ΞΕΝΟΓΛΩΣΣΕΣ ΑΝΑΚΟΙΝΩΣΕΙΣ (ABSTRACTS) ΕΛΛΗΝΩΝ ΕΡΕΥΝΗΤΩΝ

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EXPRESSION OF P16^{INK4} ON GASTRIC EPITHELIAL CELLS IN H PYLORI POSITIVE GASTRITIS BEFORE AND AFTER ERADICATION OF H PYLORI.

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Background: *H pylori(Hp)*infection induces hyperproliferation of the gastric epithelium that is considered a major factor for gastric carcinogenesis.*INK4* is a putative tumor suppressor gene and its product, *p16^{INK4}*, inhibits the kinase activity of the cdk4/cyclin D complex blocking the progression of the cell cycle. Aim: To evaluate the expression of *p16^{INK4}* on gastric epithelial cells in *Hp* gastritis before and after *Hp* eradication. Patients-Methods:Thirty patients with dyspepsia with and without ulcers were prospectively studied. At least 4 biopsies were obtained on endoscopy (antral:2,corpus:2)for CLO-test and histology(H&E, Giemsa, Alcian blue for Sydney classification). Formalin-fixed paraffin embedded tissue sections were stained by the ABC immunoalkaline phosphatase method.Rabbit polyclonal anti-*p16* (Santa-Cruz)was used. Staining intensity was graded from 0 to 2+. 23 patients were Hp(+)by both methods and 7 Hp(-)(controls).Hp eradication therapy was given to Hp(+) and all patients were endoscoped 116±9 days after; biopsies were obtained and treated as previously.Results:Hp was eradicated in 20 of 23 patients.*Normal mucosa:* Cytoplasmic staining for *p16^{INK4}* was observed on epithelial cells (proliferation zone 1+, surface epithelium 1+)with less or no stain on epithelial cells between these zones;no staining was observed on glandular epithelium.*Hp gastritis:*Intense(2+)cytoplasmic staining on epithelial cells in the proliferation zone with nuclear staining in some of them;surface epithelium and glandular cells stained like normal.*After Hp eradication* the staining pattern became like the normal mucosa.In gastric pits with intestinal metaplasia, intense(2+), only cytoplasmic staining was observed before and after eradication.Conclusions:Significance of cytoplasmic staining for *p16^{INK4}* remains unclear but staining of epithelial cells in the proliferating zone of normal mucosa suggests association with the proliferation of normal cells;expression on surface epithelium is compatible with the observation of *p16^{INK4}* accumulation in senescent cells. In Hp gastritis the pattern of staining for *p16^{INK4}* seems reasonable:substrate partners (cdk4/cyclin D,pRb)are in the nuclei and G1 arrest is needed for DNA repair mechanisms.In intestinal metaplasia,loss of nuclear *p16^{INK4}* expression may confer to the intense growth potential of these cells. *Supported by grant 70-3-2945/95ED-1909PENED/KA,Greek Ministry of Industry,Energy,Technology

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NSAIDS USE DOES NOT AFFECT H. PYLORI (HP) ERADICATION RATE IN PATIENTS WITH DUODENAL ULCER (DU) OR EROSIVE DUODENITIS (ED) WHEN TRIPLE THERAPY IS USED.

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NSAIDs use could reduce the efficacy of eradication regimens in curing HP infection. The aim of our study was to evaluate if HP eradication rate is affected by NSAID s use in patients with DU or ED, when a triple eradication regimen is used. Patients and methods: 315 patients (220 DU-95 ED, mean age 51.7±14.3 years, 175 men, 105 smokers, 125 on NSAIDs treatment at least 3 times a week), all HP positive, received a 10 days eradication regimen with omeprazole 20mg bid, clarithromycin 500mg bid, amoxycillin 1g bid. Of those initially receiving NSAIDs : 57 continued NSAIDs during eradication treatment (Group I), while 68 discontinued them (Group II). All patients initially not receiving NSAIDs did not receive NSAIDs during eradication treatment (Group III, n=190). Endoscopy was performed 4-6 weeks after treatment completion. HP eradication was verified by histology, CLO-test and ^{13}C-urea breath test. Stat: X^2 test, t-test. Results: 295 patients had a follow up endoscopy. 20 lost during follow-up, 6 discontinued treatment prematurely. Patients of Group III were younger. More smokers were included in group II. Intention to treat eradication rates were: Group I: 80.7%(CI 70.1-91.3). Group II: 79.4% (CI 69.6-89.3). Group III: 83.7%(CI 78.4-89). p>0.1. Per protocol eradication rates were: Group I: 85.2%(CI 75.4-95). Group II: 84.4% (CI 75.2-93.5). Group III: 89.8%(CI 85.3-94.3). p>0.1. There was no difference in eradication rates among the 3 groups when patients with DU and ED or smokers and non-smokers were examined separately. Conclusions: NSAIDs use seems no to affect HP eradication rates, when a triple regimen comprising omeprazole, amoxycillin and clarithromycin is received.

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HELICOBACTER PYLORI PHENOTYPES IN SYMPTOMATIC H PYLORI(+)PATIENTS AND ASYMPTOMATIC CONTROLS: CORRELATIONS WITH GASTRITIS PARAMETERS.

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Background: It has been suggested that differences in *Helicobacter pylori(Hp)*phenotypes, mainly of the Cag A, contribute to the virulence of the *Hp* and consequently to the outcome of the infection. Aims:1.To study the *Hp* phenotypes in dyspeptic *Hp(+)*patients in comparison with *Hp(+)* asymptomatic controls. 2. To investigate any correlation between the *Hp* phenotypes and the parameters of gastritis(activity,chronic inflammation-,atrophy,intestinal metaplasia,Hp density- Sydney classification). Patients and methods: a.47 dyspeptic, *Hp(+)* by CLO and histology, patients(age: 50.62±2.04SE)with duodenal ulcer(38), gastritis(5)and gastric and duodenal pathology(4); b.20 asymptomatic, *Hp(+)* by serology blood donors (age: 46.95±2SE)as controls. *Hp* phenotypes were studied in the sera using Western-blot (SORIN-IgG) for the following proteins: Cag A(p120), Vac A(p87), Urease (p63), Flagelins (p57, specific p54). In 29/47 patients (age: 50.14±2.59SE), additional biopsies from the gastric antrum and corpus were evaluated for gastritis (Sydney classification- H&E, Giemsa, Alcian blue). Results: No difference was found in the distribution of Cag A, Vac A and Urease in patients and controls. However, significant differences were found for the flagelin proteins p54 (36% vs 10%, p=0.038) and p57 (81% vs 55%, p=0.039)(table). No correlation was found between phenotypes and the gastritis parameters, as were graded. Conclusions: 1.There is significant overpresentation of flagelin proteins in patients. Flagelae contribute to the mobility of Hp and by extention to its virulence. 2. Hp phenotypes do not seem to correlate with the parameters of gastritis.

Hp phenotypes in patients and controls (percentage in parentheses)

Phenotypes	Patients (n=47)	Controls (n=20)
Cag A (+)	39/47 (83)	17/20 (85)
Vac A (+)	35/47 (76.6)	12/20 (60)
Urease (+)	42/47 (89.4)	17/20 (85)
(*) Flagelin p54	17/47 (36)	2/20 (10)
(**) Flagelin p57	38/47 (81)	11/20 (55)

(*) p=0.038, (**) p=0.039 (Fisher's exact test)

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EVALUATION OF A RAPID, NEW TEST (TESTPACK PLUS™) FOR DETECTING SERUM ANTIBODIES TO *HELICOBACTER PYLORI*.

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To initiate a "test and treat" strategy, rapid, one step, reliable, inexpensive serum tests are needed for diagnosis of *H. pylori* infection. We have evaluated the performance characteristics of the new TestPack Plus™ *H. pylori* test (Abbott Laboratories), which is a one minute qualitative test for the detection of antibodies to *H. pylori* in serum. Methods: In a prospective study 130 dyspeptics and patients with upper GI bleeding were studied. Patients were defined *H. pylori* (+) if two out of three reference tests (histology, rapid urease test, Gram staining of biopsy smears) were positive and *H. pylori* (-) if all three tests were negative. The results of TestPack Plus™ were compared with those of commercially available enzyme-linked immunosorbent assay (ELISA) kits qualitatively detecting IgG *H. pylori* serum antibodies (Pyloriset, Orion, Milenia *H. pylori*, DPC). Results: Seven patients had only one positive out of three reference tests and were excluded. Out of 123 patients (78 males, 45 females, 50, 17-85 years old) 95 were defined *H. pylori* (+) and 28 *H. pylori* (-) by the gold standard. Performance characteristics of the kits studied as compared with the gold standard are shown in Table 1. Conclusions: The sensitivity and specificity of TestPack Plus™ one step serum test are lower as compared to those of serum ELISA. However, its high positive predictive value makes it a valuable tool for in-laboratory screening purposes.

Table 1.

	Sensitivity	Specificity	PPV	NPV	ODA
TestPack	95	75	92	80	89
Pyloriset	92	82	95	74	89
Milenia	94	72	92	78	90

PPV: positive predictive value
NPV: negative predictive value
ODA: overall diagnostic accuracy

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DEVELOPMENT OF ESOPHAGITIS AFTER *H.PYLORI* ERADICATION: IMPACT OF CAGA STATUS.

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There is increasing evidence that *Helicobacter pylori (H.pylori)* eradication might predispose to refux esophagitis(RE). However, the role of CagA status in the development of RE after *H.pylori* eradication has not been studied so far. The aim of this prospective study was to examine the impact of CagA status in relation to the appearance of RE after successful *H.pylori* treatment. Methods: 50 consecutive, successfully eradicated patients, without RE at the time of *H.pylori* treatment [25 duodenal ulcer(DU) and 25 non-ulcer (NU), 30M, 20F, median age 41.5 yrs, range 21-57] were followed up for 24 months. All patients underwent upper GI endoscopy before and at 0, 6, 12, 18 and 24 months after *H.pylori* eradication or when reflux symptoms occurred. During endoscopy gastric, antral and corpus biopsies were taken for gastritis evaluation. The sum of the scores for acute [0-3 (0= absent, 3= severe)] and chronic inflammation (0-3) comprised the total gastritis score. CagA was determined by immunoblotting the sera from patients against *H.pylori* antigens. The independent variables CagA status, age, sex, smoking, alcohol intake, gastritis scores of antrum and corpus gastritis before *H.pylori* eradication, were introduced in a multivariate logistic regression analysis (MLRA) with RE during follow up as a dependent variable. Results: Among the 50 *H.pylori*(+) patients before eradication there were 22 (44%) CagA(+) patients and 28(56%) CagA(-). During follow up 12/50(24%) patients developed RE and among them there were 11 CagA(+) and 1 CagA(-) patient (p=0.0002, odds ratio 27, 95% CI 3.1-235.1). In the MLRA except for CagA(+) status, the severity of corpus gastritis before eradication exerted a significant influence (0.045) on the development of RE after *H.pylori* eradication. Conclusion: CagA(+) status and severity of corpus gastritis before *H.pylori* eradication significantly favor the development of RE after successful *H.pylori* treatment.

