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.

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

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**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: 250 U/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.
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.

**PTU-001**

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

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**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. 

**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.

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