ΠΡΟΣΚΕΚΛΗΜΕΝΕΣ ΞΕΝΟΓΛΩΣΣΕΣ ΑΝΑΚΟΙΝΩΣΕΙΣ ΕΛΛΗΝΩΝ ΕΡΕΥΝΗΤΩΝ

● *HELICOBACTER PYLORI* COLONIZATION IN PALATINE SALIVARY GLANDS

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Introduction: Recenty, a case of MALT lymphoma of the parotid gland in Sjögren's syndrome associated with localized *Helicobacter pylori* (*HP*) infection was described. Furthermore, it has found that *HP* can bind to salivary mucins, mainly those that are secreted from the palatine salivary glands because of higher degree of sulfation. However, there is no report whether this microorganism might colonize the palatine salivary glands of the oral cavity.

Aim: To investigate by immunohistochemistry the *HP* status of palatine salivary glands.

Material and Methods: Formalin-fixed, paraffin-embedded tissue blocks of palatine mucosa containing palatine salivary glands from tonsillectomy specimens for tonsillitis (26 cases) were studied. All sections were stained using hematoxylin & eosin for histologic examination and a polyclonal antibody for *HP* using an immunoperoxidase technique following heat induced antigen retrieval.

Results: Microscopically, no pathologic changes were detected in palatine salivary glands. Immunohistochemically, no distinct bacillary structures (helical and/or coccoid forms) were detected neither in the acini nor in the salivary duct system. However, anti-*HP* antibody showed immunoreactivity with acinar system antigens. Specifically, they recognized the cytoplasm of mucous acinar cells in most palatine salivary glands sections.

Conclusions: Antibody reactivity of cells in the acini of the palatine salivary glands may either be due to internalization of *HP* by these cells, or to cross-reactivity of anti-*HP* IgG with epithelial cell epitopes. The too high rates of positive reaction make the second explanation more likely, supporting the theory of an autoimmune reaction induced by *HP* infection.

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● ASPIRIN INFLUENCES MATRILYSIN (MMP-7) EXPRESSION IN GASTRIC EPITHELIAL CELLS

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Introduction: *Helicobacter pylori* (*Hp*) up-regulates matrix metalloproteinase-7 (MMP-7) expression in the gastric epithelium. This expression is more intense in CagA(+) *Hp* strains and it is suggested that this could be related to carcinogenesis.

Aims & Methods: To examine if chronic aspirin intake influences this upregulation. 142 patients (81 men) were enrolled in the study. Group I: 81 patients without aspirin consumption - 52*Hp*(+), 36 CagA(+) and Group II: 61 patients - 36*Hp*(+), 24 CagA(+) taking 100-500 mg/day of aspirin for more than 2 months. Anti-CagA status was assessed by ELISA. Immunohistochemistry was performed on paraffin embedded gastric biopsy samples using a mouse monoclonal anti-MMP-7 antibody (Clone ID2-NeoMarkers). MMP-7 staining intensity in epithelial cells of the proliferative zone of the gastric mucosa was graded blindly (0: none, 1: mild, 2: moderate, 3: very intense - cytoplasmic expression).

Results: Mean age±SD:44.9±12.8. MMP-7 intensity assessment is shown in the table. In each cell the absolute number of patients for the corresponding staining intensity score is shown in an order of 0/1/2/3.

	Antrum	**p**	**Antrum+Asp**	**Fundus**	**p**	**Fundus+Asp**
Hp(-)	19/7/3/0	$	10/3/4/8	25/4/0/0	$	12/12/1/0
p	*		NS	*		NS
Hp(+)CagA(-)	5/6/3/2	NS	2/4/4/2	8/6/1/1	NS	6/3/2/1
p	**		NS	NS		NS
Hp(+)CagA(+)	2/8/10/16	NS	3/1/11/9	13/11/8/4	NS	12/7/3/2

* $p<0.05$ between *Hp*(+) and *Hp*(-) of the same topographic distribution of each Group, ** $p<0.05$ between CagA(+) and CagA(-), $^{\$}p< 0.05$ between patients taking and not taking aspirin

Conclusion: 1) *Hp* infection enhances MMP-7 gastric epithelial expression in a CagA dependent manner. 2) Aspirin increases MMP-7 expression in *Hp*(-) both in antral and fundic mucosa.

