ΞΕΝΟΓΛΩΣΣΕΣ ΑΝΑΚΟΙΝΩΣΕΙΣ (ABSTRACTS) ΕΛΛΗΝΩΝ ΕΡΕΥΝΗΤΩΝ

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Abstract no.: 03.11

Differences in the Prevalence of EPIYA Tyrosine Phosphorylation Motifs in CagA Protein of *Helicobacter pylori* Clinical Isolates from Children and Adults

E. Panayotopoulou,* K. Papadakos,* A. Kalliaropoulos,* K. Petraki,* S. Karagiannis,† F. Dimopoulos,† S. Michopoulos,† E. Katsiyiannaki,‡ E. Roma,‡ A. Archimandritis,‡ A. Mentis* and D. N. Sgouras*
*Laboratory of Medical Microbiology, Institut Pasteur Hellenique, Athens, Greece, †Gastroenterology Clinic, Alexandras General Hospital, Athens, Greece, ‡Pediatric Gastroenterology Unit, Aghia Sophia Childrens Hospital, Athens, Greece, §First Department of Pediatrics, Athens University School of Medicine, Athens, Greece, ¶Second Department of Internal Medicine, Athens University School of Medicine, Athens, Greece

The presence of tyrosine phosphorylation motifs (TPMs) A/EPIYA/E/VNK, B/EPIYA/Q/VAKK, and C/EPIYA/T/DDLG in CagA protein have been proposed to enhance *cagA*-dependent pathogenicity. Based on the peptide identity of published CagA protein sequences, we developed degenerate polymerase chain reaction (PCR) primers and subsequently sequenced and mapped the EPIYA motifs in 124 *Helicobacter pylori* isolates (64 adults, 60 children). Strains were also typed with respect to *vacA* signal and mid-region. A higher prevalence of EPIYA-negative strains in children (20.0%) versus the adult (9.4%) population was observed, although there was no real difference in *H. pylori cagA*-negative strains (adults 14.1%, children 11.7%). The majority of strains harbored the *ABC* (adults 53.1%, children 51.7%) and the *ABCC* combination of TPMs (adults 15.6%, children 13.3%). Five strains with additional TPM-C repeats (*ABCCC*) were detected exclusively within the adult population (7.9%). A significant trend for a higher number of TPM-C in adults (see table below; χ^2 test, $p = .045$) was observed. In addition, the presence of *vacA* s2m2 isotype was associated with *cagA*-negative ($p < .0001$), but not EPIYA-negative strains. However, in children a strong association was observed between EPIYA-negative strains ($p < .0001$) and the least vacuolating *vacA* s2m2 isotype. In conclusion, strains isolated from adults are likely to harbor an increased number of phosphorylation TPM-C repeats within the CagA protein, whereas in children a significant trend for higher prevalence in EPIYA-negative *vacA* s2m2 *H. pylori* strains is observed.

TPM-C prevalence

Number of TPM-C	Adults	Children
0	6	12
1	34	31
= 2	15	8

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Positive Association Between the Presence of CagA Protein EPIYA Motifs in *Helicobacter pylori* Clinical Strains and the Severity of Histopathologic Lesions

E. Panayotopoulou,* K. Petraki,* A. Kalliaropoulos,* B. Martinez-Gonzalez,* K. Papadakos,* S. Karagiannis,† F. Dimopoulos,† S. Michopoulos,† A. Archimandritis,‡ A. Mentis* and D. N. Sgouras*
*Laboratory of Medical Microbiology, Institut Pasteur Hellenique, Athens, Greece, †Gastroenterology Clinic, Alexandras General Hospital, Athens, Greece, ‡Second Department of Internal Medicine, Athens University School of Medicine, Athens, Greece

We aimed to study the potential association between the presence of EPIYA tyrosine phosphorylation motifs (TPMs) in CagA protein of *Helicobacter pylori* clinical isolates with the clinical manifestation of the disease and the observed histopathologic lesions.

We analyzed 58 *cagA*-positive *H. pylori* clinical isolates taken from patients with gastroduodenal ulcer ($n = 39$) and nonulcer dyspepsia ($n = 19$). EPIYA motifs were determined by polymerase chain reaction (PCR) amplification and sequencing of the resulting products. *cagA* status was determined by PCR *cagA* amplification and serum CagA ELISA. *H. pylori* colonization and the associated gastritis was evaluated by the modified Sydney system and statistical analysis performed by Fischer's exact test. EPIYA-positive *H. pylori* clinical isolates in the antrum were correlated significantly with the presence of gastroduodenal ulcer ($p = .002$). The association was strong with the presence of duodenal ($p = .016$) but not gastric ulcer ($p = .091$). There was significant positive association with the severity of chronic inflammatory infiltration ($p = .037$) and the activity of chronic gastritis ($p = .033$), but not with higher levels of *H. pylori* colonization ($p = .108$). No statistical significance was observed in the fundus.

