presence of *H. pylori* crude antigen in the stool sample of infected guinea pigs. *E.coli* as negative control stained red by Evans blue dye. Occurrence of *H. pylori* crude antigen was also confirmed by ELISA.
Discussion: In recent years Mongolian gerbil has been introduced as an appropriate model for research on *H. pylori* colonization. Positive results of PCR, IFA, and stool antigen tests indicate that guinea pig could be a useful substitute for Mongolian gerbil. This study is ongoing and the methods mentioned above are going to be recruited for assessment of the efficiency of new synthetic compounds in treatment of *H. pylori*-infected guinea pigs.

P05 Other Helicobacters, Hepatobiliary Diseases, Esophageal, and Extradigestive Diseases

Abstract no.: P05.01
Dietary Factors and *H. pylori* Infection

M. Jarosz, E. Rychlik and M. Siuba
National Food and Nutrition Institute, Warsaw, Poland

Helicobacter pylori infection is very common in Poland, which is found is 85–95% of adult population. It may be at least partly related to improper lifestyle, especially diet.

The aim of the study was to examine if some dietary factors contribute to *H. pylori* infection.

Studied patients were referred for endoscopic examination of the upper digestive tract in 2002–2007 to explain the cause of dyspeptic disorders. In some patients *H. pylori* infection was diagnosed for the first time, in others reinfection occurred after successful treatment in the past. Patients who have not been infected or reinfected were included into the control group. The respondents were interviewed retrospectively on their dietary habits.

A lower frequency of fermented dairy products, vegetables, and fruit consumption was noted among persons with *H. pylori* infection as compared to the control group. In the examined group 43–47% declared to eat fermented dairy products frequently (at least five times a week) while in the control group 95–96%; in the case of vegetables consumption these percentages were 74% and 77–87% and in the case of fruit consumption 51–58% and 70–76%, respectively.

Obtained results indicate that high consumption of fermented dairy products containing probiotic bacteria, mainly *Lactobacillus*, and vegetables and fruit – source of antioxidants such as vitamin C, may decrease the risk of *H. pylori* infection.

Abstract no.: P05.02
Helicobacter felis is the Major Gastric Helicobacter Species in Dogs

M. Contreras,[*] R. Polanco,[†] V. Salazar,[*] N. Reyes,[*] M. A. García-Amado[*] and F. Michelangeli[*]
[*]Instituto Venezolano de Investigaciones Científicas (IVIC), Caracas, Venezuela; [†]Universidad Nacional Experimental "Francisco de Miranda", Falcón, Venezuela

Background: *Helicobacter* is frequently demonstrated in gastric biopsy specimens obtained from dogs; however, their role as potential pathogen in gastritis has not been clearly established.
Aim: To detect *Helicobacter* species in the gastric mucosa of dogs and to correlate the presence of *Helicobacter* infection with gastritis.
Animals: Biopsy samples were taken during gastric endoscopy from the fundus of 20 dogs with or without signs of gastrointestinal disease.
Methods: Histopathologic techniques, Helicobacteraceae family- and *Helicobacter* genus-specific polymerase chain reaction (PCR) assays, and fluorescence in situ hybridization (FISH) analysis were used to detect *Helicobacter* spp. The identity of the species was obtained by amplifying a 764-bp 16S rRNA gene sequence specific to Helicobacteraceae.
Results: Nineteen dogs showed mild to severe gastritis in the fundus, and only one had a healthy gastric mucosa. *Helicobacter* spp. DNA was detected in 18 of 19 dogs with gastritis and only one with a normal gastric mucosa. The sequences of DNA amplicons (600–711 bp) shared 99–100% identity with the 16S rRNA genes of *H. felis*, *H. salomonis*, and *Helicobacter* sp. in 79%, 10.5%, and 10.5% of the dogs, respectively. Using FISH, the presence of *Helicobacter* species was evidenced in 19 animals (100% coincidence).
Conclusions: Venezuelan pet dogs are frequently colonized by *H. felis* without a significant correlation between infection and degree of gastritis, suggesting the possibility that dogs may act as source of non-*H. pylori Helicobacter* spp. infection for humans.

Abstract no.: P05.03
Helicobacter spp. DNA in Mucosa of Swedish Patients with Cholecystitis

P. H. Karagin,[*] U. Stenram,[†] H. Nilsson,[*] T. Wadström[*] and A. Ljungh[*]
[*]Medical Microbiology and Immunology, Lund, Sweden; [†]Pathology and Bacteriology, Lund, Sweden

Background: *Helicobacter* DNA in cholecystitis mucosa has been reported with prevalence from 2% to 83% in different countries. Several authors claim that *Helicobacter* is especially found in areas of gastric metaplasia. We examined a series of patients from Southern Sweden.
Methods: Paraffin-embedded samples (5 mg tissue) from the mucosa of 55 patients with cholecystitis were studied by *Helicobacter* DNA-specific PCR assay and sequence analysis.
Results: *Helicobacter* DNA was found in nine of 36 so far examined patients. Sequence analysis displayed close similarity with *H. pullorum* in all cases and lower similarity with *H. canadensis*. There was very little gastric metaplasia.

Discussion: The prevalence was higher than in a recent German study but lower than in studies from a few non-Western countries. *H. pullorum* has been found by one previous author, *H. canadensis* by none. More cases are being examined. The paraffin-embedded specimens are composed of epithelial as well as stromal elements, and *Helicobacter* may be present only in the epithelium. The presence of non-*H. pylori* species may not be dependent on gastric metaplasia.
Conclusion: *Helicobacter* DNA in a Swedish patient material of cholecystitis mucosa was more prevalent than in Germany but lower than in several non-Western countries. The sampling strategy may have great influence on the results.

Abstract no.: P05.04
Eradication of *H. pylori* Infection in Patients with Alcohol-Induced Pancreatitis

S. Naumovski-Mihalic,[*], M. Katicic,[*] V. Maricic,[†] G. Cavric,[*] V. Colic-Cvrlje,[*] B. Sabaric,[*] M. Prskalo,[*] B. Skurla,[*] T. Filipec-Kanizaj[*] and A. Mrzljak[*]
[*]University Hospital Merkur, Zagreb, Croatia; [†]Pharmaceutical company Pliva, Zagreb, Croatia

Aim: The aim of this study was to evaluate efficacy of *Helicobacter pylori* eradication in patients with alcohol-induced pancreatitis and compared with nonalcoholic patients.
Methods: The patients with acute pancreatitis were divided into two groups. Group A: 35 patients (26 male/9 female), mean age 44 years, with alcohol-induced pancreatitis, and group B: 35 patients (20 male/15 female), mean age 41 years, with nonalcohol-induced pancreatitis. All patients had upper gastrointestinal endoscopy. *H. pylori* infection was confirmed by gastric hystology.The patient from group A drank four to eight glasses of wine or 6–8.5 L of beer per week. A triple therapy with amoxycillin (1 g two times a day), medazol (400 mg two times a day), pantoprazol (40 mg two times a day) as first cycle, or amoxycillin(1 g two times a day), claritromycin (500 mg two times a day), and lansoprazole (30 mg two times a day) as second cycle, was given to both groups for 10 days. The cure was defined as the absence of *H. pylori* infection 6 weeks after therapy.
Results: A higher *H. pylori* infection was found in group B, in nonalcoholic patients 68% versus 42% in alcoholic patients ($p > .001$). In: alcoholic patients in group A eradication of *H. pylori* infection was 82% versus 60% in nonalcoholic patients ($p > .001$). Higher wine and beer consumption were associated with an additional reduction in the risk of infection. The rate of infection decreased with time of duration of alcohol abuse.
Conclusion: *H. pylori* infection was lower in group of alcoholic patients than in control group. The eradication rate was significantly higher in patients with alcohol abuse than in control group. This study suggests that moderate alcohol consumption may facilitate spontaneous elimination of *H. pylori* infection among adults.

