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.

REFERENCES

1 Rubin GP, Hungin AP, Kelly PJ, Ling J. Inflammatory bowel disease: epidemiology and management in an English general practice population. *Alim Pharmacol Ther 2000*;14(12):1553–59

2 Stone MA, Mayberry JF, Baker R. Prevalence and management of inflammatory bowel disease: a cross-sectional study from central England. Eur *J Gastroenterol Hepatol*. 2003;15(12):1275–80

3 The IBD Standards Group. *Standards for the healthcare of people who have Inflammatory Bowel Disease (IBD). IBD Standards.* 2013 *Update.* Brighton: Oyster Healthcare Communications Ltd 2013

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