ΞΕΝΟΓΛΩΣΣΕΣ ΑΝΑΚΟΙΝΩΣΕΙΣ (ABSTRACTS) ΕΛΛΗΝΩΝ ΕΡΕΥΝΗΤΩΝ

XVth International Workshop on Gastrointestinal
Pathology & *Helicobacter*,
11-14 Sept 2002, Athens, Greece
Gut 2002;51(Suppl II):A18

3.23 ***Helicobacter pylori*** **genotypes in the Greek population**

D.N. Sgouras[1], **M. Giannaki**[2], **A. Georgopoulou**[1], **A. Kalliaropoulos**[1], **A. Natsios**[1], **I. Panagiotou**[4], **E. Roma**[4], **S. Michopoulos**[3], **K. Kallergi**[1], **A. Pangali**[2], **A.F. Mentis**[1], [1]*Laboratory of Medical Microbiology, Hellenic Pasteur Institute, Athens, Greece,* [2]*Microbiology Laboratory "Agia Sophia" Children's Hospital, Athens, Greece,* [3]*Gastroenterology Clinic "Alexandra" Hospital, Athens, Greece,* [4]*Gastroenterology Clinic "Agia Sophia" Children's Hospital, Athens, Greece*

Aim: To genotype in terms of cagA and vacA status, *H. pylori* clinical strains, isolated from children and adults in the Greek population.

Materials and Methods: 45 clinical strains from children and 56 from adult patients were isolated from gastric biopsies following gastroscopy. The presence of cagA as well as subtyping of vacA mid-and signal-region was effected by PCR on genomic DNA as described by Atherton et al, 1999 and Yamaoka et al., 1999.

Results: 64 (83%) out of 101 strains were detected positive for the presence of cagA (86% in the adult and 80% in the children population). Out of 79 strains analysed, 52 belonged to the vacAs1 and 27 to the vacAs2 subtype (66% and 34% respectively). Out of 74 strains analysed, 23 (31%) were of the vacAm1 and 48 (65%) vacAm2 subtypes while 3 (4%) remained untypeable. The combinations of vacA subtypes observed are summarized in the following table:

	s1/m1	s1/m2	s2/m1	s2/m2	s1/m1/m2
Adults	13	10	0	12	2
Children	9	10	0	15	1
Total	22	20	0	27	3

We did not detect the vacAs2/m1 subtype in the sample population.

Conclusion: A high frequency of cagA positive strains was observed in the Greek population. There was no significant difference in the frequency between the vacA mid- and signal-region subtypes. No statistically significant differences among the subtypes were detected between the adult and the children study groups.

References

[1] Atherton *et al.* (1999) *J. Clin. Microbiol.* 37(9), 2979
[2] Yamaoka *et al.* (1999) *J. Clin. Microbiol.* 37(7), 2274

XVth International Workshop on Gastrointestinal
Pathology & *Helicobacter*,
11-14 Sept 2002, Athens, Greece
Gut 2002;51(Suppl II):A32

6.08* ***H. pylori*** **in children's dental plaque: Correlation with the** ***H. pylori*** **infection status of their parents**

Y. Kafritsa[1], P. Petropoulou[2], **A. Mentis**[3], A. Tsamis[2], D. Sgouras[3], **J. Panagiotou**[1], **E. Roma**[1]. [1]*Aghia Sophia Children's Hospital, Athens, Greece,* [2]*Periodontology Department of Athens University, Athens, Greece,* [3]*Hellenic Pasteur Institute, Athens, Greece*

Aim: The aim of the study was to investigate the presence of *H. pylori* in the dental plaque of children, who underwent endoscopy for upper GI symptoms and correlate it with the *H. pylori* infection status of their parents.

Methods: The study sample consisted of 35 children aged 4 to 14 years old and 49 family members (mother and/or father). Gastric biopsies were collected from all the children for CLO-test, histology and culture. Serology was used to assess the *H. pylori* status of the parents. Subgingival plaque samples were collected from the parents and children before endoscopy. Only samples positive by two different PCR methods were considered positive for the presence of *H. pylori* in dental plaque.

