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 remision 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 I^2 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 (I^2 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 initation 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.

Methods Mucosal biospy samples were taken from ulcerative colitis patients with pouchitis (chronic pouchitis n = 9, acute pouchitis n = 4) and those without pouchitis (n = 11). Epithelial cells were isolated from biopsy tissue after incubation with DTT and EDTA. Eptihelial cell expression of ZO-1, claudin 1 and claudin 2 were measured by multicolour flow cytometry. Cytokines were assessed by multiplex ELISA of biopsy supernatants. The t-test was used for statistical analysis.
Results In acute pouchitis ZO-1 was elevated compared with both chronic pouchitis and non-pouchitis (p = 0.008), whilst in chronic pouchtis ZO-1 expression was reduced compared with non pouchitis (p = 0.006). Claudin 1 expression was reduced in chronic pouchitis (p = 0.04), but was not significantly reduced in acute pouchitis. In acute pouchitis, claudin 2 expression was elevated (p≤0.001), but was not increased in chronic pouchitis. IL6 levels were elevated in chronic pouchitis compared with non pouchitis patients (p = 0.01).
Conclusion Epithelial tight junction expression was altered in pouchitis in association with increased IL6 levels. Increased claudin 2 expression in acute, but not chronic pouchitis may represent early pathological changes in the development of pouch inflammation. In chronic inflammation the tight junction complex was deranged with reduced expression of both claudin 1 and ZO-1. Increased epithelial barrier permeability due to altered tight junction expression may be a critical mechanism in the development and perpetuation of pouch inflammation.
Disclosure of Interest None Declared.

PWE-078 A PROSPECTIVE CONTROLLED PILOT STUDY OF FECAL MICROBIOTA TRANSPLANTATION FOR CHRONIC REFRACTORY POUCHITIS

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Introduction Faecal microbiota transplantation (FMT) is an effective therapy for *Clostridium difficile* and possibly inflammatory bowel diseases (IBD). Published data of FMT for inflammatory bowel diseases are reported in case series and case reports. To our knowledge, there are no controlled studies of FMT for IBD. We aimed to conduct a prospective study of FMT for chronic refractory pouchitis.
Methods Patients with clinically, endoscopically and histologically confirmed chronic refractory pouchitis; with a pouch disease activity index (PDAI) > 7 were included. Donors were screened by clinical history and serology for HAV, HBV, HCV, HEV, Treponema, HIV, HTLV I/II and stool for M, C+S, C. difficile toxin and parasites. Fresh donor stool was collected within six hours of nasogasric administration of FMT. Stool samples were also collected from patients for analysis of coliform sensitivities before and 4 weeks after FMT. PDAI and Cleveland global quality of life score (CGQoL) were recorded prior to FMT and four weeks after FMT.
Results Eight patients with chronic refractory pouchitis who had undergone restorative proctocolectomy for ulcerative colitis underwent FMT. Three patients had ESBL resistant coliforms on stool analysis prior toFMT. Two of these patients demonstrated a change to ciprofloxacin sensitive coliform following FMT. The mean PDAI prior to FMT was 12. The mean CGQoL was 0.45. At 4 weeks following FMT, no patient had achieved a clinical remission (mean PDAI 11). No improvement in CGQoL was seen (mean 0.44).
Conclusion FMT via nasogastric administration was not effective in achieving clinical remission for chronic refractory pouchitis at 4 weeks after FMT. However, in two patients with ESBL resistant coliform, ciprofloxacin sensitivity was regained following FMT and these patients have subsequently been maintained on ciprofloxacin. This suggests FMT may alter the pouch microbiota. Further molecular microbiological analysis is being undertaken to determine the effect FMT had on these patients' microbiota. In addition, further studies of FMT are required to assess the effect of different methods of FMT that may be more efficacious for this group of patients.
Disclosure of Interest None Declared.

PWE-079 LONGITUDINAL ASSESSMENT OF EPITHELIAL AND IMMUNE CELL CHANGES FOLLOWING ILEOSTOMY CLOSURE IN PATIENTS WITH ULCERATIVE COLITIS

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Introduction The interactions between microbiota, epithelial barrier and innate immune responses are important in the pathogenesis of IBD. The ileo-anal pouch offers a unique opportunity to study these inter-relationships before the onset of disease. There are few data regarding tight junction expression or dendritic cell (DC) characteristics following restorative proctocolectomy (RPC). We aimed to assess the relationship between changes in epithelial tight junction expression, dendritic cell phenotype and mucosal cytokine production over the first year following RPC for ulcerative colitis (UC).
Methods Mucosal biopsy samples were taken from the same UC patients undergoing RPC, from the ileostomy afferent loop, the pouch pre-ileostomy closure (P0) and the pouch 6 and 12 months post-ileostomy closure (n = 5). Eptihelial cells expression of zona occludens (ZO)-1, claudin 1 and claudin 2 and DC expression of TLR 2 and 4, CCR9, β7 and CD40 were measured by multicolour flow cytometry. Cytokines were assessed by multiplex ELISA of biopsy supernatants. The paired t-test was used for statistical analysis.
Results Epithelial expression of claudin 2 was increased (p = 0.04) at 6 months and remained elevated at 12 months. No changes were seen in ZO-1 or claudin 1 expression. There was a significant increase in β7 expression on lamina propria DC (p = 0.02), but no differences in DC TLR or CD40 expresssion were seen at 6 months. DC expression of β7 was further elevated (p = 0.005) as well as significantly increased TLR 4 and CD40 expression (p = 0.04). No cytokines were found to be elevated at 6 months, but at 12 months there was a trend towards increased IL6 (p = 0.05).
Conclusion In patients with UC, altered tight junction expression with increased epithelial expression of the "pore-forming" tight junction claudin 2 was an early event after ileostomy closure that preceded the onset of mucosal inflammation. In parallel, more lamina propria DC expressed gut homing markers possibly in response to increased exposure to the changing microbial signals and a more permeable epithelial barrier. These changes in parallel may lead to increased microbial stimulation of DC with increased TLR and co-stimulatory molecule expression that could predispose to the development of inflammation.
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PWE-080 PREVALENCE OF FAECAL INCONTINENCE IN ADULTS WITH INFLAMMATORY BOWEL DISEASE

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