their General Practitioner (GP).[1,2] There is, however, limited knowledge as to how UK GPs manage this patient group and whether GP-led care meets IBD Standards.[3] This study aimed to identify factors influencing long-term follow-up of adults with IBD by GPs; achieved through examining GPs' knowledge and management of IBD, including exploration of future IBD-care models.

Methods A non-probability, convenience sample of 34 Senior Partner GPs and 130 Colleague GPs was recruited from 37 surgeries within Southampton City Primary Care Trust. Pre-piloted, closed and open-response e-questionnaires were administered to GPs asking questions on demographics, epidemiology, knowledge and management of IBD. Univariate and bivariate descriptive analyses with 90% confidence intervals were utilised. Conventional content analysis was applied to open question responses.

Results Cumulative questionnaire response rate was 50% (n = 82/164); 58% of GPs were male, with 19 mean years (SD 9.10) practicing medicine and 13 (SD 9.41) as a GP. Estimated IBD prevalence was 471:100,000. General Practitioners consulted with 2.8 adult patients (0.7%/total patients) with IBD/month and 59% independently managed those with established IBD. Short consultation times, insufficient knowledge and confidence in managing IBD and inadequate finances were identified as detrimental to GPs independently managing this patient group. Shared-care with hospital IBD services was preferred (82%).

Conclusion A high proportion of adults with stable IBD are being managed solely by GPs. General Practitioners' lack of knowledge, confidence and resources in caring for patients with IBD inevitably occurs when managing an infrequently seen chronic condition; raising clinical governance concerns. Low exposure to this patient group questions cost-effectiveness of measures to improve GPs' knowledge-base. Findings support a shared-care approach between primary and secondary care; meeting the long-term health needs of adults with IBD.

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Disclosure of Interest None Declared.

PWE-109 A METABOLOMIC PROFILING STUDY OF A CHEMICALLY-INDUCED MOUSE MODEL OF INTESTINAL INFLAMMATION

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Introduction A non-invasive, IBD-specific biomarker would be clinically useful. We have reported the changes in volatile organic metabolites (VOM) in human IBD. Human studies are limited by the variation in diet and the unpredictability of the disease. Animal models have been established to study many aspects of IBD. We report the first study of VOMs in murine DSS-colitis.

Methods C57BL/6 female mice at 9–10 weeks old were administered 4.25% dextran sulphate sodium (DSS) in their drinking water for 5 days in order to induce colitis. Clinical parameters of body weight loss, stool consistency and presence of rectal bleeding were assessed daily. Mice were culled at days 0 (n = 11), 5 (n = 11), 8 (n = 11) and 11 (n = 8); colonic, caecal, small bowel contents, mid-large bowel and distal small bowel tissue were taken. VOM profiles for each were analysed using SPME with a CAR/PDMS/DVB fibre and gas chromatography-mass spectrometry. Histology of the distal colon confirmed the presence of colitis; this was graded from none to mild or moderate/severe.

Results Histology confirmed the presence of colitis. VOM profiles for each sample at days 5, 8 and 11 were compared with day 0. The presence/absence data were used as independent variables in a chi-squared statistical test. In the colonic content, 106 compounds were identified across all groups; 13, 22 and 10 significantly varied with presence/absence between day 0 and day 5, 8 and 11, respectively. A t-test was performed on the abundance of compounds present in at least 60% of samples in one condition. A total of 29 compounds were identified; 9, 6 and 7 VOCs were present at significantly different levels between day 0 and day 5, 8 and 11, respectively. Significance levels for both chi-squared and t-tests were set at $p < 0.05$ and a fold difference of $^{3}2$. Principal component analysis (PCA) of the raw data showed clear separation between the different stages of the disease. A PCA biplot revealed that butanal, propanal, methyl propionate, ethyl acetate, ethyl propionate and 2,3-butanedione were responsible for the main separation between day 0 and day 5/8 of colitis.

Conclusion Typical clinical and histological features of colitis commenced on day 5, were maximal at day 8 and mice showed signs of recovery between days 8 and 11. The VOM results reflect this timescale, suggesting that metabolic disease profiling is able to represent the different stages of colitis. Further investigation of these differences could deepen our understanding of the pathogenesis of IBD.

Disclosure of Interest None Declared.

PWE-110 A ROLE FOR PEROXIREDOXIN-4 IN A MURINE COLITIS MODEL OF INTESTINAL INFLAMMATION

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Introduction Peroxiredoxins are a family of highly conserved antioxidant proteins. Beside its role in the reduction of peroxides, Peroxiredoxin-4 (Prdx4) has been shown to play a role in the modulation of pro-inflammatory signalling cascades. Our group has previously demonstrated that Prdx4 is expressed in the intestinal mucosa and upregulated upon stimulation with the bacterial cell wall component muramyl-dipeptide (MDP). In addition, siRNA-mediated downregulation of Prdx4 increased MDP-induced NF-κB signalling. We therefore generated a murine *Prdx4*-knockout model to address the relevance of Prdx4 in the intestinal immune response *in vivo*.

