coeliac disease.[1] Duodenal biopsies (D2) should be performed only after a positive serological test or a negative test with a high clinical suspicion by gastroenterologist. Previous studies have demonstrated that random D2 biopsies are not cost effective.[2] We aimed to analyse whether current practice is now in keeping with guidelines.

Methods This was a retrospective review of the electronic records of 422 patients who had had duodenal (D2) biopsies in 1 year. Furthermore, we collated the annual number of duodenal biopsies from 2009 to 2018 to determine if the new guidelines had made an impact.

Results The indications for endoscopy were iron deficiency anaemia (IDA) (68%), low ferritin (3%), weight loss, loose stool and non-specific gastrointestinal symptoms (29%). Only 1 patient with a negative tTG had a positive biopsy.

Prior to D2 biopsy, 192(45%) patients had no previous TTG or D2 biopsy. Of these, 9 had a positive biopsy and were subsequently found to be tTG positive. 203 (48%) patients had biopsies despite a negative tTG. 31 (7%) had previous normal D2 biopsies (12 also negative TTG).

The excess cost incurred for processing biopsies after a negative TTG was £12,180. £9882 would have been saved by carrying a TTG test in subjects having a negative biopsy.

The number of biopsies over 10 years remained largely unchanged with a low of 412 in 2012 and a high of 522 in 2018 with a median of 437 biopsies per year.

Conclusion A significant proportion of duodenal biopsies are still done in patients with a negative TTG and/or previous normal D2 biopsy. Following BSG guidelines, would have saved over £20,000 in 1 year. We suggest an IT based solution where an alert is triggered to check tTG at the same time as a referral is made for endoscopy. Furthermore, D2 biopsy samples can be delayed until a tTG is checked if not done prior to endoscopy. Finally, a point of care tTG could be utilised in GP surgeries or endoscopy units to minimise any delay. These measures will be put forward to the CCG.

REFERENCES

1. Ludvigsson JF, Bai JC, Biagi F, *et al*.Diagnosis and management of adult coeliac disease: guidelines from the British Society of Gastroenterology.*Gut* 2014;**63**:1210–1228.
2. Herrod PJJ, Lund JN. Random duodenal biopsy to exclude coeliac disease as a cause of anaemia is not cost-effective and should be replaced with universally performed pre-endoscopy serology in patients on a suspected cancer pathway. *Tech Coloproctol*. 2018;**22**(2):121–124.

Nutrition

P273 CHEMOGENETIC ANALYSIS OF HOW RECEPTORS FOR SHORT CHAIN FATTY ACIDS REGULATE THE GUT-BRAIN AXIS

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Introduction Short chain fatty acids are produced mainly by the gut microbiota. They mediate a variety of biological effects by acting on a two of G protein-coupled receptors. These receptors are expressed by various cell types, including in the gut. The exact contribution of free fatty acid 2 receptor (FFA2) in regulating gut physiology is unclear. Bolognini et al[1], recently employed a novel FFA2-Designer Receptor Exclusively Activated by Designer Drugs (DREADD) to study the physiological role of FFA2. Now we describe and further explore the physiological roles of FFA2 with a novel agonist, 4-methoxy-3-methyl-benzoic acid (MOMBA) for the FFA2-DREADD variant following transgenic expression in mice.

Methods Following an extensive screening of more than 1200 small molecules, MOMBA was identified as a potential agonist for the hFFAR2-DREADD receptor. **(1)** The selectivity of MOMBA was assesses with β-arrestin-2 recruitment assay in HEK293 cells expressing hFFA2-DREADD and hFFA2-eYFP. The effect of FFA2 activation on the release of enteroendocrine hormones (GLP-1 and PYY) was assessed on **(2)** isolated crypts and **(3)** intact colonic segments. Isolated crypts and intact colon segments were challenged with different test compound. Supernatants were subsequently collected and GLP-1 and PYY concentration was measured by ELISA. **(4)** Furthermore, role of FFA2 in sensory signalling was investigated by measuring intracellular calcium [$_{i}Ca^{2+}$] in isolated nodose ganglion (NG) and dorsal root ganglion (DRG).

Results **(1)** MOMBA is selective for hFFA2-DREAD **(2)** MOMBA (1mM-0.001mM) induces a FFA2 specific concentration dependent increase in GLP-1 secretion in colonic crypts. **(3)** Intraluminal infusion of MOMBA also resulted in a FFA2 mediated increase in GLP-1 and PYY secretion from intact colon. Furthermore, **(4)** MOMBA induced a G_q mediated increase in [$_{i}Ca^{2+}$] in cells isolated from DREADD mice. Conversely, C3 induced a G_i mediated increase in these cells.

Conclusion MOMBA specifically activates hFFA2-DREADD, hence providing a novel tool ligand to further study the physiological and pathophysiological roles of FFA2 within the gut, as well as other cell types that express this receptor.

