a database of unique marker genes, while KP strains were identified and quantified using reference-based approaches utilising genomes of 276 KP strains and 10,484 other bacterial species. Prevalence, relative abundance, and diversity of KP were tested against IBD-related clinical phenotypes, while unsupervised clustering and dimensionality-reduction approaches were used to study strain composition.
Results: Prevalence and relative abundance of KP were found to be significantly increased (p-value < $1.0*10^{-4}$) in the gut of IBD patients (prevalence ≈ 10%) when compared to the general population (prevalence ≈ 2%), with a trend of increased prevalence of KP in more severe cases of UC (pancolitis, severe colitis, and patients who underwent colonic resection). Strain identification was considered if the relative abundance of a specific strain was above 10% of the total KP content in the sample. In total, 54 strains of KP were identified and quantified, of which 33 were found only in IBD patients (18 in UC, 6 in CD and 9 in both CD and UC patients). Our results also show concordance with previous in-vitro studies: Two KP strains previously described to cause UC-like pathology and strong immune response in mice (strains KP-700603 and KP-2H7) were found to be specific to gut of UC patients in our cohorts.
Conclusion: We demonstrate the increase in prevalence of KP in the gut microbiome of IBD patients, and show that certain strains of KP are specific to IBD patients. Thus, KP may be involved in IBD and has a potential to be exploited as novel target for alleviating the severity of the disease.
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OP126 RISK OF GASTRIC CANCER AFTER *HELICOBACTER PYLORI* ERADICATION IN DIABETES MELLITUS PATIENTS: A TERRITORY-WIDE STUDY WITH PROPENSITY SCORE ANALYSIS

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Introduction: Whether diabetes mellitus (DM) increases gastric cancer (GC) risk remains controversial in prior studies due to inadequate adjustment for important risk factors including *Helicobacter pylori*(HP) infection, glycemic control, concomitant medication usage and cancer sites.
Aims & Methods: We aimed to investigate whether type II diabetes mellitus (DM) increased GC risk in patients after HP treatment.
This was a territory-wide cohort study of patients aged ≥45 years who had received clarithromycin-based triple therapy for HP between 2003 and 2012. Data were retrieved from the public electronic health database. Observation started from HP therapy to GC diagnosis, death or end of study (December 2015). Exclusion criteria included type I DM, GC diagnosed within first year of HP therapy, prior GC or gastrectomy, and failure of HP eradication. Theadjusted hazard ratio (aHR) of GCwith DM was calculated by Cox model with propensity score regression adjustmentfor 20 covariates (age, sex, smoking, alcoholism, past history of gastric and duodenal ulcers, other comorbidities [atrial fibrillation, ischemic heart disease, congestive heart failure, chronic renal failure, cirrhosis, stroke, hypertension and obesity] and medications [aspirin, non-steroidal anti-inflammatory drugs (NSAIDs), cyclooxygenase-2 (COX-2) inhibitors, and proton pump inhibitors (PPIs)]).
Results: Of 46,460 eligible patients, 6,900 (14.9%) had DM. During a median follow-up of 7.1 years (IQR:4.8-9.8) with 337,313 person-years, 153 (0.33%) developed GC at a median age of 72.4 years (IQR:63.8- 82.6). There were 31 (20.3%) cardia cancers and 88 (57.5%) non-cardia cancers, while the remaining 34 (22.2%) cases did not have site specified.DM was associated with an increased GC risk (adjusted HR:1.67; 95% CI:1.08-2.58). This association was biased towards null if concomitant medication usage was not adjusted (adjusted HR:1.30; 95% CI:0.85-1.99), with the most influential effect from statins (**Table**). On the other hand, HR increased to 1.92 (95% CI:1.28-2.90) without adjusting for comorbidities.Stratified analysis shows the risk was increased for cardia cancer only (aHR:3.40, 95% CI:1.45-7.97), in those with suboptimal DM control (time-weighted average HbA1c ≥6.0%; aHR:1.68, 95% CI: 1.07-2.63) and metformin non-users (aHR 2.59, 95% CI 1.41-4.74).
Conclusion: Type II DM was associated with an increased GC risk among HP-eradicated patients, in particular cardia GC and those with suboptimal DM control. Inadequate adjustment for concomitant medications and comorbidities could potentially bias the results in previous studies.
Disclosure: Nothing to disclose

