



**Abstract O36 Figure 1** 3D reconstruction of gastric gland with CCO positive (brown) & negative regions (blue) visible

pathology software (Qupath), to analyse clonal patch sizes. Serial tissue sectioning was performed to trace CCO/MTCO1 mutated glands of interest for 3D reconstruction. Briefly, registration using a rigid and non-rigid B-spline transformation was applied, followed by a denoising step. Segmentation of glands was done by modelling using a Gaussian distribution, extraction of closing maps and applying an ellipsoidal fitting model. Cubic interpolation was then used for 3D modelling.

**Results** Patient ages were 31–65 years. Histologically, 8 were normal, 2 had active *H. Pylori* infection, 4 had evidence of previous infection with chronic inflammation, atrophy and intestinal metaplasia. CCO and MTCO1 clones were seen as wholly mutated glands and partially mutated glands. Overall clonal expansions were small, patch size analysis showed clones were most frequently singular glands, and rarely small patches (mean patch size = 1.65 glands). *H. Pylori* infection or chronic inflammation increased the frequency and size of patches compared to non-exposed tissue. 3D reconstruction (figure 1) allowed visualisation of the structure of the oxyntic gland, and tracing of CCO lineages allowed visualisation the functional architecture.

**Conclusions** This data describes the pattern of clonal expansions occurring in normal gastric epithelium. *H. Pylori* exposure and chronic inflammation lead to an increase of up to ten fold in frequency and size of clonal expansions. We observed a smaller increase in clonal expansions with advancing age. 3D reconstruction enabled tracing of mutant lineages in oxyntic glands, demonstrating for the first time the functional 3D architecture of the gastric stem cell unit. This work may help inform a model of pre-tumour progression in the chronically inflamed stomach.

## Pancreas and neuroendocrine

O37

### SCREENING FOR PANCREATIC CANCER IN HIGH RISK INDIVIDUALS: EXPERIENCE FROM A SPECIALIST CENTRE

<sup>1</sup>Stewart Bonnington\*, <sup>1</sup>Karen Lam, <sup>1</sup>Manu Nayar, <sup>1</sup>John Leeds, <sup>2</sup>Lindsay O'Dair, <sup>1</sup>Shridhar Dronamraju, <sup>1</sup>Richard Charnley, <sup>1</sup>Kofi Oppong. <sup>1</sup>HPB Unit, Newcastle Upon Tyne Hospitals, Newcastle Upon Tyne, UK; <sup>2</sup>Clinical Genetics, Newcastle upon Tyne Hospitals, Newcastle upon Tyne, UK

10.1136/gutjnl-2020-bsgcampus.37

**Introduction** Two groups of high-risk individuals (HRI) for pancreatic ductal adenocarcinoma (PDAC) have been defined. 1) Individuals from familial pancreatic cancer (FPC) kindreds

and 2) individuals with identified genetic syndromes (GS) due to a germline mutation. Screening of HRI has been proposed to identify premalignant lesions and early stage malignancy with the aim of improving outcomes. Screening criteria have been formulated by a number of organisations including the international Cancer of the Pancreas-Screening consortium (CAPS) and the Italian Society for the Study of the Pancreas (IASP). Recent CAPS and IASP publications have reported a significant yield. A prior meta-analysis concluded that 135 patients with HRI were needed to be screened to identify one high risk lesion. The aim of this study is to review compliance with guidelines and the yield of HRI screening in our screening programme.

**Methods** The study is a retrospective review of a prospectively maintained database of HRI. EUS, was the preferred annual screening method. MRI and CT were used in some patients due to intolerance of endoscopy or preference. Data was cross-checked with the endoscopy database and electronic patient record.

**Results** A total of 110 individuals (71F) median age 46 (IQR, 41–57.75) were enrolled and underwent at least one screening procedure between January 2006 and January 2019. 108 (98.2%) met either or both CAPS/IASP criteria: 58 were classified as FPC and 50 GS. The 2 who didn't meet criteria were a patient with idiopathic juvenile onset chronic pancreatitis (CP) and a patient with idiopathic CP and one first degree relative with PDAC. 487 screening procedures were performed. 407 (83.6%) EUS, 49 (10.1%) CT and 23 (4.75%) MRI with a median of 4 [IQR, 2–6] procedures per individual and median follow up 4.3 years [IQR, 2–7.75]. 9 (8.2%) had solid or cystic abnormalities identified on EUS and underwent tissue sampling. Two patients subsequently underwent distal pancreatectomy. The first (60 yr old female with hereditary pancreatitis) had a 20 mm cystic lesion in the tail of pancreas on her 2nd EUS. Resection histology was mucinous cystic neoplasm (follow up 11 years). The 2nd (48 yr old male, FPC) had a 14 mm nodule in body of pancreas. Histology was low grade pancreatic intraepithelial neoplasia (follow up 11.5 years). There were no adverse events consequent on screening.

**Conclusions** In a large cohort of HRI undergoing screening, compliance with international criteria was good with no screening related adverse event. However, the yield to date has been low with only one high grade precursor lesion resected from 487 screening procedures.

O38

### RICOCHET: A TRAINEE-LED NATIONAL PROSPECTIVE STUDY OF THE DIAGNOSTIC PATHWAY FOR SUSPECTED PANCREATIC CANCER

RICOCHET Study Group\*. West Midlands Research Collaborative, Birmingham, UK

10.1136/gutjnl-2020-bsgcampus.38

**Introduction** Pancreatic cancer is a deadly disease with a poor prognosis. Variations in the diagnostic pathway nationally may affect outcome, therefore a prospective study is necessary to map variation.

**Methods** Trainee-led prospective UK national study of the diagnostic pathway for suspected pancreatic cancer. Including all patients presenting within a 3-month study period, with 90-days follow-up. All investigation and MDTs were recorded in the REDCap database with a unique OpenPseudonymiser