In conclusion, the severity of chronic inflammatory infiltration and the activity of chronic gastritis developed in the antrum of *H. pylori*-positive patients seem to be associated with the presence of EPIYA TPMs in CagA protein, irrespective of the levels of *H. pylori* colonization in the gastric mucosa. This association is significant in patients with duodenal but not gastric ulcer.

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Helicobacter pylori-Negative Gastric Ulcers

N. Rossolimos,* M. Theofilopoulou,* S. Balas,* P. Aggeli,* C. Vassiliou,* V. Xiromeritou,* G. Karatzikos,* G. Kafiri† and K. Papadimitriou†
*Departments of Gastroenterology and †Histopathology, Ippokration General Hospital, Athens, Greece

Objective. The prevalence of *Helicobacter pylori*-negative ulcers is highly variable geographically. In pooled series from the United States, 26% of 315 patients with uncomplicated gastric ulcers (GUs) were *H. pylori*-negative. In contrast, the reported prevalence of *H. pylori*-negative ulcers from Europe is much lower.

The aim of this study is the determination of the prevalence of *H. pylori*-negative GU among the southern Greek population.

Methods. One hundred ten patients of a median age of 51 years (21–88) who were not taking NSAIDs and underwent upper GI tract endoscopy because of epigastric discomfort were diagnosed with GUs. The methods of diagnosis used for *H. pylori* infection were firstly by histologic examination and secondly by rapid urease test.

Results. Seventy-two patients (65.5%) were *H. pylori*-positive and 38 (34.5%) *H. pylori*-negative. The percentage of *H. pylori*-negative GU is higher than that reported internationally.

The majority of these ulcers (after histologic examination) were diagnosed as "idiopathic" except for a small number, that were related to lymphoma, carcinoma, Crohn's disease, or other uncommon causes of GU.

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Lactobacillus johnsonii La1 Attenuates *Helicobacter pylori*-Associated Gastritis and Reduces the Levels of Proinflammatory Cytokines in the Gastric Epithelium of C57BL/6 Mice

D. N. Sgouras,* E. Panayotopoulou,* B. Martinez-Gonzalez,* K. Petraki,* S. Michopoulos† and A. Mentis*
*Laboratory of Medical Microbiology, Institut Pasteur Hellenique, Athens, Greece, †Gastroenterology Clinic, Alexandras General Hospital, Athens, Greece

The effect of *Lactobacillus johnsonii* La1 administration to experimental *Helicobacter pylori* infection was studied. We administered continuously over a period of 3 months, through the water supply, live La1 to *H. pylori*-infected C57BL/6 mice and followed colonization, and development of *H. pylori*-associated gastritis in the lamina propria. We also determined the levels of pro-inflammatory chemokines macrophage inflammatory protein-2 (MIP-2) and keratinocyte-derived cytokine (KC) in the serum and gastric tissue. We documented a significant attenuation both in lymphocytic ($p = .038$) and neutrophilic inflammatory infiltration ($p = .003$) in the lamina propria as well as in the circulating levels of anti-*H. pylori* IgG antibodies ($p = .003$), although we did not observe a suppressive effect of La1 on *H. pylori* colonizing numbers. Other phylogenetic-related lactobacilli did not attenuate *H. pylori*-associated gastritis to the same extent. MIP-2 serum levels were distinctly reduced during the early stages of *H. pylori* infection in the La1-treated animals and gastric mucosal levels of MIP-2 and KC were also found depressed. Accordingly, *H. pylori*-induced IL-8 secretion by human adenocarcinoma AGS cells in vitro was also significantly reduced ($p = .046$) in the presence of La1 spent culture supernatants, when neutralized to pH 6.8 without concomitant loss of *H. pylori* viability. These observations point out that during the early infection stages, administration of La1 can attenuate *H. pylori*-induced gastritis in vivo possibly, by reducing proinflammatory chemotactic signals responsible for the recruitment of lymphocytes and neutrophils in the lamina propria.

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Prevalence and Age-Related Distribution of Intestinal Metaplasia of the Gastric Mucosa

N. Rossolimos,* C. Vassiliou,* P. Aggeli,* S. Balas,* M. Theofilopoulou,* V. Xiromeritou,* G. Karatzikos,* A. Bousiotou,† G. Kafiri† and K. Papadimitriou†
*Ippokration General Hospital, Department of Gastroenterology, Athens, Greece, †Ippokration General Hospital, Department of Histopathology, Athens, Greece

Objective. The excessive consumption of antibiotics and antisecretory drugs during recent years principally by older ages and the systematic eradication of *Helicobacter pylori* worldwide (according to the criteria of Maastricht) may have caused remarkable changes in the *H. pylori* population, which is implicated in the provocation of gastric intestinal metaplasia (IM). The aim of this study is the determination of the prevalence and of the age related distribution, of gastric IM as it has developed recently.
Methods. A total of 751 patients of a median age of 54 years (22–90) who underwent gastroscopy were included in the study. None of them had history of upper GI tract surgical procedures. Biopsies were taken from the antrum, the angularis, and the body of the stomach for the diagnosis of IM according to the Houston modification of Sydney's classification.
Results. Of all the patients, 238 (31.7%) were diagnosed with IM of all types. The higher and the lower percentages of prevalence of IM were 36.0% and 13.0% in the eighth and third decades of age, respectively. The analytical results are cited below.

