bowel diseases (IBD), are typically dosed according to patient's body weight. A previous meta-analysis showed higher remission rates in patients with "therapeutic" levels of 6-thioguanine (6-TGN), but weight based dosing correlates poorly with 6-TGN levels (1). 6-TGN testing is not universally available, results are not available immediately and repeated measurements are necessary to ensure dose adequacy and adherence to therapy. Proxy measures such as mean corpuscular volume (MCV) and lymphocyte count (LC) have been advocated as markers of dose adequacy. We aimed to analyse the relationship between 6-TGN levels, MCV, LC and other putative surrogate markers of therapeutic 6-TGN levels.

Methods This retrospective study was conducted at the Royal Liverpool University Hospital. All patients who had concurrent measurements of 6-TGN and full blood count were included in the analysis. 6-TGN levels were classed as sub-therapeutic (<230), therapeutic (230–450) or supra-therapeutic (>450). The association between 6-TGN, patient demographics, MCV, LC and other putative surrogate markers was estimated using a multivariable linear regression model for continuous 6-TGN and a proportional odds logistic regression model for the ordered 6TGN levels. All results were declared statistically significant if p < 0.05.

Results A total of 106 patients (48 male, 58 female) were included and contributed 133 measurements. Of these patients 58 (55%) had Crohn's disease and 47 (44%) had ulcerative colitis. The mean azathioprine dose was 123.5 mg (SD 73.8) or 1.70 mg/kg (SD 0.67). After adjusting for other variables, a one unit increase in MCV, was associated with a 10.88 unit increase in 6TGN levels, Figure 1 (95% CI: 7.63 and 14.014, p < 0.0001) and a one unit increase in ALT was associated with a 2.67 unit decrease in 6TGN levels (95% CI: 0.36 to 4.97 p = 0.0237). There was no correlation between LC, NC, WCC or ALKPHOS and 6-TGN levels.

Conclusion MCV and 6-TGN nucleotide levels increase together. If 6-TGN levels are not available, MCV can be used as a crude but imperfect surrogate marker of dose adequacy and toxicity.

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Disclosure of Interest None Declared.

PWE-079 | **DEXA RATES AND OUTCOMES IN AN IBD POPULATION COHORT.. LESSONS LEARNT**

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Introduction Osteoporosis is one of the under diagnosed and undertreated conditions worldwide. It is estimated that 1 in 3 women and 1 in 5 men in Ireland are diagnosed with it above the age of 50 years and will be likely to become more common with the increased life expectancy.

In our study, we aimed to assess the rate of screening with DEXA scans for this condition in a prospective IBD cohort and to evaluate the results. We also studied possible factors that may influence DEXA rates.

Methods As part of two decades follow up study of a multicentre Irish IBD prospective cohort from the early 1990's that was part of European Collaborative Study on IBD (EC-IBD), patients were traced and interviewed. There were 126 IBD patients from the Greater Dublin area who consented and completed the study questionnaire, following Ethical committees' approvals. In this questionnaire we evaluated DEXA frequency and possible factors that may influence it; including patient diagnosis, age, gender, family history of IBD, courses of steroids, social class, total years of education, attending gastroenterologist or a surgeon, years of loss to follow up (LTFU) and membership to the national IBD patient support group (Irish Society of Crohn's Colitis ISCC).

Results were analysed with logistic regression and forward stepwise analysis using R program 3.0.1 software, taking p value <0.05 to be statistically significant.

Results Of the total 126 patients only 58 (46.03%) patients had DEXA scans performed. Of those 67.44% had CD and 33.73% had UC. When evaluating DEXA results osteoporosis was diagnosed in 26.67 and 21.43% of CD and UC patients respectively. Osteopenia was diagnosed in 43.33% CD and 42.86% of UC patients. About 30% of remaining scanned patients had normal DEXA. Logistic regression analysis showed that patients with positive family history of IBD and longer years of education were more likely to have had a scan with P = 0.024 and 0.049 respectively. Other possible risk factors didn't reach statistical significance. Patients who were less compliant and LTFU were less likely to get DEXA with p = 0.045 in CD and p = 0.0336 in UC group.

Conclusion Osteoporosis is an increasingly recognised condition and recently has been considered as one of the extra intestinal manifestations of IBD. Patient and doctor awareness is paramount to screening and diagnosis. Our study showed that CD patients were more likely to have DEXA scans during their disease course. The rate of osteoporosis was higher in CD patients, despite their fewer numbers when compared to UC group. Over 40% of IBD patients had osteopenia. Early detection and appropriate management will help to reduce fracture risk, improve patient quality of life in a cost effective manner.

Disclosure of Interest None Declared.

PWE-080 | **BEWARE THE INTERACTION BETWEEN THIOPURINES AND WARFARIN**

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Introduction It is not uncommon for patients to require both immunosuppression and anticoagulation and for warfarin and thiopurines to be prescribed concurrently. There is limited evidence that thiopurines can inhibit the actions of warfarin. This potential interaction needs to be addressed when monitoring patients established on warfarin as they commence thiopurines or change dose. IBD patients are increasingly changed to combination low dose thiopurine and allopurinol for high methylmercaptopurine: thioguanine nucleotide (MeMP:TGN) ratio to optimise thiopurine efficacy. However, allopurinol also may potentiate warfarin and this could further exacerbate the effect of Azathioprine/mercaptopurine dose lowering on warfarin activity.

