and time to discontinuation is shown in table 1. There were no associated factors with time to discontinuation. Maintenance frequency of 12 weeks was half as likely to be associated with discontinuation, but not statistically significant. Only 8/44 on 8-weekly maintenance frequency de-escalated to 12-weekly.

Conclusion Only a third of CD patients discontinued ustekinumab at 2 years follow-up and 5% discontinued therapy between year 1 and 2 of treatment. This suggests clinical response within the first year of treatment is likely to be sustained for another year. None of the patient, disease or drug-related factors predicted drug discontinuation.

P102 OUTCOMES OF BIOSIMILAR ADALIMUMAB SWITCHBACKS/REVERSE SWITCHING IN IBD: REAL-WORLD EXPERIENCE

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10.1136/gutjnl-2020-bsgcampus.177

Introduction Biosimilars of adalimumab are now used throughout the National Health Service (NHS). Patients who switch to biosimilars may develop adverse effects such as cutaneous reactions or disease flares, and are sometimes switched back (reverse switching) to the originator. There is limited information about the outcomes of these switchbacks, particularly in the event of disease flares. At East Sussex NHS Trust (ESHT), clinical information regarding switchback patients was collected and the outcomes analysed to gain a better understanding of the effects of reverse switching.

Methods This was a retrospective review of a database of all IBD patients who underwent switching from the originator (Humira®) to biosimilar adalimumab (Imraldi®) at ESHT. Patients who encountered adverse events post-switch were discussed at the IBD multi-disciplinary meeting and a consensus decision was made whether a switchback was appropriate. Data was collected on Harvey Bradshaw Index (HBI), Simple Colitis Activity Index (SCCAI),C reactive protein, faecal calprotectin (FC) and adverse event reports. Disease flare for this study was defined as symptoms suggestive of a flare in conjunction with a rise in HBI/SCCAI. Patients who were switched back were followed up by the IBD specialist nurse and clinical outcomes post-switchback were documented.

Results At ESHT, 113 IBD patients were switched to Imraldi®(Biogen). There were in total 17 switchbacks (12 female, 5 male) to the originator Humira®(Abbvie). 11 of the switchbacks were due to cutaneous reactions/severe pain on injection/joint pain. These 11 patients continue to remain on Humira®. 6 of the switchbacks were due to disease flare (3 patients on weekly dosing, 3 patients on fortnightly dosing). Of these 6 switchbacks, only 4 patients remain on the originator to date. 1 patient was assessed further with tests and her symptoms were found to be functional in nature. 1 patient was switched to vedolizumab. 1 patient had a further hospital admission for a Crohn's flare that required steroids but remains on Humira®. The rate of capture of clinical response was 50% if the switchback was due to a disease flare.

Conclusions As more NHS Trusts engage in switches to adalimumab biosimilars, they will increasing also face patients where a switchback may be considered. Our local experience shows that switchbacks instigated due to a disease flare should be considered with caution as the rate of clinical response is low. More research is required to ascertain the true effects of switchbacks on patient clinical outcomes to devise appropriate treatment algorithms for such situations.

P103 USTEKINUMAB: MEDIUM-TERM OUTCOMES FROM A UK MULTI-CENTRE REAL-WORLD COHORT

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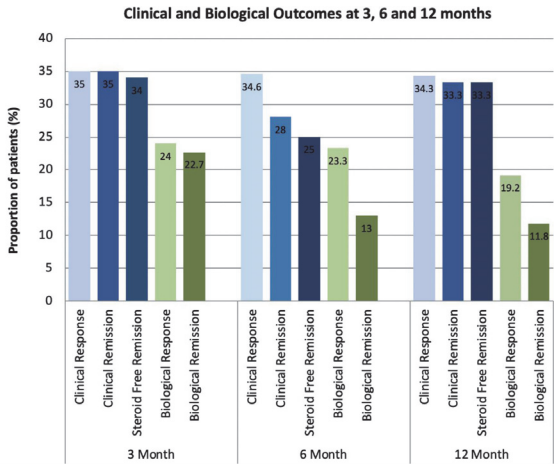
10.1136/gutjnl-2020-bsgcampus.178

Introduction Ustekinumab (UST) is effective at inducing and maintaining remission of Crohn's disease (CD). However, real-world practice varies regarding concomitant immunomodulator (IM) use and dosing regimens, with scarce data on factors predicting outcomes. We present a UK real-world (RW), multi-centre cohort.

