centres taking part in the 2015 audit, steroid exposure rates fell from 30% to 23.8% ($p=0.003$) and steroid excess from 13.7% to 11.5% ($p=NS$). Steroid exposure and excess rates for sites that had not been part of the previous audit were significantly higher (31.0% excess, 17.1% exposure, $p=0.0001$ for both). There were no significant differences in important baseline characteristics of 2 groups of sites. Logistic regression analysis revealed independent predictors of reduced risk of steroid excess, after correction for disease severity. For CD these included treatment with anti-TNF therapy ($p=0.04$), treatment in a centre with regular IBD multidisciplinary team (MDT) meetings ($p=0.01$) and treatment in an original 2015 centre ($p=0.02$). For UC treatment in a 2015 centre was also significant predictor of protection ($p=0.04$) and treatment with thiopurine monotherapy a predictor of risk of excess ($p=0.01$); usage of anti-TNF therapy in UC did not reach significance for protection from excess.

Conclusions Changes in biologic access in the UK have resulted in only modest changes in prescribing behaviour and have not yet impacted significantly on excess steroid exposure in UC, unlike in CD. Participation in an audit cycle of steroid usage was associated with a meaningful reduction in steroid excess. These data support the concept that steroid excess could be used as a key performance indicator in IBD and physicians should be engaged in this process.

ADTU-04 FAECAL CALPROTECTIN IN PSC-IBD: A NOVEL MARKER OF CHOLANGITIS

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10.1136/gutjnl-2018-BSGAbstracts.120

Introduction Primary sclerosing cholangitis (PSC) is a chronic inflammatory condition of the bile ducts leading to fibrosis and end stage liver disease. A lack of robust non-invasive biomarkers has been hindering disease monitoring and development of optimal therapies. We have previously noted that the high levels of faecal calprotectin (fcal) seen in PSC-IBD patients belie the mild or quiescent intestinal inflammation. An unsupervised proteomics study identified biliary calprotectin as a potential biomarker. Here, we test the hypothesis that fcal is a marker of biliary injury in PSC.

Methods We analysed paired endoscopic activity data (UCEIS) and fcal results of patients with PSC-IBD ($n=20$) or UC ($n=20$) who underwent colitis surveillance in the context of a colitis surveillance pilot study. Relevant clinical data was recorded prospectively. Recruiting consecutive patients attending for ERCP ($n=6$) allowed for the concomitant testing of biliary and faecal calprotectin.

Results As expected, fcal strongly correlated with severity of mucosal injury (UCEIS) in UC [$r=0.82$, 95% CI(0.58, 0.92), $p<0.0001$]. However, the correlation was weaker in PSC-IBD [$r=0.59$, 95% CI(0.19, 0.82), $p=0.006$]. Moreover, in patients with PSC-IBD and quiescent colitis (UCEIS: 0–1) fcal concentration was significantly higher in comparison to UC patients with comparable endoscopic activity [279 ug/g (10, 1560) vs. 30 (10, 161), $p=0.015$]. A trend towards abnormal liver biochemistry was seen in those PSC-IBD with higher fcal [ALP: 250IU/L (113, 561) vs. 83 (59, 170), $p=0.06$, GGT: 351 U/L

(117, 1014) vs. 51 (29, 153) $p=0.02$, AST: 53 U/L (26, 85) vs. 37 (22, 43), $p=ns$]. UC patients with quiescent colitis and fcal >150 had a higher risk of colitis relapse in 12 months [HR=7.6, 95% CI(1.8, 33.6)] in comparison to those with fcal <150. However, in patients with PSC-IBD and quiescent colitis a fcal >150 was associated instead with a higher risk of cholangitis associated complications (need for antibiotics or stent insertion), HR=6.5, 95% CI(1.3, 33.9). Strikingly, biliary calprotectin concentration showed a strong correlation with fcal concentration ($r=0.90$, $p=0.04$). Interestingly, immunostaining of biliary brushings for calprotectin demonstrated positive staining in cholangiocytes as well as neutrophils and macrophages.

