patients underwent OGDs pre-operatively and for what indication and with what result. Further sub-analysis was performed for each operation type.

**Results** 147 OGDs were done on a total of 116 bariatric surgical patients, with 23 patients having had more than one OGD each. Of these 147 OGDs 44 were pre-operative. 15 (29.5%) OGD referrals were made to investigate anaemia, 12 (27.3%) for pre-surgical screening to investigate existing symptoms of gastric ulceration, and 9 (20.5%) to investigate abdominal pain. The remaining referrals were made for interventional gastric balloon insertions and removals (6=13.6%) and to investigate symptoms of reflux (5=6.8%) and dysphagia (1=2.5%). The majority of patients (50%) were referred prior to having a roux-en-y gastric bypass operation. Most of the pre-operative OGD findings were normal (16=36.4%), but gastritis (6=15.6%), hiatal hernias (6=15.6%), gastric ulceration (2=4.5%), oesophagitis (1=2.5%) and duodenitis (1=2.5%) were noted. Of the 13 patients referred with anaemia, 9 (69.2%) had normal mucosa on OGD, but of the 12 patients who had pre-surgical screening 7 (58.3%) were found to have abnormalities, including a fundic gland polyp and antral erosions.

**Conclusion** Using a selective referral process, only 4% of all bariatric surgery cases performed required preoperative endoscopy. The commonest indication for OGD preoperatively was anaemia and the surgery cases performed required preoperative endoscopy. The BCSP has led to an increase in the number of colonoscopies compared to 26% (958/3716) of non-BCSP colonoscopies or lack of awareness of the protocol. We aim to improve adherence by increasing awareness among all endoscopy staff to ensure optimum management of malignant and suspicious lesions.

**Competing interests** None declared.

**REFERENCES**


**Inflammatory bowel disease I**

**PMO-224 NF-κB2 DELETION PROTECTS MURINE COLON AGAINST DSS-INDUCED COLITIS AND THIS IS ASSOCIATED WITH REDUCED EXPRESSION OF TNF-α AND IL14**

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**Introduction** The Nuclear Factor kappa B (NFκB) family of transcription factors signals via two pathways (classical and alternative). Classical pathway NFκB signalling has previously been implicated in the pathogenesis of inflammatory bowel diseases (IBD). However, the role of alternative NFκB pathway activation in the development of intestinal inflammation has not previously been investigated.

**Aims** To investigate the susceptibility of mice deficient in two individual NFκB family members to DSS-induced colitis and the associated molecular changes.

**Methods** Colitis was induced in adult male NFκB1-null and NFκB2-null mice and their wild-type (C57BL/6) counterparts by oral 2% DSS administration for 5 days (n=10 per group). Weight loss and Disease Activity Index (DAI) were evaluated daily. Animals were euthanased on day 6 and histological colitis severity was evaluated in H/E stained colonic sections. The colonic expression of 6 key pro-inflammatory cytokines (TNF-α, IL-1β, INF-γ, IL-6 and IL-14) was assessed by real time PCR (n=4 per group). Statistical comparisons were mostly performed by ANOVA with Bonferroni post-hoc tests, but the Kruskal–Wallis with Dunn’s multiple comparison test was used to analyse DAI and histological scores.

**Results** After oral administration of 2% DSS, NFκB1-null mice showed significantly more loss of body weight whereas NFκB2-null mice showed significantly less loss of body weight on days 5 and 6 compared to wild-type mice. DAI was also significantly higher in NFκB1-null mice and significantly lower in NFκB2-null mice compared to C57BL/6 mice. In agreement with these clinical findings, histological assessment of DSS treated animals confirmed a severely damaged and inflamed distal colon in C57BL/6 and NFκB1-null mice and minimal histological damage and significantly lower inflammation scores in NFκB2-null mice. The expression of IL-6 mRNA was significantly increased in DSS-treated NFκB1-null colon and the expressions of TNF-α and IL-14 mRNAs were significantly reduced in DSS-treated NFκB2-null colon.

**Conclusion** Disruption of the classical NFκB signalling pathway by deleting NFκB1 exacerbates colonic inflammation and tissue damage following DSS administration which may be partially mediated by IL-6. This suggests that classical NFκB pathway inhibitors may be