both questionnaires and verbal interviews. Incident cases were identified by linkage with routinely collected hospital inpatient and cancer registry data for England, Scotland and Wales. Primary clinical outcomes were GORD without oesophagitis, GORD with oesophagitis (reflux oesophagitis), Barrett's oesophagus and oesophageal adenocarcinoma. The effects of heavy manual activity on disease risk were estimated using Cox proportional hazard regression adjusted for multiple risk factors and stratified by socioeconomic status.

Results: Between 2006 and 2010, 502 men and 524 women were enrolled. Main analyses were limited to the working population with a full set of variables of interest (n=266, 453). Compared to jobs with low levels of heavy manual activity, high-level jobs had increased hazard ratios (HRs) for GORD (1.20, 95% CI 1.11-1.30), reflux oesophagitis (1.17, 95% CI 1.04-1.31) and Barrett's oesophagus (1.13, 95% CI 0.98-1.32), but not oesophageal adenocarcinoma (0.91 95% CI 0.54-1.56).

Conclusion: High levels of occupational heavy manual activity could be used as a new risk factor for GORD and reflux oesophagitis, the precursor diseases of oeosphageal adenocarcinoma.

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SO-11 Molecular evaluation of *Helicobacter pylori* infection in 470 Colombian patients with premalignant lesion and gastric cancer

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Background: Gastric cancer (GC) is the third cause of death from cancer in the world and is the first cause of death in men and the fourth in women for Colombia. The bacteria *Helicobacter pylori* (*H. pylori*) is a class I human carcinogen for GC. The presence of the gene associated with cytotoxin A (cagA) and the s1 and m1 alleles in the vacuolizing cytotoxin A (vacA) gene, are associated with the development of neoplastic lesions.

Methods: We molecularly evaluated the virulence profile of *H. pylori* strains present in 93 patients with GC and 377 patients with premalignant lesions from Ibagué-Colombia. The DNA from the antral biopsies was extracted using the DNeasy Blood & Tissue Kit of QIAGEN. The bacterium was identified by PCR amplification of a fragment of the 16s rDNA gene and the genotypes were identified by amplification of fragments of the cagA, cagE genes and the signal (s1/s2 alleles) and medium (m1/m2 alleles) regions of vacA gene. The PCRs were performed separately in a Biorad Dual-Touch 1000 thermocycler. The amplification products were visualized by electrophoresis in agarose gels.

Results: The prevalence of *H. pylori* infection was 44% and 23 genotypes of strains were identified. We observed a decrease in the number of genotypes and an increase in the frequency of the cagA/cagE/vacAs1m1 genotype according to the degree of gastric lesion increases. 41,1% (131/318) of patients with chronic non-atrophic gastritis were infected with *H. pylori*; we found the highest number of genotypes in this group of patients. 35% of those infected had the vacAs2m2 non-pathogenic genotype, 30% had the cagA/cagE/vacAs1m1 pathogenic genotype and the remaining patients had intermediate genotypes with one or two oncogenes or alleles associated with pathogenicity. 42,3% (25/59) of patients with preneoplastic pathologies were positive for infection, 8% had the non-pathogenic genotype and 64% had the pathogenic genotype. In patients with GC, 52,3% (49/93) were positive for the infection, 57% had a pathogenic genotype and only one patient had the non-pathogenic genotype.

Conclusion: We identified the cagA/cagE/vacAs1m1 pathogenic genotype and the vacAs2m2 non-pathogenic genotype reported in the literature and 21 intermediate genotypic variants. These genotypes could explain the variations in the clinic response to infection. Our results support the hypothesis of positive selection towards the genotypes considered to be pathogenic as the severity of gastric injury increases.

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SO-12 Multicentre validation of an immune-inflammation-based nomogram to predict survival in western resectable gastroesophageal adenocarcinoma: The NOMOGAST

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Background: Despite standard multimodality treatment, > 50% of operable gastroesophageal adenocarcinoma (GEA) relapse following curative-intent surgery in the West. Although treatment decision relies on established clinicopathologic features, they are flawed by a limited predictive value and inability to capture interpatient heterogeneity. We aimed at externally validating our previously-described nomogram (Salati et al. ESMO 2019) to enable a more accurate estimate of individualized risk in resected GEA.

Methods: Electronic medical records of patients undergoing curative-intent surgery for c/uT2-T4 and/or node-positive gastric and gastroesophageal junction adenocarcinoma were retrieved and variables deemed of potential interest were collected. The Modena Cancer Centre cohort served as the training cohort (TC), while the joint cohort of Cremona Cancer Centre and Forlì Cancer Centre served as the validation cohort (VC). Cox proportional hazards in univariate and multivariate regression were used to assess the effects of the prognostic factors on OS. A graphical nomogram, derived from the multivariate Cox regression model, was constructed using the package Regression Modeling Strategies (ver. 5.0-1) in R software. The performance of the prognostic model was evaluated and external validation performed.

Results: The TC and VC consisted of 112 and 319 patients, respectively. The following covariates retained independent prognostic value in the TC and were used for the construction of a nomogram estimating 3-year and 5-year OS: ECOG PS >0 (p 0 (vs 61%; p=0.24), 39% angioinvasion (vs 50%; p=0.05), and 67% were node-positive (vs 80%; p=0.08); junctional cancers account for 16% (vs 12%; p=0.15) of the overall population and 42% (vs 48%; p=0.25) of patients received adjuvant chemotherapy. The discriminatory ability of the prognostic model was evaluated with the c-Harrell index (0.78 and 0.76, in the VC and TC, respectively). Then, a 3-tier scoring system was developed through a linear predictor grouped by 25 and 75 percentiles, which strengthened the good discrimination of the model (p< 0.001). A calibration plot demonstrated concordance between the predicted survival and actual survival both in the TC and VC. Finally, a decision curve analysis was plotted that depicts the clinical utility (net benefit) of the nomogram.

Conclusion: We externally validated a prognostic nomogram to predict 3-year and 5-year OS in a joint independent cohort of resectable GEA. This tool incorporates readily-available and inexpensive patient and disease characteristics, as well as immune-inflammatory determinants. It has been shown to be accurate (well-calibrated with good discriminative ability), generalizable and clinically effective. Although a prospective validation in a larger patient population is warranted, the NOMOGAST could represent a useful tool to be implemented in the clinic to assist decision-making and clinical trial design.

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SO-13 Can we screen for pancreatic cancer? Identifying a sub-population of patients at high risk of subsequent diagnosis using machine learning techniques applied to primary care data

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Background: Ninety-four percent of pancreatic cancer patients die within 5 years of their diagnosis. Most patients experience no obvious symptoms until the tumour is well advanced. It is now possible, via a blood test, to detect pancreatic cancer before symptoms of late-stage disease are evident. However, in order to be cost-effective, this test should only be applied to a limited group of individuals.

Methods: We conducted a national, population-based, case-control study to determine whether it is possible to identify a sub-population of patients at high risk of developing pancreatic cancer. Our hypothesis was that patients with early malignancy share similar profiles of early, diffuse, warning signs which might be detectable through the application of machine learning approaches. We used the Clinical Practice