Abstract no.: P05.05
Presence of *H. pylori* Infection in Cirrhosis Patients is Related to Slower Recovery from Hepatic Encephalopathy

T. Glisic, A. Sokic-Milutinovic, M. Glisic and M. Krstic
Clinic for gastroenterology and Hepatology, Belgrade, Serbia

Background/Aims: Hepatic encephalopathy is a frequent complication of liver cirrhosis. It has been suggested that *Helicobacter pylori* contributes to hyperammonemia in cirrhosis. We aimed to investigate the relationship between *H. pylori* infection, blood ammonia concentration, and hepatic encephalopathy in cirrhosis patients.
Methods: Thirty-nine patients treated in the intensive care unit in clinical center of Serbia with decompensated liver cirrhosis entered the study (35 males, mean age 58 ± 8). They were divided into two groups based on the presence of *H. pylori* infection. Twenty-six patients were *H. pylori* positive and 13 *H. pylori* negative. Patients were evaluated for demographic and clinical data, liver impairment, blood ammonia concentration, and HE. Presence of *H. pylori* infection was determined using histology when antral endoscopic biopsies were taken or serology in those who did not undergo biopsy.
Results: HE lasted longer in *H. pylori*-positive patients (median 6 days, range 1–20 days) than in *H. pylori*-negative patients (median 3 days, range 1–11 days) $p < .05$. Higher levels of blood ammonia were also found in *H. pylori*-positive patients (*H. pylori* positive 63.5 ± 48, *H. pylori* negative 43 ± 27 μmol/L, $p > .05$) but difference was not statistically significant. No difference between groups was observed in clinical and demographic data, etiology of cirrhosis, presence of ascites, kidney function impairment, presence of gastrointestinal bleeding, or final outcome.
Conclusion: Presence of *H. pylori* infection is related to longer duration of HE, thus *H. pylori* eradication may be helpful for treatment and prevention of HE.

Abstract no.: P05.06
Detection of *H. pylori* in the Hepatobiliary System of Patients with Biliary Tract Diseases

G. Isaeva,[*] E. Abuzarova[†] and N. Frans[†]
[*]Kazan State Medical University, Kazan, Russia; [†]Kazan Institute of Biochemistry and Biophysics, Kazan, Russia

Aim: Despite several recent reports on the detection of *Helicobacter pylori* DNA in human bile, there are still uncertainties concerning the correlation of these findings with biliary tract and liver diseases.
Material and Methods: Using polymerase chain reaction (PCR), we detected the presence of *H. pylori* in bile samples, gallbladder, and liver biopsy specimens from 102 persons, of which 72 adults with hepatobiliary diseases (HBD) and liver cirrhosis. Of the 102 patients, representing the control group, 30 were without biliary diseases. Bile samples were obtained by duodenal intubations; gallbladder mucus samples were obtained by resection of the gallbladder and liver. Among the 72 patients, 42 had noncalculous cholecystitis, 19 had calculous cholecystitis and 11 with liver cirrhosis. The detection of *H. pylori* DNA was performed by polymerase chain reaction using *H. pylori*-specific primers for *ureC* according to the manufacturer's recommendation (Lytech, Russia).

Results: *H. pylori* was found in 31 samples (liver, gallbladder, bile duct, and bile) of patients with liver cirrhosis and chronic cholecystitis. In accordance with different hepatobiliary pathology, 21 (50%) samples of 42 with noncalculous cholecystitis, 8 (72.7%) samples of 11 with liver cirrhosis, and only in 2 (10.5%) of the 19 samples with calculous cholecystitis were positive. In the control group none of the samples showed the presence of *H. pylori*.
Conclusion: The presence of *H. pylori* in hepatobiliary system may tell about the influence of the bacteria in the development of hepatobiliary diseases.

Abstract no.: P05.07
The Clinical Result of *H. pylori* Eradication in Patients with *H. pylori*-positive Idiopathic Thrombocytopenic Purpura of St. Mary Hospital

G. Kim, J. Park, Y. Kim, J. Kim and S. Park
St Mary's Hospital of Catholic University, Seoul, South Korea

Background: *Helicobacter pylori* is the cause of gastric and multiple extragastric diseases. A beneficial effect of *H. pylori* eradication in patients with *H. pylori*-positive idiopathic thrombocytopenic purpura (ITP) has been reported by several investigators; however, its efficacy varies between countries. The response rate of *H. pylori* eradication in *H. pylori*-positive chronic and acute ITP patients at St. Mary Hospital was investigated
Method: Between September 2005 and April 2007, a total of 18 patients diagnosed with ITP were included in the study. The *H. pylori* infection was assessed by urea breath test. *H. pylori* eradication was performed on *H. pylori*-positive ITP patients with the amoxicillin, clarithromycin, and proton pump inhibitor regimen for 7 days. We investigated the efficacy of *H. pylori* eradication on platelet recovery in patients with *H. pylori*-positive ITP.
Result: Eighteen patients with ITP were evaluated, including 8 males with 10 females. Mean age of patients is 44.9 ± 17 years and 12 (63.2%) were positive for *H. pylori*. Six are outpatients and others are in admission state. Eradication was performed and three (25%) had a significant increase in platelet counts after treatment and all of them were located in group with successful *H. pylori* eradication. Platelet count of responders was increased from 10,000 ± 8660/μL to 111,694 ± 40,628/μL, and mean follow-up time was 24.3 ± 2.5 months.
Discussion: Our analysis shows the efficacy of *H. pylori* eradication in patients with *H. pylori*-positive ITP at St. Mary Hospital. There was no significant relationship between platelet response and clinical characteristics of *H. pylori*-positive patients.

Abstract no.: P05.08
H. pylori and Iron-Deficiency Anemia: A Meta-analysis of Case–Control Studies

J. P. Gisbert, O. P. Nyssen and A. G. McNicholl
La Princesa University Hospital and CIBEREHD, Madrid, Spain

Background: Recently, there has been a growing body of evidence suggesting a relationship between *Helicobacter pylori* gastritis and hypoferritinemia or iron-deficiency anemia (IDA).
Aim: To systematically review the role of *H. pylori* infection in hypoferritinemia and IDA, and to perform a meta-analysis of case–control studies.
Methods: *Selection of studies*: Case–control studies comparing (a) the prevalence of *H. pylori* infection in patients with and without IDA/hypoferritinemia, and (b) the prevalence of IDA/hypoferritinemia in patients with and without *H. pylori* infection. *Search strategy*: electronic and manual bibliographical searches. *Data synthesis*: Meta-analysis combining the odds ratios (OR).
Results: (a) Eight studies compared the prevalence of *H. pylori* infection in patients with (637 patients) and without (2305 patients) IDA, showing a higher prevalence of *H. pylori* infection in anemic patients [35% vs 22%; OR = 1.7; 95% confidence interval (CI) = 1.1–2.5]. Only one study compared the prevalence of infection in patients with and without hypoferritinemia (29% vs 19%; $p < .05$). (b) Seven studies compared the prevalence of IDA in patients with (1190 patients) and without (678 patients) *H. pylori* infection, showing a higher prevalence of IDA in *H. pylori*-positive patients (17% vs 14%; OR = 2.3; 95%CI = 1.2–4.3). Finally, five studies compared the prevalence of hypoferritinemia in patients with (297 patients) and without (129 patients) *H. pylori* infection, showing a higher prevalence of hypoferritinemia in *H. pylori*-positive patients (33% vs 13%; OR = 1.7; 95%CI = 1.1–2.7). Results were heterogeneous for all comparisons.
Conclusion: Epidemiologic studies suggest an association between *H. pylori* infection and lower iron stores or IDA. However, these data should be interpreted with caution due to marked heterogeneity among studies.

