

Results 114 patients were identified. 11 patients transferred care and were excluded. 91/103 (88.3%) received maintenance (>6 weeks) treatment. 47.3% had discontinued treatment while 52.7% remained on treatment. The median length of treatment was 73 (range 8–329) weeks. Only 23.3% (10/43) discontinued infliximab due to clinical remission with 34.9% (15/43) stopping because of complications and 39.5% (17/43) due to loss of response, surgery or death. The median course of treatment for those continuing on infliximab was 101 (range 8–329) weeks. 37 patients (21 female, mean age 40 years) were on maintenance infliximab treatment for over 1 year. 73% of these patients were on combined treatment with an immunomodulator and 37.8% (14/37) had required dose escalation or a reduction in dose interval. In patients continuing treatment for over 1 year, the median FC was 184µg/g (range 30–9000) with a median reduction in FC level post-treatment of 416µg/g (range –3000–7086) and a median HBI of 4 (range 0–16). 57.1% could be defined as being in clinical remission with HBI < 5.

Conclusion In our large cohort of CD patients, few patients discontinued infliximab due to clinical remission. In those continuing infliximab for over 1 year the median FC was low, suggesting good control of inflammation, and the majority of patients were in clinical remission as defined by a HBI < 5. These results support the efficacy of infliximab as maintenance therapy in CD but suggest that despite evidence of clinical remission the majority of patients continue therapy.

Disclosure of Interest None Declared.

PWE-075 ASSOCIATION BETWEEN THIOPURINE USE AND NON-MELANOMA SKIN CANCERS IN PATIENTS WITH INFLAMMATORY BOWEL DISEASE: A META-ANALYSIS

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Introduction Thiopurines are the mainstay of treatment for patients with inflammatory bowel disease (IBD). Thiopurine therapy increases the risk of non-melanoma skin cancers (NMSC) in solid organ transplant patients. The data on NMSC in patients taking thiopurines for IBD is conflicting.

Methods We searched electronic databases (PubMed, OVID, the Cochrane library, EMBASE and CINAHL) for full journal articles reporting on the risk of developing NMSC in patients taking thiopurines for IBD and hand searched the reference lists of all retrieved articles. Pooled adjusted hazard ratios and 95% confidence intervals were determined using a random effects model. Publication bias was assessed using Funnel plots or Egger's test for regression asymmetry. Heterogeneity was assessed using Cochran's Q and the I² statistic.

Results A total of 8 studies involving 60,351 patients provided data on the risk of developing NMSC in patients with IBD on thiopurines. The pooled adjusted hazard ratio of developing NMSC after exposure to thiopurines in patients with IBD was 2.275 (95% CI 1.502 to 3.446). There was significant heterogeneity (I² 76%) between the studies, but no evidence of publication bias ($P = 0.15$, intercept = –6.7 and 95% CI: –17.2 to 3.7). Meta regression analysis suggested that the population studied (hospital based versus population based) and duration of follow up (> 3 years) were the major contributors to the heterogeneity. Grouping studies according to population studied and also duration suggests that the risk was much higher in hospital based and shorter duration studies (Table).

Conclusion The risk of developing NMSC in patients with IBD on thiopurines is only modestly elevated. This effect loses significance when studies with less than 3 years follow-up are excluded. The difference in pooled risk between population based and hospital based studies suggests the possibility that ascertainment bias could have contributed to this increased risk. Use of thiopurines to treat IBD should not be limited by this marginally increased risk of NMSC.

Disclosure of Interest None Declared.

Abstract PWE-075 Table

	Pooled Hazard Ratio	95% Confidence Intervals
All Studies	2.275	1.502–3.446
Follow-up < 3 years	2.869	2.017–4.080
Follow-up > 6 years	1.876	0.868–4.056
Population based	1.828	1.196–2.795
Hospital based	7.217	3.082–16.898

PWE-076 DENDRITIC CELL CHARACTERISTICS IN POUCHITIS

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Introduction The role of dendritic cells (DC) in inflammatory bowel disease is increasingly recognised for their function in regulating intestinal immune responses. To our knowledge there are no previous studies of DC in pouchitis. We aimed to characterise changes in DC in pouchitis that may underlie the dysregulated immune response to the pouch microbiota.

Methods Mucosal biopsy samples were taken from patients with pouchitis (n = 14) and ulcerative colitis patients without pouchitis (n = 10). Lamina propria DC were isolated by collagenase digestion. DC were identified as an HLA DR+, lineage – (CD3-, CD14-, CD16-, CD19-, CD34-) population. DC expression of TLR 2 and 4, CCR9, β7 and CD40 were measured by multicolour flow cytometry. The t-test was used for statistical analysis.

Results DC expression of TLR 2 and 4 were both significantly elevated in patients with pouchitis compared with non-pouchitis patients ($p = 0.007$ and 0.008). In pouchitis patients, DC expression of β7 was increased ($p = 0.02$) and expression of CCR 9 was decreased ($p = 0.02$). DC expression of CD40 was increased in patients with pouchitis ($p \leq 0.0001$).

Conclusion In pouchitis, DC are activated and upregulate expression of microbial recognition receptors. In addition, DC expression of gut homing markers is elevated in pouchitis with a more colonic homing marker profile. Similarly to other IBD, DC are likely to be key in the initiation and perpetuation of the inflammatory response to the dysbiosis of the pouch microbiota

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PWE-077 ALTERED EPITHELIAL TIGHT JUNCTION EXPRESSION AND ELEVATED IL 6 LEVELS IN POUCHITIS

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Introduction Intestinal epithelial barrier function limits the interactions between microbial antigens and the mucosal immune system. In IBD, epithelial barrier function is impaired with altered expression of tight junctions. We aimed to assess epithelial tight junction expression and mucosal cytokines in acute and chronic pouchitis and non-inflamed pouches of patients with ulcerative colitis.