	No. of patients without DM and GC	No. of patients with DM and GC	HR	95% CI	p-value
All variables adjusted for	39,560 (GC = 117)	6,900 (GC=36)	1.67	1.08 - 2.58	0.021
Statins not adjusted for	39,560 (GC = 117)	6,900 (GC=36)	1.43	0.93 - 2.19	0.101
Statins and aspirin not adjusted for	39,560 (GC = 117)	6,900 (GC=36)	1.32	0.86 - 2.02	0.203
All drugs not adjusted for	39,560 (GC = 117)	6,900 (GC=36)	1.30	0.85 - 1.99	0.234
Comorbidities not adjusted for	39,560 (GC = 117)	6,900 (GC=36)	1.92	1.28 - 2.90	0.002
Subgroup analysis					
Metformin use					
Yes	39,560 (GC=117)	6,379 (GC=32)	1.28	0.74 - 2.20	0.378
No	39,560 (GC=117)	521 (GC=4)	2.59	1.42 - 4.74	0.002
Time-weighted average HbA1c level					
HbA1c ≥ 6.0%	39,560 (GC=117)	6,379 (GC=32)	1.68	1.07 - 2.63	0.025
HbA1c < 6.0%	39,560 (GC=117)	521 (GC=4)	1.99	0.71 - 5.54	0.188
Cancer site*					
Cardia	39,462 (GC=19)	6,876 (GC=12)	3.40	1.45 - 7.97	0.005
Non-cardia	39,513 (GC=70)	6,882 (GC=18)	1.53	0.84 - 2.78	0.161
Non-carida + unspecified site	39,541 (GC=98)	6,888 (GC=24)	1.33	0.80 - 2.23	0.271

DM, diabetes mellitus; GC, gastric cancer; HR, hazard ratio; 95% CI, 95% confidence interval; NSAIDs, non-steroidal anti-inflammatory drugs; COX-2, cyclooxygenase-2; PPIs, proton pump inhibitors; HbA1c, hemoglobin A1c * total cancer cases = 153 (non-cardia: 88, cardia: 31, unspecified: 34)

[Association between diabetes mellitus and gastric cancer (propensity score regression adjustment)]

OP127 VALIDATION OF SCORING SYSTEMS FOR DIFFERENTIATING INTESTINAL TUBERCULOSIS FROM CROHN'S DISEASE UTILIZING CLINICAL, ENDOSCOPIC AND PATHOLOGICAL FINDINGS: A MULTICENTER STUDY FROM THAILAND AND HONG KONG

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Introduction: Differentiating between Intestinal tuberculosis (ITB) and Crohn's disease (CD) is a diagnostic challenging in TB-endemic areas. Several models have been developed to distinguish these two diseases; however, no studies have externally validate these scores using data from the same cohort.
Aims & Methods: To validate existing scoring systems which used clinical, endoscopic and pathological findings in differentiating ITB from CD based on data from the same cohort.
We retrospectively collected data from patients newly diagnosed ITB and CD in 5 referral-centers in Thailand and Hong Kong. Clinical data was reviewed from medical records. Endoscopic and pathological findings were reviewed by endoscopists and pathologist blinded to the diagnosis. The data was applied to published scoring systems including score from Lee *et al* (Endoscopy 2006;38:592-7), Makharia *et al* (Am J Gastroenterol 2010;105:642-51), Jung *et al* (Am J Gastroenterol 2016;111:1156-64) and Limsrivilai *et al* (Am J Gastroenterol 2017;112:415-27). The performance of each score was evaluated with the area under the receiver operating characteristic curve (AuROC) and were compared to each other using the DeLong test.