

**Introduction Background** Mucosal healing (MH) is an increasingly important therapeutic goal in inflammatory bowel disease. Monoclonal antibody (MA) therapy aims to achieve this and faecal calprotectin (FC) concentration has been shown as a surrogate marker for MH.

**Aims** Our aim was to study the profile of Crohn's disease (CD) patients on MA therapy and evaluate whether FC levels after induction therapy with MA predicts the medium-term outcome.

**Methods** Thirty-two CD patients: infliximab n = 11, adalimumab n = 21 were identified from our MA database. Data on demography and disease characteristics were extracted from case records. A subset of CD patients with FC levels measured both at baseline and after induction therapy were analysed further for response to therapy and disease course during follow-up (n = 10). Disease activity was evaluated by modified Harvey-Bradshaw index at baseline, after induction, and at 6 and 12 months during maintenance therapy.

**Results** Of 32 patients, 22 patients were female, medium age 39.5 (range: 19–65 year), medium age at diagnosis 30.2 (range: 16–61 year), mean disease duration prior to MA was 6.1 (range: 10–22 year) and 21.8% has family history of inflammatory bowel disease. Of these, 56.2% had history of surgery prior to MA and 71.7% had concurrent immunomodulation. Disease phenotypes are shown in table. Of the 10 patients with full FC data, 6 patients normalised FC after induction (median levels 67 mg/kg, median 64 mg/kg, range 30–72). All remained in remission during follow-up median- 22 months (range 13–33 months). Four patients failed to normalise FC levels with induction therapy (median 11 months, range 6–39). Of these, 2 had operation, 2 had multiple relapses (1 treated with prolonged enteral therapy and 1 with additional oral corticosteroid courses).

**Abstract PTH-085 Table 1**

% ileal disease	3.1% (1/32)
% colonic	34.3% (11/32)
% ileocolonic	59.3% (19/32)
% isolated upper GI	3.1% (1/32)
% B1 (non stricturing/penetrating)	50% (16/32)
% B2 (stricturing)	15.6% (5/32)
% B3 (penetrating)	34.3% (11/32)
% P (perianal)	53.1% (17/32)

**Conclusion** MA therapy is used in CD patients with aggressive disease course who are treated/intolerant to immunomodulatory therapy. Normalisation of FC after induction therapy with MA is a useful marker to predict sustained clinical remission.

**Disclosure of Interest** None Declared.

**PTH-086 INVESTIGATION OF THE ANTIMICROBIAL ACTIVITY OF ESSENTIAL OILS OF CULINARY AND MEDICINAL HERBS AND SPICES AGAINST SELECTED GASTROINTESTINAL PATHOGENS**

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**Introduction** Pathogenic gut microorganisms, and dysbiosis of the gastrointestinal microbiota are a significant cause of mortality and morbidity worldwide, for instance infection with *Clostridium difficile* or *Salmonella* species can prove fatal, whereas alteration of the gastrointestinal microbiota has been implicated in irritable bowel syndrome. Due to increasing resistance of gastrointestinal pathogens to conventional antibiotics, alternative antimicrobial agents

are urgently needed. The aim of this study is to investigate whether essential oils (concentrated mixtures of aromatic compounds obtained by the distillation of plant tissues) have antimicrobial activity against selected gastrointestinal pathogens.

**Methods** We have investigated the antimicrobial activity of essential oils of a wide range of culinary and medicinal herbs against type strains of selected gastrointestinal pathogens, namely *Salmonella enterica*, *Clostridium difficile*, two strains of *Escherichia coli*, and *Candida albicans* by disc diffusion assays. Grapeseed oil was the negative control. If the essential oils inhibited the growth of the organisms, a clear halo was seen around the test discs. This was measured. The experiments were performed three times and results were analysed by two sample T Tests. Essential oils were analysed by thermal desorption gas chromatography with mass spectrometry to identify the compounds present.

**Results** Seven of the essential oils (aniseed, asafoetida, cinnamon, clove, oregano, thyme and winter savoury) produced a strong and statistically significant inhibition of the growth of all five of the organisms tested whereas a further seven essential oils (coriander, garlic, lemon balm, lemon grass, May Chang, peppermint and rosemary) markedly inhibited the growth of three or four of the organisms (and these results were also statistically significant). Batch to batch variation was evident in the antimicrobial activity of some of the essential oils. This might correlate with variations in the profile of compounds present in the essential oils.

