value of ≥200 μg/g was used to indicate active disease. The cohort was subdivided into 4 groups: active and inactive Crohn's disease (CD), active and inactive Ulcerative colitis (UC).

Results 1226 patients (771 CD, F = 65%, 455 UC, F = 35%) with matched FC and FBC data were analysed. The median age was 44y (IQR 31–57), median disease duration 102 months (IQR 31–207). Overall, 314/1226 patients (25.6%) were anaemic, 185/314 (58.9%) of which were female. Anaemia was observed more frequently in patients with active as opposed to inactive CD (110/328 [33.5%] vs 65/443 [14.7%], $p < 0.0001$). This pattern was also seen in patients with UC (129/293 [44%] vs 23/162 [14.2%], $p < 0.0001$). The prevalence of anaemia in active UC was greater than in active CD ($p = 0.014$); however, this could be explained by the higher median FC in the UC cohort (900 vs. 618, $p < 0.0001$). There was no statistically significant difference in age or Montreal location (L1 + L3 vs L2, $p = 0.16$) between the groups. ROC analysis of FC as a predictor of anaemia showed an AUC of 0.69 with a sensitivity and specificity of 0.73 and 0.57 respectively at a cut off of 200 μg/g.

Conclusion In this cohort over 25% of patients with IBD were anaemic. There is a clear correlation between disease activity and anaemia in both CD and UC, but this is unrelated to disease distribution in CD. Anaemia in asymptomatic patients should alert clinicians to the possibility of subclinical active mucosal inflammation. These data and the ROC analysis provide further support for optimising disease treatment in IBD, targeting a FC level of < 200 μg/g.

Disclosure of Interest None Declared.

PWE-095 A 10 YEAR REVIEW OF THE DEATH RATE AND CAUSE OF DEATH WITHIN A DISTRICT GENERAL COHORT OF INFLAMMATORY BOWEL DISEASE PATIENTS

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Introduction Whilst there is a growing body of data supporting the increased risk of colo-rectal cancer in patients with inflammatory bowel disease, little has been written about the overall mortality and cause of death seen in patients with this condition.

Objective To assess the death rate and cause of death in our cohort of IBD patients.

Methods A database of all known local IBD patients was established after retrospectively reviewing 37,000 colonoscopy and flexible sigmoidoscopy reports performed at the Luton & Dunstable University Hospital between 2001 and 2011. Histological assessment was then used to cross correlate all patients diagnosed with colitis. The hospital coding officer analysed the database and provided details on all deaths within that cohort over the time period and the cause of death as listed by the hospital record system.

Results In total 2640 patients with IBD were identified and 186 (7%) of these died between 2001 and 2011. The average age of death was 80 years. The cause of death ranged considerably (see chart).

Abstract PWE-095 Table

Cause of Death	Number	Percentage (%)
Pneumonia	32	17.2
Sepsis	28	15.1
Cardiac	22	11.8
Non-GI Cancer	11	5.9
Crohn's disease	11	5.9
Ulcerative colitis	9	4.8
GI Cancer	9	4.8
Stroke	8	4.3
GI Haemorrhage	6	3.2

Conclusion The Office for National Statistics stated that in 2010 the average lifespan in England and Wales, for men and women, was 85 and 89 years, respectively. The life expectancy in Luton is slightly lower than the national average by approximately 2 years (Annual Public Health Report 2012–2013). The average age of death in our IBD cohort appears substantially lower than expected, with just 41 of the 186 (22%) being related to gastrointestinal causes. Infection (sepsis and pneumonia) appeared to be the single most common cause of mortality 60/186 (37.5%), raising questions about an iatrogenic contribution.

Disclosure of Interest None Declared.

PWE-096 IS THERE AN ASSOCIATION BETWEEN PARKINSON'S DISEASE AND INFLAMMATORY BOWEL DISEASE ?