Results: Fifteen out of 35 (43%) children were considered *H. pylori* infected by at least two methods (CLO, histology, culture). *H. pylori* was detected in dental plaques of 6/15 biopsy positive children and in only 1 of the 20 biopsy negative children. The presence of *H. pylori* in the dental plaque was significantly associated with *H. pylori* detection in gastric samples from the antrum (Fisher's p=0.027). On the basis of serology, 33.3% of mothers and 45% of fathers were considered positive for *H. pylori* infection. Children who had *H. pylori* identified in their plaque belonged to families who also had *H. pylori* in dental plaque. The presence of *H. pylori* in mother's dental plaque or positive serology was associated with *H. pylori* infection in the respective child (p=0.008 and p=0.002 respectively). Although the same trend was also observed for the fathers, the difference was not statistically significant (p=0.11 and p=0.576 respectively).

Conclusions: Dental plaque of children and their families may play an important role-acting as a "reservoir" -for *H. pylori* transmission. The detection of *H. pylori* in mother's dental plaque seems to be a critical factor for the intrafamilial spread.

XVth International Workshop on Gastrointestinal
Pathology & *Helicobacter*,
11-14 Sept 2002, Athens, Greece
Gut 2002;51(Suppl II):A31

6.02* **Intrafamilial spread of** ***Helicobacter pylori*** **(*H. pylori*): A genetic analysis**

E. Roma-Giannikou[1], A. Karameris[2], B. Balatsos[3], J. Panayiotou[1], Z. Manika[4], C. Van-Vliet[1], **T. Rokkas**[5], N. Skandalis[3], C. Kattamis[1]. [1]*1st Dept. Paed. Athens University, Athens, Greece,* [2]*Laboratory of Pathology and Molecular Biology, 401 Army General Hospital, Athens, Greece,* [3]*Gastroenterology Dept, Athens General Hospital, Athens, Greece,* [4]*Histopathology Dept, Athens General Hospital, Athens, Greece,* [5]*Gastroenterology Unit, "Henry Dunant" Hospital, Athens, Greece*

Background: A high incidence of *H. pylori* among family members of children with *H. pylori* gastritis has previously been documented on biopsy material. The main objective of this study was the genetic clarification of *H. pylori* strains involved in intrafamilial dispersion.

Materials and Methods: Formalin-fixed, paraffin-embedded material of antral mucosa from 32 members of 11 families was studied for the presence of genetic homogeneity. To achieve this goal, the entire genome of *H. pylori* was studied by the RAPD fingerprinting method. Furthermore, the Urease A gene was analyzed as internal control, using a multiplex PCR-assay and the novel mutation detection method based on the Hydrolink analysis. Genetic similarities of the whole genome of *H. pylori* as well as the size and structure of Urease A gene were observed among members of the same family.

Results: Although mutations in Urease A gene were found in 22 out of 32 individuals, the RAPD fingerprinting confirmed that the same *H. pylori* strain was involved in the intrafamilial dispersion.

Conclusions: The homology of the *H. pylori* genome in members of the same family strongly supports the hypothesis of transmission of *H. pylori* from person-to-person or from a common source.

XVth International Workshop on Gastrointestinal
Pathology & *Helicobacter*,
11-14 Sept 2002, Athens, Greece
Gut 2002;51(Suppl II):A35

6.17 **The intrafamilial status of** ***H. pylori*** **infection in children with upper GI symptoms**

E. Roma[1], **Y. Kafritsa**[1], **I. Panagiotou**[1], A. Mentis[2], C. Van-Vliet[3], V. Syriopoulou[1]. [1]*Gastroenterology Unit, 1st Dept of Paediatrics, Athens University, Athens, Greece,* [2]*Hellenic Institute Pasteur, Athens, Greece,* [3]*Histopathology Dept, Aghia Sophia Hospital, Athens, Greece*

Background: *H. pylori* infection is common and its acquisition is mainly in young ages. It has been claimed that during early childhood intrafamilial contamination is more possible than from other sources outside home.

Aim: The aim of the study was to investigate the *H. pylori* status within families of children with upper GI symptoms.

Methods: 45 symptomatic children without previous eradication therapy aged 4-16 years (median 11yrs) were investigated by ^{13}C-UBT as well as all their 125 family members (parents and siblings) living in the same house. Socioeconomic status and conditions of living were also investigated.