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DIETARY, DRINKING AND SMOKING HABITS IN PATIENTS WITH DUODENAL ULCER; A CASE-CONTROL STUDY.

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Aim: To investigate the dietary, drinking and smoking habits in patients with active duodenal ulcer(DU) as compared to healthy controls. Patients-methods: 100 consecutive patients (men = 73, age: 19-84 y, mean ± SD = 48 ± 15) with endoscopically, for the first time, diagnosed DU.100 clinically healthy people without past history of peptic ulcer, absolutely matched with DU patients with regard to sex,age,and various socioeconomic parameters (income,education,area of residence,and number of rooms during the last 10y)served as controls.We studied:1.Dietary,drinking and smoking habits using suitable questionnaire;71 kinds of food grouped in 9 categories[1] were included 2.Obesity index(Obi)as estimated from the people's height and body weight 3.Hp status by serology(ELISA Ig-G).Conditional regression analysis was used for statistics.Odds Ratios (OR)were calculated after appropriate adjustments. Results:Prevalence of Hp was higher in DU patients(OR=4.31,95%CI=1.86-9.95,p=0.0006).No differences in the Hp status in relation to the studied socioeconomic factors were found in patients and controls.Smoking did not differ. Patients' height and body weight differed between DU and controls (OR=0.49, 95% CI=0.34-0.72, p<0.001 and OR=0.58,95%CI=0.41-0.81,p<0.002 respectively);Obi did not differ(p=0.06,OR=91%,95%CI=0.82-1.00).Coffee drinking did not differ,by and large,but significantly more "Greek coffee" was consumed by patients (p=0.004,OR=1.59,95%CI=1.16-2.19).In general, patients consumed significantly more spirits and controls more wine.Controls consumed significantly more legumes and light lipids including olive oil (p=0.01, OR=0.69, 95% CI=0.51-0.93 and p=0.05, OR=0.70, 95% CI= 0.49-1.01) and more potatoes (OR=0.91, p=0.77) and vegetables (OR=0.86, p=0.41). Patients consumed more cereals (OR=1.23, p=0.17), fruits (OR=1.94, p=0.0004), pastries (OR=1.29, p=0.11), meat+fish+eggs (OR=1.19,p=0.26) and dairy products(OR=1.47, p=0.03). Conclusions: 1.Obi index and smoking did not differ in patients and controls 2.Patients consumed significantly more "Greek

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REDUCED INCIDENCE OF THE NON-NSAIDS RELATED PEPTIC ULCER IN THE LAST YEARS BUT THE *HELICOBACTER PYLORI* IS STILL HERE: A 9.5 YEARS EXPERIENCE.

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Background:It has been suggested that the incidence of Peptic Ulcer(PU)is declining during the last years. Aims:α.To evaluate the incidence of non-NSAIDs related PUs in three periods through the last 9.5 years of application of *H pylori(Hp)* eradication therapies.β.To see the prevalence of Hp among them. Patients-Methods: A computer search was made from May 1st 1990 through October 31st 1999 to find out all patients with endoscopically diagnosed for the first time PUs in our institution; emergency endoscopies were also included. Patients who reported NSAIDs or aspirin intake, those who ingested caustic substances and patients with Crohn's were excluded.Three time periods were analysed.A: May 1 1990-December 31 1991 (20 months). B: May 1 1995- December 31 1996(20 months).C: January 1 1999- October 31 1999(10 months).Gastric *Hp* colonization was documented by CLO test and/or histology (H&E, modified Giemsa). Results: These are shown in the table.There is a progressive and significant reduction (up to 50%) in the incidence of PU's (DUs and GUs).The *Hp* (−) cases do not differ in the three studied periods(90-92%). Conclusions:α.There is a steady declining incidence of non- NSAIDs related DUs and GUs during the studied period. β. *Hp* colonization is constantly documented in 90-92% of the ulcers by the used methods.Thus,in spite of the significant decrease of PU's in the studied population, *Hp* remains the predominant factor in the pathogenesis of non-NSAIDs related PUs.

Incidence of PUs in each period of time (percentages in parentheses)

	Period A	Period B	Period C
Patients	7479	7475	2594
(*)PUs	904 (12)	680 (9)	151 (6)
Sex(M/F)	613/286	442/239	129/75
Age(range)	16-86	15-85	16-92
(**)DUs	589 (7.9)	461 (6.2)	97 (3.7)
(***)GUs	315 (4.2)	219 (2.9)	54 (2.4)
Hp (+) cases (%)	91	90	92

(*) (**) (***) p<0.0001

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INCIDENCE OF *HELICOBACTER PYLORI (HP)* INFECTION IN PATIENTS WITH RHEUMATIC DISEASES.

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Objective: To evaluate the seroprevelance of Helicobacter pylori (*Hp*) infection in patients(pts) with rheumatic diseases *Methods:* Serum samples of 62 pts with rheumatoid arthritis (RA), 20 pts with systemic lupus erythematosus(SLE),18 pts with seronegative spondylitis(SNS), 32 pts with ostearthritis(OA) and 36 volunteers (V) was measured for IgA and IgG antibodies against *Hp* by enzyme immunoassay (serion elisa classic). Positive were titers>30 U/ml. Western blotting was used as confirmation. No subject had received *Hp* eradication treatment, gold or sulphasalazine treatment in the past. Concurrent use of NSAIDs, steroids and antirheumatic drugs was recorded. Comparison and statistical analysis in incidence(%)and average IgA and IgG titers(M) between the subjects was performed using the x^2 test and Student's *t* test. *Results:* Comparing % and M: a) RA, SLE, SNS, OA pts to V and b) RA to OA pts, as shown in the Table revealed: 1)a greater incidence in RA pts than V for IgA(p=0.0017)and IgG(p=0.0184). 2)a greater incidence in OA pts than V(p=0.0000)for IgA antibodies. 3)a higher average titer in RA pts for IgA(p=0.0057)and IgG(p=0.0260) than V. 4)a higher average titer in OA for IgA(p=0.0338) than V. Comparison between SLE or SNS pts with V did not appeared statistically significant. *Discussion:* Patients with rheumatoid arthritis show a significantly higher *Hp* seropositivity and higher titers of IgA and IgG antibodies than healthy volunteers, whereas patients with osteoarthritis show significantly higher Hp seropositivity only for IgA antibodies. No difference in seropositivity was found between RA and OA patients. Our observations provide evidence that incidence of *Hp* infection is higher in these patients.

	RA	SLE	SNS	OA OA OA n=32	V
IgA	34/62	7/20	7/18	17/32	8/36
%	55.72	35	38.89	53.12	22.23
M ± SEM	51.2 ± 5.5	37.2 ± 10	33.4 ± 6.3	50.1 ± 7.7	30.3 ± 4.9
IgG	41/52	9/20	11/18	19/32	15/36
%	66.12	45	61.11	59.37	41.67
M ± SEM	71.0 ± 9.9	41.6 ± 10.3	44.8 ± 9.1	59.0 ± 9.3	42.2 ± 6.6

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DIAGNOSIS OF HELICOBACTER PYLORI INFECTION WITH A NEW NON-INVASIVE STOOL ASSAY BEFORE AND AFTER ERADICATION TREATMENT; THE REAL LIFE.

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Background:It has been suggested that a new H pylori(Hp)stool antigen EIA(HpSA Premier Platinum,Meridian Diagnostics, Cincinnati, USA)is reliable, easy to perform tool to diagnose Hp infection. However, after eradication of Hp the results are controversial. Aim: To assess the accuracy of this stool assay in diagnosing Hp infection before and after eradication, in comparison with the 2 mostly used in practice methods i.e. CLO test and histology. Patients-Methods:Two groups of patients with dyspepsia with and without ulcer were prospectively studied. Group A:43 consecutive adult patients who were examined for the first time: *exclusion criteria:* any treatment for Hp previously, any antibiotic in the previous 6 weeks, active GI bleeding, current treatment with corticosteroids or NSAIDs, with PPI or bismuth containing compounds during the previous 3 months or previous gastric surgery. Group B: 18 adult patients with no gastric operation or active GI bleeding who completed an eradication treatment and had not received PPIs. H_2RA, bismuth salts, antibiotics, corticosteroids, NSAIDs for the last month. Patients were endoscoped and 4 biopsies were taken (2 antrum, 2 corpus) for histology (H&E, Giemsa stain)and CLO test; patients were considered Hp(+)if either test was positive; both needed to be negative for the patient to be negative. Stool samples were collected during the first 4 days after endoscopy before any treatment, and examined by the HpSA EIA (450 nm spectrophotometry, cut-off<0.140); results were interpreted blindly. Results:These are shown in the table. Conclusion: HpSA is a reliable test in diagnosing Hp infection in patients who have not received Hp eradication treatment. Though highly specific, HpSA does not seem a reliable test to confirm Hp eradication

	Group A (n= 43)	Group B (n= 18)
Hp(+) by definition	35	6
Hp(-) by definition	8	12
HpSA(+)	31	1
HpSA(-)	5	17

HpSA(Group A): sensitivity 89% specificity 100% PPV 100% NPV 62%
HpSA(Group B): sensitivity 17% specificity 100% PPV 100% NPV 71%

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H. PYLORI INFECTION IN COMMON VARIABLE IMMUNODEFICIENCY DISEASE (CVID).

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Aim: To study the prevalence of H. pylori infection (Hp) in CVID and gastric pathology in the presence or absence of Hp. Methods This study was performed in 20 patients [11 females; mean age 35 (15-56) years] with newly diagnosed CVID but no detectable autoantibodies against parietal cells, intrinsic factor, gliadin and endomysium. Patients with non-ulcer disease matched for age, gender, social class and social habits served as controls. Patients underwent endoscopy with multiple biopsies from the duodenum and the gastric antrum, body and fundus. Hp was sought by CLO-tests, histology (Giemsa) and immunohistochemistry (rabbit IgG anti-human Hp mcAb, DAKO). Gastritis was graded for activity and severity (0-3, Houston system), Hp load (0-3). Lymphocytic infiltration was graded according to Isaacson system (0-5). Results: Twelve patients had gastric and/or intestinal nodular lymphoid hyperplasia, 15 had diffuse and 6 focal mucosal erythema and 6 had atrophic gastric rugae. Hp infection was diagnosed in 14 patients. Hp- patients had mild, chronic corpus-predominant gastritis, without any mucosal atrophy or intestinal metaplasia. Mucin depletion was minimal. A variably dense T-cell predominant chronic inflammatory cell infiltrate was seen in the upper part of the mucosa obliterating and distracting individual glands and crypts. Plasma cells were absent but primary lymphoid follicles were seen. The number of intra-epithelial lymphocytes (IEL) per 100 epithelial cells and the number of apoptotic bodies per 10 glands were significantly increased over Hp- control patients. This picture resembled gastric acute graft-versus-host disease (GvHD). In Hp+ patients a chronic pangastritis pattern with multifocal atrophy and areas of complete and incomplete intestinal metaplasia was seen. The mucosa was infiltrated with chronic inflammatory mononuclear cells: T cells were predominant and B cells or plasma cells were only rarely seen. The number of IELs and apoptotic bodies was statistically higher than in Hp+ controls. Gastritis severity and activity scores were statistically lower but the Isaacson score was higher than in controls. Eradication of Hp restored a pattern of inactive superficial gastritis in the antrum and a CvHD-like pattern in the body of the stomach. Conclusion: Hp infection seems to accelerate gastric atrophy in CVID; this may increase the potential for malignant transformation. Eradication of Hp may reverse these changes. Hp- patients have mild gastritis resembling gastric acute GvHD.