	[20,29]	[30,39]	[40,49]	[50,59]	[60,69]	[70,79]	[80,89]
n = 751	23	58	74	125	180	222	69
n = 238	3	15	20	32	64	80	24
Percentage (%)	13.0	25.9	27.0	25.6	35.6	36.0	34.8

$\chi^2 = 10,97$, $p < .10$, $Pr = 0.0892$.

Conclusions. Instead of wide eradication of *H. pylori* in the general population, gastric IM is prevalent in a high percentage of them.

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iNOS Expression in Gastric Carcinogenesis: A Molecular and Immunohistochemical Study in Tissue Microarrays (TMAs) – Correlation with *Helicobacter pylori* Infection

A. Karameris,* E. Tsiambas,† S. Gazi,‡ K. Gerontopoulos,§ G. Vilaras,¶ D. Stefanou** and T. Roikas‡†
*Department of Pathology, 417 VA Hospital, Athens, Greece, †Department of Pathology, Evangelismos Hospital, Athens, Greece, ‡EOF, Athens, Greece, §Coronary Medicine, 417 NIMTS Hospital, Athens, Greece, ¶Department of Pathology, 417 NIMTS Hospital, Athens, Greece, **Department of Pathology, University of Ioannina, Ioannina, Greece, ††Gastroenterology Clinic, Henry Dunant Hospital, Athens, Greece

Aim. To examine the role of iNOS and *Helicobacter pylori* infection in gastric carcinogenesis.
Material and Methods. Forty-five (25 *H. pylori*-positive [*Hp*+] and 20 *H. pylori*-negative [*Hp*–]) gastric carcinoma cases were studied. Twelve *Hp*+ and 10 *Hp*– carcinomas were classified as diffuse and 13 *Hp*+ *and* 10 *Hp*– as intestinal type. Intestinal metaplasia (IM) type I were observed in 5 *Hp*+ cases, IM II in 8, and IM III in 12 cases. Controls included 5 *Hp*– IM I, 4 IM II, and 10 IM III cases. The TMArrayer apparatus (Chemicon, USA) was used for the construction of TMAs. iNOS expression was determined by immunohistochemistry and differential polymerase chain reaction (PCR) in microcore samples taken by TMArrayer and properly analyzed with an Image Analysis System (DIS-200, Digital Image Systems, Hellas).
Results. Twelve of 13 of intestinal and 10/12 of diffuse *Hp*+ gastric carcinomas overexpressed iNOS. Two of 5 IM I, 8/12 IM II, and 7/8 IM III *Hp*+ cases also expressed increased iNOS levels as well as 5 diffuse and 5 intestinal-type *H. pylori* carcinomas. In addition, 0/5 *Hp*– IM I, 1/6 *Hp*– IM II, and 2/10 *Hp*– IM III cases expressed high iNOS levels. Statistically significant differences, concerning the expression of iNOS, were observed between *Hp*+ Ca and *Hp*– Ca (< .01), between IM II and III (< .05), as well as between *Hp*+ and *Hp*– IM II and III (< .01).
Conclusions. High iNOS expression is significant for the initiation and promotion of gastric carcinogenesis. High iNOS levels in IM type II and III correlated well with *H. pylori* infection and this may be a strong evidence for long-term follow-up.

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bstract no.: 11.09
igher Reinfection Rate in Celiac Disease atients After Successful *Helicobacter* radication for Peptic Ulcer Disease

D. Paraskeva,* N. Mathou,* N. Giannakou,† . Michalopoulou,† I. Ghiconti,† C. Spiliadi† and A. Karagiannis*
astroenterology Unit, Agia Olga Hospital, Athens, Greece, epartment of Pathology, Agia Olga Hospital, Athens, Greece