Abstract no.: P05.09
Effect of *H. pylori* Eradication on Iron-Deficiency Anemia: A Meta-analysis of Randomized Clinical Trials

O. P. Nyssen, A. G. McNicholl and J. P. Gisbert
La Princesa University Hospital and CIBEREHD, Madrid, Spain

Background: Reversal of iron-deficiency anemia (IDA) after successful cure of *Helicobacter pylori* infection has recently been observed in several randomized clinical trials.
Aim: To systematically review the effect of *H. pylori* eradication on hypoferritinemia and IDA, and to perform a meta-analysis of randomized clinical trials comparing *H. pylori* eradication treatment and iron administration.
Methods: *Selection of studies*: Randomized controlled trials comparing *H. pylori* eradication treatment (plus iron) versus iron administration alone for the treatment of IDA/hypoferritinemia. *Search strategy*: electronic and manual bibliographical searches. *Data synthesis*: Meta-analysis combining results of hemoglobin and ferritin increase and calculating standard mean difference (SMD).
Results: Five studies compared the increase in hemoglobin levels achieved with *H. pylori* eradication (plus iron) treatment and with iron administration alone in patients with IDA, showing a higher efficacy in the eradication group [SMD = 2.9; 95% confidence interval (CI) = 0.5–5.3]; however, two studies could not demonstrate the beneficial effect of antibiotic treatment. On the other hand, four studies compared the increase in serum ferritin concentrations after *H. pylori* eradication treatment and after iron administration in patients with IDA, showing, again, a higher efficacy in the group receiving eradication therapy

(SMD = 6.6; 95%CI = 2.7–10.4). Results were markedly heterogeneous for all comparisons.
Conclusion: Some studies have demonstrated reversal of IDA after successful cure of *H. pylori* infection, and the meta-analysis of randomized controlled trials showed that *H. pylori* eradication (plus iron) is more effective than iron administration alone for the treatment of lower iron stores or IDA. However, these data should be interpreted with extreme caution due to marked heterogeneity among studies.

Abstract no.: P05.10
Culture of *H. pylori* from Heterotopic Gastric Mucosa

N. Wueppenhorst,[*] B. Viebahn,[†] B. Hobmaier[*] and M. Kist[*]
[*]Institute of Medical Microbiology and Hygiene, National Reference Centre for Helicobacter, University Hospital Freiburg, Freiburg, Germany; [†]Gastroenterologist, Bochum, Germany

In April 2008 a 61-year-old male patient presented with tickle of the throat, globus pharyngeus, and heartburn. Endoscopy of the esophagus and stomach was performed, where subpharyngeal localized heterotopic gastric mucosa (HGM) and a chronic gastritis were diagnosed. The histologic examination was suspicious for *H. pylori* infection of the stomach and the HGM. After consulting the NRC, we tried to culture *H. pylori* from biopsies of the stomach and the HGM. *H. pylori,* susceptible to clarithromycin, levofloxacin, tetracycline, rifampicin, metronidazole, and amoxicillin with identical minimum inhibitory concentrations, was cultured from both sites. Molecular typing revealed a cagA-positive strain with a vacA S1a/m1 genotype. The patient was treated with a French triple therapy (proton pump inhibitor + amoxicillin + clarithromycin) for 10 days. All performed tests for eradication control (endoscopy with samples for rapid urease test, histology, culture and real-time PCR and stool antigen ELISA) were negative. Furthermore, the patient reported of clinical improvement. In conclusion, *H. pylori* colonization of patches of heterotopic gastric mucosa has been described in the literature. For diagnosis histologic examination is used. To our knowledge this is the first case report of positive *H. pylori* culture from HGM.

Abstract no.: P05.11
Carditis can Partially Regress After *H. pylori* Eradication but not Proton Pump Inhibitor Treatment

P. Tsibouris, K. Goula, C. Kalantzis, P. Apostolopoulos, C. Tsironis, G. Alexandrakis, A. Karameris and N. Kalantzis
NIMTS General, Athens, Greece

Aim: To define the effect of *Helicobacter pylori* eradication and antireflux treatment in carditis and intestinal metaplasia of the gastric cardia.
Patients-methods: Two hundred and forty patients with gastroesophageal reflux disease (GERD) (mean age 59 ± 15 years, 145 male) and 240 controls without GERD (mean age 58 ± 17 years, 138 male) after gastroscopy with biopsies were started on omeprazole 20 mg twice daily for 1 year plus a 10-day *H. pylori* eradication regimen if *H. pylori* positive. Finishing treatment, we repeated endoscopy with biopsies, on omeprazole and performed ^{13}C-urea breath test, off omeprazole if *H. pylori* positive. The Sydney classification was used for carditis/intestinal metaplasia.
Results: Cardiac mucosa was identified in 180 (75%) controls, 220 (92%) refluxers ($p < .001$); carditis in 102 (43%) controls, 152 (63%) refluxers ($p < .001$) [*H. pylori* positive: 50 (21%) controls, 76 (32%) refluxers ($p = .007$); *H. pylori* negative: 52 (22%) controls, 76 (32%) refluxers ($p = .01$)]. Intestinal metaplasia of the cardia was found in 45 (19%) controls, 84 (35%) refluxers ($p < .001$). Forty-one controls with carditis and 61 refluxers successfully eradicated *H. pylori*. Of them 29 controls, 31 refluxers present no carditis in the follow-up endoscopy. After 1 year, 73 (30%) controls, 123 (51%) refluxers presented carditis ($p < .001$). During follow up there was no change in the severity of intestinal metaplasia, carditis regressed in 71% (n = 29) of *H. pylori*-positive controls who eradicated *H. pylori*, 51% (n = 31) refluxers; it increased by 0.5 ± 0.1 grades in controls, 0.3 ± 0.1 in refluxers who persisted *H. pylori*, while remained rather unchanged in *H. pylori*-negative patients and controls. Five refluxers developed dysplasia.
Conclusions: 1, Both carditis and intestinal metaplasia of the gastric cardia are more frequent in GERD patients. 2, Carditis but not intestinal metaplasia can regress less frequently in *H. pylori*-positive patients than controls after *H. pylori* eradication. 3, High dose omeprazole treatment has little effect on carditis.

Abstract no.: P05.12
H. pylori Colonization of the Adenotonsillar Tissue: Fact or Fiction?