Methods A retrospective study was conducted of adult patients with CD initiated on UST between October 2016–18 at two tertiary London centres. Clinical endpoints were (i) remission (Harvey Bradshaw Index (HBI) ≤4), (ii) response, (reduction in HBI of ≥3 or sustained HBI≤4 points), at 3, 6 and 12 months. Biological endpoints were remission & response (CRP<5 mg/L in patients with a baseline CRP >5 mg/L, and 50% reduction in CRP respectively). Predictable variables were assessed by multivariate analysis using logistic regression.

Results The baseline characteristics of 120 patients were: 59 (49%) male; median age 34 yrs (IQR 26–44); median disease duration 12 yrs (7–17); 80 (66%) ileocolonic disease; 88 (73%) stricturing or penetrating disease; 61 (51%) perianal disease. 117 (98%) were biologic exposed, 34 (28%) had failed ≥3 biologics and 67 (56%) had required previous surgery. 15 (13%) patients were on steroids at baseline. Mean HBI was 5 (sd 5, n=112), CRP 15 (sd 18, n=117) and faecal calprotectin 431 (sd 808, n=46).

Clinical and biological endpoints are shown in figure 1. At 6 and 12 months, 79 (74%) and 68 (78%) patients were on escalated 8 weekly dosing. Dosing regimen did not impact outcomes.



Abstract P103 Figure 1 Clinical and biological outcomes at 3, 6 and 12 months

UST discontinuation occurred in 2 (1.7%), 13 (10.8%) and 33 (27.5%) patients by 3, 6 & 12 months. Reasons included (n): primary non-response (23), loss of response (9), sub-optimal response (4) and side effects (4). Adverse events occurred in 23 (19%) patients, including 12 patients requiring surgical intervention for progressive disease.

Concomitant IM were prescribed in 56 (47%) patients at baseline and continued in 47 (39%) and 38 (32%) at 6 and 12 months, respectively. Concomitant use did not impact outcomes at any timepoint, nor affect median treatment persistence with UST. Prior anti-TNF exposure was a negative predictor of clinical remission at one year, OR 0.32 (95% CI 0.12–0.81, p≤0.02), but there was no association with disease location, phenotype, presence of perianal disease or those with a smoking history.

Conclusions Ustekinumab is effective in the treatment of moderate-severe CD in a treatment refractory RW cohort. In keeping with trial data, prior anti-TNF exposure was a negative predictor of remission and concomitant IM use did not alter clinical or biological outcomes.

P104 FREQUENT DISEASE RELAPSE AFTER WITHDRAWAL OF INFLIXIMAB IN IBD PATIENT WITH SUSTAINED REMISSION

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10.1136/gutjnl-2020-bsgcampus.179

Introduction In the UK, NICE guidance recommends annual review of biologics, with withdrawal of therapy in all patients in remission. This study retrospectively evaluates disease course following withdrawal of infliximab in IBD patients with sustained remission.

Primary Outcome Relapse free survival.

Secondary Outcomes Identification of predictors of relapse and evaluation of response to future therapy.

Methods IBD patients from Royal London Hospital who ceased infliximab due to sustained remission were identified. The following information was obtained from electronic patient records: demographics, Montreal classification, immunomodulator use, clinician determined relapse, objective evaluation of disease activity within 3 months prior to treatment cessation and 6/12/18/24 months following cessation (CRP>5, calprotectin>50, endoscopic, radiological), steroid use at relapse, subsequent biologic use and outcome Analysis was undertaken for total IBD, CD and UC. Survival analysis and logistical regression was calculated using SPSS®.