Results: According to *H. pylori* status the index children were divided into group A: 20 (44%) who were *H. pylori* infected and group B: 25 (56%) who were negative for *H. pylori* infection. Forty four out of 57 (77%) relatives of group A were also infected, while *H. pylori* was found only in 19 out of 68 (28%) relatives of group B, (p=0.000). In group A 79% of fathers, 90% of mothers and 65% of siblings were found *H. pylori* positive, while in group B 29%, 44% and 5.2% respectively (p=0.001, p=0.01, p=0.001). There was not found any difference concerning living conditions between *H. pylori* positive and negative children.

Conclusions: The significantly higher prevalence *H. pylori* infection within families of *H. pylori* positive symptomatic children, compared to the negative ones, supports the hypothesis that transmission of *H. pylori* in children could be from the infected members of the family or the same source within house.

XVth International Workshop on Gastrointestinal
Pathology & *Helicobacter*,
11-14 Sept 2002, Athens, Greece
Gut 2002;51(Suppl II):A42

7.13 Apoptosis and cell proliferation are significantly affected by anatomic site and *Helicobacter pylori* infection of the gastric mucosa

S.D. Ladas[3], M. Chatziargyriou[1], P. Kitsanta[2], K. Triantafyllou[1], C. Barbatzas[3], S. Rapis[1]. [1]*Gastroenterology Unit, 2nd Department of Internal Medicine, Medical School of Athens, Athens, Greece,* [2]*Pathology Department of the 'Evangelismos' Hospital, Athens, Greece,* [3]*Gastroenterology Unit, Sismanogleion Hospital, Athens, Greece*

Introduction: *Helicobacter pylori* is a risk factor for gastric cancer. The association between *H. pylori* and cancer may be attributable to increased epithelial cell turnover. However, the underlying mechanisms and the response of different anatomic sites of the gastric mucosa to *H. pylori* infection remain to be defined.

Aim: To investigate the apoptosis and cell proliferation rate of different anatomic sites of gastric mucosa in *H. pylori* +ve and -ve patients.

Methods: 20 patients (M/F 10/10, age 34, 16-54 years, median, range) were studied. Six biopsies were separately obtained from the greater (GC) and lesser (LC) curvature of the antrum and corpus and the fundus and cardia. *H. pylori* status and cell proliferation were detected immunohistochemically with an anti-*H. pylori* and MIB-1 monoclonal antibodies according to the Avidin-Biotin Method. Apoptosis was measured by TUNEL method. The rate of the positive stained cells was count using image analysis technique (SABA).

Results: 11 patients were *H. pylori* –ve and 9 +ve. Median apoptotic index was significantly different among the GC 25 and LC 20 of antrum, the GC 15 and LC 13 of corpus, the fundus 12 and the cardia 15 ($P<0.001$), being significantly higher at all anatomic sites in *H. pylori* +ve patients ($P<0.001$). Median proliferation index was also significantly different among the GC 2.4 and LC 3.0 of antrum, the GC 4.9 and the LC 9.2 of corpus, the fundus 10.1 and the cardia 12.8 ($P<0.001$). However, *H. pylori* +ve patients had a significantly lower proliferation index only GC ($P=0.025$) and LC ($P=0.03$) at the corpus and the cardia ($P=0.04$).

Conclusions: Gastric cell apoptosis and proliferation are significantly affected by anatomic site and *H. pylori* infection, factors which are known to be related with gastric carcinogenesis.

XVth International Workshop on Gastrointestinal
Pathology & *Helicobacter*,
11-14 Sept 2002, Athens, Greece
Gut 2002;51(Suppl II):A44

7.23 Gastric cardia inflammation and intestinal metaplasia (IM): Role of *H. pylori* and gastroesophageal reflux disease (GERD)

N. Rosolimos[1], P. Aggeli[1], N. Sfougkatakis[1], P. Koulouris[1], K. Petraki[2], A. Nakos[1]. [1]*Hippokration General Hospital of Athens, Gastroenterology Department, Athens, Greece,* [2]*Hippokration General Hospital of Athens, Histopathology Department, Athens, Greece*

Objective: Pathogenesis of chronic inflammation and intestinal metaplasia at the gastric cardia is still unclear. We evaluated the factors involved in the pathogenesis of these histological alterations.

Methods: Totally 52 consecutive patients who underwent upper endoscopy were enrolled in the study. In each patient 2 biopsies were performed in the antrum, 2 in the gastric body and 2 at the gastric cardia in order to determine *H. pylori* infection and histological status.

