were included in this study. Before enrolment into the study, the patients were explained about the study and informed consent was obtained. The patients with unidentified colitis were excluded. The data on demographics, disease characteristics, FI (Vaizey score), and quality of life (IBD-Q) were collected. Data were analyzed using SPSS version 21.

Results There were 184 patients (women = 101, 54.9%; UC = 153, 83.2%) with a female preponderance for UC (male/female ratio = 1:1.5) and a male preponderance for CD (male/female = 2:1). Forty-eight (26%) patients reported symptoms of FI. Among the patients with FI, 70.8% were women (n = 34) and 29.2% were men (n = 14) with an average age of 52.7 years (range, 20–78 years). Average age of onset of FI was 48.6 (range, 22–74) years. Ten percent (n = 5) reported regular FI. Incontinence to flatus was seen in 33.3% (n = 16), to liquid faeces in 56.2% (n = 27), to solid faeces in 6.2% (n = 3) and to all three in 4.1% (n = 2). Twenty-one percent (n = 10) complained of disruption of their physical and social activity. There was no association between FI and type of IBD. Significant associations were found between FI and age (P = 0.005) and gender (P < 0.001). QOL in our cohort of patients was significantly affected by FI.

Conclusions In our study, nearly a quarter of patients reported FI. There was a significant correlation between FI and QOL. Therefore, enquiring about FI in IBD patients can lead to identification of this debilitating condition. This will enable early referral for continence care in this group of patients.

P160 PRE-BIOLOGIC SCREENING IN A HIGH RISK AREA: ARE WE ADHERING TO GUIDELINES?

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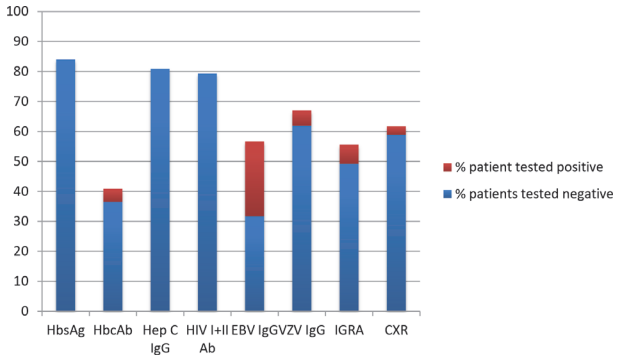
Introduction Newham University Hospital, Bart's NHS Trust, serves the London Borough of Newham which had the highest incidence of tuberculosis in the UK at 78.0 per 100,000 in 2014. Newham also has the highest average annual rate of new reported acute hepatitis B infection in the UK. There have been clear guidelines on pre-biologic screening for opportunistic infections since 2014. Our aim is to assess whether patients who are on biologic therapy have been appropriately screened prior to initiation of biologic therapy.

Methods A retrospective review of all IBD patients on the biologic database was performed in November 2019. Patients who were initiated on biologics prior to the publication of guidelines in June 2014 were excluded.

Results The total number of patients was 63. 31 patients (49.2%) had latent tuberculosis testing with Interferon-gamma release assay (IGRA) testing and 2 were positive. Screening with Chest XR (CXR) was better with 58.9% concordance. 36 patients had normal CXRs and 1 had an appearance of a granuloma.

In comparison, viral screening had higher completion rates. Hepatitis B Surface Antigen (HbsAg) was sent in 53 patients (84%) and all were negative. Hepatitis B Core Antibody (HbcAb) was sent in 23 patients (36.5%) and 1 was positive.



Abstract P160 Figure 1 Percentage of patients who received respective pre-biologic test

1 patient was HbcAb positive but HbsAg negative. In terms of Hepatitis C, 51 patients (80.9%) had Hepatitis C IgG sent and all were negative. All 50 patients (79.3%) who were tested for Human immunodeficiency virus (HIV I+II antibody) were negative. Ebstein Bar Virus IgG was sent in 20 patients (31.7%), out of which, 15 were negative. Varicella-Zoster Virus IgG was sent in 39 patients (61.9%) and 2 were positive.

An infection history was not taken, for either bacterial, fungal or viral infections and Bacille Calmette-Guerin vaccination status was not documented. No documentation was present regarding measles status. Routine vaccination status was not confirmed for diphtheria, poliomyelitis, pertussis, tetanus or Human Papilloma Virus. Prior to initiation of immunomodulation, vaccination was not considered for pneumococcal or influenza infections.

Conclusions Despite suboptimal pre biologics screening in this high risk region of East London for Tuberculosis and Hepatitis B, no cases of reactivation of either Tuberculosis or Hepatitis B have been identified to date. The results suggest that clinicans are requesting some tests but not all. Following on from these results, we will be streamlining the process for ensuring all tests are performed prior to biologic initiation with a checklist proforma for the patients notes and on our biologics database for all prescribing gastroenterologists, as per ECCO guidelines.

P161 IKKα AS A POTENTIAL THERAPEUTIC TARGET FOR THE PREVENTION OF INFLAMMATORY BOWEL DISEASE

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Introduction Current treatment options for inflammatory bowel disease (IBD) are primarily designed to suppress an established inflammatory response, but they do not prevent the initiation of the inflammatory cascade. We have previously demonstrated that the NF-κB signalling pathways play a pivotal role in murine experimental models of IBD. In particular, we showed that *nfκb2-/-* mice were protected against LPS-induced cell shedding and DSS-induced colitis compared to wild-type mice. Specifically, our data suggested that NF-κB2 signalling in intestinal epithelial cells played a more important

role than previously thought. We have now investigated whether IKKα represents a potential therapeutic target for IBD using murine epithelial intestinal organoids.

