remains unclear as does its role in treatment. Although the efficacy of vitamin D as an immunomodulator remains to be established, given current evidence it appears reasonable to screen and treat vitamin D deficiency in patients with IBD.

**Methods** Two hundred consecutive patients with vitamin D deficiency were identified from IBD clinics at Leicester General Hospital who had been seen during a 12 month period. A postal questionnaire was sent to these patients. It was anonymous and requested whether: Respondents believed they had been told of their vitamin D deficiency. General practitioners had prescribed vitamin D supplements. Vitamin D supplements had been bought at local pharmacies. Brand of vitamin D supplements taken. Compliance with treatment. Symptoms had improved with treatment. They had enough sunlight exposure.

**Results** Ninety eight of the 200 patients responded to the questionnaire, a response rate of 49%. Sixty five were English and 35 Asian. The response rate was 48% in Asians and 50% in English which is not significantly different. Seventy (71%) of the 98 patients, recalled being told they were vitamin D deficient. Thirty nine patients (40%) believed they had sufficient exposure to sunlight. 54 (71%) had been prescribed this medication by general practitioners and 22 (29%) had bought vitamin D supplements over the counter at local pharmacies or herbal stores. General practitioners predominantly prescribed Adcal D3 tablets. Sixty four (84%) patients said they were adherent to vitamin D treatment. Symptoms improved significantly in 29 (38%) patients. However, this figure rose to 45% when only those 64 patients who were compliant with therapy were considered.

**Conclusion** Vitamin D plays a significant role in intracellular functions which extends beyond its effects on bone metabolism. It is an important regulator of the immune system which may have implications for the development, severity and management of immune related disorders such as IBD. In summary the relationship between the vitamin D axis and IBD is multifaceted. It should comprise maintenance of musculoskeletal health and control of disease through immunomodulation and modification of associated malignancy.

**Disclosure of Interest** None Declared.

**REFERENCE**


**PWE-105**

**DO GASTROENTEROLOGISTS MONITOR THEIR PATIENTS TAKING 5-AMINOSALICYLATES FOLLOWING INITIATION OF TREATMENT?**

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**Introduction** 5-Amino salicylate (5-ASA) medications may rarely be associated with a significant decline in renal function and interstitial nephritis. The British Society of Gastroenterology guidelines advise regular renal function monitoring for patients (pts) taking these drugs. This study assessed whether clinicians were following best practise guidelines.

**Methods** Using longitudinal community and regional pathology databases for the East Kent population (720 k), our renal unit regularly screens a total population of 300,000 for evidence of renal disease. The data extracted is analysed using the SEIK (System for Early Identification of Kidney disease), an automated computerised system to identify pts requiring intervention for kidney disease. As part of this process, all patients taking 5-ASA medication were identified. The pathology database was studied to identify the pts on treatment who had had renal function tests and could differentiate from results initiated in primary and secondary care. Data analysis can be performed over many years of treatment.

**Results** 800 adult pts (M: 341, F 459) identified taking 5-ASA therapy (median duration 1.5 years (range 1–24 yr); mean (+/- SD) age 52.7 +/- 16.2 yr (range 18.2–94.4). The mean estimated Glomerular Filtration Rate (eGFR) on commencing 5-ASA therapy was 82 ml/min (range 25 - > 90). Pts with an eGFR < 60 were regarded as having chronic kidney disease (stage 3–5). 612 pts received 5-ASA’s for 3 months or more (median 5.2; range 1 – 24 yr) and these were included in the final analysis.

293 (48%) pts had no renal function checkes whilst on treatment. 79 (12%) pts had renal function tests less than once every 4 years and 36 pts once every 2 – 4 yrs. 204 pts had renal function measurements in 50% or more of years of treatment, of whom 116 were checked every year. 72 pts with a baseline eGFR < 60 ml/min were treated with 5-ASA’s for 3 or more months. Of these, 8 had no renal function checkes. The eGFR fell in 24 pts and in 8 by > 2ml/min/yr.

**Conclusion** The majority of pts receiving 5-ASA compounds do not have regular renal function monitoring. Some are started on treatment with abnormal results at baseline and some with identified kidney disease continued on their 5-ASA’s. Gastroenterologists are failing to follow best practise guidelines.

**Disclosure of Interest** None Declared.

**REFERENCE**


**PWE-106**

**ASSOCIATION STUDY OF IL23R AND ATG16L1 VARIANTS IN IBD MOROCCAN PATIENTS**

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**Introduction** IBD (Crohn’s disease and Ulcerative Colitis) is chronic and multifactorial disease of the gastrointestinal tract. Although several studies have tried to explore these diseases, their pathogenesis is still unclear. Recently, CD has been associated with the variants in interleukin 23 receptor (IL23R) and autophagy-related 16-like 1 (ATG16L1) genes. The aim of our study was to assess the frequency of ATG16L1 (T300A) and IL23R (L310P) variants in Moroccan IBD patients and to determine a possible effect of these variants on Disease’s phenotype and clinical course.

**Methods** we genotyped a group of 96 Moroccan IBD patients and 114 unrelated volunteers for ATG16L1 (T300A) and IL23R (L310P) variants.

**Results** Our results showed no significantly increased risk of Crohn’s disease among individuals carrying the GG genotype or the G allele for the ATG16L1 T300A polymorphism, in contrast to the IL23R L310P polymorphism which confers a protective effect. We also noticed the presence of a positive correlation between Crohn’s disease Type and ATG16L1 polymorphism. For UC, the carriage of the mutated allele in the ATG16L1 gene confers a protective effect.

**Conclusion** Our results showed a limited role of ATG16L1 and IL23R variants in the Moroccan population.

**Disclosure of Interest** None Declared.