Results: Overall inflammation of the gastric cardia was detected in 40 (78,4%) patients, *H. pylori* infection in 35 (68,6%), intestinal metaplasia of the gastric cardia in 9 (17,6%) and GERD in 18 (35,2%). The rate of *H. pylori* infection was not found higher in patiens who had inflammation of the gastric cardia compared to those who had not [28/40 (70%) vs 5/9 (55,6%); p>0,1], while the prevalence of GERD symptoms was also not different between the two groups [15/40 (37,5%) vs 2/9 (22,2%); p>0,1]. Regarding the intestinal metaplasia, *H. pylori* infection rate was also no higher in patients with IM compared to those without [5/8 (62,5%) vs 30/43 (69,8%); p>0,1], while no difference concerning the prevalence of GERD symptoms was found between the two groups [2/9 (22,2%) vs 16/42 (38,1%); p>0,1]. No difference in age and sex emerged between patients with or without histological alterations of the gastric cardia.

Conclusions: no relation was found between both inflammation and intestinal metaplasia at the gastric cardia and *H. pylori* infection and GERD.

XVth International Workshop on Gastrointestinal
Pathology & *Helicobacter*,
11-14 Sept 2002, Athens, Greece
Gut 2002;51(Suppl II):A55

9.12* Role of growth factors, apoptosis and COX-1 and COX-2 expression in gastric carcinogenesis - Correlation with *H. pylori* (Hp) infection

A. Karameris[1], **T. Rokkas**[2]. [1]*Dept of Pathology, 417 VA Hospital, Athens, Greece,* [2]*Gastroenterology Unit, "Henry Dunant" Hospital, Athens, Greece*

Introduction: Several epidemiologic studies demonstrated a correlation between gastric carcinogenesis and infection with Hp. The exact mechanism that is responsible for the transformation of the gastric mucosa still remains obscure. It is postulated that excessive mucosal cell proliferation may eventually result in gastric atrophy, mutation and transformation of epithelial cells in a more malignant phenotype. These processes seems to be completed by the expression of COX-2 as an inflammatory enzyme to release excessive amounts of PGE2, leading to further proliferation, reduction of apoptosis, angiogenesis and tumor growth. So the aim of this study was to investigate the possible role of Growth Factors such as TGFa and HGF in gastric carcinogenesis in correlation with Hp infection, as well as to study the expression of genes such as COX-1 and 2 and apoptosis related molecules Bax and BCL-2.

Material and Methods: Bioptic specimens and serum from 50 patients with gastric carcinoma were used. Molecular biology techniques were implicated for the study of the expression of TGFa, HGF, COX-1 and 2, and immunohistochemistry for the detection of Bax and BCL-2 proteins. Negative controls were used.

Results: 40/50 patients were positive for Hp. CagA+ strains were detected in 35/40 Hp (+) cases. HGF and TGFa are expressed more frequently in gastric carcinomas than in normal tissues (10 and 15-folds respectively). No statistically significant difference was observed in the COX-1 expression between diseased and normal tissues. On the other hand, COX-2 expression was exclusively seen among gastric carcinoma cases. Only the Bax protein correlated with 50% of the Hp (+) cancer cases.

Conclusion: Infection with Hp, especially with strains expressing CagA+, seems to be a well documented pathway in developing gastric carcinoma. Upregulation of growth factors and COX-2, and dysregulation of the Bax/BCL-2 system in Hp infected patients, may contribute to neoplastic transformation.

XVth International Workshop on Gastrointestinal
Pathology & *Helicobacter*,
11-14 Sept 2002, Athens, Greece
Gut 2002;51(Suppl II):A59

10.02 *Helicobacter pylori* related gastric carcinogenesis and immunohistochemical expression of FHIT gene product

K.H. Katsanos, A. Skopelitou, M. Michail, O. Baltayannis, E.V. Tsianos. *Medical School of Ioannina, Ioannina, Greece*

Background: Changes in the fragile carcinogenic region FRA3B of Fragile histidine triad (Fhit) gene have been proposed as an early event in gastric carcinogenesis. We investigated the immunohistochemical expression of Fhit gene product (protein) in biopsies of patients diagnosed with chronic gastritis [*Helicobacter pylori* (Hp) related or not], gastric epithelial dysplasia, intestinal metaplasia and gastric adenocarcinoma

Materials and Methods: We performed immunohisto-chemistry in archival material of formalin-fixed, paraffin-embedded tissues of 135 gastric biopsies (76 endoscopical and 59 surgical), using the anti-Fhit antibody (rabbit anti-Fhit polyclonal antibody, ZYMED) and the streptavidin-biotin peroxidase method (Dakopatts). Biopsies from normal gastric epithelium were used as controls. Depending on the strength of staining for Fhit all biopsies were characterized as negative (0) up to strongly positive (3+) ones. Statistical analysis was performed with Fischer's exact test and McNemar's x2 test.