S. Vilarinho,[*] R. M. Ferreira,[†‡] N. Guimarães,[†§] B. Gomes,[†] W. Xiaogang,[¶] F. Oliveira,[*] D. Freitas,[*] B. Rodrigues,[*] E. Atraca,[*] G. Pereira,[*] N. Marçal,[*] F. Carneiro,[†‡¶] A. Marques,[**] T. Godinho[*] and C. Figueiredo[†‡]
[*]Serviço de Otorrinolaringologia, Hospital de S. Marcos, Braga, Portugal; [†]IPATIMUP – Institute of Molecular Pathology and Immunology of the University of Porto, Porto, Portugal; [‡]Faculty of Medicine University of Porto, Porto, Portugal; [§]IBB – Institute for Biotechnology and Bioengineering, Centre of Biological Engineering, Universidade do Minho, Braga, Portugal; [¶]Serviço de Anatomia Patológica, Hospital de S. João, Porto, Portugal; [**]Serviço de Imuno-Hemoterapia, Hospital de S. Marcos, Braga, Portugal

Gastric infection with *Helicobacter pylori* is the most common chronic infection worldwide. One of the routes of transmission of the infection is the oral route. Molecular techniques have allowed the detection of *H. pylori* DNA in samples of the oral cavity, although culture of *H. pylori* from these types of samples has been sporadic. Studies have tried to demonstrate the presence of *H. pylori* in adenotonsillar tissue, with contradictory results. Our aim was to clarify whether the adenotonsillar tissue may constitute an extragastric reservoir for *H. pylori*.

Sixty-two patients proposed for adenoidectomy or tonsillectomy were enrolled. A total of 101 samples, 55 adenoid and 46 tonsils, were obtained. Patients were characterized for the presence of anti-*H. pylori* antibodies by serology. On each surgical sample rapid urease test, immunohistochemistry, PCR-DEIA directed to the *vacA* gene of *H. pylori*, and FISH with a peptide nucleic acid probe for *H. pylori* were performed.

In the study population, 33% of the individuals had anti-*H. pylori* antibodies. Rapid urease test was positive on samples of three patients, all with positive serology. Immunohistochemistry was positive on two patients, all with negative serology. All positive cases by rapid urease test or immunohistochemistry were negative by FISH. PCR-DEIA directly in adenotonsillar tissue was negative in all samples.

In conclusion, the adenotonsillar tissue does not constitute an extragastric permanent reservoir for *H. pylori* infection, at least in this population from the North of Portugal.

Abstract no.: P05.13
Does Eradication of *H. pylori* Infection Delay the Development Lung Cancer?

T. U. Kosunen,[*] E. Pukkala,[†‡] S. Sarna,[§] K. Seppälä,[¶] A. Aromaa,[**] P. Knekt[**] and H. Rautelin[*††‡‡]

[*]Department of Bacteriology and Immunology, Haartman Institute, University of Helsinki, Helsinki, Finland; [†]Finnish Cancer Registry, Helsinki, Finland; [‡]School of Public Health, University of Tampere, Tampere, Finland; [§]Department of Public Health, University of Helsinki, Helsinki, Finland; [¶]Department of Medicine, Helsinki University Central Hospital, Helsinki, Finland; [**]National Institute for Health and Welfare, Helsinki, Finland; [††]Helsinki University Central Hospital Laboratory, Helsinki, Finland; [‡‡]University and University Hospital of Uppsala, Uppsala, Sweden

Background: In several studies *Helicobacter pylori* infection has been associated with lung cancer. Nearly all *H. pylori*-infected subjects have permanently elevated levels of circulating antibodies. Rapidly falling antibody titers after antimicrobial medications indicate cure of infection.

Methods: Altogether 26,705 consecutive patients tested in 1986–1998 for *H. pylori* antibodies were allocated to three subcohorts: 1, seropositive patients without confirmation of cure (Hp+NOCURE); 3, seropositive patients with rapidly falling antibody titers (Hp+ERADICATED); and 3, seronegative patients (Hp–). Subsequent lung cancers were identified from the Finnish Cancer Registry until the end of 2006. The risk ratios (RRs) with 95% confidence intervals (95%CI) were defined in a Poisson regression analysis using the Hp+NOCURE as the reference.

Results: Among 11,633 Hp+NOCURE, 3650 Hp+ERADICATED, and 11,422 Hp– patients, followed for a mean of 9.3, 10.4, and 10.9 years, subsequent lung cancers were found in 161, 33, and 55 patients, respectively. For the Hp+ERADICATED, the RR was 0.49 (95%CI 0.27–0.89) for the first five follow-up years but increased from the sixth follow-up year to 0.96 (95% CI 0.65–1.42). The RRs for Hp– were 0.63 (95%CI 0.40–0.99) and 0.50 (95%CI 0.33–0.77) for the same periods, respectively.

Conclusions: Cured *H. pylori* infection led to a significantly decreased incidence of lung cancers for the first five follow-up years. During the whole follow up, significantly fewer lung cancers were found in the Hp– cohort than in the Hp+NOCURE cohort.

Abstract no.: P05.14
Reflux Esophagitis in Children: Does Endoscopy Predict the Disease?

E. Tutar,[*] D. Ertem,[*] C. Celikel,[†] E. Pehlivanoglu[*] and E. Pehlivanoglu[*]

[*]Marmara University School of Medicine, Div. Pediatric Gastroenterology and Nutrition, Istanbul, Turkey; [†]Marmara University School of Medicine, Department of Pathology, Istanbul, Turkey

The concordance between gastroesophageal reflux (GER) symptoms and endoscopic and histopathologicfindings is still obscure in children. The aim was to evaluate the correlation between symptoms of GERD and endoscopic and histopathologic esophagitis in children who underwent endoscopy and to define the role of *H. pylori* gastritis on the reflux esophagitis as well.

A total of 59 subjects who had complaints suggesting GERD underwent endoscopy. Reflux symptoms were evaluated by using either Orenstein or Manterola scoring systems depending on the age of patients. The endoscopic and histopathologic diagnosis and severity of reflux esophagitis were assessed by Savary–Miller and Vandenplas grading systems, respectively. The updated Sydney score was used to assess *H. pylori* gastritis.

Mean age of the study group was 8.9 ± 4.4 years, and 31 (52.5%) had GERD according to the symptom scores. Thirty-one (52.5%) children had endoscopic and 47 (79.7%) patients had histopathologic esophagitis. The correlation between symptom score and endoscopic findings was not significant. However, the correlation between symptom score and histopathologic esophagitis was significant ($p < .01$). Eighteen of 28 (64.3%) endoscopically normal patients had histopatologic esophagitis. *H. pylori* was positive in 74.6% of the patients, and 29 of 44 *H. pylori*-infected children had gastritis. There was no correlation between *H. pylori* gastritis and histologic esophagitis in our group.

The clinical findings are very important for the suspicion of GER in childhood. Since endoscopic findings are not very leading in children, esophageal biopsies should be obtained in every child during endoscopy. *H. pylori* gastritis is not correlated with reflux esophagitis in this study.

Abstract no.: P05.15
The Expressed Interrelation of Heartburn with Esophagitis, Barrett Esophagus, and *H. pylori*-Associated Peptic Ulcer in Patients with Various Age–Sex Characteristics Among Inhabitants of Eastern Siberia

D. V. Kuklin,[*] V. Tsukanov[*] and V. O. Timoshenko[†]

[*]Scientific Medical Research Institute for Northern Problems of Siberian Division of Russian Academy of Medical Sciences, Krasnoyarsk, Russia; [†]Krasnoyarsk State Medical University, Krasnoyarsk, Russia

Aim: To study interrelation of heartburn, esophagitis, and *Helicobacter pylori*-associated peptic ulcer in representatives of various age–sex groups.

