contamination. The frequency of *H. pylori* appearance in Caucasians and Mongols was about 95.2–97.8%, prevalence of ulcerative disease: 8.9% in Caucasians and 4.5% in Mongols. Ethnical features were revealed in diffusion of *H. pylori* strains: in Caucasian population ulcerative disease was associated with *s1* and *s2* subtypes VacA, in Mongol-CagA. Increased proliferation of epithelium cells in antral part of a stomach was shown in Mongoloid patients comparing to Caucasians. Immunophenotype study of cellular infiltrate of lamina propria also allowed to reveal ethnic differentiation: more sufficient activation of T-cell and humoral immune responses in Mongols compared to Caucasians. Apoptosis rate was sufficiently higher in patients with ulcerative disease comparing to healthy donors in both ethnical groups; though proliferation/apoptosis ratio was statistically higher in sick and healthy Mongols compared to appropriate groups of Caucasians.

Thus, that two populations of people from different origin infected with *H. pylori* and living on the same territory are characterized with different pathology rates and different types of infect-macroorganism interactions.

### Abstract no.: P053 Interactions between *H. pylori* and Host Susceptibility Factors among Iranian GI Patients

M. Mohammadi,* Y. Talebkhan,* A. Nahajvijo,*1 N. Mohajerani,* A. Oghalaie,* E. Smaiel,* S. Saberi Kashani,* E. M. Hosseini1 and M. A. Mohagheghi1

1Biotechnology Research Center, Pasteur Institute of Iran, Tehran, Iran; 2Cancer Research Center, Medical Sciences/University, Tehran, Iran; 3Azad University of Karaj, Tehran, Iran; 4Endoscopy Unit, Amiralam Hospital, Medical Sciences/University of Tehran, Tehran, Iran; 5Cancer Research Center, Medical Sciences/University of Tehran, Tehran, Iran

**Introduction.** *Helicobacter pylori* is reported as an etiological agent in gastric cancer (GC) development. This infection is more prevalent in developing countries including Iran. Virulence factors such as CagA and VacA are implicated in the pathogenesis of *H. pylori*. This study aims to explore the etiological role of *H. pylori* in GC development.

**Methods.** *H. pylori* status was determined by culture, histology, and RUT. Three hundred and thirty-two participants referring for endoscopy and 144 GC patients undergoing gastric resection were included in this study. Immunoblotting, ELISA and specific PCRs were performed on collected samples.

**Results.** *H. pylori* infection was detected in 72.6% of all subjects composed of 71.7% of dyspeptic patients and 75% of GC cases. Seroreactivity toward *H. pylori* was detected in 82.6% of non-GC and 86.5% of GC groups. vacA s1m1 and s1m2 genotypes were significantly prevalent in GC and non GC patients, respectively. Serological data revealed that unlike cagA genotyping, which revealed no association with either group, the presence of anti-CagA antibody can be a predictive marker in GC development. Through host genotyping it was found that L1/L2 genotype of *il-1* has the same frequency among *H. pylori*-infected and noninfected subjects. In both *H. pylori*-positive and -negative groups the mean and standard deviation of pepsinogen I and II levels were approximately the same.

**Conclusion.** This study showed that although the majority of the studied subjects are infected with *H. pylori* and other etiologic factors seem to play roles in GC development in the Iranian population.

### Abstract no.: P054 Long-Term Follow-up of Patients with Low Bacterial Density of *H. pylori* after Eradication Therapy: Hypothesized Spontaneous Eradication

S. I. Pimanov, E. V. Makarenko and M. E. Maveenko

State Medical University, Vitebsk, Belarus

**Objectives.** The objectives of this study were to assess gastric histopathology changes and to detect the presence of *Helicobacter pylori* infection in 3–5 years after its failed eradication treatment. The study was dedicated to patients with low bacterial density after eradication treatment.

**Methods and Patients.** Thirty patients with *H. pylori*-associated duodenal ulcer before, 2 months, and 3–5 years after eradication were studied. Updated Sydney system for histopathological examination was used. Morphologic results were compared in 2 months and 3–5 years after eradication treatment. In 2 months after eradication these patients had negative rapid urease test (RUT), decreased bacterial density according to morphological score, which constituted nearly +1, positive result of *H. pylori* in biopsy specimens by PCR method.

**Results.** In 3–5 years after eradication 7 out of 30 patients had positive RUT, PCR, and high bacterial density according to morphological score. Inflammation, activity, atrophy, and lymphoid follicles in the antral mucosa did not change in these patients. Twenty-three patients had negative results according to all used methods of *H. pylori* detection. In this group with successful eradication inflammation, activity, atrophy, and lymphoid follicles in the antral mucosa fell from 1.63 ± 0.50 to 0.96 ± 0.30 (*p < .001*); 1.13 ± 0.38 to 0.46 ± 0.33 (*p < .001*); 0.86 ± 0.67 to 0.89 ± 0.60 (*p = 1.0*); 0.65 ± 1.0 to 0.13 ± 0.46 (*p = .114*), respectively.

**Conclusion.** Most patients with low bacterial density in 3–5 years after eradication treatment are free of *H. pylori* infection and show the improvement of gastric morphology.