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HELICOBACTER PYLORI INFECTION AMONG PATIENTS WITH ALCOHOLIC AND NON-ALCOHOLIC CIRRHOSIS.

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The prevalence of Helicobacter pylori (H.P) in patients with alcoholic and non-alcoholic cirrhosis is uncertain. The aim of this study was to determine the prevalence of H. P infection among cirrhotic patients and to evaluate its relationship to demographic data, etiology of cirrhosis and liver function. Methods: Upper GI endoscopy was performed in 45 consecutive cirrhotic patients. The existence of varices, gastritis and H. pylori infection was estimated. H. P infection was determined by CLO-test and with antral biopsies (n=5). No patient received NSAIDs, anti-secretory drugs or antibiotics for at least the last 3 months. Etiology of cirrhosis was classified as alcoholic and non-alcoholic. The grade of H. pylori infection was related to age, sex, etiology of cirrhosis, gastritis and Child-Pugh grading. H. pylori gastritis was estimated according to modified Sydney classification. Chi-Square test was used for the statistical analysis. Results: Twenty alcoholic and 25 non-alcoholic (HBV: 14, HCV: 8, PBC: 3) cirrhotics (m: 34, F: 11),with a median age of 59 years (range: 34-83) were included. Overall H. pylori prevalence was 62,2%. The prevalence varied from 52,2% to 72,7% in those younger and older than the median age (p:NS) and from 58,8% to 72,7% in men and women (p:NS) respectively. 50% of alcoholic and 72% of non-alcoholic cirrhotic patients were H.pylori(+) (p:NS). Among all patients, 24 (53,3%) were of grade A, 13 (28,9%) of grade B and 8 (17,8%) of grade C, according to Child-Pugh grading and 37,5%, 84,6% and 100% were infected respectively [p< 0,01 (A vs B), P< 0,01 (A vs C), P:NS (B vs C)]. Esophageal varices were found in 16 (80%) of alcoholic and in 17 (68%) of non-alcoholic cirrhotics. Endoscopic gastritis and histologically determined congestive gastropathy were found in 11 (55%) and 8 (40%) of alcoholic patients and in 18 (72%) and 17 (68%) of non-alcoholic patients respectively (p:NS). In cirrhotics with endoscopic gastritis H.P infection was histologically confirmed in 80% of alcoholic and in 100% of non-alcoholic patients. Conclusions: a)The prevalence of H.P infection in cirrotics was 62,2%. b) H. P infection was not related to age, gender or etiology of cirrhosis c) Endoscopic gastritis was histologically related especially to H.P gastritis and not to congestive gastropathy d) The severity and activity of gastritis was related to H. P grade and not to congestive gastropathy e) Patients with more severe cirrhosis (grade C and B) had more severe H. P infection grade in comparison with grade A cirrhotics.

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PROLONGATION OF OMEPRAZOLE (O) TREATMENT AFTER H. PYLORI (HP) ERADICATION INFLUENCES THE INCIDENCE OF ESOPHAGITIS.

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The need for prolongation of antisecretory treatment after HP eradication is disputable. The aim of our study was to evaluate if prolongation of antisecretory treatment after successful eradication could influence the incidence of esophagitis. Patients and methods: 181 patients with duodenal ulcer (DU) and 74 with erosive duodenitis (ED), all positive for HP were randomly assigned to continue O 20mg qd for 7 (Group I-85 patients), 21(II-85 patients) or 35 days (III-85 patients) respectively, after a 10 days regimen with O 20mg bid, clarithromycin 500mg bid, amoxycilline 1g bid. Groups were comparable for sex, age smoking and bleeding at the entry. Endoscopy was performed 3 months after treatment. HP eradication was verified by histology, CLO-test and ^{13}C UBT. Esophagitis was graded according to the Savary-Muller. Stat: X^2 test, t-test. Results: 242 patients had a follow up endoscopy (mean age 50.3±15 years, 140men, 76smokers). Intention to treat eradication rates were: Group I: 82.4%(CI 74.1-90.6), II: 81.2% (CI 72.4-89.7), III: 84.7%(CI 76.9-92.5)- p>0.1. Pre-treatment (E_0) and post-treatment (E_3) esophagitis and changes in esophagitis status at a 3 month period after successful eradication are shown in the table: Ag: aggravation or de novo appearance, NC: no change, Im: improvement or disappearance of esophagitis. 32/152 with DU and 2/62 with ED presented E_3 (p=0.002). 16/56 with bleeding at entrance endoscopy and 18/158 without bleeding also presented E_3 (p=0.05). Of those who did not eradicate HP, two patients of Group III presented esophagitis (p>0.1)both pre and post-treatment. Age, sex smoking, hiatus hernia, post-treatment gastritis and commorbid diseases did not influence the incidence of E_3. Conclusions 1) Prolongation of antisecretory treatment for 35 days could aggravate esophagitis 3 months after successful HP eradication. 2) DU and bleeding at entrance endoscopy could be considered as aggravating factors increasing the incidence of post-treatment esophagitis.

	E_0	E_3	Ag	NC	Im
I	12	14	10	54	6
II	12	10	6	57	6
III	8	22	18	50	4
p	>0.1			<0.001	

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DOES CURE OF THE H. PYLORI INFECTION INDUCE GASTRO-ESOPHAGEAL REFLUX SYMPTOMS IN PATIENTS WITH DUODENAL ULCER?

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The development of gastroesophageal reflux disease (GERD) symptoms following eradication of H. pylori (H.p.) in patients with duodenal ulcer (DU) remains controversial. Aim: To evaluate the development of symptoms from upper GI tract in patients with DU after cure of H.p. infection. Methods: 93 consecutive patients [72 men/ 21women, age (mean) 55 (37-77) years] with endoscopically proved H.p. (+) DU who were treated with omeprazole, clarithromycin and amoxycillin and H.p. was eradicated were enrolled in the study. All patients underwent follow-up endoscopy at least 1 month after the end of the treatment and none of them had reflux esophagitis. At the end of the treatment all patients entered follow-up phase with visits every 3 months. Upper GI symptoms such as heartburn, regurgitation, epigastric pain, nausea, vomiting, early satiety, postprandial fullness and belching were assessed pre-entry and at every visit. Each symptom was scored on a fourth graded scale (0= abscence, 1= mild, 2= moderate, 3= severe). None of the patients had reflux or dysmotility-like symptoms at the pre-entry assessment. Results: Mean period of follow-up was 35 (28-44) months. 60/93 (64,5%) pts developed symptoms during follow-up. The cumulative mean symptoms-free interval was 25 (95% CI : 22-27) months. Only 6 (6,5%) patients developed epigastric pain, but dysmotility-like symptoms (post-prandial fullness, early satiety, belching) developed at 36 (38,7%) pts (mild / moderate : 30 / 6) and the cumulative mean dysmotility-like symptom-free interval was 30 (95% CI: 27-33) months. Heartburn and/or regurgitation developed at 42/93 (45,2%) pts (mild / moderate : 30 / 12) and the cumulative mean reflux symptoms-free interval was 29 (95% CI: 25-32) months. The time of presentation of GERD symptoms was not correlated with their severity [mild / moderate : 12 (95% CI: 10-14) / 16 (95% CI: 10-21), log rank 1,8, p= 0,18]. On the contrary moderate dysmotility-like symptoms were developed earlier than mild one [moderate / mild : 9 (95% CI: 7-10) / 13 (95% CI: 12-15), log rank 15,07, p= 0,001]. All twelve patients with moderate reflux symptoms underwent gastroscopy and 7 of them had esophagitis (5 degree I / 2 degree II of Los Angeles classification). Conclusions: About half of patients with H.p. (+) duodenal ulcer seems to develop reflux or dysmotility-like symptoms after eradication of the H. pylori.

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4/43 **The Peroxidation of Polyunsaturated Fatty Acids (PUFAs) in the Pathogenesis of Chronic Gastritis.**

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Objective: To define the role of PUFAs peroxidation in the pathogenesis of chronic gastritis.

Methods: Eighty seven patients -53 male, 34 female, 64.5±19.4 yrs- with symptoms of chronic gastritis were subject to gastroscopy; gastric juice and biopsies from the body, antrum and fundus were taken. Peroxides were determined in gastric juice by the thiobarbiturate assay as malonodialdeyde (MDA) analogues.

Results: In five pts with median MDA of 60mM no gastritis was documented. Fifteen pts were HP(+), 13 presenting with diffuse gastritis. Among them MDA respective to activity scores 1, 2 and 3 were 779.4±395.6, 2014±484.5 and 2262.5±500mM. Among 30 HP(-) pts with diffuse gastritis respective MDA to activity scores 1, 2 and 3 were 1350±990, 1874.2±1316.6 and 4795.5±1612.5mM and among 20 HP(-) pts with gastritis of the antrum respective MDA to activity scores 1, 2 and 3 were 2258.3±361.2, 2039.6±1781.3 and 4125±125mM. A positive correlation was documented between the extent of the mucosal involvement and the MDA concentrations in both HP(-) pts (r: +0.294, p:0.05) and HP(+) pts (r: +0.694, p: 0.026).

Conclusions: Peroxidation of PUFAs is involved in the pathogenesis of chronic gastritis independent of the presence of *Helicobacter pylori* being related to the extent of the inflammation of the gastric mucosa.

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*3504

TIME TREND FOR THE APPEARANCE OF ESOPHAGITIS AFTER *HELICOBACTER PYLORI* ERADICATION.

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Successful *Helicobacter pylori (H.pylori)* treatment has been reported to predispose to the development of reflux esophagitis (RE). However, despite this evidence, to date no attention had been paid to the time trend for the appearance of RE after *H.pylori* treatment. This information could be of importance in dealing with patient management after eradication. The aim of this prospective study therefore was two fold. Firstly, to examine the RE rate after *H.pylori* eradication and secondly, to study the time trend for the appearance of RE. Methods: 50 consecutive, successfully eradicated patients, without RE at the time of *H.pylori* treatment [25 duodenal ulcer (DU) and 25 non-ulcer (NU), 30M, 20F, median age 41.5 yrs, range 21-57] were followed up for 24 months after *H.pylori* eradication or when reflux symptoms occurred. The chi-square test for trend and the life table analysis were performed for calculating the incidence and time trend for RE appearance after *H.pylori* eradication. Patients were censored if the end point was reached or if RE was noted at endoscopy. Results: Among the 50 *H.pylori*(+) successfully treated patients, 12/50(24%) developed RE during the 24 month follow up period. In 9/12 patients(75%), RE developed during the first year of the follow up, at months 5,6,7,9,10,11 and 12 (respective percentages of patients without RE 98,96,92,90,88,86 and 82%) as opposed to only 3/12(25%)patients (p<0.0001) in whom RE appeared during the second year of the follow up, at months 16,17,18 (respective percentages of patients without RE 80,78 and 76%). Conclusion: A substantial proportion of *H.pylori* successfully treated patients develop RE and the time trend suggests an increased risk during the first year after *H.pylori* eradication

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11/3 **Reduced Incidence of the Non-NSAIDs Related Peptic Ulcer In the Last Years but the *Helicobacter pylori* is Still Here; A 9.5 Years Experience.**

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Background: It has been suggested that the incidence of Peptic Ulcer (PU) is declining during the last years.

