tract. It has been suggested that faecal calprotectin might be a useful screening tool in distinguishing organic from functional bowel disease, especially in children. We report, for the first time, elevated faecal calprotectin in patients with diarrhoea due to pancreatic insufficiency.

Method: A total of 115 patients (77 women: 38 men; age 19–85) with chronic non bloody diarrhoea were screened in this study. Both pancreatic elastase and calprotectin were assayed by ELISA in faecal samples (normal : calprotectin < 30 µg/g, elastase >150 µg/g.) Pancreatic imaging in those with low faecal elastase was carried out by a combination of CT scanning, endoscopic pancreatograms and endoscopic ultrasonography.

Results: Twenty six (21%) patients had low faecal elastase. Of these, 5 were confirmed radiologically to have chronic pancreatitis. All 5 patients were shown to have abnormally high faecal calprotectin levels (90–534 µg/g). 14 patients had suspected pancreatic insufficiency in the absence of other causative pathology. 13 (93%) of these patients had elevated faecal calprotectin (73–799 µg/g). 7 patients with low faecal elastase and 'watery' faecal samples were subsequently shown to have probably normal pancreatic function and an alternative explanation for symptoms (adenoma, Clostridial colitis, small bowel bacterial overgrowth, diverticular disease). Of these patients, 2 (29%) had abnormally high faecal calprotectin (176 µg/g=adenoma, 2670 µg/g=C.diff colitis) (table 1).

Abstract 223, Table 1

	Total patients	Low elastase	High calprotectin
Confirmed pancreatic disease	5	5 (100%)	5 (100%)
Possible pancreatic disease	14	14 (100%)	13 (93%)
No pancreatic disease	96	7 (7%)	42 (44%)

Conclusion: Pancreatic insufficiency may be associated with high faecal calprotectin in the absence of neoplastic or inflammatory intestinal disease. Low faecal elastase may also be associated with watery diarrhoea secondary to inflammatory intestinal disease, itself associated with an elevated faecal calprotectin concentration.

Symposium: Guts, Liver and Bones: 224–225

224 GUIDELINES FOR OSTEOPOROSIS PREVENTION: HOW FAR HAVE WE YET TO GO?

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Background: The British Society of Gastroenterology has published guidelines for the prevention and treatment of osteoporosis in patients with coeliac disease and inflammatory bowel disease (IBD) (*Gut* 2000;46(suppl 1):i1-i8). This has important implications for the planning of clinical services, especially with the limited availability of DEXA scanning. We have undertaken an assessment of the need for DEXA scans in our gastroenterology out-patient population.

Methods: In a district general hospital, population 460,000, we identified all patients with coeliac disease and IBD attending all gastroenterology out-patient clinics (3.5 consultant gastroenterologists, total 6 clinics per week) over a one month period. We assessed the number of these patients who required DEXA scanning and further investigation according to the BSG guidelines.

Results: Over a four week period 124 of a total 204 patients with either coeliac disease or IBD were found to require a DEXA scan according to the guidelines. The mean age of those requiring a scan was greater than those who did not (54.1 (18.6)y vs 38.6 (12.3)y (age (sd)). All 21 patients with coeliac disease required a scan. Of those with Crohn's disease 43/72 required a scan as did 60/111 patients with ulcerative colitis. Reasons for scans in patients with IBD are given in the table. DEXA scans had already been requested in 12 patients and 5 patients were on treatment (table 1).

Conclusions: Currently we are achieving BSG guidelines in less than 10% of our patient population. Introducing guidelines will mean that almost 2/3 of patients with Crohn's disease and more than half our ulcerative colitis patients will require a DEXA scan. Subsequently scanning will be required for patients with a new diagnosis of IBD,

Abstract 224, Table 1

	On steroids	Post-menopausal or male >55y	Both steroids and age criteria
Crohn's disease	21	21	1
Ulcerative colitis	14	32	14

those requiring long courses of steroids and those reaching the age thresholds, as well as those on bone protection treatment. Patients with coeliac disease who require a scan at diagnosis place less pressure on resources.

225 PREVALENCE OF FRAGILITY FRACTURE IN COELIAC DISEASE AND INFLAMMATORY BOWEL DISEASE

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Background: Osteoporosis is a common complication of both inflammatory bowel disease and coeliac disease, but there is little information on the prevalence of fragility fracture in these patients.

Methods: Ambulant patients with coeliac disease (102), Crohn's disease (88) and ulcerative colitis (130) completed a questionnaire detailing their fracture history. Fractures involving major trauma e.g. Road Traffic Accidents, falls from greater than three feet and sports injuries were excluded from analysis.

Results: 42% of patients with coeliac disease had experienced a previous fracture. Commonest fractures involved the distal radius (32%) and ankle (16%). Prevalence was higher in females than males (46% vs 33%). Females were younger than males (52.3yrs vs 59.1yrs) and were diagnosed at an earlier age (45.9yrs vs 52.3 yrs). In neither sex was age at diagnosis related to history of fracture. 99 patients were diagnosed after the age of 25, when peak bone mass is achieved. 130 patients with ulcerative colitis (47 male, 83 female, mean age 45.7yrs) and 88 patients with Crohn's disease (31 male, 57 female, mean age 38.7yrs) completed the questionnaire. 29% of patients with ulcerative colitis and 30% of patients with Crohn's disease had suffered a fracture. Patients incurring fractures after diagnosis of ulcerative colitis had earlier onset of disease (mean 28.6yrs vs 37.9yrs) and longer duration (14.0yrs vs 7.7yrs) than patients without fractures. Fracture risk was unrelated to disease extent, use of systemic corticosteroids, smoking and family history of fragility fracture.

Conclusions: Fractures are very common in patients with coeliac disease and inflammatory bowel disease. Efforts to prevent, detect and treat low bone mineral density may reduce this prevalence.

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226 IMMUNE RESPONSES IN BARRETT'S METAPLASIA

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Introduction: Barrett's metaplasia (BM) is a chronic inflammatory disorder of the oesophagus in response to persistent gastro-oesophageal reflux and is associated with two distinct complications: benign oesophageal strictures and a 125-fold increased risk of developing an oesophageal adenocarcinoma. Benign strictures usually form in proximal areas of BM whereas adenocarcinomas arise distally. The characteristics of the inflammation and its role in the development of these complications have however been unclear.

Hypothesis: We propose that different immune responses are involved in the pathogenesis of these complications and that inflammation in BM is not a homogenous condition.

Methods: Endoscopic biopsies were obtained from normal oesophageal mucosa (n=10), oesophagitis (n=10) and paired biopsies from proximal and distal BM (n=20). The samples were assessed using immunohistochemistry, immunofluorescence, western blotting, and PCR amplification of DNA isolates from paraffin-embedded biopsies with consensus primers TCRB, TCRG and IgH.