Conclusion In patients with PSC-IBD and quiescent colitis the identification of a raised fcal is likely to herald complications of inflammation in the bile ducts rather than the colon. In this setting, fcal may be a valuable prognostic biomarker of cholangitis. Additionally, our data suggest that in PSC, the source of raised fcal may also be the damaged biliary epithelium.

ADTU-05 GUT MICROBIAL COMPOSITION IN THE MIGRANT SOUTH-ASIAN IBD POPULATION IN UK

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10.1136/gutjnl-2018-BSGAbstracts.121

Introduction Epidemiological studies have highlighted that the South Asian migrant population in UK have a comparable risk of developing IBD but with a more aggressive phenotype than the white Caucasian population. It remains unclear if this is due to environmental/lifestyle factors or differences in host genetics. The human gut microbiota is impacted by health status and diet and therefore represents a potentially adaptive phenotype that is influenced by the environment. As the gut microbiota has been shown to be different in the native South Asian population compared to those in developed countries, we aimed to investigate if there were ethnic differences in the microbiota in IBD patients.

Methods Stool samples were collected from South Asian ($n=20$) and Caucasian patients ($n=46$) with IBD attending outpatient clinics at University Hospital Birmingham along with healthy controls ($n=17$). DNA was extracted and the V4 hyper-variable region of the 16S rRNA gene amplified and sequenced. Analysis was performed on the QIIME pipeline using the GreenGenes database. Diversity analysis was corrected for false discovery rates.

Result Patients with IBD had a significantly different gut microbial composition in comparison to healthy controls as expected ($p=0.01$). Gut microbial diversity was reduced in IBD ($p=0.001$). A significant decrease in *Firmicutes* phylum was observed in patients with IBD in comparison to healthy controls which was primarily due a reduced abundance of communities from genus *Faecalibacterium praeunitionis*, *Lachnospiraceae*, *Ruminococcus* and *Blautia* ($p<0.002$). Within the IBD cohort, the alpha or beta diversity of gut microbiota was very similar for South Asians and Caucasian patients. No significant differences were seen at any taxonomic levels.

Published literature have previously demonstrated that the gut microbiota in healthy South Asians living in the subcontinent was enriched with populations of *Lactobacillus*, *Ruminococcus*, and *Bifidobacterium* bacteria. In our South Asian IBD cohort these microbial communities were significantly reduced.

Conclusion This is the first study to date investigating the gut microbial composition in the migrant population with IBD in UK. The gut microbiota in South Asian IBD patients is similar to Caucasian IBD patients living in UK. Our findings suggest the need to explore further the role environmental factors in the development of IBD associated dysbiosis but also the role of host factors in pathogenesis.

ADTU-06 'REAL WORLD' OUTCOMES OF VEDOLIZUMAB OVER 12 MONTHS: THE CROSS PENNINE MULTICENTRE EXPERIENCE

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10.1136/gutjnl-2018-BSGAbstracts.122

Background Vedolizumab (VDZ) is a gut selective $\alpha 4\beta 7$ anti-integrin approved for the treatment of UC and CD. We aimed to assess one year clinical and safety outcomes of VDZ.

Methods We previously reported 12 week outcomes using retrospectively collected demographic, clinical, and adverse effects data of patients treated with VDZ at 8 UK centres since 2014. We now report longer term outcomes, evaluating clinical response at week 12 and 52, using Physician Global Assessment, Harvey Bradshaw Index (HBI) and partial Mayo Score (pMS).

Results Of 203 patients, 135 had CD (96% anti-TNF experienced), and 68 UC (66% anti-TNF experienced). 101 received concomitant immunomodulator therapy and 97 received steroid bridging therapy.

