than oesophageal cancer (0.735 vs. 0.900, p < 0.001). Inflammatory scores in GOJ cancers were lower than in gastric cancer and higher than in oesophageal cancer.

**Conclusion** This study provides direct evidence for marked differences in the gastric mucosal phenotype in the patients with oesophageal versus gastric non-cardia cancer, with the former being healthy and uninflamed, but the latter atrophic and inflamed. The background gastric mucosa of GOJ cancer supported them being two distinct aetiologies, one group resembling oesophageal adenocarcinoma and other gastric non-cardia cancer.

**Disclosure of Interest** None Declared.

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**PTU-165** WORLDWIDE EPIDEMIOLOGICAL EVIDENCE SUPPORTS A COMMON FACTOR PREDISPOSING TO NON-CARDIA GASTRIC CANCER AND PROTECTING FROM OESOPHAGEAL ADENOCARCINOMA

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**Introduction** During last three decades, global incidence of oesophageal adenocarcinoma has increased more rapidly than any other cancer. A concurrent reduction in the incidence of gastric cancer has been reported from some populations. We aimed to examine the geographical pattern of oesophageal adenocarcinoma versus gastric non-cardia cancer across the world where reliable cancer registry data were available.

**Methods** Data were abstracted from “Cancer Incidence in Five Continents” Volume 10. Oesophageal and gastric cancers were selected based on ICD-10 codes C15 and C16, respectively. Oesophageal adenocarcinomas were identified by ICD-O morphology codes. Datasets reporting >500 cases for total gastric cancer and >100 for total oesophageal cancer were selected. We examined correlation between age-standardised Incidence rates (ASR) of oesophageal adenocarcinoma and non-cardia gastric cancer using Spearman’s non-parametric correlation coefficient (CC). We also allocated cancer sites into oesophageal and non-cardia gastric adenocarcinoma categories based on gender ratio for oesophageal adenocarcinoma and non-cardia gastric cancer in each dataset.

**Results** Out of 424 datasets from 290 cancer registries, 206 datasets covering 40 countries met the selection criteria. There was a strong inverse correlation between oesophageal adenocarcinoma and gastric non-cardia cancer in males (CC = -0.768, p < 0.001) and females (CC = -0.705, p < 0.001). After dividing cardia cancer into two subtypes with potentially oesophageal or gastric origin and adding them to original oesophageal adenocarcinoma or gastric non-cardia groups, the inverse correlation remained strong in males (CC = -0.660, p < 0.001) and females (CC = -0.536, p < 0.001). Oesophageal adenocarcinoma only showed a rise when incidence of non-cardia gastric cancer fell below 9/100,000 person-years for males and 4.5/100,000 person-years for females.

**Conclusion** This cross-sectional study is consistent with a common underlying factor predisposing to non-cardia gastric cancer and protecting from oesophageal adenocarcinoma, such as H. pylori atrophic gastritis. If this is the case, then the incidence of non-cardia gastric cancer would need to fall to substantially lower levels than currently seen in Far Eastern populations before any rise in oesophageal adenocarcinoma would be apparent.

**Disclosure of Interest** None Declared.