Abstract PTU-077 Figure 1 Gating strategy to identify three subsets of dendritic cells in a colonic biopsies from an individual unaffected by IBD.

Conclusion Our novel multi-parameter analyses of live cells prepared from fresh colonic and ileal biopsies enable precise examination of the disease-inducing and effector populations that drive UC and CD. We are using the methods described here to dissect the immunological mechanisms driving inflammation in patients with IBD. We anticipate that our on-going comparisons of each of these immune cell populations in blood and intestinal biopsies from unaffected individuals, CD, and UC patients will reveal important details about pathogenic mechanisms controlling intestinal inflammation.

Disclosure of Interest None Declared.

PTU-078 | **THE APPLICATION OF A MARKOV MODEL FOR EVAUATING ASA THERAPY FOR ULCERATIVE COLITIS**

[1]V Mukhekat, [2]I Ahmad, [1]Y Merali, [2]D Aldulaimi*. [1]Warwick Business School, University of Warwick, Coventry, UK; [2]General Medicine, Worcestershire Acute Hospitals NHS Trust, Worcestershire, UK

Introduction We developed a Markov model for ulcerative colitis that calculated the costs of two different treatment strategies for 1000 patients and healthcare providers and ran the model over a ten year cycle to evaluate each treatment arm.

Methods Healthcare costs were calculated from the published study of costs of ulcerative colitis at Aintree University Hospital, Liverpool, UK. Effects were quantified with the EQ-5D visual analogue scale and Work Productivity and Activity Impairment (WPAI) study.

Lost income for patients was calculated assuming that the patient was self employed, earning the national average wage and had no sickness benefits.

Results Cohort simulation is used to present the results. At the end of exercise, 277 patients were in the phase of remission in ASA continuing arm and 232 patients were in the same phase in ASA discontinuing arm.

Total costs to the healthcare providers for providing treatment to the patients were found to be £15,390,139.81 (discounted-£12,825,018.45) for ASA continuing arm and that for ASA discontinuing arm were £12,911,557.43 (discounted-£10,752,633.35). These costs were incurred to gain QALYs of 31,682.14 (discounted-26713.79) for the ASA continuing arm and of 31,307.73 (discounted-26,405.89 for ASA discontinuing arm).

Primary outcome in terms of ICER (Incremental Cost-Effectiveness Ratio) is £6730.55 per QALY (acceptable range < £20,000–30,000). Secondary outcomes such as Discounted total earnings lost due to illness and Discounted total earnings affected due to work impairment were found to be in favour of ASA continuation by 19.43% (£3.7 million) and 11.15% (£7.8 million) in favour of ASA continuing arm.

Sensitivity analysis was conducted to challenge both the cost and effect assumptions. CEAC (Cost Effectiveness Acceptability Curve) revealed the probability of 1 is reached with the cost-effectiveness acceptability limit of £40,000 ceiling limit.

Conclusion Markov models have been used extensively to study the cost-effectiveness of healthcare interventions in chronic diseases.

Our Markov model considered costs for both patients and healthcare providers, rather than solely considering the costs for healthcare providers.

Results not only revealed the cost-effectiveness of ASA for the healthcare provider but also potential benefits for the broader economy.

Disclosure of Interest None Declared.

PTU-079 | **THE TPMT AND ABCB1 POLYMORPHISMS IN IBD PATIENTS IN CRETE: IMPACT ON DISEASE AND RESPONSE TO TREATMENT**

C Coucoutsi, O Sfakianaki, G Emmanuel, E Marinidou, A Voumvouraki, I Koutroubakis, E Kouroumalis*. Gastroenterology, University of Crete Medical School, Heraklion, Greece

Introduction It is well known that polymorphisms of the TPMT gene (coding for thiopourine methyl-transferase), influence response to treatment with azathioprine. Polymorphisms of the ABCB1 gene (coding for p-glycoprotein 170) has been associated with IBD and resistance to treatment but results are conflicting. The aim of this study was to determine the frequencies of TPMT and ABCB1 gene polymorphisms in IBD patients from Crete, a population genetically homogeneous, and how these polymorphisms might influence response to treatment and disease behaviour.

Methods A total of 222 IBD patients records were reviewed for intake of azathioprine, possible adverse reactions, response to treatment and need for colectomy. All patients were genotyped for TPMT gene polymorphisms, that have been related to intolerance to azathioprine (G238C, G460A and A719C) as well as ABCB1 gene polymorphisms (G2677T/A and C3435T), using a PCR-RFLP method. The same polymorphisms were also determined in 119 age and sex healthy controls.

Results Allele frequencies of TPMT gene in our study population were found to be in concordance with those reported in other Caucasian populations. 76 IBD patients were identified receiving azathioprine, of whom 16 were discontinued (10 CD, 6 UC) due to adverse reaction. 2 of them were found to carry the G460A and A719G alleles (TPMT 3A genotype) (12.5%).

For the ABCB1 gene, G2677T/A allele frequencies were found to be similar to those reported in the literature. There was no association of G2677T/A or C3435T with clinical phenotype, or resistance to treatment. However, 77.3% of 22/222 patients who did not respond to therapy and required surgery, where found to carry both the C3434T and the G2677T mutation.

Conclusion Our study was conducted in a genetically homogenous population in the island of Crete. No correlation of any single SNP was found with either clinical activity or response to treatment. However, most patients who carried both the G2677T and C3435T mutations were refractory to treatment, a finding which implies that resistance to treatment in IBD patients is a more complex issue, which requires the presence of a genetic locus rather than a single SNP.

