steroids and 29 received immunomodulatory therapy (27 azathioprine, 3 methotrexate, 1 cyclosporin, 2 6MP, 10 infliximab, 6 adalimumab). Seven deficient patients (4 CD, 3 UC) required a colectomy and the mean 25-OHD level in this group was 10.7 (range 4.0–20.1 ng/ml). Mean time to surgery was 3.6 years (range 0–8 years). Of the CD patients 1 had a subtotal colectomy, 3 had hemicolectomy and of the UC patients 2 had subtotal colectomy and 1 had total colectomy.

Conclusion There was a high prevalence of Vitamin D deficiency although assessment was suboptimal and probably reflective of a wider experience. Patients with vitamin D deficiency appeared to have a more aggressive disease course. The role of vitamin D in IBD is a science in evolution underpinning exciting implications for research. Meanwhile vitamin D deficiency is under-recognised and consequently undertreated with likely implications for adequate disease control in this potentially vulnerable group.

Disclosure of Interest None Declared

PTU-062 VALUE OF MRI IN PREDICTING SEVERE OUTCOME IN SMALL BOWEL CROHN'S DISEASE

doi:10.1136/gutjnl-2013-304907.154

^{1,*}C Rutter, ¹S Bhatt, ²I Sequeiros, ²P Burn, ¹P D Thomas. ¹Gastroenterology; ²Radiology, Taunton and Somerset NHS Foundation Trust, Taunton, UK

Introduction The optimal approach to managing terminal ileal (TI) Crohn's disease remains to be defined. It is unclear at what stage surgery or biological therapy should be offered and current clinical and biochemical parameters offer poor prediction of disease course. Small bowel MRI scanning (SBMRI) has been correlated with endoscopic and histological disease severity in Crohn's disease and may offer better global assessment of the extent and severity of disease. We aimed to determine which MRI features might predict the need for surgery or biological therapy.

Methods 48 sequential patients with Crohn's disease who underwent SBMRI in a 20 month period to Feb 2011 were identified from a radiological database. 8 patients were excluded due to predominant colonic disease. All remaining 40 patients had confirmed isolated TI disease. Standard management with escalation of therapy via immunomodulors, biological agents and surgery based on clinical follow up was applied. Patients were followed for a minimum of 2 years after the initial MRI. MRI scanning was performed using oral fluid load, IV buscopan, T1/2 axial, coronal and dynamic post contrast sequences. The images were reviewed by a radiologist blinded to outcome of cases and key abnormal features recorded (mesenteric abnormalities, wall thickness > 6 mm, disease extent > 15 cm or proximal dilatation > 25 mm). Patients were then divided into 2 groups, those requiring biological therapy or surgery (severe) and those managed with 5ASA or immunomodulators alone (non severe).

Results The characteristic of the two groups is shown in the table. Means given unless stated.

Abstract PTU-062 Table

	n	Age			Years post diagnosis	Previous surgery (%)	Immunomodulator use (%)
A Non severe	20	36	77	19	9.9	45	45
B Severe	20	42	120	30	9.8	45	75

6/20 patients in the non-severe group (A) had two or more adverse radiological features compared with 12/20 in the severe (B) group (p = 0.06). However, only 3/20 patients had lumen > 25 mm or extent > 15 cm in A compared with 15/20 in B (p < 0.001). Wall thickness and mesenteric involvement were not associated with a

severe outcome. Disease extent and proximal luminal diameter were significantly associated with surgery (p = 0.02 and p = 0.0001). 85% of patients who eventually required surgery had either proximal lumen > 25 mm or disease extent > 15 cm.

Conclusion Two or more adverse radiological MRI features are associated with with the need for surgery or biological therapy. Small bowel dilatation > 25 mm proximal to the disease segment and disease extent > 15 cm are particularly associated with the need for surgery. These MRI findings may be helpful in deciding appropriate longer term strategies for managing these patients. **Disclosure of Interest** None Declared

PTU-063 ASSESSMENT OF SLEEP IMPAIRMENT IN PATIENTS WITH CROHN'S DISEASE: RESULTS FROM THE USTEKINUMAB CERTIFI STUDY

doi:10.1136/gutjnl-2013-304907.155

^{1,*}C Gasink, ¹D Chan, ¹L-L Gao, ²B Schenkel, ³C Han. ¹Janssen R&D, LLC, ²Janssen Scientific Affairs, LLC, Spring House, ³Janssen Pharmaceutical Services, Malvern, United States

Introduction To describe the extent of sleep impairment reported in CERTIFI (Ph2 evaluating UST in inducing & maintaining clinical response & remisison)using Jenkins Sleep Evaluation Questionnaire (JSEQ) & establish a clinically meaningful improvement threshold for JSEQ.