radication regimes for *Helicobacter pylori* infection have en proven particularly effective and with low reinfection (or rudescence) rate that is estimated ~1–2% annually.
Aim of the study was to assess maintenance of the eradication in tients with celiac disease. In 29 patients (16 male, 13 female, age nge 19–67 years) with celiac disease, all in clinical and histologic mission on a gluten-free diet, benign peptic ulceration (20 odenal, 9 gastric) was diagnosed endoscopically. None was on ng-term NSAIDs.
Of the 29 patients, 23 were *Hp*+ by histology and *Campylobacter*-e organism (CLO) test and received for both healing and . *pylori* eradication first-line triple schemes (PPI, clarithromycin, noxycillin) and those who failed (7/23) to become *Hp*– second-e quadruple schemes (PPI, bismuth compounds, amoxycillin, etronidazole), both at the recommended dose and duration. All d their ulcers healed, 20 of 23 became *Hp*– by histology/CLO st and/or ^{13}C-UBT, whereas three of 23 remained *Hp*+. *Hp*– tients were reevaluated for their *H. pylori* status with ^{13}C-UBT ter a mean observation period of 55 months (range 22–68 months). velve of twenty (60%) remained *Hp*–, whereas 8/20 (40%) came *Hp*+. Reinfection rate for matched nonceliac patients and r similar observation period was ~10%. Nine of twenty patients, ur *Hp*– and five *Hp*+, were re-endoscoped because of dyspeptic mptoms and in three of nine, all *Hp*+, recurrence of duodenal cer was diagnosed.
Even if the number of patients studied is small, it seems that ients with celiac disease have higher than expected reinfection e. Genetic factors influencing susceptibility to *H. pylori* ection by modulating the host immune response might be plicated to explain this observation.

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EFFECTIVENESS OF ENDOSCOPIC BALLOON DILATATION AS FIRST LINE TREATMENT IN PATIENTS WITH A BULB STENOSIS DUE TO DUODENAL ULCER

P. Tsibouris[1], G. Tsianos[2], A. Vassilopoulos[2], A. Babanis[2], T. Galeas[3], C. Lappas[3]
[1]Gastroenterology, [2]Surgery, [3]Medicine, Trikala General Hospital, Trikala, Greece

INTRODUCTION: Surgical treatment is the gold standard for pyloric stenosis. Endoscopic balloon dilatation is reserved for high-risk patients. Aim of the study: To evaluate mid term effectiveness of endoscopic balloon dilatation to treat stenosis of duode nal bulb.
AIMS & METHODS: Aim of the study: To evaluate mid term effectiveness of endoscopic balloon dilatation to treat stenosis of duodenal bulb. Patients and methods: Between 1/1/2002 and 31/12/2004, 40 consecutive patients (Group-I, mean age 72±11, 25 male, 10 smokers - 31±47 pack-years, 14 in excess alcohol consumers, 20 NSAIDs consumers) with duodenal bulb stenosis due to a duodenal ulcer (mean diameter of bulb lumen 3 mm) offered and underwent an endoscopic balloon dilatation (by Flex-EZ Balloon Dilator Hobbs Medical Inc, Transtein, Germany) of the stenosis 48 hours after admission (pantoprazole 8 mg/h iv was started on admission). After discharge pantoprazole 40 mg daily was prescribed for 2 months. Endoscopy was repeated after 3 months, when H. pylori status was checked and eradication treatment was prescribed in H. pylori positive patients. Endoscopy was repeated every 6 months for the first year and yearly thereafter. Results were compared with 40 consecutive patients (Group-II, mean age 65±12 years, 26 men, 11 smokers - 33±38 pack-years, 13 in excess alcohol consumers, 19 NSAIDs users) offered surgery as first line treatment between 1/1/1999 and 31/12/2001 and had the same follow-up for the same period as endoscopically treated patients (Mean follow-up: Group-I: 22±14 months, Group-II: 23±13 months). Statistics: t-test, X2.
RESULTS: Intention to treat: Group I: 39 (98%) patients, Group II: 30 (75%) patients (p = 0.004). Per protocol analysis: Group-I: 39/40 (98%), Group-II: 30/30 (100%) (p = 0.39). During follow-up 5 (13%) Group-I and 1 (3%) Group-II patients relapsed (p = 0.17). Mean hospital stay Group-I: 2±3 days, Group-II: 7±8 days (p = 0.02). One Group-I patient had perforation and 3 presented aspiration pneumonia despite gastric lavage (one died). No Group-II patient had a major complication. In 4/5 Group-I and 1/1 Group-II patients relapse was attributed in NSAIDs use (3/5 Group-I patients presented with multiple relapses) and in 1/5 Group-I patients to inadequate H. pylori eradication. Multiple Group-I relapsers finally needed an operation, while all other relapsers were effectively treated by balloon dilatation. Although 16/40 (40%) Group-I patients presented oesophagitis before dilation 3/40 (8%) had oesophagitis during follow-up (2 less severe than pre-dilatation endoscopy 1 de novo oesophagitis). 12/30 (40%) Group-II patients who had an operation, presented oesophagitis preoperatively and 10/30 (33%) had oesophagitis during follow-up (4 cases with more severe disease, 4 with de novo oesophagitis, 2 with less or equal severe disease). During follow-up 5/30 (17%) Group-II patients presented dumping.
CONCLUSION: Endoscopic balloon dilatation is a quite safe and effective treatment for duodenal bulb stenosis due to duodenal ulcer. Thus surgery should be relieved for endoscopic treatment failures and multiple relapsers.