**Conclusion** Some of the essential oils studied might be therapeutically useful against gastrointestinal pathogens. Quality control of the oils would be necessary and further work is needed to identify the active antimicrobial compounds in the oils.

**Disclosure of Interest** None Declared.

**PTH-087 FAM5C, A POSSIBLE CAUSAL MOLECULE IN ULCERATIVE COLITIS REVEALED THROUGH A TRANSCRIPTOMIC ANALYSIS OF THE BOWEL MUCOSA**

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**Introduction** Abnormalities of the colonic mucosa have been implicated in the pathogenesis of ulcerative colitis (UC). We investigated mRNA profiles of macroscopically non-inflamed mucosal biopsies from the colon in patients with UC, Crohn's disease (CD) and control subjects without gastrointestinal disease (HC), to identify genes that might be involved in the aetiology of the disease.

**Methods** Paired biopsies were taken for histology and mRNA extraction from macroscopically non-inflamed mucosa in the ascending and descending colon, and the rectum, from 24 patients with UC, 14 with CD and 27 HCs undergoing routine colonoscopy. Patients were in complete clinical remission and were either on no treatment or on 5-aminosalicylates ± azathioprine. rRNA was hybridised to Illumina HumanHT-12 v4 Expression Beadchips. Array expression data were log transformed and normalised. Only probes with a detection p-value < 0.01 were analysed. Differential gene expression analysis between groups (using p < 0.05 FDR correction) and outlier analysis (p < 0.005, fold change (FC) ≥ 1.5) were performed at each location using customised software. Results were verified by qPCR and candidate molecules were examined in an independent cohort of UC patients.

**Results** In group comparisons, of the 26,261 expressed probes, Family with Sequence Similarity 5, member C (*FAM5C*) was the only gene to be significantly under-expressed in UC, both in the rectum (FC = -1.58, p = 0.0008) and the descending colon

(FC = -1.64,  $p = 0.0011$ ). Outlier analysis showed that *FAM5C* was also grossly under-expressed in the ascending colon in 37.5% of UC patients, demonstrating that its expression is abnormal throughout the colon in a significant proportion of individuals. Expression levels were not abnormal in CD. Expression of *FAM5C* in UC did not correlate with the known markers of inflammation, *IL-8*, *S100A8*, *DEFA5* and *DEFA6*, or with treatment. The under-expression of *FAM5C* in UC was confirmed in biopsies of non-inflamed rectal mucosa from an independent cohort of patients (FC = -1.68,  $p = 0.0073$ ) and by qPCR ( $p < 0.001$ ).

**Conclusion** This is the first description of the under-expression of *FAM5C* in UC. As these observations were made in non-inflamed mucosa, low levels of this protein might be involved in the pathogenesis of the disease. Indications that *FAM5C* may function as tumour suppressor [1], could link to the observed predisposition to colonic malignancy in UC.

**Disclosure of Interest** None Declared.

## REFERENCE

1. Kuriowa T *et al.* (2009) *Oncol Rep*, 1005–11.

## PTH-088 INCIDENTAL DIAGNOSIS OF INFLAMMATORY BOWEL DISEASE IN A BRITISH BOWEL CANCER SCREENING COHORT: A MULTI-CENTRE STUDY

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**Introduction** The UK Bowel Cancer Screening Programme (BCSP) was launched in 2006 to cover the entire population of England and Wales. It screens individuals aged 60–69 years with a Faecal Occult Blood test (FOBT) followed by a screening colonoscopy if FOBT positive. We aimed to quantify the incidental diagnosis of Inflammatory Bowel Disease (IBD) and patient outcome in this cohort.

**Methods** A retrospective review of BCSP outcomes was conducted from launch in February 2007 to August 2012. Screening data included patients invited, number screened (FOBT “normal” or “abnormal”) and colonoscopies performed. In those diagnosed with IBD at colonoscopy confirmed on histology, clinical data (demographics, disease characteristics, treatment and outcome) were obtained from case note and electronic record review.