Results 75 patients were identified. CD:UC = 43:32. F:M = 34:41, median age = 31.3 years (IQR 41.15–40.75), median duration of follow up = 21.1 months (IQR 11.1–44.2), Asian:Black:Caucasian:Unknown = 16:3:47:9. The median relapse free survival for CD was 12.4 months (IQR 10.4–14.4) and for UC was 18.2 months (IQR 10.5–25.9). Relapse rates for patients who had completed follow up for each time point are presented in table 1:

Abstract P104 Table 1

	6 months	12 months	18 months	24 months
CD	23.7% (9/38)	51.4% (19/37)	63.3% (19/30)	75% (15/20)
UC	20% (5/25)	31.6% (6/19)	52.6% (10/19)	78.6% (11/14)
IBD total	22.2% (14/63)	44.6% (25/56)	59.2% (29/49)	76.5% (26/34)

In univariate analysis, perianal disease and L3 disease were negatively associated with relapse at 1 year for patients with CD (perianal OR 0.87 CI 0.09–0.81 p=0.03 and L3 OR 0.18 (comparator L1) CI 0.04–0.87 p=0.03). However significance was lost when multivariate analysis was undertaken. Following relapse, 43.1% (19/44) required steroids and 88.6% (39/44) restarted a biologic, 69.2% (30/44) restarting infliximab. Of those who restarted infliximab, 56.7% (17/30) responded to standard therapy, with 10% (3/30) requiring dose escalation. 33.3% (10/30) required alternative therapy.

Conclusion Within 24 months of cessation 76.5% patients relapsed. The majority of these restarted a biologic. However, only 56.7% patients who restarted infliximab responded to standard dose. With additional costs of newer biologics and morbidity of disease flare and steroid use, routine withdrawal of TNF antagonists should only occur after careful consideration.

P105 DENDRITIC CELLS IMPRINT PRO-INFLAMMATORY α4β7 +CLA+ T CELLS WITH POTENTIAL FOR GUT AND SKIN HOMING

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10.1136/gutjnl-2020-bsgcampus.180

Background Integrin α4β7 induced on T cells during activation in intestinal lymphoid enables selective homing to the intestinal mucosa. Retinoic acid (RA) produced by the activating dendritic cell (DC) induces α4β7 and also inhibits the fucosyltransferase FUC-T VII that otherwise generates the selectin ligand CLA required for skin homing. Therefore, antigen experienced T cells are generally either gut tropic (α4β7 +CLA-) or skin tropic (α4β7-CLA+). We hypothesised the existence of additional 'dual tropic' (α4β7+CLA+) T cells, generated in the gut but with capacity to traffic to skin; such cells could explain skin inflammation in inflammatory bowel disease (IBD). Here, we report the generation of dual tropic cells *in vitro* and characterise the population in blood.

Methods Using flow cytometry, expression of α4β7 and CLA was assessed on *ex vivo* T-cells in whole blood and on proliferating cells generated by stimulation of naïve CD4+ T cells with monoclonal antibodies (anti-CD3/28/2), or with allogeneic colonic or monocyte-derived DC (moDC). Cultures were in the presence or absence of serum, monoclonal antibodies, RA receptor (RAR)α antagonist, or conditioned media. Expression of FUCT-VII was assessed by qRT-PCR.

Results T-cells activated with antibodies expressed β7 but not CLA. Inhibition of RARα signalling and removal of serum reduced β7 expression and induced both CLA and FUCT-VII expression, suggesting endogenous RARα signalling shapes homing phenotype in these cultures. In contrast, activation with DC (colonic or RA-generating moDC), generated CLA+ T cells, including a population which co-expressed β7. Conditioned medium from DC stimulated cultures did not induce CLA on antibody-activated cells.

Activation by DC or in the presence of the RARα antagonist both led to increased expression of FUCT-VII. However,