### Abstract no.: P055 Water-Exposed *H. pylori* Presents Decreased Virulence Properties

N. M. Guimarães,* N. Azevedo,* M. J. Vieira* and C. Figueiredo†

*Center of Biological Engineering, Braga, Portugal; †IPATIMUP, Porto, Portugal; ‡Medical Faculty, University of Porto, Porto, Portugal

*Helicobacter pylori* transmission has been associated in epidemiological studies with water. *H. pylori* has been identified in this environment using molecular techniques. As water may be an environmental reservoir for *H. pylori*, and because there is lack of information regarding the capacity of water-exposed bacteria to induce a response in host cells, we assessed the cultivability of
water-exposed *H. pylori* and determined whether these bacteria retain the ability to adhere to and to induce inflammation.

We used *H. pylori* strain 26,695 and AGS cell line. Bacteria were grown in TSA with 5% sheep blood and incubated for 48 hours at 37 °C under a microaerophilic atmosphere. After that, *H. pylori* was exposed to water at 25 °C, in aerobic conditions for different time periods. *H. pylori* cultivability was determined by standard plating methods. Adhesion to and Interleukin-8 production by AGS cells were assessed by ELISA, using an anti-*H. pylori* antibody and a commercially available kit, respectively.

Our results showed that, after 24 hours water exposure, *H. pylori* was no longer cultivable. Water exposure of *H. pylori* led to a significant decrease of its ability to adhere to AGS cells. Also, significantly lower IL-8 secretion was observed in AGS cells cultured with water-exposed *H. pylori* than with unexposed bacteria. Altogether, these results suggest that after being exposed to water, planktonic *H. pylori* presents decreased virulence properties. As such, additional mechanisms of protection in water, such as inclusion in biofilms, might be needed for the maintenance of the infectious ability by this bacterium.

Acknowledgements: Fundação Ciência Tecnologia (SFRH/BD/24579/2005).

---

**Abstract no.: P056**

**Association of MALT Lymphoma with *H. pylori* Infection**

A. Filipovic,* J. Gligorijevic† and T. Backovic‡

*Ministry of Health, Belgrade, Serbia; †Institute of Pathology, Nis, Serbia

MALT lymphoma, at an early stage, is confined to the gastric mucosa and its growth depends critically on *Helicobacter pylori*-mediated immunological stimulation. Sequential serological studies and retrospective studies of archival gastric biopsy material have shown that the infection by *H. pylori* precedes the development of lymphoma. Subsequent studies have suggested an association of MALT lymphoma with *H. pylori* infection in 62%. Host immune gene polymorphisms and gastric acid secretion produces ability of *H. pylori* to colonize gastric niche. The host immune response to *H. pylori* induces and sustains an actively proliferating B-cell population. Protracted remission may be induced by antibiotic therapy for *H. pylori*. 

**Abstract no.: P057**

**H. pylori against Host Immune System**

T. Backovic,* A. Filipovic† and A. Backovic‡

†Institute of Pathology, Nis, Serbia; †Ministry of Health, Belgrade, Serbia; ‡Institute of Molecular Biology, Pisa, Italy

Infection by *Helicobacter pylori* causes gastritis, peptic ulcers, and other serious outcomes such as gastric cancer. Disease outcome is the result of the interplay between the host and bacterial virulence factor (citotoxin-associated antigen CagA), which seem to modulate the host immune system and is implicated in pathogenesis of the carcinoma of stomach. Gastric mucosal biopsies from *H. pylori*-infected humans showed an increased concentration of various types of leukocytes. IgA antibodies were detectable in gastric juice. This study has shown that the infection by *H. pylori* reflects the host immune response, which could be used in a way of developing new anti *H. pylori* therapy.

---

**P06 Pathology and Pathophysiology**

**Abstract no.: P058**

**Influence of *H. pylori* on the Progression of Gastric Mucosa Atrophy in Patients with Portal Gastropathy**

L. B. Lazebnik,* S. G. Khovermi,* I. Koviazina* and A. G. Zhukov†

*Central Research Institute of Gastroenterology, Moscow, Russia; †Municipal Clinical Hospital #62, Moscow, Russia

**Introduction.** Portal gastropathy (PG) is a common finding in patients with portal hypertension caused by liver cirrhosis, but influence of *Helicobacter pylori* on pathogenesis and natural history of PG is not cleared up.

The goal of the study was to evaluate histological changes in gastric mucosa and to establish role of *H. pylori* in development of PG.

**Methods.** Gastric biopsy was obtained from 54 patients. The diagnosis of liver cirrhosis was confirmed in 39, and chronic hepatitis in 15 patients. *H. pylori* was revealed in 37 patients. Four groups of the patients were discharged, according to severity of portal hypertension. The evaluation of chronic gastritis grade and stage of gastric mucosal atrophy was carried out according new scale. Proliferative activity in gastric mucosa was evaluated by immunostain for Ki-67.

**Results.** Eighty-five percent of patients from I–II groups were *H. pylori* positive and had chronic gastritis G-II and G-III with stage of PG positive. Local discirculation and concomitant inflammation in gastric mucosa at *H. pylori*-positive patients exacerbates severity of atrophy.

**Conclusion.** *H. pylori* promotes faster development of atrophic processes in the gastric mucosa at early stages of PG via