

parallel to the muscle layer. Gastric submucosal dissection and oesophageal submucosal resections/tunnelling procedures were performed on 5 consecutive 60kg pigs. All cases were video recorded. The time taken to complete resection/tunnelling, complications encountered and power settings used were recorded. Two animals were euthanized immediately (termination study – TS) and three animals were recovered for 3 days (survival study = SS). Submucosal defects and excised flaps were measured and assessed histologically.

**Results** Five (3TS, 2SS) consecutive gastric submucosal dissections, 5 oesophageal resections {4 (3TS, 1SS) semi and 1 (SS) full circumferential oesophageal mucosal resections} and 2 submucosal tunnelling procedures {1 (SS) with partial myotomy and 1 (TS) with no myotomy} were performed. The median time to complete a gastric resection was 46 min range (21–83min) using RF cutting 35 W and 41 min range (12–50 min) using RF cutting 25W for the oesophageal excision/tunnelling procedure. Median gastric defect size was 55 mm, range 35–70 mm and median oesophageal defect size was 47 mm, range 35–70 mm. Microwave coagulation was applied for either minor bleeding or visible vessels on 57 occasions (mean energy 7.5 W). No endoscopic or histologic perforations were noted. All excised flaps were appropriate for histological assessment apart from one oesophageal flap that was mildly heat damaged. Gastric and oesophageal muscle layers/serosa were intact and viable. In three oesophageal cases, there was a mild muscle cell alteration but contiguity was retained. In one gastric resection, another dissection knife assisted the last ribbon cut.

**Conclusion** This initial evaluation of “Speedboat-RS2” in the upper GI tract suggests that it facilitates rapid and safe en-bloc mucosal resection in the oesophagus and stomach. It also appears promising for safe and rapid submucosal tunnelling in the oesophagus and has potential to be utilised for POEM.

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## Colon and anorectum

### PWE-001 FIELD CANCERISATION THEORY IN COLORECTAL CANCER (CRC): WHAT ROLE DO FIBROBLAST GROWTH FACTORS HAVE?

<sup>1</sup>A Patel\*, <sup>2</sup>N Williams, <sup>3</sup>C Nwokolo, <sup>1</sup>G Tripathi, <sup>1,3</sup>R Arasaradnam. <sup>1</sup>CSRI, University of Warwick, UK; <sup>2</sup>Colorectal Surgery, University Hospital Coventry and Warwickshire, Coventry, UK; <sup>3</sup>Gastroenterology, University Hospital Coventry and Warwickshire, Coventry, UK

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**Introduction** Characterisation of the molecular field defect around colorectal cancer (CRC) could enable identification of novel biomarkers that could be used for early detection of CRC. Previous studies have suggested fibroblast growth factor 19 (FGF19) may play a role in CRC formation through interaction with the B-catenin/wnt signalling cascade. The role of fibroblast growth factor 7 (FGF7) however remains controversial. The aim of this study was to determine if there are differences in FGF19 and FGF7 gene expression in cancer tissue and the adjacent ‘normal tissue’ compared with normal colonic tissue.

**Methods** Mucosal pinch biopsies were taken from the rectum and caecum at time of colonoscopy for healthy controls. For CRC

patients, tissue samples were taken from the tumour, adjacent to the tumour and at the resection margin of the colectomy specimen. Healthy controls were age and sex matched to CRC patients. Quantitative real time PCR was used to determine gene expression of FGF19, its receptor FGFR4, FGF7 and its receptor, FGFR2. Results were further validated using immunohistochemistry. Serum levels of FGF19 were measured using the Quantikine ELISA kit (RandD systems, UK).

**Results** 49 patients were recruited (28 M: 21 F, median age 71 years (range 48–86 years)); 18 patients with CRC and 32 healthy controls. There was no overall difference in gene expression of FGF19/FGFR4 or FGF7/FGFR2 between cancer patients and healthy controls. There was upregulation of FGFR4 in mucosa adjacent to the tumour (mean fold change 1.23 vs. 0.93,  $p = 0.38$ ) and the tumour itself (mean fold change 1.49 vs. 1.04,  $p = 0.700$ ) in patients whose tumour expressed FGF19 compared to those that did not. Patients with upregulation of FGF19/FGFR4 had a significantly lower fasting serum FGF19 level (119 pg/ml versus 208 pg/ml,  $p = 0.05$ ).

FGF7 was upregulated in 6/19 cancers; this was associated with a significant upregulation in FGF7 in adjacent mucosa compared with cancers where FGF7 was downregulated (mean fold change 3.62 vs. 0.95,  $p = 0.018$ ). There was a non-significant trend towards upregulation of the receptor (FGFR2) in mucosa adjacent to the cancer and the tumour tissue itself.

**Conclusion** Upregulation of FGFR4 in patients whose tumours expressed FGF19 corresponded inversely with serum FGF19 suggesting its potential as a putative biomarker. Significant upregulation of FGF7 in ‘normal’ mucosa adjacent to only tumours that express FGF7 lends support to the field theory of colorectal carcinogenesis.

**Disclosure of Interest** None Declared.

### PWE-002 THE POSITIVE PREDICTIVE VALUE OF A COLONOSCOPIST LABELLING A LESION AS CANCER – WHAT SHOULD WE TELL THE PATIENT?

AM Verma\*, RE Smith, A Dixon, AP Chilton. Gastroenterology, Kettering General Hospital NHS Foundation Trust, Kettering, UK

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**Introduction** Colonoscopy is the modality of choice for bowel cancer screening and investigation of iron deficiency anaemia. Hence colonoscopists are most likely to diagnose colorectal cancer (CRC). It is desirable to inform a patient post endoscopy they have CRC but a colonoscopist may fear giving an incorrect diagnosis.

All patients with CRC are discussed at the weekly multi-disciplinary team meeting. A delay in treatment is often caused by the patient not being aware of a CRC diagnosis. This may require an additional appointment to inform the patient before the appointment with a Surgeon or Oncologist to discuss treatment.

This delay can increase the risk of progression of CRC and reduces the time the patient and family have to adjust to a CRC diagnosis and its consequences. We aim to test the positive predictive value (PPV) of colonoscopists diagnosing CRC and audit if patients were informed.

**Methods** 8561 colonoscopies undertaken at Kettering General Hospital (KGH), if “tumour/cancer” was recorded this was correlated to outcome. The reporting software gives an option to record if patient “informed of cancer” or “informed of lesion”.

**Results** “Tumour/cancer” recorded 350 times (4.09% of colonoscopies)