n=69; p<0.001) and ≥10 mm polyps (21%, n=63; p<0.001). The histological features of the polyps ≤5 mm were predominantly tubular adenomas (TA) (80%, n=86/107), please see Abstract PWE-110 table 1 for rest of histology. Only 38% (n=66) of polyps ≤5 mm were hyperplastic in nature. In polyps ≤5 mm in size, one TA with high grade dysplasia (0.6%) was seen but no carcinoma compared to 11 (3.6%) carcinomas/high grade dyspasia in ≥6 mm size polyps (p<0.001). Three cancers were seen, two in polyps ≥10 mm size and one in the 6–9 mm size. 51% were left sided, 14% were right sided, location not specified in 35%, this difference was not statistically significant.

**Conclusion** In patients who are positive for FHH the incidence of diminutive polyps is considerable and importantly there is a significant proportion of adenomatous polyps. Based on these findings we believe that diminutive polyps should be resected, histologically evaluated and followed-up appropriately.

**Competing interests** None declared.

# Small bowel II

## PWE-111 LIPOPOLYSACCHARIDE INDUCES SMALL INTESTINAL EPITHELIAL CELL APOPTOSIS AND SHEDDING WHICH IS REGULATED BY NFκB SIGNALLING

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**Introduction** The gut barrier is formed by a single cell thick, simple columnar epithelium consisting of intestinal epithelial cells (IECs) held together by tight junctions. In the small intestine, IECs are generated in the crypt, migrate up the crypt-villus axis and are shed at the villus tip. When IECs detach from the villus during the process of shedding, discontinuities occur in the epithelium. IEC shedding increases in response to pro-inflammatory cytokines, resulting in larger discontinuities and increased intestinal permeability. Understanding the mechanisms which regulate IEC shedding may allow development of therapeutic strategies which ameliorate its detrimental effects on gut barrier function in intestinal disease states.

**Aims** To investigate whether IEC apoptosis and shedding are increased during induction of endotoxic shock, and the genetic regulation of this process.

**Methods** Lipopolysaccharide (LPS) from *Escherichia coli* O111:B4, was administered to adult female C57BL/6 mice at 10 mg/kg by intraperitoneal injection (n=6 per group). Animals were euthanased 1–6 h after LPS administration. The duodenum, jejunum and ileum were fixed in formalin. Histopathological examination of H&E stained sections, quantification of villus length, and immunolabelling of IECs for active caspase 3 was performed. Dose response experiments were performed using 0.125 to 20 mg/kg LPS, sacrificing mice (n=4 per group) at 1.5 h. Transgenic mice (NFκB1−/−, NFκB2−/− and cREL−/−) were administered 10 mg/kg or 0.125 mg/kg LPS and euthanased at 1.5 h (n=6). Statistical comparisons were made by t-test with Bonferroni correction.

**Results** In C57BL/6 mice, 10 mg/kg LPS caused a 20–50-fold increase in IEC apoptosis and shedding, with consequent villus atrophy, which was greatest in the duodenum and jejunum at 1.5 h, and in the ileum at 2 h. IEC apoptosis and shedding had subsided by 6 h after LPS administration. LPS at 0.125 mg/kg caused an attenuated response in C57BL/6 mice in terms of villus injury and amounts of apoptosis and shedding at 1.5 h, whereas at this dose NFκB1−/− mice were severely affected, while NFκB2−/− mice were minimally affected.

**Conclusion** These findings suggest that endotoxic shock causes dynamic and hyperacute selective injury to the murine small intestine, prior to obvious pathological alterations in other organ systems. The induced apoptosis and cell shedding occurs within a well defined time period, and varies in intensity and time course in different intestinal segments. The differing sensitivity to LPS in transgenic mice implicates NFκB signalling in the pathogenesis of this type of intestinal injury.

**Competing interests** None declared.

## PWE-112 PREVALENCE OF OSTEOPOROSIS IN A LIVERPOOL COELIAC COHORT SUPPORTS ROUTINE USE OF BONE MINERAL DENSITY ASSESSMENT

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**Introduction** Coeliac disease may be associated with osteoporosis and increased fracture risk. Osteoporosis is a significant public health problem with major consequences for patients and health care systems.[1] Debate exists concerning the utility of routine bone mineral density (BMD) assessment in patients with coeliac disease.[2] We aimed to identify the prevalence of osteoporosis in patients with coeliac disease as defined by BMD assessment.

**Methods** Dietitian led data sets are currently maintained for all coeliac patients under active follow-up at University Hospital Aintree. We retrospectively analysed this information to determine (1) the frequency of BMD assessment in coeliac patients and, (2) the results of BMD assessment. Osteoporosis was defined as a T score of ≤−2.5 SDs below mean at either lumbar spine or hip.

**Results** The data sets for 232 patients were available for analysis. Demographics: 70% female, 30% male, mean age at diagnosis 52 (range 3–79 years). BMD assessment was undertaken in 211 (91%). The indication for this assessment in all cases was a clinicopathological diagnosis of coeliac disease. Of those undergoing BMD assessment, 26% had osteoporosis. On questioning at outpatient assessment 141 (67%) patients reported participation in regular weight bearing exercise. Of these patients 33 (24%) had osteoporosis compared to 10/30 (33%) not documented to undertake weight bearing exercise (p=0.26). Of those undergoing BMD assessment, 128 (61%) had been prescribed calcium supplements. 49/128 (38%) patients documented to be taking calcium supplements had BMD measurements consistent with osteoporosis compared to 4/82 (5%) patients not taking calcium supplements.

**Conclusion** At this UK centre, where over 90% of patients with coeliac disease underwent BMD assessment, 26% had osteoporosis. This is comparable to the rate demonstrated by Fitzgerald *et al* (25%)[3] and provides further support for the routine use of BMD assessment in coeliac disease to screen for osteoporosis. A lesser proportion of patients who participated in regular weight bearing exercise had osteoporosis at BMD assessment, but this finding was not statistically significant.

**Competing interests** None declared.

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