Aims:1. To evaluate the incidence of non-NSAIDs related PUs in three periods through the last 9.5 years of application of *H. pylori* (Hp) eradication therapies.2. To see the prevalence of Hp among them.

Patients-Methods: A computer search was made from May 1st 1990 through October 31st 1999 to find out all patients with endoscopically diagnosed for the first time PUs in our insonition; emergency endoscopies were also included. Patients who reported NSAIDs or aspirin intake, those who ingested caustic substances and patients with Crohn's were excluded. Three time periods were analysed. A: May 1 1990–December 31 1991 (20 months). B: May 1 1995- December 31 1996 (20 months).C: January 1 1999- October 31 1999 (10 months).Gastric Hp colonization was documented by CLO test and/or histology (H&E, modified Giemsa).

Results: These are shown in the table.There is a progressive and significant reduction (up to 50%) in the incidence of PUs (DUs and GUs). The Hp (+) cases do not differ in the three studied periods (90–92%).

Conclusions:1. There is a steady declining incidence of non- NSAIDs related DUs and GUs during the studied period. 2. Hp colonization is constantly documented in 90–92% of the ulcers by the used methods. Thus, in spite of the significant decrease of PUs in the studied population, Hp remains the predominant factor in the pathogenesis of non-NSAIDs related PUs.

Incidence of PUs in each period of time (percentages in parentheses)

	Period A	Period B	Period C
Patients	7479	7475	2594
(*) PUs	904 (12)	680 (9)	161 (6)
Sex (M/F)	618/286	442/238	129/76
Age (range)	16–86	16–85	16–92
(**) DUs	588 (7.9)	461 (6.2)	97 (3.7)
(***) GUs	316 (4.2)	219 (2.9)	64 (2.4)
Hp (+) cases (%)	91	90	92

(*), (**), (***): p < 0.0001

11/8 NSAIDs' Use Does not Affect *H. pylori* (HP) Eradication Rate in Patients with Duodenal Ulcer (DU) or Erosive Duodenitis (ED) when Tripla Therapy is Used.

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NSAIDs' use could reduce the efficacy of eradication regimens in curing HP infection.

The aim of our study was to evaluate if HP eradication rate is affected by NSAID's use in patients with DU or ED, when a triple eradication regimen is used.

Patients and Methods: 315 patients (220 DU-95 ED, mean age 51.7±14.3 years, 175 men, 105 smokers, 125 on NSAIDs' treatment at least 3 times a week), all HP positive, received a 10 days eradication regimen with omeprazole 20mg bid, clarithromycin 500mg bid, amoxycillin 1g bid. Of those initially receiving NSAIDs': 57 continued NSAIDs' during eradication treatment (Group I), while 68 discontinued them (Group II). All patients initially not receiving NSAIDs' did not receive NSAIDs' during eradication treatment (Group III, n=190). Endoscopy was performed 4–6 weeks after treatment completion. HP eradication was verified by histology, CLO-test and ^{13}C-urea breath test.

Stat: X2 -test, t-test.

Results: 295 patients had a follow up endoscopy. 20 lost during follow-up, 6 discontinued treatment prematurely. Patients of Group III were younger. More smokers were included in group II. Intention to treat eradication rates were: Group I: 80.7% (CI 70.1–91.3), Group II: 79.4% (CI 69.6–89.3), Group III: 83.7% (CI 78.4–89)- p>0.1. Per protocol eradication rates were: Group I: 85.2%(CI 75.4–95), Group II: 84.4% (CI 75.2–93.5), Group III: 89.8% (CI 85.3–94.3)- p>0.1. There was no difference in eradication rates among the 3 groups when patients with DU and ED or smokers and non-smokers were examined separately.

Conclusions: NSAIDs' use seems no to affect HP eradication rates, when a triple regimen comprising omeprazole, amoxycillin and clarithromycin is received.

12/4* Development of Esophagitis after *H. pylori* Eradication: Impact of CagA status.

T. Rokkas[1], S.D. Ladas[2], C. Triandafyllou[2], C. Liatsos[1], E. Petridou[1], G. Papatheodorou[1], A. Karameris[1], S.A. Raptis[2]. [1]*401 Army Gen. Hospital University of Athens: Athens, Greece;* [2]*University of Athens: Athens, Greece*

Background: There is evidence that *Helicobacter pylori* (*H. pylori*) eradication might predispose to reflux esophagitis. However, the role of CagA status in the development of esophagitis after *H. pylori* eradication has not been studied so far. The aim of this prospective study therefore was to examine the impact of CagA status in relation to the appearance of esophagitis after successful *H. pylori* treatment.

Methods: The study included 50 consecutive, successfully eradicated patients, without esophagitis at the time of *H. pylori* treatment [25 duodenal ulcer (DU) and 25 non-ulcer (NU), 30M, 20F, median age 41.5 yrs, range 21–57]. All patients underwent upper GI endoscopy before and at 0, 6, 12, 18 and 24 months after eradication or when reflux symptoms occurred. During endoscopy antrum and corpus biopsies were taken for gastritis evaluation. The sum of the scores for acute [0–3 (0 = absent, 3 = severe)] and chronic inflammation (0–3) comprised the cumulative gastritis score. CagA was determined by immunoblotting the sera from patients against *H. pylori* antigens. The independent variables age, sex, smoking, alcohol intake, CagA status and gastritis scores of antrum and corpus gastritis before H. pylori eradication were introduced in a logistic stepwise regression analysis with esophagitis during follow-up as the dependent variable.

Results: Prior to eradication, among the 50 *H. pylori* (+) patients 22 (44%) were CagA(+) and 28 (56%) were CagA(-). During follow-up 12/50 (24%) patients developed esophagitis of whom 11 (91.7%) were CagA(+) and 1 (8.3%) CagA(-) (p=0.0002). In the life-table analysis the estimated incidence of esophagitis within 24 months in CagA(+) patients was 50% (11/22) as opposed to 9.64% (1/28) in CagA(-) patients ($p < 0.0001$, logrank test). Multiple logistic regression analysis showed that CagA(+) status (odds ratio 13.75, 95% CI 1.4 – 134.53, p=0.0243) and severity of corpus gastritis before eradication (odds ratio 2.88, 95% CI 1.03 – 8.06, p=0.0436) were risk factors for the development of esophagitis after *H. pylori* eradication.

Conclusion: CagA(+) status and severity of corpus gastritis before *H. pylori* eradication significantly favor the development of esophagitis after successful *H. pylori* treatment.

12/3* Reflux Oesophagitis Before and After Eradication of *H. pylori* in Patients with Duodenal Ulcer.

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Objective: Patients with duodenal ulcer (DU) may develop reflux oesophagitis (RO) after eradication of Hp. However, the prevalence of pre-existing RO and its outcome after eradication of Hp in DU patients have not been adequately studied. Outside clinical trials reflux symptoms are usually underestimated or overshadowed by the dominant ulcer symptoms. In addition, a detailed and thorough description of the LOS area is usually omitted when the endoscopist is satisfied by the findings of an ulcerated duodenal bulb; this makes retrospective studies unreliable. Thus, the aim of this prospective trial was to extend previous reports (Gut 1998;43:A96) on the prevalence of pre-existing RO in patients with active DU and its outcome after Hp eradication.

Methods: The LOS area was carefully examined and any abnormal findings were recorded in every dyspeptic patient undergoing endoscopy but only patients with an active DU were enrolled in this study. Oesophagitis was graded (0–3). Biopsies were taken from the antrum, body and fundus and on both sides of the Z-line starting 1cm proximal to the gastric rugae. Hp was sought by histology (Giemsa, H&E), immunohistochemistry and CLO-tests on gastric biopsies before and 10 weeks after completion of OAC500 for 10 days. Eradication of Hp was confirmed by UBT in the last 30 patients of the study. Follow up endoscopy with biopsies was performed 1 year later or whenever dyspeptic symptoms recurred.

Results: 75 DU patients were enrolled in the study [45 males, 60 smokers, none regular NSAID user). All were infected by Hp. Thirty patients (40%) had concurrent RO grade I (n=25) or II (n=5) and were all smokers; of these, 25 patients (33%) had a small (n=20) or larger (n=5) hiatus hernia. A grade II RO was only seen in the patients with a large hiatus hernia. DU patients with RO had usually distorted, oedematous, obliterated duodenal bulb when compared with DU patients without RO. OAC500 healed all ulcers and eradicated Hp in 56 patients including all 30 patients with pre-existing RO. However, recurrence of RO was seen 4–8mo after eradication of Hp in 5 patients who before treatment had a large hiatus hernia and a grade II RO.

Discussion: A considerable proportion of patients with DU obliterating the duodenal bulb may have concurrent RO as a result of gastric outlet obstruction. This RO may easily escape or not be recorded during routine endoscopy. However, it is effectively treated by Hp eradication regimen and does not recur unless there is an underlying large hiatus hernia.

12/18 Time Trend for the Appearance of Esophagitis after *H. pylori* Eradication.

T. Rokkas[1], S.D. Ladas[2], C. Triandafyllou[2], S.A. Raptis[2]. [1]*401Army Gen. Hospital University of Athens: Athens, Greece;* [2]*University of Athens: Athens, Greece*

Successful *Helicobacter pylori* (*H. pylori*) treatment has been reported to predispose to the development of reflux esophagitis (RE). However, despite this evidence to date no attention has been paid to the time trend for the appearance of RE after *H. pylori* treatment. This information could be of importance in dealing with patient management after *H. pylori* eradication.

The aim of this prospective study therefore was two fold; firstly, to examine the RE rate after *H. pylori* eradication and secondly to study the time trend for the appearance of RE.

Methods: 50 consecutive, successfully eradicated patients, without RE at the time of *H. pylori* treatment [25 duodenal ulcer (DU) and 25 non-ulcer (NU), 30M, 20F, median age 41.5 yrs, range 21–57] were followed up for 24 months. All patients underwent upper GI endoscopy before and at 0, 6, 12, 18 and 24 months after *H. pylori* eradication or when reflux symptoms occurred. The chi-square test for trend and the life table analysis were performed for calculating the incidence and time trend for RE appearance after *H. pylori* eradication. Patients were censored if the end point was reached or if RE was noted at endoscopy.

Results: Among the 50 *H. pylori* (+) successfully treated patients, 12/50 (24%) developed RE during the 24 month follow-up period. In 9/12 patients (75%), RE developed during the first year of the follow up at months 5, 6, 7, 9, 10, 11, 12 (respective percentages of patients without RE 98, 96, 92, 90, 88, 86, and 82%) as opposed to only 3/12(25%) patients ($p < 0.0001$) in whom RE appeared during the second year of the follow-up at months 16, 17, 18 (respective percentages of patients without RE 80, 78 and 76%).

Conclusion: A substantial proportion of *H. pylori* successfully treated patients, develop RE and the time trend suggests an increased risk during the first year after *H. pylori* eradication.