Of 135 CD patients, 9 discontinued VDZ prior to week 12. At week 12, 38.5% were in PGA remission and 40.0% had a PGA response. Between week 12 and 52, a further 35 patients discontinued VDZ. At week 52 PGA remission and response were seen in 39.2% and 24.3% respectively. Mean HBI decreased from 9.2 (baseline) to 5.1 (week 12) and 5.2 at week 52 ($p < 0.01$).

Of 68 UC patients, 3 discontinued VDZ prior to week 12. At week 12, 48.5% were in PGA remission and 42.6% had a PGA response. Between week 12 and 52, a further 9 patients discontinued VDZ. At week 52, PGA remission and response were seen in 67.2% and 16.4% respectively. Mean pMS decreased from 6.1 (baseline) to 3.1 (week 12) and 2.2 (week 52; $p < 0.01$).

Adverse events were reported in 48 cases (24%) Of these 29 were infection related. Overall incidence of infection was 11.1 per 100 person-years of VDZ exposure.

Conclusion In our cohort of refractory (predominantly anti-TNF experienced) patients, VDZ proved to be safe and effective. Although at 12 weeks response/remission rates were similar in CD and UC, VDZ appeared to be more effective at maintaining remission for UC compared to CD. The incidence of infectious complications was lower than that seen with anti-TNF therapies (average 14 per 100 person-years).

PTU-001 TOFACITINIB, AN ORAL JAK INHIBITOR, IN THE TREATMENT OF ULCERATIVE COLITIS: OPEN-LABEL, LONG-TERM EXTENSION STUDY

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10.1136/gutjnl-2018-BSGAbstracts.123

Introduction Tofacitinib is an oral, small molecule JAK inhibitor that is being investigated for ulcerative colitis (UC). The efficacy and safety of tofacitinib was demonstrated as induction and maintenance therapy in 3 Phase 3, randomised, placebo-controlled studies (OCTAVE Induction 1, NCT01465763; OCTAVE Induction 2, NCT01458951; OCTAVE Sustain, NCT01458574) in patients (pts) with moderate to severe UC.¹

Methods We present interim safety and efficacy data up to 3 years of treatment (as of 8 July 2016) from an ongoing Phase 3, multicentre, open-label, long-term extension study (OLE; NCT01470612) in pts who had completed or demonstrated treatment failure in OCTAVE Sustain, or who were non-responders after completing OCTAVE Induction 1 or 2. Pts in remission at Week 52 of OCTAVE Sustain received tofacitinib 5 mg twice daily (BID); all others received 10 mg BID. At Month 2, all pts underwent endoscopy, and non-responders from Induction were mandated to withdraw if no evidence of clinical response was shown. Remission was defined by a Mayo score ≤ 2 with no individual subscore > 1 , and rectal bleeding subscore of 0. Binary efficacy endpoints were derived from Mayo score, based on local-read endoscopic subscore.

Results 914 pts (5 mg BID, $n=156$ [17.1%]; 10 mg BID, $n=758$ [82.9%]) received ≥ 1 dose of study drug; 381 pts (41.7%) discontinued. The most frequent AE leading to discontinuation was worsening of UC. The most frequent treatment-emergent AEs by system organ class (both doses) were 'infections/infestations' and 'gastrointestinal disorders', and by preferred term were 'nasopharyngitis' and 'worsening of UC'. Serious infections AEs were reported in 4 (2.6%) and 14 (1.8%) pts with 5 and 10 mg BID, respectively. Malignancies excl. NMSC were reported in 9 (1.2%) pts in the 10 mg BID group (no clustering of malignancy type); none were reported in the 5 mg BID group. No new safety risks were identified. Data 'as observed' for remission and mucosal healing at Months 2, 12 and 24 are shown.

Conclusions In pts with moderate to severe UC who remained in the OLE study, no new safety concerns emerged compared with those observed with tofacitinib in rheumatoid arthritis. Efficacy results from this OLE study support sustained efficacy with tofacitinib 5 and 10 mg BID.

Funded by Pfizer Inc.