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Introduction The BRAAK theory of Parkinson's disease believes that the aetiology may all start in the bowel with a "slow virus" entering the central nervous system after passing through the intestinal mucosa {Hawkes C.H., 2007}. There is already work confirming an increased frequency of H. pylori infections (requiring treatment) in the 5 years prior to Parkinson's disease being diagnosed {Nielsen H.H. 2012}. Recently a gene associated with the inherited form of Parkinson's disease (leucine-rich repeat kinase 2 - LRRK2), has been shown to regulate the transcription factor NFAT1 (nuclear factor of activated T cells 1), which in turn appears to regulate cells in the immune system, including macrophages, dendritic cells and T cells. Higher quantities of NFAT1 activity are seen in the colonic mucosa of Crohn's patients, where the total quantity directly correlates to the severity of the disease {Liu Z., 2011}. Objective: With these theories in mind, we aimed to assess whether a higher than expected association between inflammatory bowel disease (IBD) and Parkinson's disease (PD) could be found.

Methods A cross correlation analysis was performed using the IBD and PD databases at the Luton & Dunstable University Hospital. A retrospective analysis was also performed using medical notes and the internal electronic results system to assess the disease severity of these two conditions.

Results The prevalence of IBD and PD within the UK population is said to be 225/100,000 (UC 150/100,000 + CrD 75/100,000) and 140/100,000, respectively. The L&D catchment area covers 330,000 and so one would have expected approximately 742 IBD and 462 PD patients, respectively. The databases had 2783 IBD patients (median age = 51) and 350 PD patients (median age = 79) listed. Probability analysis predicted that we would find just 1 patient with concomitant PK and IBD, however, we found 6 subjects with these conditions concomitantly. This translates into 0.2% of IBD patients having PD and 1.72% of PD patients having IBD. Mild-to-moderate PD was noted in 3 patients, and all 3 had mild-to-moderate IBD. Three of the PD patients were scored as having moderate-to-severe disease, and 2 of these also had moderate-to-severe IBD.

Conclusion The proportion of PD patients having concomitant IBD is considerably higher than one would have expected by chance. This raises possible issues around genetic association, but also lends some credence to theories that PD may owe its origins to the bowel and infective translocation across bowel mucosa. Those patients with more significant IBD also appeared to have more severe PD.

Disclosure of Interest None Declared.

PWE-097 THE EFFECT OF ETHNICITY ON THE PREVALENCE OF INFLAMMATORY BOWEL DISEASE WITH THE LUTON AND DUNSTABLE CATCHMENT REGION, IN UK

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Introduction The incidence and prevalence of inflammatory bowel disease in Asia, is generally lower than what we see in the West, however, in recent years there has been a significant increase. Currently there is little information available regarding the impact of ethnicity on inflammatory bowel disease (IBD) prevalence within the UK. One study has suggested that young Asians born in Britain are at a significantly higher risk of developing IBD than the indigenous European population.
Objectives To assess the prevalence and disease distribution amongst our ethnically diverse local population.
Methods Data from the Office for National Statistics 2010 was used to establish the general make up of the local population. The regional IBD excel database was analysed for differences in the ethnic diversity seen in our IBD cohort as compared to that predicted within the local population. In addition, differences in disease type were also assessed for between the different IBD ethnic groups, using the Mann Whitney unpaired t-test.
Results The local population is made up by White 68% Asian 19% (Indians 4.4% Pakistanis 10.6% and Bangladeshis 4%), Black 7%, Mixed 3% and Chinese + others 3%. The IBD database contains 2755 patients, but 270 did not want to disclose their ethnicity. Of the remaining 2485, 2059 (83%) were White, 303 (12%) were Asian, (Indians 111, Pakistanis 133 and Bangladeshis 35), 92 (4.5%) were Black, 28 (1%) were Mixed and 3 were Chinese. IBD was less common within the Asian cohort, but there was a statistical differences seen in the type of IBD suffered by the local Caucasian and Asian populations ($p = 0.0141$). Asian patients had proportionally more UC and less Crohn's, with the exception of the Bangladeshis who had a higher (but not statistically significant) prevalence of Crohn's disease.
Conclusion IBD is less common in the Asian community. Proportionally UC is more frequently experienced than Crohn's within the Asian population. This may be related to the known increased genetic predisposition of Southern Asians (Indians, Pakistanis and Bangladeshis) to ulcerative colitis.
Disclosure of Interest None Declared.