Results: Total absence or minimal expression of Fhit protein was noticed in 79% of Hp positive(+) gastritis, in 76.4% of chronic gastritis with low or high grade dysplasia and in 56% of gastric adenocarcinomas. The absence of Fhit protein expression correlated with the presence of Hp ($p=0.0001$) and epithelial dysplasia ($p=0.01$) but not with enteric metaplasia. Moreover, a correlation was noticed between the absence of Fhit expression and the infiltrating phenotype of gastric adenocarcinoma ($p=0.02$) as well as with its histological and clinical staging ($p=0.01$).

Conclusions: Our results strongly indicate that absent or reduced expression of Fhit protein in Hp(+) gastritis or chronic gastritis with dysplasia, may be an early event in gastric carcinogenesis, probably through direct or indirect changes in the fragile region of the Fhit gene.

XVth International Workshop on Gastrointestinal
Pathology & *Helicobacter*,
11-14 Sept 2002, Athens, Greece
Gut 2002;51(Suppl II):A60

10.05 Inflammation and intestinal metaplasia of cardiac mucosa in patients with duodenal ulcer disease and reflux esophagitis

S. Michopoulos[1], K. Petraki[2], M. Sotiropoulou[3], A. Natsios[1], N. Katsakos[1], G. Manthos[1], G. Stamatis[1], D. Sgouras[4], N. Kralios[1], A. Mentis[4]. [1]*Gastroenterology Unit of 'Alexandra' Hospital, Athens, Greece,* [2]*Pathology Unit of 'Metropolitan' Hospital, Athens, Greece,* [3]*Pathology Unit of 'Alexandra' Hospital, Athens, Greece,* [4]*Microbiology Department of Pasteur Institute, Athens, Greece*

Aim of the study: To evaluate possible differences in inflammation and intestinal metaplasia in patients with duodenal ulcer (DU) and reflux esophagitis (RE).

Patients and Methods: 90 patients with DU, all Hp(+) and 98 patients with RE, 54 Hp(+) and 44 Hp(-) were enrolled in the study. Biopsy specimens obtained from gastric antrum (A), fundus (F) and cardia(C) were examined with Heamatoxylin-Eosin, modified Giemsa, PAS-Alcian Blue and HID-Alcian Blue stains. The grade and activity of inflammation were evaluated according to updated Sydney System. Serology for CagA characterization was performed in all Hp(+) patients. Stat: chi-square.

Results: The number of patients with the different degrees (0/1/2/3) of the grade and the activity of inflammation as well as the presence and the extent of intestinal metaplasia (IM) are shown in the table. CagA seropositivity was 90% for DU patients but only 40% for REHp(+).

	Grade	Activity	IM
DU	0/16/40/34	4/14/32/40	64/16/10/0
p	*	**	NS
REHp(+)	2/12/34/6	7/17/24/6	38/6/8/2
p	**	**	NS
REHp(-)	2/28/14/0	34/8/2/0	32/10/2/0

* p<0.01, ** p<0.001.

Conclusions: 1) The grade and activity of the inflammation of the cardia are more severe in DU than in REHp(+) patients. 2) The grade and activity of the cardia inflammation are more severe in REHp(+) than in REHp(-) patients. 3) There is no difference in the presence of IM neither between DU and REHp(+) nor between REHp(+) and REHp(-) patients. 4) the prevalence of CagA seropositivity is higher in DU than in REHp(+) patients

---

XVth International Workshop on Gastrointestinal
Pathology & *Helicobacter*,
11-14 Sept 2002, Athens, Greece
Gut 2002;51(Suppl II):A94

15.15 *Helicobacter pylori* infection in upper gastrointestinal bleeding in patients with hereditary hemorrhagic disorders