**Results** Of 477,553 patients invited, 219,705 were screened, representing an uptake of 46.01% and FOBT positivity of 2.35%. Colonoscopy was performed in 5350 patients (female 2287). Polyps were detected in 2344 (39.86%), cancer in 339 (5.77%) and 1383 (23.52%) had a normal examination. Endoscopic appearance suggestive of IBD in 112 patients was confirmed at histology in 66. Eleven patients were excluded as the diagnosis of IBD preceded screening. Twenty-one of 55 incidental cases were female. Median age at diagnosis was 64. Sixteen patients had Crohn’s disease (CD), 33 ulcerative colitis (UC) and 6 had IBD-type unclassified (IBDU). Follow-up data was available in 42 patients (mean follow-up 23.9 months). Twenty patients (47.6%) were asymptomatic at diagnosis. Seven (35.0%) of the asymptomatic patients became symptomatic during the follow-up period. Treatment included steroids (11), 5-ASA (34), immunomodulators (azathioprine 6; methotrexate 1) and anti-TNF (infliximab 2; adalimumab 1). None required surgery. In those requiring escalation of therapy (14.3%) the median time to immunomodulation was 21 months (range 5–30 months). Those requiring immunomodulators and/or anti-TNF therapy (male 4; female 2) had asymptomatic extensive UC, symptomatic left-sided UC, symptomatic left-sided IBDU, symptomatic Crohn’s colitis and symptomatic stricturing terminal ileal CD (2) at diagnosis.

**Conclusion** An incidental diagnosis of IBD is not uncommon. With the advent of bowel cancer screening this number is set to increase. A proportion of these patients demonstrate rapid disease progression. Such patients may present an important model for study of early disease with novel insights and evolving treatment paradigms.

**Disclosure of Interest** None Declared.

## PTH-089 IMPACT OF SEASONAL VARIATION ON COURSE OF INFLAMMATORY BOWEL DISEASE AND EFFECT OF DATE OF BIRTHS ON THE ONSET OF DISEASE: A FACT OR A MYTH!

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**Introduction** Effect of seasonal variation on the natural history of the inflammatory bowel disease (IBD) is now well known. Also births in certain time of the year may have an impact on the onset of inflammatory bowel disease later in life. We reviewed our cohort with inflammatory bowel disease over the last four years to look for any such association.

**Methods** Data collection was retrospective over the last 4 years using IBD database and medical records. Clinical and demographic details of newly diagnosed patients with IBD were recorded.

**Aims** Our aim was to identify any evidence of seasonal variability on natural history of IBD and to identify any link between the onset of IBD symptoms and the date of births.

**Results** We had 279 newly diagnosed cases of inflammatory bowel disease during the last 4 years (2008–2011). There was incremental rise in the incidence of disease during this period and majority of the cases had UC (70% UC, 30% CD).

There was no consistent correlation of incidence of IBD in any particular season over the last four years ( $p$  value = 0.065). Furthermore, there was no consistency in the data for the birth dates pattern and the onset of disease symptoms in our cohort.

**Conclusion** In our retrospective cohort study we could not demonstrate seasonal variability or impact of date of birth on disease onset but ongoing prospective data collection over a longer period of time may help explore this association.

**Disclosure of Interest** None Declared.

## REFERENCES

- Lewis JD, Abernethy FN, Lichtenstein GR, Bilker WB, Brensinger C, Strom BL. Seasonal variation in flares of inflammatory bowel disease. *Gastroenterology*. 2004 Mar; 126(3):665–73.
- Haslam N, Mayberry JF, Hawthorne AB, Newcombe RG, Holmes GK, Probert CS. Measles, month of birth, and Crohn’s disease. *Gut*. 2000 Dec; 47(6):801–3.

## PTH-090 EFFICACY AND SAFETY OF DOUBLE-DOSING OR DECREASING THE INTERVAL OF ANTI-TNF THERAPY IN CROHN’S DISEASE WHO HAVE SHOWN LOSS OF RESPONSE TO STANDARD ANTI-TNF DOSING REGIMEN - A DGH EXPERIENCE

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**Introduction** Loss of response to Infliximab or Adalimumab therapy is commonly encountered during the course of treatment in patients with refractory Crohn’s disease (CD). The aim of this study was to evaluate the safety and efficacy of dose intensification; defined as either double-dosing or decreasing interval of anti-tumour