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● EFFECT OF *H. PYLORI* ERADICATION ON ESOPHAGEAL ACID EXPOSURE AND OESOPHAGEAL MOTILITY IN PATIENTS WITH GASTROESOPHAGEAL REFLUX DISEASE (GERD)

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Aims & Methods: To investigate the impact of *Helicobacter pylori* eradication on the oesophageal acid exposure and esophageal motility in patients with GERD. Thirty-seven patients (M:25, W12, mean age 42 years, range 19-65) with GERD *Hp*(+) underwent Endoscopy-biopsy, manometry and 24-h pH-metry. All patients received standard eradication treatment, and they were re-examined with pH-metry, manometry, endoscopy-biopsy. The statistical analysis was made with ANOVA and Pearson tests.

Results: Pre-treatment endoscopy showed reflux oesophgitis in 18 patients (Savary Miller grade 1:14, 2:4) and Negative Endoscopy Reflux Disease (NERD) in 19. All patients had antrum predominant gastritis. The mean De Meester score was 47.1. The mean Lower Oesophageal Sphincter Pressure (LOSP) was 13.2 mmHg. The motility of the esophageal body was normal in 33 patients (all NERD and grade 1 oesophagitis). The 4 patients with grade 2 oesophagitis had ineffective motility of oesophageal body and LOSP<7 mmHg. After successful *HP* eradication both gastritis and oesophagitis were healed in all patients. The mean De Meester score after eradication was 14.2, a significant decrease compared to the pretreatment score (p<0.001). After eradication a slight increase in LOSP (14.2 mmHg) was observed (NS). In the 4 patients with grade 2 oesophagitis and ineffective motility the oesophageal corpus activity returned to normal with no change in LOSP.

Conclusions: 1. The eradication of *Hp* infection may lead to a decrease in gastroesophageal reflux, and may have no effect on LOSP and oesophageal body motor activity. 2. The type of gastritis of our patients may have influenced the results.

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● CAGA POSITIVE *HELICOBACTER PYLORI* STRAINS ARE ASSOCIATED WITH INCREASED RISK OF PEPTIC ULCER BLEEDING AMONG NSAID USERS. A MULTICENTER CASE-CONTROL STUDY

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Introduction: The role of *Helicobacter pylori* infection and especially of the cytotoxin associated gene product strain in peptic ulcer bleeding among chronic NSAIDs users remains controversial.

Aims & Methods: A case-control study was carried out of 191 consecutive chronic NSAIDs users admitted because of peptic ulcer bleeding. Peptic ulcer was verified by endoscopy. Controls were 196 chronic NSAIDs users without signs of bleeding of similar age and gender as cases. Multivariate regression analysis was performed for further evaluation of the interaction among *H. pylori*, CagA status and other risks factors.

Results: *H. pylori* infection was present in 121 (63.4%) cases compared with 119 (60.7%) controls (OR=1.14, 95%CI, 0.76-1.72). CagA positive strains were found to be significantly more frequent in cases than controls (65/106 versus 41/99 p=0.008). Current smoking (OR=2.65; 95%CI, 1.14-6.15; p=0.02) CagA status (OR=2.28; 95%CI, 1.24-4.19; p=0.008), dyspepsia (OR=6.89; 95%CI, 1.84-25.76; p=0.004) and past history of peptic ulcer disease (OR=3.15; 95%CI, 1.43-6.92; p=0.004) were associated significantly with increased risk of bleeding peptic ulcer.

Conclusion: Results suggest that CagA - positive *H. pylori* infection is associated with a more than 2-fold increased risk of bleeding peptic ulcer among chronic NSAIDs users.