12/14 Prolongation of Omeprazole (O) Treatment after *H. pylori* (Hp) Eradication Influences the Incidence of Esophagitis.

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The need for prolongation of antisecretory treatment after HP eradication is disputable.

The aim of our study was to evaluate if prolongation of antisecretory treatment after successful eradication could influence the incidence of esophagitis.

Patients and methods: 181 patients with duodenal ulcer (DU) and 74 with erosive duodenitis (ED), all positive for HP were randomly assigned to continue O 20mg qd for 7 (Group I-85 patients), 21(II-85 patients) or 35 days (III-85 patients) respectively, after a 10 days regimen with O 20mg bid, clarithromycin 500mg bid, amoxycillin 1g bid. Groups were comparable for sex, age smoking and bleeding at the entry. Endoscopy was performed 3 months after treatment. HP eradication was verified by histology, CLO-test and ^{13}C UBT. Esophagitis was graded according to the Savary-Muller.

Stat. X^2-test, t-test.

	E_0	E_3	/	Ag	NC	Im
Group I	12	14	/	10	54	6
Group II	12	10	/	6	57	6
Group III	8	22	/	18	50	4
P	>0.1		/		< 0.001	

Ag: aggravation or de novo appearance. NC: no change. Im: improvement or disappearance of esophagitis.

Results: 242 patients had a follow up endoscopy (mean age 50.3±15 years, 140 men, 76 smokers). Intention to treat eradication rates were: Group I 82.4% (CI 74.1–90.6), II: 81.2% (CI 72.4–89.7), III: 84.7% (CI 76.9–92.5)- p>0.1. Pre-treatment (E_0) and post-treatment (E_3) esophagitis and changes in esophagitis status at a 3 month period after successful eradication are shown in the table.

32/152 with DU and 2/62 with ED presented E_3 (p=0.002). 16/56 with bleeding at entrance endoscopy and 18/158 without bleeding also presented E_3 (p=0.05). Of those who did not eradicate HP, two patients of Group III presented esophagitis (p>0.1)both pre and post-treatment. Age, sex, smoking, hiatus hernia, post-treatment gastritis and commorbid diseases did not influence the incidence of E_3.

Conclusions 1) Prolongation of antisecretory treatment for 35 days could aggravate esophagitis 3 months after successful HP eradication. 2) DU and bleeding at entrance endoscopy could be considered as aggravating factors increasing the incidence of post-treatment esophagitis.

15/19 *H. pylori* Infection in Common Variable Immunodeficiency Disease (CVID).

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Objective: To study the prevalence of *H. pylori* infection (Hp) in CVID and gastric pathology in the presence or absence of Hp.

Methods: This study was performed in 20 patients [11 females; mean age 35 (15–56) years] with newly diagnosed CVID but no detectable autoantibodies against parietal cells, intrinsic factor, gliadin or endomysium. Patients with irritable bowel syndrome matched for age, gender, social class and social habits served as controls. Patients underwent endoscopy with multiple biopsies from the duodenum and the gastric antrum, body and fundus. Hp was sought by CLO-tests, histology (Giemsa) and immunohistochemistry (rabbit IgG anti-human Hp mcAb, DAKO). Gastritis was graded for activity and severity (0–3, Houston system), Hp load (0–3). Lymphocytic infiltration was graded using the Isaacson's score (0–5).

Results: Twelve patients had gastric and/or intestinal nodular lymphoid hyperplasia. 15 had diffuse and 6 focal mucosal erythema and 6 had atrophic gastric rugae. Hp infection was diagnosed in 14 patients. Hp- patients had mild, chronic corpus-predominant gastritis, without any mucosal atrophy or intestinal metaplasia. Mucin depletion was minimal. A variably dense T-cell predominant chronic inflammatory cell infiltrate was seen in the upper part of the mucosa obliterating and distracting individual glands and crypts. Plasma cells were absent but primary lymphoid follicles were seen. The number of intra-epithelial lymphocytes (IEL) per 100 epithelial cells and the number of apoptotic bodies per 10 glands were significantly increased over Hp- control patients. This picture resembled gastric acute graft-versus-host disease (GvHD). In Hp+ patients a chronic pangastritis pattern with multifocal atrophy and areas of complete and incomplete intestinal metaplasia was seen. The mucosa was infiltrated with chronic inflammatory mononuclear cells; T cells were predominant and B cells or plasma cells were only rarely seen. The number of IELs and apoptotic bodies was statistically higher than in Hp+ controls. Gastritis severity and activity scores were statistically lower but the Isaacson's score was higher than in controls. Eradication of Hp restored a pattern of inactive superficial gastritis in the antrum and a GvHD-like pattern in the body of the stomach.

Discussion: Hp- patients with CVID have mild gastritis resembling gastric acute GvHD. Hp infection is associated with more intense gastric atrophy; this may increase the potential for malignant transformation.

13/20 Incidence of *Helicobacter pylori* Infection in Patients with Rheumatic Diseases.

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Objective: To evaluate the seroprevalence of *Helicobacter pylori* (Hp) infection in patients (pts) with rheumatic diseases.

Methods: Serum samples of 62 pts with rheumatoid arthritis (RA), 20 pts with systemic lupus erythematosus (SLE), 18 pts with seronegative spondylitis (SNS), 32 pts with osteoarthritis (OA) and 36 healthy volunteers (V) were measured for IgA and IgG antibodies against Hp by enzyme immunoassay. Positive were titers >30 U/ml. Western blot was used as confirmation. Pts had not received Hp eradication treatment, or sulphasalazine or gold for arthritis, in the past. Concurrent use of NSAIDs, steroids or antirheumatic drugs was recorded. Comparison and statistical analysis in average IgA and IgG titers (M=mean±SEM) and incidence (%) between the subjects was performed using chi-test and student's t-test.

Results: Comparison between RA pts and V, SLE pts and V, SNS pts and V and OA pts and V and between RA and OA pts revealed, as shown in table: a greater incidence in RA than V for IgA (p=0.002) and IgG (p=0.018); a greater incidence in OA than V for IgA antibodies (p=0.000); a higher average titer of IgA (p=0.006) and IgG (p=0.026) in RA than V and a higher average titer of IgA (p=0.034) in OA than V.

	RA n=62	SLE n=20	SNS n=18	OA n=32	V n=36
IgA	34/62	7/20	9/18	17/32	6/36
%	55.72	35	38.89	53.12	22.23
M	51.2 ±5.5	37.2±10	30.4 ±6.3	50.1 ±7.7	30.3 ±4.9
IgG	41/62	9/20	11/18	19/32	15/36
%	66.12	45	61.11	59.37	41.67
M	71.9 ±9.9	41.8±10.3	44.8 ±9.1	59.0 ±9.3	42.2 ±8.0

Discussion: Patients with rheumatoid arthritis show a significantly higher Hp seropositivity and higher titers of IgA and IgG antibodies than volunteers. Patients with osteoarthritis show significantly higher Hp seropositivity only for IgA antibodies. This data indicates a higher incidence of Hp infection in RA patients.

15/35 Rabeprazole 7-days Vs Rabeprazole 10-days Triple Therapy in the Eradication of *H.Pyport* Infection – A Randomized Study.

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

Methods: Ninety-seven *H. pylori* positive patients (CLO-test,histology) (median age 48, range 18–79) with peptic ulcer (n=59) or non-ulcer dyspepsia (n=38) were randomized to receive RAB 400mg bid, Clarithromycin (CL)500mg bid, and Amoxycillin (AMO) 1gr bid for 1 week (Group A, n=49), or RAB 400 mg bid, CL 500mg bid and AMO 1gr bid for 10 days (Group B, n=48). *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 (66/97, 68%) *H. pylori* strains.

Results: The regimen failed to eradicate three (4.5%) *H. pylori* strains (one in Group A and two in Group B) which exhibited primary Cl resistance. The eradication rates according to intention to treat analysis (ITT) were: 40/49(81.6%) in Group A and 40/48 (83.3%) in Group B, and according to per-protocol (PP)analysis: 44/49 (89.8%) in Group A and 43/48(89.6%) in Group B. Side effects in both groups were mild and no patient discontinued treatment due to adverse effects.

Discussion: We conclude that both (1-week vs 10-days) triple therapies based in rabeprazole proved equally effective and safe to eradicate *Helicobacter pylori* infection.

We also could observe that k of the Hp and Ss mixes are similar to that of Hp and statistically higher than the Ss one

Discussion: The CO_2 rate constant allows the differentiation of Hp from the other urease producer bacterias that are present in the oral cavity. These findings would allow the detection of Hp in the oral cavity in a faster and easier way and therefore may contribute to clarify the oral-oral mode of transmission of this infection.

XIIIth International Workshop on Gastroduodenal Pathology & *Helicobacter pylori*, 11-14 October 2000, Rome, Italy
Gut 2000;47(Suppl I):A124

17/7 **Cystic Fundic Gland Polyps (CFGP): Relation to *H. pylori* Infection and Familial Polyposis coli (FAP) Syndrome.**

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Objective: CFGP are small sessile polyps of the secretory gastric mucosa which are usually found in middle-aged women. They are sporadic but can be associated with FAP, chronic treatment with proton pump inhibitors (PPIs) but not Hp infection. This study aims at seeking any association between CFGP and Hp, FAP or sporadic colorectal adenomas.

Methods: Between 1992 and 2000, 20 patients with CFGP were identified [(15 female), mean age 35 (range 25–72) years]. On endoscopy, biopsies were taken from the antrum, body and fundus for grading of gastritis (Houston score). Hp was sought by the means of histology, CLO-tests, and immunohisto-chemistry. Larger CFGP were excised whereas biopsies were taken from smaller polyps. Positive and negative risk factors for colorectal adenomas or cancer were recorded, including a family history, regular use of NSAIDs, hypercholesterolaemia, a previous cholesystectomy, etc. Serum gastrin levels were measured. Patients underwent total colonoscopy and any polyps found were excised and sent for histology.

Results: None of the patients was on chronic PPI treatment. None of the excised CFGPs was dysplastic. Hp was detected in 14/20 (70%) patients. The majority of Hp+ patients showed a mild chronic active superficial antral predominant gastritis without atrophy. Elderly Hp+ patients had changes of atrophic gastritis without evidence of dysplasia. Gastrin levels were normal. No case of FAP was detected. Two solitary sessile proximal colonic adenomas were detected in 2 female patients, 45 and 52 years respectively; one had a family history of colorectal adenomas. No regression of any CFGP was noticed within 2.5 years following eradication of Hp in 10 patients.

Discussion: In this study CFGPs were sporadic. However, the fact that 2/20 patients had concurrent colonic adenomas raises an issue for association. No association was found with chronic PPI therapy. Contrary to other reports, the vast majority of our patients were infected by Hp to a degree similar to that reported in the general population of similar age in our country.

8th United European Gastroenterology Week (UEGW)
November 2000, Brussels, *Gut 2000;47(Suppl III):A79*

P.141 **REDUCED INCIDENCE OF THE NON-NSAIDS RELATED PEPTIC ULCER IN THE LAST YEARS BUT THE HELICOBACTER PYLORI IS STILL HERE: A 9.5 YEARS EXPERIENCE**

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Background: It has been suggested that the incidence of Peptic Ulcer(PU)is declining during the last years.