Disclosure of Interest None Declared.

PTU-080 | **CORRELATION OF FDG PET SCANNING WITH ENDOSCOPIC FINDINGS IN PATIENTS WITH CROHN'S DISEASE**

[1]E Russo*, [2]S Khan, [3]AP Brown, [3]N Keat, [3]W Hallett, [3]R Janisch, [3]RN Gunn, [3]EA Rabiner, [1]PM Matthews, [1]TR Orchard. [1]Medicine, Imperial College London, UK; [2]Radiology, Imperial College Healthcare NHS Trust, UK; [3]Imanova Centre for Imaging Sciences, London, UK

Introduction [18]F- Fluoro-deoxy-glucose Positron Emission Tomography (FDG-PET) scanning is gaining popularity in the assessment of several inflammatory disorders including Crohn's Disease (CD).

Methods 11 patients with established CD,underwent [18]F- FDG-PET scanning that was compared with recent endoscopic data, provided treatment had been unchanged between the two tests. Patients were fasted for 6 h and received 185 MBq of iv FDG followed by 800 ml of oral 2.5% mannitol. A low dose CT scan of the abdomen was performed, followed by PET, at 60 min post FDG injection. PET data was acquired over a maximum of 3 bed positions (10 min/ bed position).

Analysis involved dividing the gut into 6 segments on CT (terminal ileum, ascending, transverse, descending and sigmoid colon and rectum). Segmental maximum standardised uptake value (SUV_{MAX}) and SUV intestine-to-liver ratio ($SUV_{ITL}= SUV_{MAX}/$ Liver SUV_{MEAN}) were calculated. A segment is defined as abnormal (PET +ve) when its SUV_{MAX} > Liver SUV_{MEAN} as per previous literature.[1–3] SUV_{MAX} and SUV_{ITL} of endoscopically abnormal versus endoscopically normal PET +ve segments were compared using the Mann-Whitney test.

Results 11 patients (52 gut segments) had PET within a median of 1 month of endoscopy. 21/52 segments were active on endoscopy. Of these 20/21 were also PET +ve. However, 17/31 of endoscopically negative segments were alsoPET+ve suggesting a sensitivity of 95% and a specificity of 45% in our cohort.

Raising the SUV_{MAX} threshold for defining a PET +ve segment from the existing (>SUV_{LIVER}) to > 3.5x SUV_{LIVER} reduced sensitivity from 95% to 86%, but improved specificity from 45% to 82% compared to the gold-standard of endoscopy.

Conclusion FDG-PET appears to be up to 95% sensitive in identifying segments with endoscopically active CD.

Several 'false positive' segments are also observed conferring a low specificity.

A threshold of segmental SUV_{MAX} signal > 3.5 x SUV_{LIVER} greatly improves sensitivity with a minimar reduction in specificity.

Abstract PTU-080 Table 1 SUV_{MAX} and SUV_{ITL} in endoscopically normal (17) versus endoscopically abnormal (20) PET +ve segments

	PET +ve segments	
	Endoscopically normal	Endoscopically abnormal
SUVmax (mean ± SD)	5.4 (±4.1)	10.4 (±4.2)
p value		p = 0.0004
SUV_{ITL} (mean ± SD)	3.0 (±1.9)	5.5 (±1.8)
p value		p = 0.0003

Segments which demonstrate FDG signal but are negative on endoscopy may reflect disease undetected by endoscopy, or may be false positives. A comparison with histological activity is required to clarify this.

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Disclosure of Interest None Declared.

PTU-081 | **FAECAL CALPROTECTIN AND ILEAL CROHN'S DISEASE: CORRELATION WITH A SMALL BOWEL MRI SCORE FOR DISEASE ACTIVITY**

[1]F Fascì-Spurio*, [2]NA Kennedy, [3]L Wong, [3]P MacLean, [2]J Satsangi, [3]S Glancy, [2]CW Lees. [1]Gastroenterology Unit, Sapienza University, Rome, Italy; [2]Gastroenterology Unit, Western General Hospital, Edinburgh, UK; [3]Department of Radiology, Western General Hospital, Edinburgh, UK

Introduction Small bowel MRI (SBMRI) is the current standard for assessing ileal inflammation in Crohn's disease. Faecal calprotectin (FC) is closely correlated with colonic inflammation, but is thought to be of less utility in ileal disease. Interpretation of existing data linking FC with SBMRI findings have been confounded by the presence of colonic inflammation. We therefore aimed to ascertain how FC best reflects MRI findings exclusively in the small bowel.

Methods 150 SBMRI studies with matched FC results (±30 days) were identified from the Edinburgh FC Register (2008–12; n = 18,138). Scans were entered into an anonymous 'teaching' list on PACS and each re-read independently by 2 expert GI radiologists blind to all clinical and lab data. Technical, quality and disease parameters were recorded onto standard proformas. Scans rated by one or other radiologist as being of poor quality were excluded (n = 31/150). 7/13 disease parameters were excluded due to poor interobserver variability (Cohen's kappa <0.5). A 6 item simple MRI score (range 0–10) was derived from assessment of the worst segment (bowel wall thickness, oedema, and relative enhancement, mesenteric oedema and pre-stenotic dilatation) plus total disease extent (overall kappa = 0.85). For comparisons with FC, studies where the radiologists reported upper GI or colonic inflammation were excluded (27/119).

Results 150 SBMRI scans were re-evaluated from 123 patients with purely ileal Crohn's (Montreal L1, n = 109; L3 + previous panproctocolectomy, n = 14; 65% female; median age at MRI