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● EFFECT OF LACTIC ACID BACTERIA ON GASTRIC INFLAMMATION AND HUMORAL IMMUNE RESPONSE TOWARDS *H. PYLORI* IN THE SSI MOUSE INFECTION MODEL

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We have evaluated the effect of individual lactobacilli (LAB) belonging to different species (L. johnsonii La1, L. amylovorus DCE 471 and L. acidophilus IBB 801) for their potential ability to affect *H. pylori (Hp)* colonization and the associated gastritis in C57BL/6 mice infected by the *Hp* SS1 strain.

We administered LAB continuously through the animal drinking water and found that a daily dose of 10^8 cfu/animal did not eradicate or reduce *Hp* colonization at 6 and 12 weeks postinfection as assessed by viable cultures. However, there was a significant reduction in chronic inflammation (updated Sydney system) at 6 weeks ($p=0.003$, Wilcoxon rank sum test) and 12 weeks ($p=0.038$) in the La1-treated animals compared to *Hp*-infected controls, but only at 6 weeks postinfection in the two other LAB-treated groups (DCE 471 $p=0.047$ and IBB 801 $p=0.014$). Moreover, in all the Lab-treated animals, we observed a striking attenuation of chronic active gastritis, as a significantly reduced number of neutrophils invaded the lamina propria, throughout the whole 12-week period (La1 $p=0.003$, DCE 471 $p=0.011$ and IBB 801 $p=0.011$. Anti-*Hp* IgG antibodies in the serum were depressed significantly only in the case of La1-treated *Hp*-infected animals ($p<0.05$, Students t-test), although a tendency for reduction in antibody titers was also observed in the other two LAB-treated animal groups. These results point out that factors either secreted by a expressed on live LAB can influence both the anti-*Hp* humoral response and the neutrophilic infiltration in the lamina propria, a hallmark of *H. pylori* infection.

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● IS *HELICOBACTER PYLORI* ERADICATION THE TREATMENT OF CHOICE FOR PEPTIC ULCER DISEASE IN PATIENTS WITH LIVER CIRRHOSIS?

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Introduction: *Helicobacter pylori* eradication is the standard treatment to cure peptic ulcer disease. The role of *H. pylori* in peptic ulcer in patients with liver cirrhosis has not been studied.

Aims & Methods: To investigate the natural course of peptic ulcer disease in cirrhotics, after healing with either *H. pylori* eradication or omeprazole (OME) therapy.

Consecutive patients with liver cirrhosis and peptic ulcer were studied. Patients had never received *H. pylori* eradication, and had not used NSAIDs or proton pump inhibitors the past two months before entering the study. All underwent endoscopy at entry with *H. pylori* testing (histology). *Helicobacter pylori* +ve patients received one week triple eradication therapy followed by 20 mg/d OME for three weeks, and *H. pylori* -ve patients were treated with 20 mg/d OME for four weeks. Follow up endoscopies were performed four weeks after completion of therapy and at 12 and 24 months or when symptoms recurred. Those patients with ulcer recurrence were treated with 20 mg/d OME maintenance therapy.

Results: Out of 28 patients (24 male, 4 female, aged 61, 48-71 years, 18 alcoholics, 10 non-alcoholics, Child: A=13, B=10, C=5), 18 had a duodenal and 10 a gastric ulcer. In 17/18 *H. pylori* +ve patients *H. pylori* was successfully eradicated. All ulcers were healed at 4 weeks after eradication or OME therapy. During the 24 months follow up period ulcer relapsed in 17 (8/18 *H. pylori* +ve, 9/10 *H. pylori* -ve, p=0.041) out of the 28 patients. There was no ulcer relapse while on OME maintenance therapy. Multivariate regression analysis showed that a negative *H. pylori* status at entry to the study (x2=11.4, p<0.001), older age (x2=7.7, p=0.005) and the severity of cirrhosis (Child) (x2=4.8, p<0.03) were significantly related to ulcer recurrence. Sex, etiology of cirrhosis and type of ulcer were rejected by the model.

Conclusion: *Helicobacter pylori* eradication does not protect *H. pylori* +ve cirrhotics from ulcer recurrence. Therefore, omeprazole maintenance treatment is mandatory, irrespectively of *H. pylori* status.

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