Aims: 1. To evaluate the incidence of non-NSAIDs related PUs in three periods through the last 9.5 years of application of H pylori(Hp) eradication therapies. 2. To see the prevalence of Hp among them.

Patients/Methods: A computer search was made from May 1st 1990 through October 31st 1999 to find out all patients with endoscopically diagnosed for the first time PUs in our institution; emergency endoscopies were also included. Patients who reported NSAIDs or aspirin intake, those who ingested caustic substances and patients with Crohn's were excluded.Three time periods were analysed. A: May 1 1990- December 31 1991 (20 months). B: May 1 1995 - December 31 1996 (20 months). C: January 1 1999 - October 31 1999 (10 months). Gastric Hp colonization was documented by CLO test and/or histology (H&E, modified Giemsa).

Results: These are shown in the table. There is a progressive and significant reduction (up to 50%) in the incidence of PUs (DUs and GUs).The Hp (+) cases do not differ in the three studied periods(90-92%).

Incidence of PUs in each period of time [percentages in parentheses]

	Period A	Period B	Period C
Patients	7439	7475	2594
(*) PUs	904 (12)	680 (9)	161 (6)
Sex(M/F)	618/286	442/238	129/76
Age(range)	16-86	16-85	16-92
(**) DUs	588 (7.9)	461 (6.2)	97 (3.7)
(***) GUs	316 (4.2)	219 (2.9)	64 (2.4)
Hp (+) cases (%)	91	90	92

(*), (**), (***): p<0.0001

Conclusions: 1. There is a steady declining incidence of non- NSAIDs related DUs and GUs during the studied period. 2. Hp colonization is constantly documented in 90-92% of the ulcers by the used methods. Thus, in spite of the significant decrease of PUs in the studied population, Hp remains the predominant factor in the pathogenesis of non-NSAIDs related PUs.

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01/05 **MOLECULAR ANALYSIS OF B-CELL CLONALITY IN HELICOBACTER PYLORI GASTRITIS**

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Background: Helicobacter pylori (Hp) gastritis is a risk factor for gastric mucosa associated lymphoid tissue (MALT) lymphoma. Clonal B-cell populations have been detected in a variable proportion (0-40%) of patients with Hp-gastritis, but their clinical significance is still uncertain.

Aim: To identify the presence of clonal B-cell population in Hp-gastritis and assess histological factors that may affect the development of B-cell clones.

Methods: Twenty-five patients (aged 19-79 years, mean 52, 13 males) with Hp-gastritis (H-E and modified Giemsa) were studied. Twenty Hp-negative dyspeptic subjects served as controls. Routinely fixed paraffin embedded blocks of two antral and two corpus biopsies from patients and controls were analysed for B-cell clonality by a seminested polymerase chain reaction (PCR) using FRIIA and FRIIIA primers for amplification of the variable region of the immunoglobulin heavy chain gene (V-IgH gene). The histological findings were evaluated according to the Sydney classification system of gastritis and the Wotherspoon-Isaacson (W-I) scoring system for gastric lymphoid infiltrates.

Results: Amplified DNA was obtained from all samples and revealed monoclonal or polyclonal PCR amplification patterns. Clonal bands were observed in 10 (2/8 W-I grade 1, 2/13 W-I grade 2, and 2/4 W-I grade 3 lesions) and polyclonal smears in 15 cases (6 W-I grade 1, 7 W-I grade 2 and 2 W-I grade 3). Four additional W-I grade 2 samples with clonal bands were associated with a background polyclonal smear and were not reproducible. Clonal bands were not recorded in samples from Hp negative controls.

Conclusion: PCR-detectable monoclonality was found in about 25% (6/25) of our Hp-gastritis patients, it is independent of the W-I score and cannot be taken as evidence of an existing neoplastic lesion.

8th United European Gastroenterology Week (UEGW)
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P.148 **DIETARY HABITS IN PATIENTS WITH DUODENAL ULCER: A PROSPECTIVE STUDY**

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Aim: To investigate the dietary, drinking and smoking habits in patients with active duodenal ulcer(DU) as compared to healthy controls. **Patients-methods:** 100 consecutive patients (men=73, age: 19-84 y, mean ± SD = 48 ± 15) with endoscopically, for the first time, diagnosed DU, 100 clinically healthy people without past history of peptic ulcer, absolutely matched with DU patients with regard to sex, age, and various socioeconomic parameters (income, education, area of residence, and number of rooms during the last 10 y) served as controls. We studied: 1. Dietary, drinking and smoking habits using suitable questionnaire; 71 kinds of food grouped in 9 categories 1 were included 2. Obesity index (Obi) as estimated from the people's height and body weight 3. Hp status by serology (ELISA IgG). Conditional regression analysis was used for statistics. Odds Ratios (OR) were calculated after appropriate adjustments. **Results:** Prevalence of Hp was higher in DU patients (OR = 4.31, 95%CI = 1.86-9.95, p = 0.0006). No differences in the Hp status in relation to the studied socioeconomic factors were found in patients and controls. Smoking did not differ. Patients' height and body weight differed between DU and controls (OR = 0.49, 95%CI = 0.34-0.72, p < 0.001 and OR = 0.58, 95%CI = 0.41-0.81, p < 0.002 respectively); Obi did not differ (p = 0.06, OR = 91%, 95%CI = 0.82-1.00). Coffee drinking did not differ, by and large, but significantly more "Greek coffee" was consumed by patients (p=0.004, OR=1.59, 95%CI = 1.16-2.19). In general, patients consumed significantly more spirits and controls more wine. Controls consumed significantly more legumes and light lipids including olive oil (p = 0.01, OR = 0.69, 95%CI = 0.51-0.93 and p = 0.05, OR = 0.70, 95%CI = 0.49-1.01) and more potatoes (OR = 0.91, p = 0.77) and vegetables (OR = 0.86, p = 0.41). Patients consumed more cereals (OR = 1.23, p = 0.17), fruits (OR = 1.94, p = 0.0004), pastries (OR = 1.29, p = 0.11), meat + fish + eggs (OR =1.19, p = 0.26) and dairy products (OR = 1.47, p = 0.03). **Conclusions:** 1. Obi index and smoking did not differ in patients and controls 2. Patients consumed significantly more "Greek Coffee", spirits, fruits and dairy products; controls consumed significantly more legumes, wine and light lipids + olive oil. 3. Some foods and spirits may be involved in the pathogenesis of DU; otherwise, patients prefer some foods that may not induce or may ameliorate symptoms.

P 170 **IS THERE A RELATIONSHIP BETWEEN H.PYLORI AND BLEEDING, IN NSAIDS USER PATIENTS, WITH PEPTIC ULCER?**

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The role of H. pylori (H.p.) in bleeding ulcers in NSAIDs users remains controversial. **Aim:** To investigate, prospectively, the role of H.p. in bleeding and non-bleeding peptic ulcers in NSAIDs users.

Patients/method: Eighty-five patients who presented at the emergency room with upper GI bleeding and have been receiving NSAIDs during the last week (Group 1), as well as, 51 symptomatic, NSAIDs users (during the last week) [32 M/19 F, age (mean±SD) 49±16 (range 20-81) yrs], with endoscopically detected peptic ulcer (Group 2), were included in the study between March 1998 and September 1999. H. p. status has been evaluated by serology, rapid urease test and histology. The distribution of ulcers (gastric, duodenal), the type of the NSAIDs (aspirin, non-aspirin), smoking and alcohol consumption have been recorded.

Results: In group 1, duodenal ulcer was detected in 29 (34 %) pts, gastric ulcer in 56 (66 %) pts, 47 (55 %) pts reported aspirin consumption and 34 (40%) pts presented with dyspeptic symptoms before bleeding. In group 2, duodenal ulcer was detected in 27 (53 %) pts, gastric ulcer in 24 (47 %) pts, 26 (51 %) pts reported aspirin consumption. H.p. was positive in 60 (71%) pts of group 1 versus 25 (49%) pts of group 2 (p= 0,01). In duodenal ulcer patients H.p. positivity was significantly related to bleeding (22/29 pts vs 13/27, p=0,03). In gastric ulcer pts H.p. was frequently positive in bleeders though this observation did not reach statistical significance (38/56 pts *vs* 12/24pts, p=ns). The type of the NSAIDs, smoking and alcohol consumption were neither related to bleeding nor H.p. status.

Conclusions: A severe complication such as bleeding in NSAIDs users is significantly correlated with H.p. status.

P.181 **EFFICACY OF HELICOBACTER PYLORI ERADICATION REGIMENS IN FUNCTIONAL DYSPEPSIA – COMPARISON WITH PEPTIC ULCER DISEASE**

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H. pylori (HP) infection is often diagnosed in patients with functional dyspepsia (FD) or non-ulcer dyspepsia. The aim of this retrospective study was to report any differences in HP eradication rate between patients with and without endoscopic findings using various treatment regimens.

Methods: Among 248 patients with HP infection (F:92, M:156 with mean age 52.5 and range:20-79 yrs) 106 (group A; F:56, M:50 with mean age 51.9) had FD with normal endoscopy or minimal endoscopic findings and 142 (group B; F:61,M:81 with mean age 52.2 yrs) had peptic ulcer or erosive gastroduodenitis. Regimens were based in either proton pump inhibitor (PPI) or ranitidine bismouth citrate (RBC) in combination with two antibiotics from: clarithromycin(250 or 500mg), amoxicillin(1g), tetracycline-HCl(500mg), metronidazole(500mg) and tinidazole(500mg), all given bid for 1 week. Endoscopy was repeated 4 weeks after the end of the treatment and HP eradication was considered successful if both rapid urease test and histology from antrum and corpus were negative.

Results: There were no significant differences between the 2 groups with respect to age, gender, alcohol and NSAID's consumption; however, patients in group B were more likely to be smokers(p<0.001). HP was eradicated in 195/248 patients(78.6% with 95% CI's 73.5%-83.7%). Eradication rate was higher in group B compared to group A(81.7% vs 73.6%) but the difference was not significant (χ^2 test; p>0.05). Moreover, there was no significant difference in HP eradication rate between patients in the 2 groups treated either with a PPI or a RBC based regimen.

Conclusion: Patients with functional dyspepsia appear to respond equally successfully to HP eradication therapies compared to patients with peptic ulcer disease.

P.286 **SYMPTOMS FROM THE DIGESTIVE SYSTEM AND HELICOBACTER PYLORI INFECTION IN HOSPITAL WORKERS**

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The aim of this study was to compare the incidence of various symptoms from the digestive system in healthy hospital workers positive or negative for Helicobacter pylori (Hp).

Subjects and Methods: 432 healthy employees of our hospital (nurses and members of the administrative staff) completed a special questionnaire concerning various symptoms mainly from the digestive system. Information was also obtained concerning surgical operations and chronic diseases involving various systems. The Hp positivity was confirmed by estimation of an IgG antibody in the serum. All data were entered on a special statistical package and were analyzed using parametric and non-parametric tests.