Methods Pts with moderate-to-severe CD(CDAI \geq 220 & \leq 450) who had previously failed or were intolerant to \geq 1 anti-TNF were randomised to PBO/UST induction at wk0. Primary endpt was clinical response (\geq 100-pt reduction in CDAI from BL) at wk6. Sleep impairment assessed using JSEQ (total score 0–20; higher scores indicate greater sleep impairment) at BL&wk6. Relationships between BL sleep impairment, clinical disease activity (CDAI), & HRQoL impact(IBDQ) evaluated using Pearson correlation. Clinically meaningful improvement threshold of JSEQ was established with the anchor-based [clinical response by reduction in CDAI of \geq 70-pt or clinically meaningful improvement (\geq 16-pt) in IBDQ] & distribution-based (change by one-half of the standard deviation [SD] of BL JSEQ score) methods. Prop of pts who achieved clinically meaningful improvements in JSEQ at wk6 was determined & compared.

Results At BL, both grps(n = 526) experienced similar degree of moderate sleep impairment, with JSEQ scores (mean±SD) of 11.0 ± 4.30(PBO)&11.0 ± 4.59(UST),resp. About 60% were "waking up feeling tired and worn out"; about 30-35% of pts had trouble falling asleep, staying asleep, & woke up several times during the night for 15-30 days in the previous month. At BL, JSEQ was correlated with CDAI (r = 0.19, p < 0.0001)&IBDQ(r = -0.39, p < 0.0001). Using the anchor-based method, pts who achieved vs didn't achieve clinically meaningful improvements in CDAI or IBDQ at wk6 vs BL, reported improvements(mean±SD)from BL in JSEQ of -2.52 ± 4.43 vs $-0.58 \pm 3.51 \otimes -2.68 \pm 4.16$ vs -0.16 ± 3.45 , resp. Using distribution-based method, the JSEQ clinically meaningful improvement threshold was 2.25(SDof BL JSEQ = 4.50). 2 potential thresholds for clinically meaningful improvement in JSEQ (eg. reduction of > 2 or > 3 points from BL JSEQ score at wk6) were derived. More pts who received UST induction achieved both thresholds at wk6(Table).

Abstract PTU-063 Table

Treatment Group	Improvement in Total JSEQ > 2 at Wk6	Improvement in Total JSEQ > 3 at Wk6
PB0 (n = 130)	26.2%	18.5%
UST combined (n = 390)	34.6% (p = 0.084)	27.2% (p = 0.048)
Absolute difference between UST and PBO	+8.5% for UST	+8.7% for UST

Conclusion Pts experience significant sleep problems as measured by JSEQ; magnitude of impairment correlates with disease activity. Both anchor- &distribution-based methods derive similar thresholds representative of clinically meaningful improvements in JSEQ. UST induction resulted in a greater proportion of pts achieving clinically meaningful improvements in sleep impairments.

Disclosure of Interest C. Gasink Employee of: Janssen R&D, LLC, D. Chan Employee of: Janssen R&D, LLC, L.-L. Gao Employee of: Janssen R&D, LLC, B. Schenkel Employee of: Janssen Scientific Affairs, LLC, C. Han Employee of: 3. Janssen Pharmaceutical Services

PTU-064 IDENTIFICATION OF SYNE1 AND FOXE1 HYPERMETHYLATION TO IMPROVE DIAGNOSIS AND MANAGEMENT OF COLERECTAL NEOPLASIA IN INFLAMMATORY BOWEL DISEASE

doi:10.1136/gutjnl-2013-304907.156

^{1,2,*}C Papadia, ³J Louwagie, ⁴P Del Rio, ⁵M Novelli, ²G de'Angelis, ⁶W Atkin, ⁷C Bordi, ⁸P Bassett, ¹A Forbes. ¹Gastroenterology & Nuitrition, University College London, London, UK; ²Gastroenterology, Parma University Hospital, Parma, Italy; ³Molecular Diagnostics,, Novartis, Basel, Switzerland; ⁴Surgery, Parma University Hospital, Parma, Italy; ⁵Pathology, University College London; ⁶Cancer Prevention Unit, Imperial College London, London, UK; ⁷Pathology, Parma University Hospital, Parma, Italy; ⁸Statsconsultancy, University College London, UK

Introduction Colitis-associated colorectal cancer (CAC) affects individuals with inflammatory bowel disease (IBD) more often and younger than cancer in the general population. Colonoscopy provides the surveillance gold standard. Changes to surveillance intervals have been made given data demonstrating that endoscopic appearance is an important predictor of future dysplasia or cancer, but adjuvant, non-invasive clinical tools are still warranted to improve surveillance outcomes and to assist in management and interpretation of dysplasia. Methylation markers may be able to do this. Material and methods

Methods using reexpression profiles of colon cancer cell lines, candidate genes were identified; promising markers were tested on tissue using the Base5 methylation-profiling platform. Promoter sequences were linked with gene expression to identify epigenetically silenced genes. Marker candidates were screened using methylation specific PCR assays to assess the methylation status of 2 gene promoters (FOXE1, SYNE1) in biopsies from 93 longstanding IBD patients and 30 healthy controls. Samples included colitis-associated colorectal adenocarcinomas (n = 25); IBD-associated dysplastic lesions (n = 29); adenomas arising on a background of UC (n = 8); samples from IBD patients with no neoplasia (n = 31) and healthy controls (n = 30).