Methods In this study, two different *Prdx4*-knockout mouse lines were used: A constitutive $Prdx4^{-/-}$ knockout strain, in which global Prdx4 expression was deleted and a conditional mouse line that specifically lacked Prdx4 in the intestinal epithelium ($Prdx4^{\Delta IEC/\Delta IEC}$). Intestinal inflammation was induced by administration of dextran-sodium-sulfate (DSS) in the drinking water of $Prdx4^{-/-}$, $Prdx4^{\Delta IEC/\Delta IEC}$ and respective littermate control

mice. Mice were assessed for weight loss, disease severity, histopathology and endoscopic appearance.
Results We found that during DSS-induced colitis *Prdx4*$^{-/-}$ mice lost significantly more weight and had a more pronounced disease activity than their wild-type littermates. However, no such differences were observed in *Prdx4*$^{\Delta IEC/\Delta IEC}$ mice, compared to their *Prdx4*$^{floxed/floxed}$ littermates. Likewise, colon histopathology and endoscopy did not reveal significant differences. We next examined Prdx4 expression in dissociated intestinal segments and found that Prdx4 levels in the lamina propria exceeded those of the intestinal epithelium (foldchange >2). In addition, already under basal conditions, lamina propria immune cell composition differed significantly between *Prdx4*$^{-/-}$ and wild-type mice.
Conclusion Our data assign a protective role of Peroxiredoxin-4 in intestinal inflammation which does not arise from the intestinal epithelium but presumably from the lamina propria. Further studies will be needed to determine the functional basis and molecular mechanisms of the observed effects.
Disclosure of Interest None Declared.

PWE-111 HIGHER RED BLOOD CELL METHOTREXATE POLYGLUTAMATES CORRELATE WITH INCREASED DISEASE ACTIVITY, AND ARE USEFUL IN ASSESSING ADHERENCE

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Introduction Methotrexate (MTX) is commonly used in patients with inflammatory bowel disease (IBD). Within red blood cells (RBC), MTX is activated by sequential addition of glutamic acid residues to form polyglutamates (MTXPG $_{1–5}$). In rheumatoid arthritis, low [MTXPG] has been associated with active disease, whereas other studies have demonstrated an inverse relationship, including the only published data in IBD. The aim of this study was to determine if RBC [MTXPG] reflect clinical response in IBD patients and whether they are useful in assessing adherence.
Methods This was a single-centre, retrospective pilot study of 21 IBD patients treated with weekly MTX. RBC $MTXPG_{1–5}$ was measured using high-performance liquid chromatography. Clinical status (active disease or remission) was assessed by 2 IBD physicians blinded to [MTXPG], using a combination of prospectively recorded clinical activity indices (Simple Colitis Activity Index, Harvey Bradshaw Index), endoscopy, faecal calprotectin and C reactive protein (CRP). Pearson correlation coefficient, r was calculated to assess the relationship between MTX dose and [MTXPG]. Association between [MTXPG] and clinical response was analysed with unpaired t-test.
Results 4/21(22%) patients (3 of whom admitted non-adherence) had undetectable MTXPGs and were excluded from further analysis. $MTXPG_{2–4}$ were detected in all adherent patients. PG_3 was the predominant polyglutamate accounting for a mean of 43% of total MTXPG. A linear relationship between dose of MTX and $PG_{1–5}$ was observed. 12/21(57%) patients were assessed as having active disease. No significant difference in mean [$MTXPG_n$] was observed between those with active disease and remission. For each $MTXPG_n$, a non-significant trend towards a higher concentration was observed in patients with active disease.
Conclusion In this study, the largest to date in IBD, measuring RBC MTXPG was useful in assessing adherence to MTX. A trend towards higher PG concentrations was associated with active disease confirming the findings in the only other study in IBD. Whether this is confounded by higher doses being used in patients with more active disease warrants further study in larger, prospective trials.

REFERENCE

Disclosure of Interest: None Declared.

PWE-112 MANAGEMENT OF IRON DEFICIENCY ANAEMIA IN THE OUTPATIENT INFLAMMATORY BOWEL DISEASE COHORT

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Introduction Iron deficiency is the commonest micronutrient deficiency in IBD and causes impaired quality of life. 35% of patients have been reported to be iron deficient and 65% require iron replacement over the course of their disease. We analysed the diagnosis and treatment of iron deficiency in our local IBD cohort and compared this to BSG guidelines. They state that all IBD patients should have an annual full blood count and if anaemic (Hb <12 g/dl for women, Hb <13 for men) iron studies should be undertaken. If disease is inactive and ferritin <30 or there is active disease and ferritin <100, the patient should be on iron. This should be the recommended type of iron; IV iron in severe anaemia (haemoglobin <10) or severe intestinal disease activity, concomitant therapy with an erythropoietic agent, or patient preference. Hb should be rechecked after 4 weeks and if it does not rise by 2g/dl or normalise, IV iron should be started. If Hb <10 and there is no response to IV iron therapy within 4 weeks EPO should be given.
Methods A prospective study was undertaken in the Royal Berkshire NHS Foundation Trust of patients attending IBD clinics in December 2012. 100 patients attending clinic consecutively were recruited.

Abstract PWE-111 Table 1 Correlation of methotrexate dose to MTXPG and clinical outcome

MTX PGn	Correlation between MTX dose and MTXPG, r, (p)	Active disease: [PGn] (nmol/RBC 8 × 1012), mean, SD	Remission [PGn] (nmol/RBC 8 × 1012) mean, SD	p value
PG1	0.96 (p = 0.01)	22 ± 16	15 ± 1	0.28
PG2	0.92 (p = 0.008)	24 ± 3.6	17 ± 2.3	0.17
PG3	0.98 (p = 0.003)	51 ± 9.8	36 ± 6.7	0.26
PG4	0.94 (p = 0.019)	19 ± 4.9	12 ± 1.7	0.25
PG5	0.67 (p = 0.219)	4.5 ± 1.5	1.3 ± 0.73	0.09