Results: Statistically significant differences between positive and negative subjects were observed for the following variables: reflux symptoms ($X2=6.62$, df=1, p<0.01) tendency for vomiting ($X2=3.46$ df=1, p<0.06) chronic disorders of other systems excluding digestive ($X2=8.950$, df=1, p<0.003) and history of "peptic ulcer" and / or "gastritis" ($X2=7.87$, df=1, p<0.005). Marriage people had statistically significantly more chances to acquire the infection ($X2=4.07$, df=1, p<0.04) than the single persons. It was of interest that the consumption of Greek coffee was significantly related to Hp infection ($X2=6.06$, df=2, p<0.05). Various other parametric variables such as years of smoking, number of cigarettes smoked per day, alcohol consumption and number of bowel movements per day, were not statistically related to Hp infection.

Conclusion: Infection by Hp in hospital workers is significantly related to some symptoms from the upper GI tract as well as with chronic disorders from other organs or systems.

P.297 **FACTORS INFLUENCING THE SEROCONVERSION OF THE PREVIOUSLY HELICOBACTER PYLORI NEGATIVE HOSPITAL WORKERS**

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Factors influencing the seroconversion of previously negative for Helicobacter pylori (Hp) infection groups of the normal population are unknown

The aim of this study was to provide information concerning the influence of various epidemiological and clinical parameters on the transformation from serum negative to serum positive for Hp hospital workers (a well-known group of people with increased chance of acquiring Hp infection).

Subjects and Methods: Out of 437 hospital workers studied on 1994 for Hp infection, 84 (19.2%) initially negative for Hp infection became positive after 4 years of follow-up (1998). A large number of epidemiological and clinical parameters (55 non-parametric and 14 parametric variables) were studied on 1994 and in the same group of persons on 1998, in order to find-out those having staustical significance. The analysis of the results was done using a special statistical package.

Results: The only clinical symptom which was found to have statistical significance was abdominal bloating and dietetic restrictions, which were followed by the subjects themselves ($X_2=3.309$, df=1, P<0.05). Other non-parametric variables such as sex, educational level, marriage, smoking, alcohol and coffee consumption, surgery for various reasons, and chronic gastrointestinal disorders, were not significant. The ANOVA analysis for various parametric variables such as age, number of brothers and sisters, number of cigarettes smoked per day and number of bowel movements per day were no statistically significant.

Conclusion: Abdominal bloating and dietetic restrictions were the only factors predicting positivity for Hp over time. Further studies, in other groups of the normal population are needed in order to find out possible predictive factors for Hp positivity.

P.318 P27KIP1 EXPRESSION IN HELICOBACTER PYLORY GASTRITIS AND INTESTINAL METAPLASIA

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Aim: The study of p27Kip1 expression by human gastric epithelial cells in Helicobacter pylori (Hp) gastritis before and after eradication of the microorganism. The expression of p27Kip1 in intestinal metaplasia (IM) was also studied.

Patients and methods: a. 25 patients, 21Hp(+) without IM and 4Hp(-). All patients were endoscoped for the evaluation of dyspepsic symptoms. 17/21 had duodenal ulcer (DU), 1/21 gastric ulcer(GU) and 3/21 mixed lesions (DU+GU). Patients were endoscoped again, after the administration of triple anti-Hp therapy, in 116±9d. b. 5 Hp(+) patients with IM. Biopsies were taken from antrum and corpus (anterior-posterior wall) for CLO test and histologic evaluation of gastritis (Sydney classification). The immunohistochemical studies were performed on paraffin-embedded tissue sections with a mouse anti-human p27 antibody (DAKO); quantitative evaluation (percentage of positive cells) according to three distinct zones was done (zone 1=surface+upper 1/3 of the pit, zone 2=the rest 2/3 of the pit, zone 3=glands).

Results: a. Normal mucosa: p27 is expressed by few cells (0-2/pit), in zone 2 (proliferation zone). No expression was found on surface and glandular epithelium (zones 1,3). A lot of p27(+) lymphocytes in lamina propria and in the cortex of lympoid follicles (when present) were detected; no expression by lymphocytes in the germinal center. b. Hp gastritis: Increase, not statistically significant, of the p27(+) epithelial cells in zone 2; no expression in zones 1,3. c. After eradication: p27 expression quantitatively and spatially almost like normal mucosa. d. Intestinal metaplasia: Significant (Mann Whitney, $p<0.05$) increase of p27(+) epithelial cells in zone 2 as compared with normal and Hp(+) without IM mucosa.

Conclusion: The terminal differentiated cells of the surface epithelium do not express p27Kip1. The relative increase of positive cells during inflammation and the significant increase of p27 expression in Hp(+) IM may represent a defensive mechanism of gastric mucosa against Hp infection.

P.348 EVALUATION OF RAPID, NEW TEST FOR DETECTING SERUM ANTIBODIES TO HELICOBACTER PYLORI

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Background. Rapid one step, reliable, inexpensive serum tests are needed for serodiagnosis of H. pylori infection. We have evaluated the performance characteristics of the new TestPack Plus H. pylori test (Abbott Laboratories), which is a one minute qualitative test for the detection of antibodies to H. pylori in serum.

Methods. In a prospective study 130 dyspeptics and patients with upper GI bleeding were studied. Patients were defined H. pylori (+) if two out of three reference tests (histology, rapid urease test, Gram staining of biopsy smears) were positive and H. pylori (-) if all three tests were negative. The results of TestPack Plus were compared with those of commercially available ELISA kits detecting IgG H. pylori serum antibodies (Pyloriset, Orion, Milenia H. pylori, DPC).

Results. Seven patients had only one positive out of three reference tests and were excluded. Out of 123 patients (78 males, 45 females, 50, 17-85 years old) 95 were defined H. pylori (+) and 28 H. pylori (-) by the gold standard. Performance characteristics of the antibody tests studied as compared with the gold standard are shown in the table below.

	Sensitivity	Specificity	PPV	NPV	ODA
TestPack	65	75	92	60	83
Pyloriset	92	82	95	74	89
Millenia	94	72	92	76	90

Conclusions. The sensitivity and specificity of TestPack Plus one step serum test are lower as compared to those of serum ELISA. However, its high positive predictive value makes it a valuable tool for in-laboratory screening purposes.

P.344 DIAGNOSIS OF HELICOBACTER PYLORI INFECTION WITH A NEW NON-INVASIVE STOOL ASSAY BEFORE AND AFTER ERADICATION TREATMENT

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Background: It has been suggested that a new H. pylori(Hp)stool antigen EIA(HpSA Premier Platinum,Meridian Diagnostics, Cincinnati, USA)is reliable, easy to perform tool to diagnose Hp infection. However, after eradication of Hp the results are controversial. Aim: To assess the accuracy of this stool assay in diagnosing Hp infection before and after eradication, in comparison with the 2 mostly used in practice methods i.e. CLO test and histology. Patients-Methods:Two groups of patients with dyspepsia with and without ulcer were prospectively studied. Group A:43 consecutive adult patients who were examined for the first time; exclusion criteria: any treatment for Hp previously, any antibiotic in the previous 6 weeks, active GI bleeding, current treatment with corticosteroids or NSAIDs, with PPI or bismuth containing compounds during the previous 3 months or previous gastric surgery. Group B: 28 adult patients with no gastric operation or active GI bleeding who completed an eradication treatment and had not received PPIs, H2RA, bismuth salts, antibiotics, corticosteroids, NSAIDs for the last month. Patients were endoscoped and 4 biopsies were taken (2 antrum, 2 corpus) for histology (H&E, Giemsa stain)and CLO test; patients were considered Hp(+)if either test was positive; both needed to be negative for the patient to be negative. Stool samples were collected during the first 4 days after endoscopy before any treatment, and examined by the HpSA EIA (450 nm spectrophotometry, cut-off <0.140); results were interpreted blindly. Results: Group A: Sensitivity 89%, Specificity 100%, PPV 100%, NPV 67%. Group B: Sensitivity 30%, Specificity 100%, PPV 100%, NPV 72%. Conclusion: HpSA is a reliable test in diagnosing Hp infection in patients who have not received Hp eradication treatment. Though highly specific, HpSA does not seem a sensitive test to confirm Hp eradication.

P.349 EVALUATION OF A NEW UREASE TEST FOR THE DETECTION OF HELICOBACTER PYLORI INFECTION

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Background. Rapid urease tests are often used during upper gastrointestinal endoscopy to detect Helicobacter pylori infection.

Aims. We have evaluated the performance characteristics of a new, urea-soaked paper with graded chromatic index (Pronto test) and compared it with a urea-gel with chromatic index (CLO test). Both tests are relied on the color change, due to the Helicobacter pylori urease production.

Methods. We studied 103 consecutive in and out patients (61 males and 42 females, 19-85 years old) referred for upper gastrointestinal endoscopy. Twenty-five patients (24%) had upper gastrointestinal hemorrhage and 35 (34%) were on proton pump inhibitor therapy. During gastroscopy 3 biopsies obtained from the antral mucosa and used for histology and urease tests (CLO and Pronto). Rapid urease test results were examined at 30-min intervals for two hours.

Results. The two test results are presented at the table below.

	Sens	Spec	PPV	NPV	ODA
Pronto (30 min)	48%*	100%	100%	48%	65%
CLO (30 min)	10%	100%	100%	%35%	40%
Pronto (120 min)	65%#	100%	100%	59%	77
CLO (120 min)	62%#	100%	100%	57%	75%

(PPV. Positive predictive value, NPV. Negative predictive value, ODA. Overall diagnostic accuracy, * $p< 0.001$, # $p=0.6$)

Patients that had not upper gastrointestinal hemorrhage and those not taking antisecretory therapy and had Pronto and CLO sensitivity of 79% and 76% respectively at 120 min. There was a linear correlation of the estimated Helicobacter pylori colonization density of the antral mucosa between histology and Pronto color grade results ($y=-0.12+0.8x$, $p< 0.001$).

Conclusions. Pronto and CLO-test results are comparable at two hours. However, Pronto is cheaper than the CLO test and gives a positive result at 30 min in 48% of patients, offering a more rapid diagnosis. It also provides a semiquantitative estimation of Helicobacter pylori colonization density of the antral mucosa.

P.353 **IS THE INTAKE OF SECOND SAMPLE OF ^{13}C UREA BREATH TEST IN 20 MINUTES SUITABLE TO MONITOR THE H.PYLORI INFECTION STATUS?**

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^{13}C Urea breath test (UBT) is one of the best methods for the diagnosis of H.Pylori infection. Basal breath samples and samples in 30 minutes, after urea intake, are usually obtained. The elimination of the time of the second sample intake in 20 minutes would have the advantages of higher simplicity and speed.

Aim: To test the efficacy of UBT for H.Pylori infection, when second sample obtained in 20 minutes, compared to the results taken in 30 minutes in the same patients.

Methods: UBT was performed in 164 consecutive patients (mean age: 47.29±13,47. 67 males, 97 women). 64% after H.Pylori eradication therapy administration. Basal samples and samples at 20 and 30 minutes after taking 100mg of urea labeled with 13C, were obtained in each patient. A citric acid solution was used prior to the urea intake. All gas samples were analyzed for excess 13C02/12C02 ratio, which was expressed as d‰. d UBT (DOB) being considered positive when >4‰.

Results:32.8% of the patients showed H.Pylori infection in both samples in 20 and 30 minutes. The study showed a 100% concordance between the two samples in 20 and 30 minutes. Mean values (±SD) of dUBT in 20 and 30 minutes in patients with positive results in 30 minutes gas sample, were respectively 16,9070±9,0027 and 20,7045±11,3806.