N.P. Eleftheriadis[1], S. Makri[2], C. Aggouridaki[3], K. Pistevou-Gompaki[4], P. Makris[2]. [1]*Aristotles University of Thessaloniki, Thessaloniki, Greece,* [2]*Aristotles University of Thessaloniki, Hemostatic Unit of The First Propedeutic Dept, Greece,* [3]*Aristotles University of Thessaloniki, Immunological Laboratory, Greece.* [4]*Aristotles University of Thessaloniki, AHEPA Hospital, Greece*

Background: In patients with hereditary hemorrhagic disorders (HHD) upper gastrointestinal bleeding (UGB)presents a life-threatening complication, while the role of *Helicobacter pylori* (HP) infection in these patients has not been fully clarified in the literature. The aim of this study was to evaluate the role of HP infection and dental status in UGB in patients with HHD.

Methods: Thirty-seven patients with HHD, mean age 42±16 years, [18 patients (17 males, one female) and 19 patients (12 males, 7 females) with and without history of UGB respectively], and 52 patients without HHD, who came for elective gastroscopy, due to dyspeptic symptoms, mean age 61±16, 32 males, 20 females, induced into the study. Endoscopy was performed to all patients with UGB and to controls. ELISA was used to detect 1) IgG (Pharmacia& Upjohn, normal<10U/ml) 2) anti-CagA (RADIM, normal<10 RU/ml) and 3) IgA (Novum Diagnostica, normal<20 NU/ml) serum and saliva HP-antibodies of patients and controls. Moreover, dental status was examined using the decayed/missing/filled teeth index (DMFT) in patients and controls. χ^2-test was used for statistical analysis.

Results: 24/37 (64,8%) patients and 34/52 (65,4%) controls had HP-IgG serum antibodies positive (p>0,1, NS), while 20/37 (54,05%) patients and 18/52 (34,6%) controls had serum anti-CagA positive (NS). However, 15/18 (83%) and 5/19 (26,3%) HHD patients' with and without UGB respectively, had serum anti-CagA positive (p<0,01), while serum HP-IgG was positive in 13/18 (72%) and 11/19 (58%), respectively (NS). Furthermore, saliva HP antibodies and the DMFT calculated index did not differ between the two subgroups.

Conclusions: Although there was not found statistically significant difference in HP infection between HHD patients' and controls, CagA strain appeared more frequently in those hereditary bleeding patients' with history of HGB. According to our results and in combination with the increased bleeding complications, anti-CagA screening and therapy is recommended to all patients with HHD.

XVth International Workshop on Gastrointestinal
Pathology & *Helicobacter*,
11-14 Sept 2002, Athens, Greece
Gut 2002;51(Suppl II):A62

10.13 Evolution of endoscopic incidence of gastroduodenal ulcer (GDU) and esophagitis (E) after the wide application of *Helicobacter pylori* (Hp) eradication regimens

S. Michopoulos[1], M. Sotiropoulou[2], K. Petraki[3], A. Natsios[1], N. Katsakos[1], G. Manthos[1], G. Stamatis[1], M. Zisis[1], N. Kralios[1]
[1]*Gastroenterology Unit of 'Alexandra' Hospital, Athens, Greece,* [2]*Pathology Unit of 'Alexandra' Hospital, Athens, Greece,* [3]*Pathology unit of 'Metropolitan' Hospital, Athens, Greece*

There are several recent reports suggesting a decrease in the incidence of GDU. The aim of our study was to evaluate if there are changes in the prevalence of endoscopic diagnosis of GDU or E after the wide application of Hp eradication regimens.

Patients & Methods: We analyzed all the endoscopic reports and related files of all the patients who had an UGI endoscopy during the years 1993, 1997 and 2001. Our department is situated in a central hospital of Athens, it belongs to the National Health System (NHS) and it has an open access for all beneficiaries of NHS.

Results: Total number of fiberoscopies (UGI), gastric/duodenal ulcers (GDU) and E with the corresponding LA classification (A/B/C/D) are shown on the table. The groups were comparable for age, tobacco and alcohol consumption. Valuable information on Hp status in patients with endoscopic lesions were available in only 58% during 1993, while this increased dramatically during 1997 (96%) and 2001 (97%). Among them the % of Hp(+) patients was 50%, 42% and 31% for E, 77%, 65% and 73% for GU and 90%, 89% and 85% for DU for the 3 periods respectively. NSAID's consumption was not modified according to the files' data. Complications decreased only in 2001 (205, 196, 68).