PWE-098 MECHANISMS UNDERLYING THE DEVELOPMENT OF CHRONIC INFLAMMATION IN INFLAMMATORY BOWEL DISEASE: DEFINING THE ROLE OF THE RAGE PATHWAY USING COMPUTATIONAL AND BIOLOGICAL ANALYSIS STRATEGIES

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Introduction Inflammatory bowel disease (IBD) is a chronic inflammatory disease with an estimated annual cost to the NHS of £720 million. Patients typically present with established disease and this makes it difficult to determine the underlying aetiology: knowledge that would aid early diagnosis and treatment.
Methods To better define factors underlying the development of IBD that might be used as diagnostic aids for treatment/prevention of IBD we have analysed the early immune response in mice that will develop colitis using a validated infection model of colitis. Microarray analyses of colon tissue were conducted using the Puma and Tigre packages for Bioconductor to determine gene expression and investigate the transcription factor pathways involved.
Results Microarray analysis identified an early and rapid increase in expression of the receptor for advanced glycation end-products (RAGE) in colitic prone (susceptible) mice. In contrast, mice that clear the infection (resistant mice) had no increase in RAGE. In addition, the transcription factor analysis revealed a downregulation of colitic protective factors in the RAGE signalling pathway. Immunohistochemistry data showed high RAGE expression in the gut epithelium prior to the onset of colitis. Previous work from our group has shown that epithelial cells promote dendritic cell recruitment associated with resistance and clearance of parasite infection. In the colitis model, infection also induced the rapid recruitment of macrophages and dendritic cells (DC) into the gut and lymph nodes in resistant mice but not susceptible mice. By day 31 post-infection, resistant mice had resolved the infection and inflammation whereas susceptible mice had significantly higher immune cell accumulation and colitis. Current work is addressing the expression of RAGE blocking ligands and the regulation of RAGE in the guts of resistant and susceptible mice.
Conclusion RAGE has been associated with chronic IBD in patients however our data implicates RAGE in the development and propagation of IBD. We propose that the RAGE pathway is an early indicator of IBD and may be useful therapeutically and in determining efficacy of IBD therapy.
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PWE-099 FAECAL CALPROTECTIN: HELP OR HINDERANCE IN EVALUATING PATIENTS WITH LOWER GI SYMPTOMS

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Introduction Several studies demonstrate the potential of faecal calprotectin measurement in distinguishing inflammatory bowel disease (IBD) from irritable bowel syndrome (IBS). It is unclear, however, to what extent such measurements alter patient investigation and management over standard history taking, examination and routine blood tests.
Methods We reviewed all faecal calprotectin results from samples submitted by adults not previously known to have IBD between February 2010 and April 2012. Using the health board's Clinical Workstation relevant outpatient letters, results of subsequent investigations and clinical outcomes were reviewed. A calprotectin value of > 60ug/g was considered elevated.
Results Cinical data was missing for 13 of the 266 patients. Of 155 with a **normal result** management was unaltered in 126, of whom 50 were referred for lower GI endoscopy before their result was known. In another 5 patients IBD was later found despite a normal result. A normal result may have obviated the need for colonoscopy or capsule endoscopy in 17, and otherwise altered management in 12 other patients. Outcomes for the 98 with an **elevated result** are summarised in the table. Patient management was unaltered in 60. There were 33 for whom an elevated result prompted colonoscopy and/or capsule endoscopy, and results were normal in 27. Five others still await colonoscopy or capsule.
Conclusion This study casts doubt on the value of faecal calprotectin in the routine evaluation of patients presenting with lower GI symptoms. Invasive investigations prompted by elevated results proved normal much more often than abnormal. The assay seems

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