outcome was clinical response defined by a fall in PGA. Secondary outcomes were drug persistence and fall in CRP or faecal calprotectin (FC). Statistical analysis was performed by Wilcoxon signed rank test in R v.3.4.4.

**Results** 84 patients (mean age 42 years, sd 14.7, mean disease duration 12.3 years, sd 8.9) were treated to December 2018. 82 (98%) had previous exposure to anti-TNF therapy, 35 (42%) to both anti-integrin and anti-TNF and 2 were naïve to biologics. 39 (46.4%) were on immunomodulator co-therapy and 6 (7%) on steroids. Post-induction treatment was 12 weekly (9), 8 weekly (74) or 6 weekly (1).

Post-induction (median 14.7 weeks, IQR 8.03) and 1 year (median 44.4 weeks, IQR 13.4) reviews were available for 72 and 47 patients respectively. Post-induction clinical response occurred in 38 (53%) and remission in 5 (7%). At 1 year response occurred in 34 (72%) and remission in 7 (15%). Of 15 non-responders post induction left on UST, 9 responded by 1 year. Steroid-free response occurred in 35 (48%) post-induction and 31 (65%) at 1 year.

By 1 year 8 patients stopped UST (median 256 days, range 62–329); 1 cutaneous reaction, and 7 primary non-response or secondary loss of response. Only 1 of 8 given a second intravenous dose in an attempt to recapture response had a sustained benefit.

For those with an elevated biomarker at baseline (CRP $>10$ or FC $>250$) and repeat measurement post induction, mean CRP improved from 37 (11–96) to 28 (3.8–116) (p=0.074) and FC 2260 (292–6000) to 1152 (24–5084) (p=0.089). For paired results at baseline and 1 year mean CRP improved from 29 (11–96) to 19 (3–77) (p=0.045) and mean FC 2019 (823–6000) to 1070 (42–5528) (p=0.011). There was no significant change in normal baseline biomarkers.

**Conclusions** UST was well tolerated in a complex UK CD cohort and demonstrated benefit in terms of clinical response, improvement of biomarkers and in some full clinical remission. Due to delayed response continuation beyond 3 months is justifiable in some non-responders. Reloading to recapture response had as described elsewhere was of limited benefit.

**Results** In the majority of non-IBD controls, co-culture of colonic lymphocytes with HEK293T cells co-transduced with intestine specific BTNL3+BTNL8 proteins, resulted in profound TCR down-regulation in a subset of γδT cells (specifically those expressing the Vγ2/3/4 chains). αβ7+ is a lymphocyte marker of epithelial residence present on the vast majority of γδT cells in health. αβ7+ + γδT cells make TCR down-regulation responses to BTNL3+8, whereas the subset of γδT cell αβ7+ cells, which are in the minority, had grossly attenuated or absent assay responses. In many IBD patients, there is significantly reduced αβ7+ expression on γδT cells, and a severe attenuation or loss of BTNL-dependent TCR down-regulation. To assess whether the inflammatory milieu of IBD contributes to this dysregulation; addition of pro-inflammatory cytokines IL-12 and IL-18 (but not IL-1β and IL-23) to an organ culture system lead to down regulation of αβ7+ on γδ T cells and a consequent attenuation of response to BTNLs. This clearly implicates specific cytokines in the disruption of the γδ-BTNL axis which is evident in disease. Further characterisation of αβ7+ + γδT cells demonstrated an activated pro-inflammatory phenotype in comparison to more quiescent αβ7– γδT cells.

**Conclusion** The BTN-L-γδT cell axis, is a novel and important mechanism by which epithelial cells and a specific subset of the intraepithelial γδT cell compartment communicate, and is often dysregulated in IBD. While our data suggests that IL-12 blockade may help to restore this axis, IL-23 may be redundant in this setting, with implications for emerging therapeutic strategies. αβ7+ is a current target of therapeutic blockade, which has the potential to disrupt this axis and ultimately may exacerbate disease given the pro-inflammatory nature of αβ7– γδT cells.

**PTh-083** FROM PARIS TO MONTREAL: EVOLUTION OF CROHN’S DISEASE LOCATION AND BEHAVIOUR FROM CHILDHOOD TO ADULTHOOD

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**Introduction** Crohn’s disease (CD) is a progressive disorder but the natural history among paediatric onset CD appears different to that of adult onset CD. The long term disease evolution among paediatric onset CD beyond the childhood years is not well characterised. We conducted a single centre cohort study of all paediatric CD patients transitioned to adult care to assess the long term evolution of CD.

**Method** We conducted a retrospective observational, study of all CD patients diagnosed in childhood who were subsequently transferred to the care of an adult gastroenterology unit and had a minimum follow up of 2 years. We examined the case notes for evolution of disease location and behaviour. Disease location and behaviour was characterised using Paris classification at diagnosis and Montreal classification at last follow-up. In addition, we examined variables associated with the need for CD related intestinal resection. We used a paired McNemar’s test to compare disease location and behaviour at diagnosis and most recent follow-up.

**References**