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SOLUBLE TRIGGERING RECEPTOR EXPRESSED ON MYELOID CELLS (STREM-1): A NEW MEDIATOR INVOLVED

V. Koussoulas[1], S. Vassilliou[1], E.J. Giamarellos-Bourboulis[2], G. Tassias[1], M. Demonakou[3], M. Mouktaroudi[2], H. Giamarellou[3], C. Barbatzas[1]
[1]Gastroenterology,SismanoglionGeneralHospital,[2]4thDept.ofInternal Medicine,AthensMedicalSchool,[3]Pathology,SismanoglionGeneralHospital, Athens, Greece

INTRODUCTION: Triggering receptor expressed on myeloid cells (TREM-1) is a promoter of cytokine production triggered by microbial components. To investigate the significance of sTREM-1 for the pathogenesis of peptic ulcer, sTREM-1 was associated with parameters of gastritis and lipid peroxidation.
AIMS & METHODS: Forty patients with dyspepsia were enrolled; twenty with peptic ulcer and 20 controls without any macroscopic abnormalities. All patients were endoscoped; gastric juice was aspirated and biopsy specimens were collected from antrum and body. sTREM-1 was estimated by a hand-made enzymeimmunoassay. Lipid peroxidation, indexed by malondialdehyde (MDA), was estimated by the thiobarbitouric assay, after passage through an HPLC system.
RESULTS: Mean (±SE) of sTREM-1 of controls and patients with ulcer was 5.53±0.78 pg/ml and 174.02±98.28 pg/ml respectively (P = 0.006). Mean (±SE) of sTREM-1 concentrations in subjects without evidence of gastritis, H. pylori positive gastritis and H. pylori negative gastritis were 47.31±16.67 pg/ml, 77.36±43.38 pg/ml and 125.95±103.35 pg/ml, respectively (PNS between patients). sTREM-1 was positively correlated with the degree of mucosal atrophy (P = 0.009) but it was not correlated either with the activity of gastritis or the degree of intestinal metaplasia or MDA levels or with the density of Helicobacter pylori.
CONCLUSION: Results revealed that sTREM-1 might be an independent factor leading to the ulcerative inflammatory process.

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EPITHELIAL CELL TURNOVER IN NON-DYSPLASTIC GASTRIC MUCOSA ADJACENT TO EARLY AND ADVANCED GASTRIC CANCER

K. Triantafyllou[1], P. Kitsanta[2], D.G. Karamanolis[3], C. Kittas[4], S.D. Ladas[1]
[1]HepatogastroenterologyUnit,AttikonUniversityGeneralHospital,Athens, Greece, [2]Histopathology Department, Sheffield Teaching Hospitals, Sheffield,UnitedKingdom,[3]GastroenterologyDepartment,Tzaneion HospitalofPiraeus,Piraeus,[4]LaboratoryofHistology-Embryology,Medical School, Athens University, Athens, Greece

INTRODUCTION: Epithelial cell turnover alterations, as well as, p53 and Bcl-2 protein expression play important role during gastric oncogenesis.
AIMS & METHODS: The aim of the study was to investigate cell apoptosis and proliferation rates, p53 and Bcl-2 protein expression in non-dysplastic tissue (NDT) adjacent to early (EGC) and advanced gastric carcinomas (AGC).
We examined 17 EGC and 15 AGC, and NDT specimens five cm from the margin of each tumor. Cell proliferation, p53 and Bcl-2 expression were detected immunohistochemically using MIB-1, anti-p53 and anti-Bcl-2 monoclonal antibodies. Apoptosis was measured by TUNEL method. The rate of the positive stained cells was count using image analysis technique (SABA).
RESULTS: No differences were observed of either median apoptotic (2 vs 2) or median proliferation (8 vs 12) index between NDT adjacent to early and advanced tumors. While both indices were significantly higher in tumors than in the NDT, no difference was observed of either apoptotic (10 vs 10) or proliferation (35 vs 25) index between EGC and AGC, as well. However, both p53 (4 vs 2, p = 0.004) and Bcl-2 (15 vs 5, p = 0.05) protein expression was higher in the NDT adjacent to advanced tumors. H. pylori positive as compared to H. pylori negative gastric mucosa showed higher both p53 (3 vs 1, p = 0.01) and Bcl-2 (15 vs 10, p = 0.05) immunoreactivity.
CONCLUSION: 1. Cell turnover is not different between NDT adjacent to EGC and AGC. 2. Both p53 and Bcl-2 protein accumulation is more intense in NDT adjacent to AGC and 3. p53 and Bcl-2 immunoreactivity is related to H. pylori infection.