Methods: We carried out cross-section epidemiologic study using the modified questionnaire of Mejo Clinic (Locke G.R., Talley N.J. et al., 1994). Prevalence of heartburn was examined in 506 men

of military age, 375 men aged 40 to 60 years and 296 women aged 18 to 30 years. All subjects underwent upper digestive tract endoscopy with biopsy. Morphologic research included microscopic examination after staining by hematoxylin and eosin and definition of *H. pylori* dissemination after Gimsa staining.

Results: In military age men with heartburn rate of esophagitis was 7.1%, Barrett esophagus – 3.9%; peptic ulcer – 11.8%; in persons without heartburn – 0.3% ($p < .001$); 0% ($p = .0002$); 1.9% ($p < .001$) accordingly. In men aged 40–60 years with heartburn rate of esophagitis was 7.4%; Barrett esophagus – 5.7%; peptic ulcer – 12.5%; in patients without heartburn – 1.5% ($p = .005$); 0.5% ($p = .003$); 4.5% ($p = .005$), accordingly. In young women with heartburn these parameters was 2.7%; 1.4%; 6.8%, and in women without heartburn – 0% ($p = .01$); 0% ($p = .08$); 0.9% ($p = .005$), accordingly.

Conclusion: The expressed interrelation between heartburn, esophagitis, and *H. pylori*-associated peptic ulcer was registered in patient groups with various age–sex characteristics.

Abstract no.: P05.16
H. pylori Chronic Infection and Alzheimer Disease: Vascular or/and Inflammatory Association?

C. Roubaud-Baudron,* P. Krolak-Salmon,† F. Mégraud* and N. Salles*

*INSERM U853, University Victor Segalen Bordeaux 2, Bordeaux, France; †Centre de Mémoire, de Ressource et de Recherche, University Claude Bernard, Lyon, France

Recent case–control studies reported an association between *Helicobacter pylori* infection and Alzheimer dementia (AD). To explore this association, we compared vascular and inflammatory factors, in a group of *H. pylori*-infected and noninfected AD patients.

We studied serum and cerebrospinal fluid (CSF) samples from a group of AD patients. We assessed: 1, vascular comorbidities and cognitive status; 2, C-reactive protein level (CRP), homocysteine level, and cytokines [interleukin (IL)-1beta, IL-6, IL-8, tumor necrosis factor (TNF)-alpha] for serum; 3, cytokines, phospho-Tau protein (pTau), and amyloid betapeptide levels from LCR; and 4, brain magnetic resonance imaging data (Fazekas scale). *H. pylori* infection was defined by a positive ELISA and/or immunoblot test (CagA).

A total of 53 patients were included (23 men and 30 women, mean age: 68.5 ± 8.7 years). *H. pylori* infection was diagnosed in 37 (69%) patients, including 32 (86.5%) CagA+. *H. pylori* infection was associated with a decreased pepsinogen ratio ($p = .008$) and was significantly associated with vascular factors, i.e. positive correlation with homocysteinemia ($r = 0.44$, $p = .001$) and increased MRI white substance hyper-intensities ($p = .04$). *H. pylori* infection was also associated with chronic inflammation, i.e. positive correlation with CRP ($r = 0.31$, $p = .03$) and fibrinogen ($r = 0.34$, $p = .02$) levels and increased CSF TNF-alpha ($p = .02$) and IL-8 ($p = .004$) levels. *H. pylori* infection was statistically associated with the presence of neurodegeneration markers, i.e. increased pTau ($p = .01$).

H. pylori infection is associated with AD by two mechanisms which are probably linked: systemic and CSF inflammation and increasing brain vascular lesions. Chronic infection might be correlated to neurodegeneration lesions via both of these factors.

Abstract no.: P05.17
Symptoms of Gastroesophageal Reflux in the Population: Association with Serum Biomarkers

O. V. Reshetnikov,* S. A. Kurilovich,* S. A. Krotov,† V. A. Krotova† and E. D. Pylenkova*

*Institute of Internal Medicine, Novosibirsk, Russia; †Vector-Best Joint-Stock Company, Novosibirsk, Russia

Gastroesophageal reflux (GER) was associated with serum pepsinogen level in some studies; however, its interrelationship with *Helicobacter pylori* infection is still a matter of controversy.

Material and Methods: Two hundred and sixty-four subjects randomly selected from the general population were studied in Novosibirsk, Western Siberia (127 males, 137 females aged 45–69 years, mean age 59.4 years). Bowel Disease Questionnaire was used to study GER symptoms. Serum biomarkers (*H. pylori* antibodies, pepsinogen I, and gastrin-17) were assessed with Gastropanel (Biohit, Finland). Anti-*H. pylori* IgG CagA antibodies were evaluated using domestic ELISA kits (Joint-Stock Company Vector-Best, Novosibirsk, Russia).

Results: GER symptoms (at least once a month) were reported by 27.3% not depending on sex and age. In univariate analysis, presence of GER symptoms was associated with elevated pepsinogen I concentration (mean 123.2 vs 94.1 ng/mL, $p < .0001$), low gastrin-17 concentration (median 4.48 vs 6.34 pmol/L, $p = .014$), and CagA negativity (76.1 vs 60.9%, $p = .016$), but not with *H. pylori* status.

Multivariate analysis showed independent association of GER with pepsinogen I concentration > 100 ng/mL (OR = 3.0, 95% CI 1.6–5.5, $p = .0004$), and CagA negativity (OR = 2.3, 95% CI 1.2–4.3, $p = .01$).

Conclusion: Serologic evidence of nonatrophic, non-CagA-infected gastric mucosa is associated with gastroesophageal reflux symptoms in a population sample.

Abstract no.: P05.18
Is There Any Role of *H. pylori* in the Pathogenesis of Bronchiectasis?

T. Aydin Teke,* Y. Akyon,* E. Yalcin,* H. Ozen,* N. Cobanoglu,* S. Pekcan,† M. Kose,‡ D. Dogru,* N. Kiper* and U. Ozcelik*

*Hacettepe University, Ankara, Turkey; †Meram University, Konya, Turkey; ‡Erciyes University, Kayseri, Turkey

The aim of this study was to find out whether or not *Helicobacter pylori* is responsible for lung injury in pediatric patients with bronchiectasis.

The study group consisted of 29 patients with noncystic fibrosis bronchiectasis. The control group was consisted of nine individuals who did not have any documented evidence of pulmonary infection or bronchiectasis. Bronchoalveolar lavage (BAL) and fasting gastric aspirate samples (FGAS) were used to detect *H. pylori* by culture and polymerase chain reaction (PCR). Urea breath test (UBT) was performed. Gastroesophageal reflux (GER) was evaluated in *H. pylori*-positive patients.

BAL culture and/or PCR was positive in 11 of 29 cases of study group. BAL culture was negative in all individuals in the control group, but PCR was positive in two of nine individuals. The FGAS cultures and/or PCR analysis were positive in 12 patients in the

study group, while four cases were positive in the control group. Six of 11 patients were positive for *H. pylori* both in BAL and in FGAS. GER was detected in two of these patients. In the control group one of the two individuals was positive for *H. pylori* both in BAL and in FGAS, but none of these individuals had GER. UBT was positive in eight of 17 study group patients with *H. pylori* positivity while it was positive in one of five patients with *H. pylori* positivity in the control group.