Aim To raise awareness of risk of harm from a clinically important interaction between thiopurines and warfarin +/- allopurinol.

Methods We studied 8 patients, identified from our anticoagulation database, who had been previously established on warfarin, and then commenced azathioprine or mercaptopurine for inflammatory bowel disease (2), systemic lupus erythematosus (1), nephritic syndrome (1), Wegener's granulomatosis (1), polyarteritis nodosa (1), dermatomyositis (1) and renal transplant (1). The effect of thiopurine on international normalised ratio (INR), and warfarin dose prior to and following commencement of thiopurine was recorded.

Results In 6/8 patients, following introduction of azathioprine or mercaptopurine, the warfarin dose had to be significantly increased (100% [18–500], Median [range]) in order to maintain a therapeutic INR. Any subsequent reductions in thiopurine dose were mirrored by a rise in INR and lower requirement for warfarin.

In 2 IBD patients, each with a high warfarin requirement, thiopurine metabolites were measured. In both patients MeMP: TGN ratio was >11. Thiopurine dose was reduced to 25% and allopurinol 100 mg added. INR was carefully monitored. In both cases INR increased within a week (to 6.9 and 11.2) and warfarin doses were subsequently reduced by ½ and 2/3 respectively to regain therapeutic INR.

Conclusion It is important for clinicians to be aware of the potential inhibitory action of thiopurines on warfarin's anticoagulant effect. Close INR monitoring is essential when initiating thiopurines and especially when reducing their dose and/or adding allopurinol. Failure to recognise the latter could result in bleeding due to over-anticoagulation. The high MeMP:TGN ratio in 2 of our patients also raises the possibility that thiopurine metabolites may play a role in the interaction between thiopurines and warfarin.

Disclosure of Interest None Declared.

PWE-081 | **DIAGNOSTIC PERFORMANCE OF FAECAL CALPROTECTIN IN PRIMARY CARE**

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Introduction Faecal calprotectin is recommended by NICE[1] for distinguishing between irritable bowel syndrome (IBS) and inflammatory bowel disease (IBD) in patients with lower gastrointestinal (GI) symptoms in primary care. If cancer is suspected in these patients and 'red-flag' symptoms such as anaemia or bleeding, they should be referred to Gastroenterology in accordance with the NICE suspected cancer guideline.[2] We use calprotectin in secondary care and will be extending the service to primary care providers. However, a number of GPs have been requesting faecal calprotectin on an ad-hoc basis for 1 year, giving us valuable insight into how the test performs in primary care.

Methods An audit was carried out of primary care calprotectin data in a 1 year period (Dec 12–Dec 13). This data was compared to an audit of 1 month of secondary care data (Jun 13). Clinical details, such as endoscopy and histology results were extracted from electronic patient records.

Results In total 198 requests for calprotectin came from primary care in 1 year and 40 were unsuitable for analysis (wrong sample type or delayed arrival in lab). Of the remaining 158 calprotectin requests, 76% were considered appropriate, having clinical details including symptoms described by NICE. Worryingly, 17%

of requests had inappropriate clinical details such as bleeding; such patients' referral to Gastroenterology was potentially delayed by requesting calprotectin. In 7% of requests no reason for request was discernable.

Of the primary care requests, 29% results were consistent with intestinal inflammation (>50 µg/g). If GPs use our proposed algorithm which suggests only referring patients with a calprotectin >50 µg/g, and those where strong clinical suspicion remains, there is potential for up to 71% reduction in patients referred to Gastroenterology with 'IBS/IBD' symptoms.

Diagnostic performance of calprotectin compared with endoscopy and histology diagnosis in secondary care is excellent with a sensitivity of 100% and a specificity of 91%. In primary care the corresponding data gives a sensitivity of 93% and a specificity of 79%.

Conclusion We received a large number of unsuitable samples. In addition GPs appear to be inappropriately requesting calprotectin in patients with symptoms such as bleeding, therefore it is critical to offer the service in a controlled way as part of a locally agreed care pathway. We are producing a GP information leaflet to advise on appropriate sample collection, result interpretation and the proposed patient pathway. We will re-audit primary care data once this is introduced to investigate whether a targeted approach leads to improved diagnostic performance of calprotectin in primary care.

REFERENCES
1 Faecal Calprotectin diagnostic tests for inflammatory diseases of the bowel, NICE DG11
2 Referral for suspected cancer, NICE CG27

Disclosure of Interest None Declared.

PWE-082 | **THE IMPACT OF NOD2 VARIANTS ON GUT MICROBIOTA IN CROHN'S DISEASE AND HEALTHY CONTROLS**

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Introduction Crohn's disease (CD) is now understood to be caused by the interaction between genetic and environmental factors with dysregulation of gut microbiota playing a pivotal role. NOD2, the strongest genetic risk factor for CD, encodes a pattern recognition receptor and plays an important role in epithelial defence. Studies of NOD2-knockout mice have demonstrated shifts in gut microbiota. Human studies to date have been limited by relatively small numbers of individuals homozygous for NOD2 mutations without accurate matching of controls.

Methods Individuals with CD of known NOD2 status were identified from the UK IBD genetics consortium. Patients in clinical remission were selected if they carried 2 of the common NOD2 variants (homozygotes or compound heterozygotes). Each NOD2 mutant patient was matched to a NOD2 wild-type