Title	High prevalence of intestinal metaplasia in a high gastric cancer risk region in China
Author(s)	Wong, BCY; Lam, SK; Ching, CK; Ho, J; Yuen, ST; Hu, HC; Lai, KC; Ong, LY; Gao, Z; Chen, JS; Chen, BW; Jiang, XW; Hou, XH; Lu, JY; Wang, QH
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cases two years after *H. pylori* eradication. Statistical ... eradicators and non-eradicators was stated from the time of 12 months after eradication.

Conclusions: The cure of *H. pylori* infection significantly reduces the presence of antral IM. Regression of IM appears to be a long-term process taking many months after *H. pylori* eradication.

P456 High prevalence of intestinal metaplasia in a high gastric cancer risk region in China

B.C.Y. Wong, S.K. Lam, C.K. Ching, J. Ho, S.T. Yuen, W.H.C. Hu, K.C. Lai, L.Y. Ong, Z. Gao, J.S. Chen, B.W. Chen, X.W. Jiang, X.H. Hou, J.Y. Lu, Q.H. Wang. *Department of Medicine, University of Hong Kong, Hong Kong; Public Health Bureau, Changle, China*

Background: Intestinal metaplasia (IM) is considered to have pre-malignant potential. There is inconclusive evidence on the association between intestinal metaplasia and *Hp* infection.

Aim: We studied the development of IM in normal subjects focusing on the association with *Hp* infection in China with a high gastric cancer mortality (75/105 population) compared it with Hong Kong (mortality $7.5/10^5$).

Method: Antral and body mucosa biopsies were examined for the presence of (IM) in asymtomatic or dyspeptic subjects (n = 534) selected at random from 2,434 volunteers in Changle with no history or endoscopic evidence of ulcer during a gastric cancer screening program. 94 normal or dyspeptic subjects in Hong Kong were used as control. Extent of IM was semiquantitatively scored from 0 to 3 (0 = no IM, 1 = mild, 2 = moderate, 3 = severe). Hp infection was determined by rapid urease test, histology and serology (anti-*Hp* IgG antibody (Bio-Rad).

Results: IM was found in 4% of both *Hp*+ and *Hp*− body mucosa. The prevalence of IM was higher in antral than body mucosa (24% v 4%; $p < 0.001$)

Antral	Changle		Hong Kong	
	Hp+ (%)	Hp−(%)	Hp+ (%)	Hp−(%)
IM-0	321 (75)	87 (82)	53 (90)	34 (97)
IM-1	60 (14)	10 (9)	5 (8)	1 (3)
IM-2	39 (9)	6 (6)	1 (2)	0
IM-3	8 (2)	3 (3)	0	0

There is no correlation between *Hp* status and presence of IM in both Changle and Hong Kong (p = ns). Changle has a higher prevalence of IM than Hong Kong ($p < 0.001$) in both *Hp*+ and *Hp*−subjects.

Conclusion: The overall prevalence of antral IM in both *Hp*+ and *Hp*−subjects are higher in Changle than in Hong Kong. We failed to show, however, a significant association between IM and *Hp* infection in Changle. The relatively high prevalence of IM in *Hp−ve* subjects in this high gastric cancer risk region may mean that apart from *Hp* infection, other factors like diet or pollution may play an important role in carcinogenesis.

P457 Relationship between *H. pylori* infection, autoimmunity and gastritis in patients with sjögren's syndrome and dyspepsia

D. Sorrentino, G. Faller, G.F. Ferraccioli, S. DeVita, A. Labombarda, A. Ponzetto, S. Kahlow-Toussaint, E. Bartoli. *Dept of Internal Medicine, University of Udine, Italy; Department of Pathology, University of Erlangen- Nürnberg, Germany*

Background: Patients with Sjögren's syndrome are often affected by an ill-defined chronic gastropathy. In this regard, the relationship between H. pylori infection, autoimmunity and gastritis is uncertain.

Methods: 54 dyspeptic subjects with Sjögren's syndrome and 150 dyspeptic controls were retrospectively evaluated for the prevalence of autoantibodies against human gastric mucosa and of H. pylori infection/virulence and pathogenetic role.

Results: Prevalence of H. pylori was similar in the two groups: 31/54 (57%) vs 93/150 (62%). Prevalence of anti cag-A was also similar in the two groups while eradication rate was lower in Sjögren's syndrome patients (56% vs 86%, χ^2 test: $p < 0.02$). In the latter, several months after eradication, dyspepsia persisted in the majority – 86% vs 13% of controls ($p < 0.001$). Autoantibodies against gastric mucosa were present in 29% of

P458 Atrophic gastritis and *Helicobacter py* patients treated with proton pump inh

C.J. Larkin, R.G.P. Watson, J. Sloan, J. Ardill, C. Johnst K.D. Buchanan. *Department of Medicine, Queen's Univ Ireland*

Background: Long term proton pump inhibition is st conditions such as uncomplicated Barrett's oesophag have suggested that acid suppression in the presence of infection is associated with increased risk of atrophic g

Aims: To investigate the prevalence and associatio atrophic gastritis in patients taking long term proton pu

Methods: 41 patients with Barrett's oesophagus, gas flux disease and non-ulcer dyspepsia were recruited. Fa levels were recorded. Biopsies taken from the gastric l for atrophy by the Sydney System. H. pylori status histology and by 13 carbon urea breath testing.

Results:

Atrophic gastritis	No. infected with H. pylori	Mean serum gastrin
Present (n = 15)	13	323
Absent (n = 26)	4	81

Approximately one third of patients on treatment v atrophic gastritis. Eighty-seven percent of these had The rate of H. pylori infection differed significantly b gastritis group and the non-atrophic gastritis group ($p <$ level was significantly higher in the atrophic gastritis There was no significant difference in the length of tim PPIs.

Conclusions: The development of atrophic gastrit is strongly associated with H. pylori infection and m elevation in fasting serum gastrin. It is not associat therapy.

P459 The −308 polymorphism in the TNFα associated with increased risk of duo

A.E. Griffiths, M.M. Walker[1], D. Mantafounis, M.R. T [1]*Departments of Medicine and Histopathology, Imperi Medicine at St Mary's, London, UK*

Background: It is likely that host genetic factors play the outcome of chronic *H. pylori* (Hp) infection. One gene is that encoding the important pro-inflammatory cytokine has been found in significantly greater concer juice and mucosa of Hp positive compared with Hp r the highest levels of TNFα correlate with more severe gene is polymorphic at position −308 where a G to A an allele (termed TNF2) which is associated with inc TNFα compared with the more common TNF1 allele.

Aim: The aim of this study was to test the hypothes common in duodenal ulcer (DU) patients than contro higher levels of TNFα contributing to the pathogenesi

Methods: A 107 base-pair segment of the TNFα amplified using the polymerase chain reaction. TN distinguished by the loss of a Nco1 restriction site in t

Results:

Genotype	DU	HP+ NUD	Hp− NUD
1,1	84	15	39
1,2	32	10	13
2,2	3	0	3
Total	119	25	55

For DU v Hp+ NUD the odds ratio (OR) resultin TNF2 = 0.63 (0.24–1.72); For Hp+ v Hp− OR = 1.1

Conclusions: The possession of the TNFα −30 increase the risk of duodenal ulcer, nor the risk of *H.*