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PREVALENCE OF HELICOBACTER PYLORI INFECTION IN PATIENTS WITH CORONARY HEART DISEASE. CORRELATION WITH RISK FACTORS

C.T. Tsimpidakis[1], S. Karatapanis[1], N. Papantoniou[2], P. Lisgos[1], G. Terezakis[1], A. Farmakidis[1], D. Kipraios[1], M. Kornelakis[1], K. Komnianides[1], Y. Papantoniou[2], P. Ponzetto[3]
[1]1stDept.ofInternalMedicine,[2]Dept.ofGastroenterology,RhodesGeneral Hospital,Rhodes,Greece,[3]Dept.ofGastroenterology,UniversityofTurin,Turin, Italy

INTRODUCTION: Helicobacter pylori (H. pylori) infection has been implicated in the pathogenesis of many extra-gastric conditions including coronary heart disease (CHD). However the role of H. pylori in the pathogenesis of CHD is still controversial
AIMS & METHODS: To evaluate the prevalence of H. pylori infection among patients with acute myocardial infraction (AMI) as compared with a control group of blood donors. We also tried to investigate any possible correlation of classic risk factors for AMI with H. pylori infection in those patients who were H. pylori positive and compared these findings to those in the patients without the infection. 212 males (mean age 59.3, range 44-68) with AMI were included in our study. 310 male blood donors matched for age were used as a control group. Diagnosis of AMI was based on the established WHO criteria.(symptoms, ECG changes, elevated enzymes). The presence of H. pylori was assessed by serology (ELISA). All patients completed a questionnaire which included demographic data and known risk factors for coronary heart disease.
RESULTS: In patients with AMI H. pylori was present in 187/212 (88.2%) as compared with 189/310 (59.0%) in the control group (P < 0.00001). The calculated odds ratio (OR) for AMI in patients with H. pylori was 5.19 (95%CI: 3.15-8.59), after correction by Mantel-Haenszel. This significant difference was noted in all age groups.
There were no significant differences between the two groups (AMI patients with or without H. pylori) regarding the prevalence of known risk factors for AMI (cholesterol, hypertension, smoking, fibrinogen, diabetes).
CONCLUSION: In the present study we observed a significant correlation between the prevalence of H. pylori infection and coronary heart disease. H. pylori may play a role in the pathogenesis of AMI which does not seem to be mediated by the classic risk factors.

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M1474

High Detection Rate of the Cytomegalovirus (cmv) Genome in Gastric Adenocarcinoma Tissue Samples. a Possible Role in the Pathogenesis of the Disease and the Relationship With Helicobacter Pylori (HP)

Dimitrios Dimitroulopoulos, Maria Christodoulou, Dimitris Xinopoulos, Klisthenis Tsamakidis, Aikaterini Fotopoulou, Alexandra Papadakiontopoulou, George Genimatidis, Nikolaos Apostolikas, Evi Panotopoulou, Emmanouil Paraskevas

Aim: Estimation of possible role of CMV, alone or correlated with HP, in the pathogenesis of gastric adenocarcinoma.
80 nonimmunocompromised patients (gastric cancer: 40 - premalignant lesions: 40) and 80 individuals with nonendoscopic evident gastric disease (control group), were enrolled in the study. Bioptic specimens were obtained from the malignant and the premalignant lesions, 3 cm away from the lesions and from endoscopically healthy mucosa from the patients and the control group. Polymerase chain reaction (PCR) was used to identify the CMV genome. The presence of HP was investigated with CLO-test and histological examination of mucosal gastric samples from the antrum and/or the corpus of the stomach. Multivariate statistical analysis correlated the results with epidemiological parameters which may be involved in the disease's pathogenesis.
The viral genome was detected in 11/40 (27.5%) tissue samples from malignant lesions, 15/40 (37.5%) samples from premalignant lesions, but in none of the bioptic specimens from endoscopically healthy mucosa either from patients or control group. HP was detected in 15/40 (37.5%), 24/40 (60%), 37/80 (46.2%) of each group respectively. X^2-statistical analysis revealed significant difference in the detection rate between the control population and the two patients' groups ($p<0.001$). No-significant statistical correlation was observed between the detection rate of viral genome and the detection rate of HP in the two examined patients' groups ($p = 0.999$, $p = 0.317$ respectively) and between the detection rate of CMV and the epidemiological parameters involved in gastric carcinogenesis. No-significant statistical correlation was observed between the detection rates of HP of the two patients' groups ($p = 0.074$).
Our results indicate a possible role of CMV in gastric adenocarcinoma pathogenesis as independent factor in a patients'subgroup.

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T958

Rabeprazole 7-Days Vs. Rabeprazole 10-Days Vs. Rabeprazole 14 Days Triple Therapy in the Eradication of *H.pylori* Infection-A Randomized Study.

Stylianos Karatapanis, Sotiris Georgopoulos, Lambrini Skorda, Nikitas Papantoniou, Konstantinos Komnianides, Philip Lisgos, Chrysostomos Psellas, Andreas Mentis, Haridimos Tsibidakis, Hara Kouvidou, Konstantinos Lois, Dimitrios Kipreios, Vasilios Artikis

Objective: To evaluate the efficacy and safety of three triple therapies based on rabeprazole (RAB).

