

cases two years after *H. pylori* eradication. Status of 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 7.5/10⁵).

Method: Antral and body mucosa biopsies were examined for the presence of (IM) in asymptomatic 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	
	<i>Hp+</i> (%)	<i>Hp-</i> (%)	<i>Hp+</i> (%)	<i>Hp-</i> (%)
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-* 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. Autoantibodies against gastric mucosa were present in 29% of

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

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

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

Aims: To investigate the prevalence and association of atrophic gastritis in patients taking long term proton pump inhibitors.

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

Results:

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

Approximately one third of patients on treatment with proton pump inhibitors had atrophic gastritis. Eighty-seven percent of these had *H. pylori* infection. The rate of *H. pylori* infection differed significantly between the atrophic gastritis group and the non-atrophic gastritis group (p < 0.05). The level was significantly higher in the atrophic gastritis group. There was no significant difference in the length of time on PPIs.

Conclusions: The development of atrophic gastritis is strongly associated with *H. pylori* infection and mucosal atrophy and elevation in fasting serum gastrin. It is not associated with long term PPI therapy.

P459 The -308 polymorphism in the TNF α associated with increased risk of duodenal ulcer

A.E. Griffiths, M.M. Walker¹, D. Mantafounis, M.R. Taylor. *¹Departments of Medicine and Histopathology, Imperial College School of Medicine at St Mary's, London, UK*

Background: It is likely that host genetic factors play a role in the outcome of chronic *H. pylori* (*Hp*) infection. One such factor is that encoding the important pro-inflammatory cytokine TNF α . TNF α has been found in significantly greater concentrations in the duodenum and mucosa of *Hp* positive compared with *Hp* negative subjects. The highest levels of TNF α correlate with more severe disease. The TNF α gene is polymorphic at position -308 where a G to A substitution creates an allele (termed TNF2) which is associated with increased TNF α compared with the more common TNF1 allele.

Aim: The aim of this study was to test the hypothesis that the TNF2 allele is more common in duodenal ulcer (DU) patients than controls and that higher levels of TNF α contribute to the pathogenesis of DU.

Methods: A 107 base-pair segment of the TNF α gene was amplified using the polymerase chain reaction. TNF α genotypes were distinguished by the loss of a NcoI restriction site in the TNF α gene.

Results:

Genotype	DU	<i>HP+</i> NUD	<i>HP-</i> 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) resulting from the TNF2 allele was 0.63 (0.24-1.72); For *HP+* v *HP-* OR = 1.1.

Conclusions: The possession of the TNF α -308 polymorphism does not increase the risk of duodenal ulcer, nor the risk of *H. pylori* infection.