Methods Intestinal crypts were harvested from C57BL/6J mice (n=3) and cultured into enteroids in 3D using a Matrigel matrix. Enteroids were either untreated or administered 0.6 μM of IKKα inhibitor SU1433 on day 3 after passage and stimulated with 30 ng/ml TNF on day 4. Enteroid morphological changes were assessed daily using the Enteroid Circularity Score. Immunohistochemistry for active caspase-3 and Ki-67 was performed to assess apoptosis and cell proliferation. Enteroids were harvested at 3, 6, 24, and 48 hours after stimulation and RT-qPCR was performed to determine the expression profiles of selected inflammation-related and non-canonical NF-κB related target genes.

Results Enteroids pre-treated with SU1433 and then stimulated with TNF were protected against enteroid rounding compared to TNF treatment alone. There was also a marked decrease in active caspase-3 positive apoptotic cells 48 hours following TNF in the SU1433 pre-treated group compared to the TNF only group. At the 3, 6, 24 and 48 hour time points NF-κB2, TNF, CXCL9 and ICAM-1 expressions were significantly increased compared to TNF-naïve groups, however there was no significant difference between the SU1433+TNF and TNF only treatment groups.

Conclusions TNF induced enteroid rounding and promoted the expressions of several inflammation related genes in the NF-κB signalling pathways. Pharmacological inhibition of IKKα prevented TNF-induced enteroid rounding. This was associated with a significant decrease in apoptosis on histology. Together, our findings suggest that IKKα may be a potential therapeutic target for the prevention of IBD relapse.

P162 COMPARATIVE EFFECTIVENESS OF VEDOLIZUMAB AND USTEKINUMAB IN ANTI-TNF REFRACTORY CROHN'S DISEASE: MULTI-CENTRE RETROSPECTIVE COHORT STUDY

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Introduction Anti-tumour necrosis factor (TNF) agents are effective in Crohn's disease (CD), but up to 30% of patients fail to respond or develop intolerance and require alternative biological therapy. Both vedolizumab and ustekinumab are licensed to treat anti-TNF refractory CD patients. Clinical trials of vedolizumab and ustekinumab in anti-TNF refractory patients seem to suggest comparable efficacy, but no real-life data exist to facilitate decision-making. We conducted a multicentre retrospective cohort study to assess the comparative effectiveness of vedolizumab and ustekinumab in treating anti-TNF refractory CD.

Methods Anti-TNF exposed CD patients then treated with vedolizumab or ustekinumab were included. Disease activity was monitored serially by calculation of Harvey-Bradshaw index (HBI) for up to 12 months. Faecal calprotectin (FC) at baseline and subsequent visits were recorded if available. Clinical response was defined as decrease in HBI ≥3 and remission by HBI <5. We compared the effectiveness of ustekinumab and vedolizumab on an intention to treat basis.

Abstract P162 Table 1

		Vedolizumab n = 85	Ustekinumab n = 45	Fisher's Exact Test (P value)
Response	2 months	35%	49%	0.138
	4 months	39%	56%	0.095
	6 months	39%	49%	0.351
	12 months	44%	53%	0.356
Remission	2 months	16%	36%	**0.017***
	4 months	21%	40%	**0.038***
	6 months	39%	49%	0.351
	12 months	44%	53%	0.356
Steroid-Free Remission	2 months	12%	29%	**0.028***
	4 months	20%	38%	**0.036***
	6 months	15%	38%	**0.008***
	12 months	25%	42%	**0.047***

Results After exclusion of patients without evaluable data, 85 patients commencing vedolizumab and 45 commencing ustekinumab therapy were included. Baseline characteristics (age, disease location, behaviour, smoking status and baseline FC) were comparable in both cohorts. 29 (34%) of patients receiving vedolizumab and 6 (13%) receiving ustekinumab stopped treatment within 12 months due to adverse events or lack of response. Clinical response rates were similar between treatments. Clinical remission rates, however, were greater at 2 and 4 months in patients treated with ustekinumab. Steroid-free remission rates were greater in patients treated with ustekinumab at all time points (see table 1 - Response and remission rates for vedolizumab and ustekinumab).

Significance set at p = 0.05. *indicates statistical significance reached

Conclusions Vedolizumab and ustekinumab effectiveness was broadly comparable to that seen in their landmark clinical trials. A higher proportion of patients receiving vedolizumab needed to change treatment within 12 months. Higher rates of steroid-free remission were seen in patients treated with ustekinumab at all time points.

P163 EMERGENCE/EXACERBATION OF INFLAMMATORY BOWEL DISEASE IN PATIENTS RECEIVING SECUKINUMAB FOR ANKYLOSING SPONDYLITIS -CASE SERIES

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Secukinumab is an IgG monoclonal antibody against interleukin-17A and is licenced for the treatment of ankylosing spondylitis (AS), psoriatic arthritis and plaque psoriasis. IL-17 is one of the pro-inflammatory cytokines involved in the pathogenesis of above inflammatory conditions and blocking it has proved beneficial in their management.

National guidance advises for Secukinumab to be used with caution due to a risk of exacerbation or development of new onset IBD. However, safety results of