Methods : 142 *H.pylori* positive patients (CLO-test, histology) (median age 48, range 18-79) with peptic ulcer (n=79) or non-ulcer dyspepsia (n=63) were randomized to receive RAB 20mg bid, Clarithromycin (CL) 500mg bid, and Amoxycillin (AMO) 1gr bid for 1 week (Group A, n=47), or RAB 20mg bid, CL 500mg bid and AMO 1gr bid for 10 days (Group B, n=48) or RAB 20mg bid, CL 500mg bid and AMO 1gr bid for 14 days. *H. pylori* eradication was assessed 4 weeks after completion of treatment (by CLO-test and histology). Clarithromycin sensitivity tests were carried out in the cultured pre-treatment (96/142, 67.6%) *H.pylori* strains.

Results : The eradication rates according to intention to treat analysis (ITT) were 35/47 (74.4%) in Group A, 38/48 (79.1%) in Group B and 43/47 (91.5%) in Group C (P<0.05 between Group A and C), and according to per-protocol analysis (PP) 37/47 (78.7%) in Group A, 40/48 (83.3%) in Group B and 44/47 (93.6%) in Group C (P<0.05 between group A and C). Primary CL resistance was 9/96 (9.4%). The eradication rate in the CL resistant *H.pylori* strains was 3/9 (33.3%), whereas in the CL sensitive strains 81/87 (93.1%) P<0.0005. Side effects in all groups were generally mild and only two patients discontinued treatment due to adverse effects (1 in Group B and 1 in Group C).

Conclusion : We conclude that 14-days triple therapy based on rabeprazole proved more effective than the 7-days regimen in the eradication of *Helicobacter pylori* infection.

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The Impact of Eradication of *Helicobacter Pylori* on the Evolution of Intestinal Metaplasia and Gastric Dysplasia

Haridimos Tsibidakis, Nikitas Papantoniou, Stylianos Karatapanis, Gerasimos Terezakis, A Farmakidis, Philip Lisgos, A Porizeno

Aim: The study aims to investigate the evolution of intestinal metaplasia and gastric dysplasia in patients after successful eradication of *helicobacter pylori* (HP).

Patients and Methods: 103 patients (61males, 42 females, mean age 65 years) with HP positive chronic atrophic gastritis were included in our study. All patients underwent upper GI endoscopy and biopsies were taken from the antrum and body of the stomach to estimate the degree of atrophy, intestinal metaplasia,(IM),and dysplasia.. The presence of HP was established by histology (Giemsa), serology (ELISA) and with the urea breath test (UBT-test). All patients received HP eradication therapy with triple regimens. Eradication was assessed 4 weeks after completion of treatment by UBT and HP histology. All patients were re-examined endoscopically at 6, 12, and 24 months after successful eradication of HP to assess the evolution of intestinal metaplasia and gastric dysplasia.

Results: At initial endoscopy we noted type I intestinal metaplasia in 74/103 patients (72%), type IIa in 7/103 (7%), and type IIb in 22/103 (21%). A slight degree of dysplasia was also observed in 15/103 patients (14%). 24 months after the eradication of HP, in the patients with IM of type I ,we found regression of IM in 11/74, a change to type IIb in 2/74, and to IIa in 3/74 while in 58/74 IM remained unchanged. In the patients with IM of type IIa we observed regression in 3/7, change to IIb in 2/7 while in 2/7 IM remained unchanged. In the patients with IM of type IIb we noted regression in 3/22, change to IIa in 3/22, change to type I in 7/22, and no change in 9/22. At 24 months after successful eradication of HP we noted disappearance of dysplasia in 14/15 patients.

Conclusions: The data of our study show that after successful eradication of HP there is regression of intestinal metaplasia and dysplasia of the stomach in a significant number of patients.

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GENETIC ANALYSIS OF CAGA AND VACA GENES OF H. PYLORI STRAINS IN CRETE

C.S. Petraki[1], V.A. Lemonomichelaki[2], I.E. Koutroubakis[3], E. Kouroumalis[3]

[1]Primary Health Care Centre of Anogia, Regional Medical Office of Zoniana, Crete, Greece, [2]Department of Social Medicine, School of Medicine, University of Crete, Heraklion, Crete, Greece, [3]Department of Gastroenterology, School of Medicine, University of Crete, Heraklion, Crete, Greece