According to our findings, we could suggest that *H. pylori* could have a role in the development and/or progression of bronchiectasis and that GER might mediate in the transfer of *H. pylori* to the lungs.

Abstract no.: P05.19
Occurrence of *H. pylori* Infection in the Esophageal Mucosa of Symptomatic Subjects

M. Contreras,[*] V. Salazar,[*] M. A. García-Amado,[*] N. Reyes,[*] M. Aparcero,[†] O. Silva,[‡] D. Castro,[‡] R. Romero,[§] P. Gueneau[*] and F. Michelangeli[*]

[*]Instituto Venezolano de Investigaciones Científicas (IVIC), Caracas, Venezuela; [†]Unidad de Gastroenterología del Hospital Pérez Carreño, Caracas, Venezuela; [‡]Centro de Control de Cáncer Gastrointestinal, San Cristóbal, Venezuela; [§]Policlínica Santiago de León, Caracas, Venezuela

Over the past few years, interest in *Helicobacter pylori* has extended from its role in the etiology of diseases of the stomach and duodenum to its possible role in the etiology of diseases of the esophagus. While in developing countries, the prevalence of *H. pylori* gastric colonization is known to be high (46 to 95%) in symptomatic subjects, there are few reports of the presence of *H. pylori* in the esophageal mucosa. The aim of this study was to assess the *H. pylori* infection in the esophagus of 85 Venezuelan symptomatic patients. Infection was detected through polymerase chain reaction (PCR) analysis of DNA extracted from esophageal and gastric biopsies, using genus- and species-specific primers. Infection in the esophagus was found by PCR in 52 patients (61%), of which 50 (59%) were *cagA*(+) while infection in the stomach was detected in 42 patients (49%) of which 38 (44%) were *cagA*(+). *H. pylori* infection in the esophagus was confirmed in 65 patients (76%) by FISH to visualize the bacteria within intact tissue samples. The presence of *H. pylori*, independently of method employed, was identified in 72 patients (85%) of which 45 (53%) have coincident positive tests, while 27 patients (32%) have positive but not coincident tests. According to our results, the prevalence of infection by *H. pylori* found in the esophageal mucosa is higher than that in the gastric mucosa, suggesting that *H. pylori* colonizes the esophagus when the esophageal mucosa is substituted by columnar epithelium.

Abstract no.: P05.20
Risk Factors Associated with Rosacea

K. Abram,[*] H. Silm,[*] H. I. Maaroos[†] and M. Oona[†]

[*]Clinic of Dermatology, University of Tartu, Tartu, Estonia; [†]Department of Polyclinic and Family Medicine, University of Tartu, Tartu, Estonia

Introduction: Although rosacea is a very common disease, the cause of disease is still a mystery – *Helicobacter pylori* infection, genetic predisposition, climatic factors, and detrimental habits are implicated as triggers of rosacea. The aim of current study is to evaluate several suspected risk factors coincidently.

Methods: Patients with rosacea from a dermatology clinic and skin-healthy controls from a randomly selected employees population enrolled the study. Skin status was evaluated by one and same dermatologist. Participants were queried for age, gender, sun-reactive skin type, and detrimental habits using a questionnaire; blood samples for detecting *H. pylori* serostatus were collected.

Results: Totally 145 skin-healthy controls and 172 subjects either with flushing episodes or established rosacea included the study. In multivariate analysis, rosacea patients had significantly higher chance to have photosensitive skin types [odds ratio (OR) 1.75; 95% confidence interval (CI) 1.01–3.04; $p = .007$], positive family history to rosacea (OR 4.31; 95% CI 2.34–7.92; $p < .0001$), or previous smoking status (OR 2.01; 95% CI 1.07–3.80; $p = .031$) compared with skin-healthy controls. There were no statistically significant differences either in gender, *H. pylori* serostatus, caffeine intake, alcohol consumption, occupational environment, or education level between rosacea patients and controls.

Conclusion: Rosacea is foremost associated with familial predisposition. There is no association between *H. pylori* infection and rosacea in current study.

Abstract no.: P05.21
Caga-Positive Strains of *H. pylori* may Play a Role in Pre-eclampsia and Poliabortivity through a Cross-Reactivity with Trophoblast Cells

F. Franceschi,[*] N. Di Simone,[†] G. Gigante,[*] D. Roccarina,[*] B. Giupponi,[*] G. De Marco,[*] R. Castellani,[†] M. D'Alessio,[†] F. Di Nicuolo,[†] S. D'Ippolito,[†] C. Tersigni,[†] G. Gasbarrini,[*] A. Gasbarrini,[*] A. Caruso[†] and N. Gentiloni Silveri[*]

[*]Policlinico Gemelli, Internal Medicine, Rome, Italy; [†]Policlinico Gemelli, Obstetrics and Gynecology, Rome, Italy

Background: The role of bacterial and viral infections in trophoblast diseases, such as pre-eclampsia and poliabortivity, has been extensively studied in the past few years. Interestingly, while trophoblast cells show an endothelial phenotypic profile, a study from our group has shown that antibodies anti-CagA cross-react with endothelial cells, possibly playing a role in some vascular diseases. We hypothesized that anti-CagA antibodies may recognize antigens of trophoblast cells, thus impairing their function.

Materials and Methods: Placenta samples were obtained from healthy women. Trophoblast cells were cultured for 72 hours in a medium containing increasing concentration of polyclonal anti-CagA antibodies (from 6 to 200 μg/mL). Binding of anti-CagA antibodies to trophoblast cells was verified through flow cytofluorimetry and immunoflurescence, while the invasive potential of these cells was assessed by using a membrane invasion culture system and measuring of MMP-2 activity. Trophoblast lysate was also prepared for immunoprecipitation using polyclonal anti-CagA antibodies.

Results: Anti-CagA antibodies recognized antigens of trophoblast cells of all samples, showing a dose-dependent binding, both at cytofluorimetry and at immunofluorescence. Incubation of trophoblast cells with increasing doses of anti-CagA antibodies

significantly reduced their invasiveness. Furthermore anti-CagA antibodies specifically precipitated two high molecular weight proteins from trophoblast lysate.
Conclusions: This study shows, for the first time, that anti-CagA antibodies recognize antigens expressed on the surface of trophoblast cells, reducing their invasiveness ability. These data give biologic plausibility to the theory that CagA-positive strains of *H. pylori* may play a role in trophoblast-related diseases.