Results The presence of the 2 genes significantly varied between the groups. Both were increasing likely with increased disease severity. Neither occurred in controls, whilst 60% of CAC patients had FOXE1, and 80% of CAC patients had SYNE1.

Conclusion FOXE1- SYNE1 methylation markers panel demonstrated significantly increased expression in neoplastic tissue. Syne1 was highly represented in CAC. Methylation of these promoter genes might be considered a potentially useful pathology marker of neoplasia in longstanding inflammatory bowel disease.

Disclosure of Interest None Declared

PTU-065 INFLUENZA VACCINATION UPTAKE IN INFLAMMATORY BOWEL DISEASE- IS THERE ROOM TO IMPROVE?

doi:10.1136/gutjnl-2013-304907.157

^{1.} D Cheema, 'R Muhammed. '*Department of Paediatric Gastroenterology, Birmingham Children's Hospital, Birmingham, UK*

Introduction The aim of our study is to assess the seasonal influenza vaccination uptake in patients with inflammatory bowel disease (IBD)

Methods We have conducted a telephonic survey of our IBD patients in February 2012 to assess the influenza vaccination uptake for winter 2011–2012.

Results 140 children had responded to this survey (61.6% of our IBD patients). 84 children had Crohn's disease, 35 had Ulcerative colitis and 21 had IBD unclassified. Majority of these children (90/140) were on immunosuppressive treatments. 61 children (44%) had received seasonal influenza vaccination in that winter. 21 of them received in October, 20 in November, 13 in December and 3 in January. Out of the 79 children who have not received the influenza vaccine, 42 were not aware of the need for vaccination and did not have the influenza vaccine in the previous winters as well. 10 children were aware of the need for the influenza vaccine; however they opted not to receive the vaccine. 14 children intended to receive the vaccine, however this was deferred due to various reasons like intercurrent illness, family bereavement and difficulties experienced the General Practice surgery. Only one IBD patient needed hospitalisation in 2011 and 2012 with Influenza infection, however this was in July before the vaccination had started.

Conclusion Department of Health advises influenza vaccination for immunosuppressed individuals and also for children with medical conditions, who may need treatment with steroids for more than a month. European Crohn's and Colitis Organisation (ECCO) recommend influenza vaccination for IBD patients on immunomodulators. Experience from Philadelphia, Boston and Poland show that good, but variable, antibody response occurs after influenza vaccination in children and better protection occurs against type A strains. Side effects, both local and systemic, are generally mild. Experience from Australia and Germany show that the seasonal flu vaccination uptake in IBD patients are generally low, 10% and 16% respectively. We would like to hear from other centres about their experience of influenza vaccination uptake in IBD patients. Further efforts need to be done to increase the awareness of influenza vaccination in patients with IBD

Influenza vaccination uptake in our IBD patients are better than reported from other centres, however further work needs to be done both locally and nationally to improve the influenza vaccination uptake.

Disclosure of Interest None Declared

PTU-066 NEW INSIGHT INTO THE MUCOSAL PROFILE OF EICOSANOID MEDIATORS IN ULCERATIVE COLITIS

doi:10.1136/gutjnl-2013-304907.158

^{1,*}D S Pearl, ^{2,3}M Masoodi, ³M Eiden, ⁴J K Shute, ⁵P C Calder, ¹T M Trebble. ¹Department of Gastroenterology, Portsmouth Hospitals NHS Trust, Portsmouth, UK; ²Nestle Institute of Health Sciences, Lausanne, Switzerland; ³Elsie Widdowson Laboratory, Medical Research Council, Cambridge; ⁴Institute of Biomedicine and Biomolecular Sciences, University of Portsmouth, Portsmouth; ⁵Human Development and Health Academic Unit, Faculty of Medicine, University of Southampton, Southampton, UK

Introduction Ulcerative colitis (UC) is a relapsing remitting disorder of the colon with a recognised role for certain eicosanoid mediators derived from polyunsaturated lipid substrates. However, a detailed characterisation of the eicosanoids involved in UC is currently lacking. Using a comprehensive lipidomics approach, we profiled eicosanoids that could exhibit both pro- and anti-inflammatory function in inflamed and non-inflamed colonic mucosal biopsies from UC patients.

Methods Biopsies were taken from inflamed and nearby noninflamed colonic mucosa (69 patients, 54 with paired inflamed and non-inflamed mucosa) from patients with symptomatic relapses. Inflammation was scored endoscopically and histologically. Mucosal lipid mediators were determined by LC-MS/MS lipidomics analysis. Univariate and multivariate statistical analyses were used to investigate the association of lipid mediators with the disease state