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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[1,2,*]J Landy, [2]H O Al-Hassi, [2]E R Mann, [3]S T Peake, [4]S D McLaughlin, [5]Z L Perry-Woodford, [6]P J Ciclitira, [7]J Nicholls, [8]S K Clark, [2]S C Knight, [1]A L Hart. *[1]St Mark's IBD Unit, St Mark's Hospital; [2]APRG, Imperial College; [3]IBD Unit, St Mark's Hospital, London; [4]Gastroenterology, Royal Bournemouth and Christchurch Hospitals, Bournemouth; [5]Pouch Care, St Mark's Hospital; [6]Gastroenterology, St Thomas' Hospital; [7]Biosurgery and Surgical Technology, Imperial college; [8]Colorectal Surgery, St Mark's Hospital, London, UK*

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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[1,2,*]J Landy, [2]H O Al-Hassi, [2]E R Mann, [3]S T Peake, [4]P J Ciclitira, [5]J Nicholls, [6]S K Clark, [2]S C Knight, [1]A L Hart. *[1]St Mark's IBD Unit, St Mark's Hospital; [2]APRG, Imperial College; [3]IBD Unit, St Mark's Hospital; [4]Gastroenterology, St Thomas' Hospital; [5]Biosurgery and Surgical Technology, Imperial college; [6]Colorectal Surgery, St Mark's Hospital, London, UK*

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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