Abstract no.: P05.22
Autoimmune Thyroid Disease and *H. pylori* Infection

G. Di Cairano, M. Campagna, M. Ghezzi, F. Pacini, R. Nuti, N. Giordano, L. D'Onofrio, S. Lardiello and N. Figura
Department of Internal Medicine, Endocrino-Metabolic Sciences and Biochemistry, Policlinico Le Scotte, Siena, Italy

Aim: *Helicobacter pylori* infection may be associated with autoimmune thyroid disease (ATD). Our aim was to verify whether the overall *H. pylori* and the CagA-positive (CagA+) *H. pylori* infection could influence the systemic levels of thyroid hormones, autoantibodies to thyroglobulin (TGA) and microsomial peroxidase (MPA) and pro- and anti-inflammatory cytokines in patients with ATD.
Methods: We enrolled 44 patients with ATD (mean age 49 [range 18 to 75] years) and 70 controls without ATD matched for age, gender, and social class. *H. pylori* infection and CagA status were determined serologically. All patients and controls underwent thyroid echography and determination of fT3, fT4 (pg/mL), TSH (μ/mL), TGA, MPA (U/mL), IL-1beta, IL-6, TNF-alpha, and IL-10 (pg/mL). Statistics was performed by Mantel–Haenszel chi-square test and Mann–Whitney U test.
Results: Fifteen patients (34.0%) and 14 controls (20.0%) were infected [$p = .09$, odds ratio (OR) = 2.07, 95% confidence interval (CI) 0.81–5.31]; seven infected patients and eight infected controls were CagA+ (NS). The mean levels of ft4 in CagA+ and CagA– infected patients were 8.23 [standard deviation (SD) 1.89] and 7.18 (SD 1.05) ($p = .05$). The difference between the levels of TGA in infected and uninfected patients was almost significant (595.21 [SD 1043.57] vs 277.02 [SD 693.56], $p = 0.173$). TNF-alpha concentrations in CagA+ patients were significantly increased with respect to CagA– infected and uninfected patients (1.90 [SD 2.47] vs 0.46 [SD 0.49] and 0.51 [SD 0.66], $p = .05$ and $p = .028$, respectively). IL-10 levels were 157 (SD 61) and 124 (SD 73) in infected and uninfected patients ($p = .042$).
Conclusion: *H. pylori* infection may influence the inflammatory response of ATD patients.

Funded by Siena University PAR 2006.

Abstract no.: P05.23
H. pylori Infection is Negatively Associated with Reflux Esophagitis: A Cross-Sectional Case–Control Study of 5616 Health Check-up Koreans

S. Chung,* J. Choi,† D. Kim,* H. Jung† and I. Song†
*Seoul National University Hospital Healthcare System Gangnam Center, Helathcare Institute, Seoul, South Korea; †Department of Internal Medicine, Seoul National University College of Medicine, Seoul, South Korea

Background: The relationship between *Helicobacter pylori* infection and gastroesophageal reflux disease remains controversial.
Aim: To investigate this relationship in a large Korean population-based study.
Methods: A cross-sectional case–control study of 5616 health check-up subjects undergoing upper gastrointestinal endoscopy was conducted (2808 cases with reflux esophagitis versus age- and sex-matched controls). *Helicobacter pylori* infection was determined by measuring anti-*H. pylori* IgG.
Results: The prevalence of *H. pylori* infection was significantly lower in subjects with reflux esophagitis than in controls (38.4% vs 58.2%, $p < .001$). After adjusting for the effects of smoking, alcohol, waist circumference, and body mass index *H. pylori* infection is negatively associated with reflux esophagitis [odds ratio (OR) 0.44, 95% confidence interval (CI) 0.39–0.49]. There was significant inverse correlation between the severity of esphagitis and the prevalence of *H. pylori* infection. Subjects infected with *H. pylori* showed a significant increase in the risk of gastric atrophy (OR 3.22, 95% CI 2.79–3.70).
Conclusions: *H. pylori* seropositivity was independently associated with the reduced risk for reflux esophagitis and correlates with the milder grade of esophagitis in Korean population, which suggest that *H. pylori* infection may play an inhibitory role for reflux esophagitis.

Abstract no.: P05.24
Persistency of *H. pylori* in Inferior Third of Esophagus Epithelium in Patients with GERD

O. S. Rzhavicheva* and V. V. Tsukanov†
*Krasnoyarsk Railway Station Hospital, Krasnoyarsk, Russia; †Scientific Medical Research Institute for Northern Problems of Siberian Division of Russian Academy of Medical Sciences, Krasnoyarsk, Russia

Aim: To study prevalence of *Helicobacter pylori* in epithelium of inferior third of esophagus in patients with gastroesophageal reflux disease (GERD).
Methods: Sixty-three patients with GERD (48 men and 15 women) aged 23 to 52 years were examined. All subjects underwent upper digestive tract endoscopy with biopsy and description of esophagus changes on Los Angeles classifications (1994). Morphologic research of esophagus epithelium included microscopic examination after staining by hematoxylin and eosin, and also definition of *H. pylori* dissemination after Gimsa staining.
Results: The erosive esophagitis was determined in 90.5% patients. The metaplasia of esophageal inferior third epithelium

was diagnosed in 19.0% persons. *H. pylori* was recorded in 19.0% patients. *H. pylori* was observed in 91.7% (11 of 12) patients with Barrett esophagus and in 2.0% persons (1 of 51) without metaplasia of esophagus ($p < .001$).
Conclusion: *H. pylori* was found often in metaplasied epithelium of esophagus and can render the influence on development of this disease.

Abstract no.: P05.25
Relationship Between *H. pylori* Infection, Gastric Atrophy, and the Risk of Esophageal Squamous Cell Carcinoma in Germany

M. Venerito,[*] S. Kohrs,[*] T. Wex,[*] K. Mönkemüller,[*] D. Schubert,[†] D. Adolf[‡] and P. Malfertheiner[*]
[*]Department of Gastroenterology, Hepatology and Infectious Diseases, Magdeburg, Germany; [†]Department of Surgery, Magdeburg, Germany; [‡]Institute for Biometrics, Magdeburg, Germany

Background: Recent studies from Sweden and Japan have shown a positive association between gastric atrophy and an increased risk for developing esophageal squamous cell carcinoma (OSCC). However, this findings need to be confirmed in other ethnic groups due to the wide geographic variation of this cancer and the changing prevalence of the *Helicobacter pylori* infection.
Aim: To investigate whether *H. pylori* infection and gastric atrophy are associated with an increased risk for OSCC using a case–control study in Germany.
Methods: Fifty-eight consecutive patients (40 males and 18 females, mean age 65 ± 9 years) with OSCC, and 116 sex- and age-matched controls with dyspepsia, were enrolled prospectively. Antrum and corpus atrophy were evaluated by histology of biopsy specimens and serology. Pepsinogen (PG)-I level < 30 µg/mL and PGI/II ratio < 2.5 were indicative for corpus atrophy, gastrin (G)-17 values of < 1 pmol/L were suggestive for the presence of antrum atrophy. Fasting serum was analyzed for G17, PGI, PGII, and *H. pylori* antibodies using specific EIA tests (GastroPanel; Biohit, Plc). Anti-CagA antibodies were analyzed by immunoblot assay.
Results: *H. pylori* infection, assayed by either serology or anti-CagA antibodies, was not associated with an increased risk for OSCC ($p > .05$, Fisher exact test). The presence of gastric corpus and/or antrum atrophy diagnosed by means of histology, PGI, PGII, and/or G1-7 was also not associated with an increased risk for OSCC ($p > .05$).
Conclusions: Neither *H. pylori* infection nor gastric atrophy was associated with an increased risk for OSCC.