	UGI	GDU	E
1993	2170	148/243	75/38/10/3
1997	2576	94/293	66/43/7/2
2001	2407	90/323	96/70/11/2

Conclusions: Since 1993, 1) The total number of UGI procedures has not changed, 2) The accurate control on Hp status has dramatically increased, 3) Neither the prevalence of GDU and E at endoscopy nor their Hp status were modified, 4) Non variceal hemorrhage decreased during 2001, regardless of the same NSAID's consumption.

XVth International Workshop on Gastrointestinal
Pathology & *Helicobacter*,
11-14 Sept 2002, Athens, Greece
Gut 2002;51(Suppl II):A102

15.48 Omeprazole plus azithromycin and either tinidazole or amoxycillin for the eradication of *Helicobacter pylori* infection

G. Anagnostopoulos, P. Kostopoulos, G. Margantinis, S. Tsiakos, D. Arvanitidis. 251 *Air Force and Veterans Hospital, Athens, Greece*

Introduction: Rising antimicrobial resistance is a major determinant of *Helicobacter pylori* eradication regimens' failure in addition to other factors such as poor compliance,cagA negativity etc. Thus, shortening of treatment and the use of alternative antimicrobial agents remains the goal of treatment strategies. Azithromycin is an attractive agent for the eradication of *H. pylori* due to its excellent inhibitory activity and long biological half-life.

Materials and Methods: We prospectively investigated 160 outpatients with dyspeptic symptoms who were referred for upper gastrointestinal endoscopy. *Helicobacter pylori* status was determined by rapid urease test and histological assessment. The patients were randomized to receive A) Omeprazole 20 mg bid and amoxycillin 1 gr bid for 7 days plus azithromycin 500 mg od for the first 3 days of the therapy (OAzAm group) or B) Omeprazole 20 mg bid for 7 days plus azithromycin 500 mg od and tinidazole 500 mg bid for the first 3 days of the therapy (OAzT group). Five to six weeks after concluding the therapy eradication was assessed by endoscopy and ^{13}C-urea breath test and was defined as negative results on both tests.

Results: *H. pylori* was eradicated in 50 of 80 patients of OAzT group (ITT 62.5%)and in 57 of 80 patients of OAzAm group (ITT 71.2%). The difference was not statistically significant.

Treatment results

	OAzT group	OAzAm group
No of patients	80	80
No of drop-out	0	0
Cure rates		
PP%(a)	62.5%(50/80)	71.2%(57/80)
95% CI	54-71%	63.2-79.1%
ITT%(a)	62.5%(50/80)	71.2%(57/80)
95% CI	54-71%	63.2-79.1%

Conclusion: The eradication rates achieved with the above regimens were low. Possible reasons for the sub-optimal eradication rates could be connected with antimicrobial resistance. It seems that no ideal therapy exists that leads to a 100% eradication rate. In the future bacterial resistance may increase more and lead to a big problem. Further studies are needed to compare the efficacy of short-term regimens for *Helicobacter pylori* infection and to evaluate the role of different proton pump inhibitors.

10th United European Gastroenterology Week (UEGW)
19-23 October 2002, Geneva, Switzerland,
Gut 2002;51(Suppl III):A209

PREVALENCE OF HELICOBACTER PYLORI INFECTION IN PATIENTS WITH REFLUX OESOPHAGITIS

A.A. Gatopoulou, K. Mimidis, G.I. Leontiadis, N. Lirantzopoulos, G.I. Minopoulos
Endoscopy Unit, Department of Surgery, Demokritus University of Thrace, Alexandroupolis, Greece

INTRODUCTION: The role of Helicobacter pylori (HP) in oesophagitis is still undefined. It has been suggested that it may be protective against oesophagitis.

AIMS & METHODS: We intended to evaluate the relationship between HP prevalence and oesophagitis in endoscopy, as well as between HP and histological diagnosis. In this prospective study 50 consecutive patients were subjected to upper gastrointestinal endoscopy for the evaluation of reflux symptoms. The presence of HP was estimated by CLO test. Lugol chromoendoscopy-directed biopsies of the oesophagus were taken in all cases.

