptoms (*Gut* 1996; 38: 322–7). In contrast with the asymptomatic patients of Di Stefano *et al* and that of Mazure *et al*¹ all of our patients had clinical symptoms of coeliac disease and none of them had been identified during serological screening. All our patients had clinical suspicion of coeliac disease, although 34 of 63 did 'only' have dyspepsia or biochemical abnormalities. Di Stefano *et al* do not report the degree of mucosal changes in their patients. All our patients had severe mucosal changes in the proximal small bowel of grade III or IV on the basis of Alexander's classification.²

This difference in patient selection may explain the discrepancy in the degree of osteopenia in our studies.

Di Stefano *et al* report that '85% of their patients diagnosed in their third decade fail to reach a normal peak bone mass'. Does this mean T score -1 or -2 ? Mora *et al*³ did report low bone mass in 33 children with untreated coeliac disease (mean age 8.95 years). Our eight patients who were 17–25 years old when diagnosed did not (as a group) have reduced bone mineral density, but five of them had Z score slightly below zero (Fig 1, *Gut* 1996; 38: 322–7). Maybe the patients reported by Mora *et al* had a greater degree of malabsorptive state than our patients as they were diagnosed at a lower age. Molteni *et al*⁴ did not report osteopenia in coeliac children (as cited) but found normal bone mineral density in patients with coeliac disease treated since childhood.

Our patients younger than the age of 25 did not have reduced bone mineral density but if

left untreated they would probably be at risk of osteopenia. Dietary treatment is therefore important for coeliac patients of all ages.

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Acute idiopathic pancreatitis

EDITOR,—We have read with great interest the article by Ballinger and colleagues. The authors conclude that the prognosis is good after a first episode of acute 'idiopathic' pancreatitis and that specialised investigation is unnecessary (*Gut* 1996; 38: 293–5). We entirely disagree with their conclusions.

We performed recently¹ a study of the aetiology of acute pancreatitis in the region of Nice. During one year, we included prospectively 121 cases of acute pancreatitis. In 19 cases, no cause was found at admission (patients without history of alcohol abuse, without gall bladder or common bile duct calculi on ultrasound examination and with normal computed tomograms except signs of acute pancreatitis). These patients were reinvestigated two months later: calcaemia, phosphoraemia, triglycerides, echoendoscopy, and ERCP searching for biliary cholesterol crystals. Finally, a definite cause of acute pancreatitis was found for 10 patients giving a percentage of 'idiopathic pancreatitis' changing from 15.7 to 7.4%. The diagnoses found were four cases of 'occult lithiasis' (two patients with biliary cholesterol crystals, one patient with microlithiasis in the common bile duct at ERCP and without stones in the gall bladder, one patient with microlithiasis occurring in the gall bladder during the follow up), one drug induced pancreatitis (proved by unintentional rechallenge), one patient with hyperparathyroid adenoma (with normal calcaemia at the time of the first episode), three patients with acute pancreatitis revealing a carcinoma (two pancreatic carcinoma, and one antral carcinoma involving the main pancreatic duct) and one cryptosporidial infection of the biliary tract resulting from HIV. For one of the cancers, ERCP after the first episode showed only the presence of a pancreas divisum, and a sphincterotomy of the minor papilla was performed. After a second episode, a plastic stent was put in the santorini duct but we note at the time of writing a stenosis at the corporeocaudal junction (the scanner showed only the presence of a small pseudocyst of the pancreatic tail). So we decided to perform surgery that finally revealed a small carcinoma. There was 20% of recurrence during the follow up (median 111 days) but only one recurrence after the correct diagnosis was made.

The important point is that these 19 cases were labelled 'idiopathic' at admission and