BACKGROUND/AIM: Helicobacter pylori (H. pylori) causes different gastroduodenal pathologic conditions. Certain genotypes have been suggested to be associated with the virulence of this pathogen. The aim of this study was to investigate the frequency of H. pylori vacA and cagA genotypes and their association with gastroduodenal disease in Crete.
METHODS: Polymerase chain reaction (PCR), Clo-test and histology examination was performed in fifty gastric biopsies taken by dyspeptic patients who were undergone routine endoscopy.
RESULTS: H. pylori was found in 24/50 patients in whom infection was detected by PCR. Of the 24 cases 9 (37.5%) had vacA signal sequence s2 strains, 13 (54.17%) had s1a strains and one (4.17%) had s1b strain. One (4.17%) case contained multiple s-subtypes. VacA middle region sequences m1 and m2 were detected in 4 (16.67%) and 16 (66.67%) strains respectively. One patient (4.17%) contained multiple m-subtypes whereas in three (12.5%) cases the m-type was undetectable. CagA was present in 7/24 (29.17%) patients. The cancer strain was cagA+/vacAs1 type. There was a correlation between cagA positivity and vacAs1. There was no difference between the cagA+/vacAs1 strains and the presence or absence of ulcers.
CONCLUSION: cagA status and vacA genotyping was successful with tissue samples taken directly from gastric biopsies. The predominant vacA strains in Crete are s1a/m2 type, strains which produce moderate amounts of vacuolating toxin. The cagA status and the s1 vacA allele are unreliable as single markers in determining the risk of developing peptic ulcer disease. It is important for primary care physicians to know the genetic heterogeneity among H. pylori strains in order to select an appropriate therapy.

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RABEPRAZOLE REGIMENS FOR THE ERADICATION OF H.PYLORI

S. Karatapanis[1], M. Kornelakis, S. Georgopoulos[2], L. Skorda[2], N. Papantoniou[1], K. Komianides[1], F. Lisgos[1], C. Psellas[1], A. Mentis[3], V. Artikis[2]

[1]General Hospital of Rhodes, [2]Department of Gastroenterology, "ELPIS" General Hospital, [3]Hellenic Pasteur Institute, Athens, Greece

BACKGROUND/AIM: To evaluate the efficacy and safety of three triple therapies based on rabeprazole (RAB).
METHODS: 142 H.pylori positive patients (CLO-test, histology) (median age 48, range 18-79) with peptic ulcer (n=79) or non-ulcer dyspepsia (n=63) were randomized to receive RAB 20mg bid, Clarithromycin (CL) 500mg bid, and Amoxycillin (AMO) 1gr bid for 1 week (Group A, n=47), or RAB 20mg bid, CL 500mg bid and AMO 1gr bid for 10 days (Group B, n=48) or RAB 20mg bid, CL 500mg bid and AMO 1gr bid for 14 days. H. pylori eradication was assessed 4 weeks after completion of treatment (by CLO-test and histology). Clarithromycin sensitivity tests were carried out in the cultured pre-treatment (96/142, 67.6%) H.pylori strains.
RESULTS: The eradication rates according to intention to treat analysis (ITT) were 35/47 (74.4%) in Group A, 38/48 (79.1%) in Group B and 43/47 (91.5%) in Group C (P<0.05 between Group A and C), and according to per-protocol analysis (PP) 37/47 (78.7%) in Group A, 40/48 (83.3%) in Group B and 44/47 (93.6%) in Group C (P<0.05 between group A and C). Primary CL resistance was 9/96 (9.4%). The eradication rate in the CL resistant H.pylori strains was 3/9 (33.3%), whereas in the CL sensitive strains 81/87 (93.1%) P<0.0005. Side effects in all groups were generally mild and only two patients discontinued treatment due to adverse effects (1 in Group B and 1 in Group C).
CONCLUSION: We conclude that 14-days triple therapy based on rabeprazole proved more effective than the 7-days regimen in the eradication of Helicobacter pylori infection.

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INFECTION BY H.PYLORI AMONG PATIENTS PRESENTING WITH DYSPEPSIA

E. Rovithis, E. Markakis, M. Rovithis, N. Tsakountakis, A. Fthenakis, R. Pateli, G. Fountakis, C. Daskalaki, E. Tzortzis, N. Ploumis, C. Apostolakis, G. Varkarakis, I. Saridou, G. Balahoutis

Health Centre of Kastelli, Heraklion, Crete, Greece

BACKGROUND/AIM: To estimate the positive predictive value (PPV) for H. pylori infection of a positive answer to a simple question that denotes symptomatic dyspepsia.

METHODS: Patients with self-reported or evident (by repeated prescriptions of anti- ulcer medications) dyspepsia, formed the population of the study. Patients then asked to give either a positive or a negative (yes or no) answer to the following statement: "I often have problems with my stomach". All patients were subsequently tested for H. pylori infection using as a "gold standard" a prescription diagnostic breath test kit (HELICOBACTER INFAI ®)

RESULTS: Sixty seven patients gave a positive answer to the above statement. Twelve of them were found to be infected with H. pylori. The Positive Predictive Value of the statement was calculated to be 17.9%.

CONCLUSION: Reported sypmtoms of dyspepsia have a low PPV as a "diagnostic tool" for H. Pylori infection. Consequently primary care physicians should not base their clinical decision as to order a H. Pylori laboratory examination on the presence of dyspeptic symptoms alone.