RESULTS: The overall prevalence of HP in 50 GORD patients was 35/50 (70%). Among the 50 patients: 29 (58%) had normal endoscopic findings (presence of HP in 20/35, 57.1%; absence of HP in 9/15, 60%; p=ns),13 patients (26%) had grade A oesophagitis(HP+ in 11/35, 31.4%; HP- in 2/15, 13.3%; p<0,05), 6 patients (12%) had grade B oesophagitis (HP+ in 2/32, 5.7%; HP- in 4/15, 26.7%; p<0.05), 2 patients (4%) had grade C oesophagitis (HP+ 2/35 5.7%, HP- 0%). Furthermore, the overall prevalence of HP in biopsies was 34/48 (70.8%). The histological diagnosis were as follows: 2/48 (4.2%) normal (2/34, 5.9% HP+; 0/14HP- 0%;p=ns), 11/48 (22.9%) low grade oesophagitis (8/34 23.5% HP+; 3/14 21.4% HP-; p=ns),35/48 (72.9%) high grade oesophagitis (24/34 70.5% HP+; 11/14 78.5% HP-;p=ns)

CONCLUSION: A statistically significant correlation was observed between the presence of HP and grade A oesophagitis in endoscopy, as well as between absence of HP and grade B oesophagitis. All cases of grade C oesophagitis were HP(-). There was no correlation between histological diagnosis and HP presence. The aforementioned probably indicate that the absence of Helicobacter pylori may lead to more severe oesophagitis in endoscopy.

10th United European Gastroenterology Week (UEGW)
19-23 October 2002, Geneva, Switzerland,
Gut 2002;51(Suppl III):A210

APOPTOSIS AND EXPRESSION OF THE REGULATORY PROTEINS BCL-2, BCL-X AND CYCLIN-D1 IN HELICOBACTER PYLORI INDUCED GASTRITIS

J.A. Karagiannis[1], **N. Giannakou[2]**, K.D. Paraskeva[1], P. Mathiou[2], P. Kitsanta[2], A. Tsinga[2], G. Gaga[2], D. Theodosiou[2], C. Spiliadi[2]
[1]Gastroenterology Unit, [2]Histopathology, "Agia Olga" Hospital, Athens, Greece

INTRODUCTION: Several cell cycle regulatory proteins have been studied extensively in a wide range of diseases of the gastrointestinal tract because of their regulatory effect on cell proliferation and particularly for their implication in carcinogenesis.

AIMS & METHODS: Aim of the study was to assess apoptosis and the expression of the cell cycle regulatory proteins Bcl-2, Bcl-x and Cyclin D1 in patients with HP gastritis before and after successful HP eradication. Multiple endoscopic biopsies from the body and antrum of the stomach from 30 adult patients with histologically confirmed HP gastritis were studied. Apoptotic activity was assessed with the TUNEL method and Bcl-2, Bcl-x and Cyclin D1 expression with immunoperoxidase staining. Bcl-x expression was studied before HP eradication only while 10 patients with reactive NSAID-induced gastritis were used as controls for Bcl-x expression.

RESULTS: Mean value of apoptotic index before and after HP eradication was 3.5% (0.6-11.7%) and 12% (4.8-32.9%) for the body and 5.02% (2.03-19.29%) and 7.4% (1.9-16%) for the antrum respectively (p<0.05). Bcl-2 expression was observed in the antrum only in 8/30 (27%) and 26/30 (87%) patients before and after HP eradication respectively (p<0.05). Cyclin D1 expression was found in ~1% of cells, both before and after HP eradication, being increased (in ~5-10% of cells) only in foci of regenerative activity. Bcl-x expression was observed in 10/10 (100%) patients with Grade 3 and in 6/8 (75%) with Grade 2 gastritis and G3 and G2 HP load respectively. All patients (12/12, 100%) with mild (Grade 1) gastritis were negative for Bcl-x expression as were the 10/10 (100%) control patients with NSAID-induced reactive gastritis.

CONCLUSION: Although if the number of patients studied is relatively small, it seems that after successful HP eradication: a) apoptotic activity remains increased, particularly in the body of the stomach, b) expression of the anti-apoptotic protein Bcl-2 is statistically significantly increased in the antral mucosa only, c) cyclin D1 expression remains unchanged and d) Bcl-x expression is associated with high HP load and severe gastritis, being negative in reactive (NSAID-induced) gastritis.