P274 EFFECTS OF A BOWEL PREPARATION DIET ON THE GUT MICROBIOME

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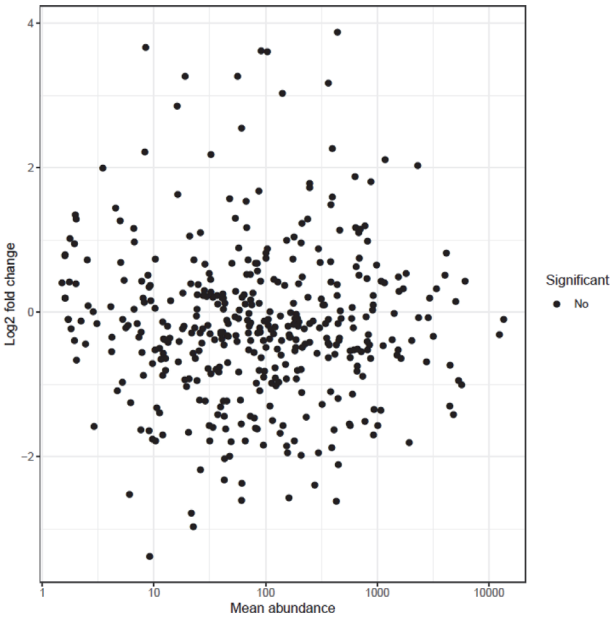
Introduction Prior to colonoscopy, patients undergo a bowel preparation regimen to clear bowel contents and optimize view of the bowel wall. In the UK, the bowel preparation regimen may involve patients undertaking either a 3-day low-fibre/low-residue diet (LRD) or a 1 day clear liquid diet (CLD) before their procedure. The day before their procedure, all patients are required to take a laxative. A low-fibre diet has been associated with reduced gut microbiome richness and diversity. A low-fibre diet has also been associated with a *Bacteroides* enterotype and an enrichment of *Alistipes* and *Parabacteroides* genera. A transition from a high-fibre to a low-fibre diet has been shown to result in changes in the gut microbiome within 24 hours. We present the results of an

evaluation of the effects of a 3-day LRD on the gut microbiome.

Methods Faecal samples were obtained from 5 healthy subjects. Samples were collected while participants were on their regular diets and immediately after undertaking a 3-day LRD. DNA was extracted from samples using PSP spin stool DNA plus kit (Stratec). The concentration of extracted DNA was determined using fluorometry. PCR was carried out on extracted DNA samples and gel electrophoresis was performed to determine the integrity of amplicons. A 2 step PCR strategy was utilized to produce amplicons for DNA sequencing specific to genes encoding highly conserved bacterial 16S ribosomal RNA samples. This yielded 25 microliter aliquots with a DNA concentration of 2 ng/microliter which were sequenced on the Illumina HiSeq platform. Statistical analysis was carried out using R (v.3.5.0). Gut microbiome compositions of participants before and after a 3-day LRD were represented using relative taxa abundance plots. The gut microbiomes of participants before and after a 3-day LRD were compared using fold change analysis to identify any differentially increasing or decreasing taxa. The gut microbiome richness of participants was determined and alpha diversity was calculated using Shannon diversity index. The beta diversity of the gut microbiomes of participants was determined using weighted unifrac distance matrix and plotted two-dimensionally using non-metric distance scaling (NMDS).

Results After a 3-day LRD, there were no significant taxa abundance changes that were consistent between subjects (figure 1). A 3-day LRD did not have a significant effect on the richness and alpha diversity of the gut microbiomes of subjects. A 3-day LRD did not have a significant effect on the beta diversity of the gut microbiomes of subjects ($p = 0.981$).



Abstract P274 Figure 1

Conclusions A 3-day LRD did not have a significant effect on the gut microbiomes of participants. Combined with literature showing that laxatives have no long-lasting effects on the gut microbiome, our study shows that the bowel preparation regimen has no significant effects on the gut microbiome.

P275 ANALYSIS OF EXCLUSIVE ENTERAL NUTRITION FORMULAS IN CROHN'S DISEASE – NEW INSIGHTS INTO DIETARY TRIGGERS

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Introduction/Background Exclusive enteral nutrition (EEN) is an effective treatment for Crohn's disease (CD).

Aims We hypothesised that ingredients of EEN formulas are less likely to initiate a disease flare and their dietary elimination is not essential for disease amelioration.

Methods We performed compositional analysis of EEN formulas with evidence of efficacy in management of active Crohn's disease. Macronutrient content was compared against the dietary reference values (DRV), the UK National Diet and Nutrition Survey (NDNS), and intake of Crohn's disease children. Food additives were cross-referenced against the FAO/WHO database.

Results 61 formulas were identified with variable composition (carbohydrates [22.8–89.3%], protein [7.8–30.1%], fat [0–52.5%]). Maltodextrin, milk protein and vegetable/plant oils were the commonest macronutrient sources. Their n-6:n-3 fatty acid ratio varied from 0.25–46.5. 56 food additives were identified (median per formula: 11). All formulas were lactose, gluten-free and 82% lacked fibre. The commonest food additives were emulsifiers, stabilisers, antioxidants, acidity regulators, and thickeners, figure 1. Food additives, implicated in Crohn's disease aetiology, were present in formulas [modified starches (100%), carrageenan (22%), carboxymethyl cellulose (14%) and polysorbate 80 (5%)], figure 1. Remission rates did not differ between EEN formulas with and without those food additives. Analysis including only formulas from RCTs retained in the latest Cochrane meta-analysis produced similar findings. EEN formulas contained less energy from saturated fat than NDNS intake. Crohn's disease children consumed more sugars, total/saturated fat than the EEN content.

Conclusions We have identified food ingredients which are less likely to trigger Crohn's disease activity. We hereby challenge current perceptions surrounding the role of these ingredients in Crohn's disease management