Abstract no.: P05.26
Helicobacter Eradication Therapy in Idiopathic Parkinsonism with Anti-nuclear Antibody: Implications for Better Methods of Detecting Low Density Infection

S. M. Dobbs,[*†] R. Dobbs,[*†] A. J. Lawson,[‡] R. J. Owen,[‡] I. T. Bjarnason,[†] C. Weller[*] and A. Charlett[*§]
[*]Psychiatry and Psychological Medicine, Institute of Psychiatry, London, UK; [†]Department of Gastroenterology, King's College Hospital, London, UK; [‡]Laboratory of Gastrointestinal Pathogens, Centre for Infections, Health Protection Agency, London, UK; [§]Statistics Unit, Centre for Infections, Health Protection Agency, London, UK

Background: Apparent *Helicobacter* eradication (to level of polymerase chain reaction on paired biopsies) converted malignant idiopathic parkinsonism (IP) to benign, marked deterioration accompanied proven failure (*Helicobacter* 2008;13:309–22).
Methods: Antinuclear antibody (ANA) was sought, before and 1.5 years after anti-*Helicobacter* therapy in IP probands (sera processed in single batch, blind to sequence). Duplicate measurements of primary outcome mean –stride length were made before (two occasions) and post-therapy (≥ 6 weekly intervals). Any background antiparkinsonian medication was constant, the short-$t^1/_2$ levodopa an exclusion.
Results: The context is a quarter of 126 IP probands being ANA-seropositive. In the urea breath test positive (29.6%), neutrophil and lymphocyte counts were not higher, CD8+ count was 28 [95% confidence interval (CI) 6, 54)%, $p = .01$]. In 21 culture-positive probands, ANA was present in 8 before anti-*H. pylori* therapy, 10 after. Eradication failure was demonstrated in six (2 UBT-positive, 4 on culture/PCR only), of whom 5 were ANA-positive afterwards: ANA tended to mark continuing infection ($p = .06$). Following therapy [median 358 (interquartile range 148, 498) days with 11 (8, 20) visits] in 20 probands, stride length deteriorated markedly in the ANA positive [by 149 (95% CI 13, 284) mm/year], in contrast ($p = .03$) to improvement in ANA-negative [77 (–1, 156) mm/year]. Effects of ANA status and eradication success/failure were intrinsically linked. A low CD8+ count was associated with deterioration ($p = .02$), tended to be with eradication failure ($p = .06$).
Discussion: Prognostic indicators are needed because *Helicobacter* is difficult to eradicate in IP, even when antimicrobial sensitivities are known/compliance monitored (*Helicobacter* 2008;13:309–22). Reduced cytoxicity may impair clearance of residual organisms. ANA may alert to continuing low-density infection not detected by PCR on only two biopsies.

Abstract no.: P05.27
Signaling Pathways Involved in Hepatic Stellate Cell Activation are Regulated by miRNAs

C. J. Guo, Q. Pan and G. D. Guo
Digestive Disease Laboratory and Department of Gastroenterology, Shanghai Jiaotong University, Shanghai, China

Activation of hepatic stellate cells (HSCs), which is regulated by multiple signal transduction pathways, is the key event in liver fibrosis. Moreover, members of these pathways are important targets for microRNAs (miRNAs). To better understand the critical pathways of HSCs activation, we carried out comprehensive comparative bioinformatics analysis of microarrays of quiescent and activated

HSCs. The analysis revealed that 13 pathways were upregulated and 22 pathways were downregulated by microRNA. Furthermore, mitochondrial integrity based on highly upregulated Bcl-2 and downregulated caspase 3, 9 was confirmed in HSCs and fibrotic livers by immnofluorescence assay, semiquantitative RT-PCR, qRT-PCR, and Western blot. These findings provide in vitro and in vivo evidence that the mitochondrial pathway of apoptosis plays a significant role in the progression of liver fibrogenesis via HSCs activation.

Abstract no.: P05.28
The Prevalence of *H. pylori* Infection in the Patients with Different Gastroesophageal Reflux Disease Groups

E. V. Onuchina* and V. V. Tsucanov†
*Irkutsk State Medical University, Irkutsk, Russia; †Scientific Research Institute of Medical Problems of North, Krasnojarsk, Russia

Aim: To study the prevalence of *Helicobacter pylori* infection in patients with GERD belonging to different age groups.

Material and Methods: A total of 722 patients were investigated: 326 patients older than 60 years and 244 patients under 60 years with GERD; the control group consisted of 152 patients without GERD. The severity of esophagitis was evaluated using the Los Angeles classification. The diagnosis of *H. pylori* was carried out with histologic method and rapid urease test.

Results: *H. pylori* infection in GERD patients is revealed more seldom than in the control group. The chances ratio (CR) of association of *H. pylori* infection and GERD in patients older than 60 years were 0.28 (95%CL: 0.10–0.43), $p = .00001$, in the patients under 60 years it was – 0.59 (95%CL: 0.14–1.03), $p = .00001$. The analysis of clinical forms of GERD revealed that infection was the least in Barrette esophagus (BE) in both age groups. The analysis shows that in young patients the prevalence of *H. pylori* was higher in ERD, in elderly patients – in NERD. In increasing severity of esophagitis in the elderly, decrease of helicobacteriosis spreading was noted, while in young patients the spread of helicobacteriosis increased.

Conclusion: Level of helicobacteriosis not depending on age had distinct reverse connection with the presence of BE in patients of elderly group with severity of GERD.

P06 Molecular Genetics and Genomics, Virulence Factors and Pathogenesis I

Abstract no.: P06.01
Prevalence of *cagA* and *jhp0947* Genes in *H. pylori* Isolates from First-Degree Relatives of Gastric Cancer Patients

S. Massarrat,* F. Siavoshi,† H. Ghadiri,† M. Mirzaei* and M. Zamani†
*Digestive Disease Research Center, Shariati Hospital, Medical Sciences/University of Tehran, Tehran, Iran; †Microbiology Department, Faculty of Sciences, University of Tehran, Tehran, Iran

Introduction: *Helicobacter pylori* gastritis is a dynamic and progressive process. Studies suggest that a combination of different virulence genes determine the severity of inflammation. The aim of this study was to investigate the prevalence of *cagA* and *jhp0947* genes in the first-degree relatives of gastric cancer patients (FDRCPs) and their correlation with different types of gastritis.

Methods: One hundred and forty-three *H. pylori* strains were isolated from antral gastric biopsies of FDRCPs. All of the patients had gastritis. Three types of gastritis according to pathologic findings were: antral-predominant gastritis, corpus-predominant gastritis, and pangastritis. Genomic DNAs from isolates were subjected to PCR-based genotyping of *cagA* and *jhp0947* genes. Primers were designed for amplification of these genes.

Results: The prevalence of the *cagA* and *jhp0947* among 143 *H. pylori* isolates from FDRCPs.

Type of gastritis			
Pangastritis	Antral predominant	Corpus predominant	Genotype status
54 (%73)	50 (%78.1)	5 (%100)	*CagA* positive
38 (%51.4)	35 (%54.7)	1 (%20)	*jhp0947* positive
74 (%51.7)	64 (%44.7)	5 (%3.6)	Total

Discussion: *cag A*-positive and *jhp0947*-positive *H. pylori* strains were predominant in FDRCPs. However, we could not find any association between genotypes of *H. pylori* strains and different types of gastritis. Although a number of putative *H. pylori* virulence genes have been associated with risks of a clinical outcome, none have clearly been linked to one specific *H. pylori*-related disease. Further studies with more isolates and candidate genes might help to predict the clinical outcome of *H. pylori* infection with certain genotypes.