Conclusion: When performing 13 C-urea breath test, it seems sufficient to obtain second sample at 20 minutes, which further simplifies the diagnostic method.

P.373 **DOES GASTRITIS AFTER H. PYLORI ERADICATION IN PATIENTS WITH DUODENAL ULCER CORRELATE WITH DYSPEPTIC SYMPTOMS?**

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Gastroesophageal reflux disease (GERD) symptoms after H. pylori (H.p.) eradication, in patients with duodenal ulcer (DU), is a known debate. Dyspeptic symptoms after eradication remain under investigation.

Aim: To evaluate the development of dyspeptic symptoms in patients with DU after H.p. eradication and their correlation with the histologic features of gastritis.

Methods: 93 H.p. (+) DU consecutive patients (72 M/ 21 F, age (mean) 55 (37-77) yrs) in whom H.p. was eradicated were enrolled in the study. All patients, underwent endoscopy before and 1 month after the end of the treatment and none of them had reflux esophagitis. Two biopsies from gastric antrum and two from gastric corpus were obtained at each endoscopy. H.pylori status was evaluated with CLO-test and histology. Each biopsy specimen was evaluated according to Sydney classification. At the end of the treatment all patients entered follow-up phase with visits every 3 months. Upper GI symptoms (heartburn, regurgitation, epigastric pain, nausea, vomiting, early satiety, postprandial fullness) were assessed pre-entry and at every visit using a fourth graded scale. None of the patients had reflux or dysmotility-like symptoms at the pre-entry assessment.

Results: Mean period of follow-up was 35 (25-44) months. 60/93 (64,5%) pts developed symptoms during follow-up. The cumulative mean symptoms-free interval was 25 (95% CI: 22-27) months. Dysmotility-like symptoms (post-prandial fullness, early satiety) developed at 36 (38,7%) pts and the cumulative mean dysmotility-like symptom-free interval was 30 (95% CI: 27-33) months. Heartburn and/or regurgitation developed at 42/93 (45.2%) pts and the cumulative mean reflux symptoms-free interval was 29 (95% CI: 25-32) months. The time of presentation of GERD symptoms was not correlated with their severity. On the contrary, moderate dysmotility-like symptoms were developed earlier than mild one [moderate / mild: 9 (95% CI: 7-10) / 13 (95% CI: 12-15), log rank 15,07, p= 0,001]. The degree of gastritis post-treatment was significantly lower in patients who developed symptoms [1,02±0,18 vs 1,48±0,21 (mean±SE), p< 0,05] but it was not correlated with the development of specific symptoms (GERD vs dysmotility-like 1,12±0,16 vs 1,01±0,12, p=ns). There was no correlation between other histologic characteristics and the development of symptoms.

Conclusions: About half of patients with H.p. (+) duodenal ulcer seem to develop reflux or dysmotility-like symptoms after eradication of the H. pylori and this probably correlated with the decline of the degree of gastritis in the early post-eradicated period.

P.375 **PREVALENCE OF REFLUX OESOPHAGITIS IN PATIENTS WITH HELICOBACTER PYLORI INFECTION**

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Introduction: Increased prevalence of oesophagitis has been reported after eradication of Helicobacter Pylori (HP). The aim of this study was to examine the relationship between HP infection and reflux oesophagitis before and after eradication treatment.

Methods: We retrospectively studied 241 consecutive patients (102 M-139 FM) with HP infection, during the period among 1994-1999. The diagnosis was established by histological examination of samples taken from gastric antrum and corpus. The patients divided in three groups according to the endoscopical findings. Duodenal ulcer (group A) was diagnosed in 147pts (60.9%), oesophagitis (group B) in 38pts (15.7%) and other diseases or the endoscopy was normal (group C) in 56pts (23.2%). After eradication therapy the patients underwent gastrointestinal endoscopy and the incidence of reflux oesophagitis in each group was recorded.

Results: After eradication therapy 189pts (78,4%) were HP negative(121pts of group A, 29pts of group B and 39pts of group C). Post therapy oesophagitis was found in 5pts of group A (4.1%), in 4pts of group B (13.7%) and in 1pt of group C (2.5%).

	HP positive	Eradication	Post eradication oesophagitis
Group A	147 (60.9%)	121 (82.3%)	5 (4.1%)
Group B	38 (15.7%)	29 (76.3%)	4 (13.7%)
Group C	56 (19.3%)	39 (69.6%)	1 (2.5%)

Conclusions: The results of our study indicate that peptic ulcer disease is closely associated with HP infection and that there is no significant difference in HP prevalence between patients with reflux oesophagitis and those with other diseases or with normal endoscopic findings. They do not also support the hypothesis that HP eradication therapy has a predicitve role in the manifestation of gastroesophageal reflux disease.

P.387 **HELICOBACTER PYLORI INFECTION (Hp-I) AND GLAUCOMA (GLA)**

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Objective: To determine the frequency of Hp-I in GLA patients and in controls.

Design: Prospective, non randomized, comparative study.

Patients and Methods: 41 GLA patients (32 with chronic simple-open angle glaucoma (CSOA-GLA) and 9 with pseudo-exfoliative glaucoma (PE-GLA)] and 30 age matched controls were investigated. Upper gastrointestinal endoscopy was performed to evaluate macroscopic abnormalities, and gastric mucosal biopsy specimens were obtained for the presence of Hp-I tested by rapid urease slide test (CLO test) and Crezyl fast violet and/or Giemsa staining. The presence of gastritis was classified in accordance with the Sydney system using hematoxylin and eosin stain. In addition, intestinal metaplasia was evaluated with Alcian blue stain. Saliva samples were also tested by CLO. Serum was analyzed for the presence of Hp specific IgG antibodies by ELISA.

Results: In 87.8% of the GLA patients and in 46.7% of the controls Hp-I was confirmed by the presence of Hp bacteria histologically (odds ratio 8.22, x2=14.075, P=0.0002). Hp was detected by urease test: (a) in the gastric mucosa in 73.2% of the GLA patients and 46.7% of the controls (P=0.02), and (b) in the saliva in 41.5% of the GLA patients and 30% of the controls (P>0.05). Sixty-eight percent of the GLA patients and 30 % of the controls were seropositive for Hp (P=0.002). When compared with controls, GLA patients exhibited less often endoscopic normal appearance of gastric mucosa (P=0.01), and more often antral gastritis (P=0.0004) or peptic ulcer disease (P=0.01). Histologic grade 3 gastritis was only observed in the GLA patients (P=0.03).

Conclusions: Hp-I seems to be related to GLA. If a causal link between Hp-I and GLA is confirmed this may have a major impact on the pathophysiology and management of GLA.

P.409 **MICs OF RABEPRAZOLE, A RECENTLY DEVELOPED PROTON PUMP INHIBITOR, AND OMEPRAZOLE, AGAINST HELICOBACTER PYLORI**

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Several studies have shown that the proton pump inhibitors (PPIs) of the benzimidazole type (omeprazole, lansoprazole, pantoprazole) exert antibacterial in vitro activity against *Helicobacter pylori (H. pylori)*. However, few in vitro data exist on Rabeprazole, the newest PPI.

The aim of this study therefore, was firstly, to compare rabeprazole MICs against *H. pylori* with those of omeprazole and secondly, to examine whether these MICs were influenced by *H. pylori* susceptibility or resistance towards commonly used antibiotics for *H. pylori* eradication such as metronidazole, clarithromycin and amoxycillin.

Material and Methods: Fifty *H. pylori* strains were tested. All strains were recent clinical isolates from different patients with chronic gastritis and/or peptic ulcer. In addition one reference strain, *H. pylori* CCUG38771, was also included in the study. For each component examined the MIC_{50} (the MIC at which 50% of strains were inhibited) and MIC_{90} (the MIC at which 90% of strains were inhibited) were determined by agar dilution.

Results: MICs (μg/mL) for omeprazole were within the range of 32–128 with MIC_{50} at 32 and MIC_{90} at 64. The respective values for rabeprazole were markedly lower (range 4–16, MIC_{50} 4 and MIC_{90} 16). *H. pylori* resistance percentages for metronidazole, clarithromycin and amoxycillin were 45%, 9.8% and 0% respectively (E-test). Antibiotic resistance did not influence MICs of either PPI tested.

Conclusion: Rabeprazole? the newest PPI, is more effective than omeprazole in vitro against *H. pylori* clinical isolates and this effectiveness is not influenced by *H. pylori* resistance to commonly used antibiotics for *H. pylori* eradication.

P.400 **RANITIDINE BISMUTH CITRATE BASED REGIMENS FOR HELICOBACTER PYLORI ERADICATION. WHICH IS THE OPTIMAL TREATMENT DURATION AND ANTIBIOTICS COMBINATION?**

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H. pylori (HP) eradication therapies based on ranitidine bismuth citrate (RBC) have recently been introduced in clinical practice. The aim of this study was to compare the efficacy of 2-week vs 4-week regimens with RBC in combination with 2 antibiotics during the 1st week.

Methods: 104 patients with HP infection (F: 51, M: 53 with mean age 51.5 and range: 22-79 yrs) were offered 4 eradication regimens: group A (n=27) RBC 400mg bid for 2 weeks plus clarithromycin (CLA) 500mg and metronidazole (MET) 500mg bid for one week, group B (n=23) RBC 400mg bid for 2 weeks plus HCl-tetracycline (TET) 500mg and MET 500mg bid for one week, group C (n=27) RBC 400mg bid for 4 weeks plus CLA 500mg and MET 500mg bid for one week and group D (n=27) RBC 400mg bid for 4 weeks plus TET 500mg and MET 500mg bid for one week. In all patients endoscopy was repeated one month after cessation of treatment and eradication was considered successful if both rapid urease test and histology were negative.

Results: HP was eradicated overall in 85/104 pts (81.7% with 95% CI's 74.2%-89.3%) while symptoms improved in 72.5%. The 4 groups were comparable with respect to age, gender, smoking, alcohol and NSAID's consumption and peptic ulcer prevalence. HP eradication rates were 92.6% (75.7-99), 60.8% (38.5-80.3), 88.9% (70.8-97.6) and 81.5% (61.9-93.7) in groups A,B,C, and D, respectively. Eradication rate was significantly lower in group B patients compared to groups A,C and D (χ^2 test: p=0.021). There were no significant differences between groups concerning ulcer healing, resolution of symptoms and side effects.

Conclusions: 1) A 2-week regimen with RBC combined with CLA and MET during the 1st week is highly efficacious in HP eradication but substitution of CLA with TET reduces efficacy significantly. 2) Both 4-week regimens with RBC achieve high eradication rates regardless the antibiotics combination used in this study.

P.428 **EFFICACY AND SAFETY OF THREE 'TRIPLE'-DRUG REGIMENS IN H.PYLORI ERADICATION – FORERUNNING ANNOUNCEMENT**

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Background: There are many drug regimens of H.p. eradication. Several studies try to compare the efficacy and safety of these regimens.

Aim: To show the effectiveness and safety of the three ten-days 'triple' drug regimens in H.pylori eradication.

Methods: 134 outpatients, 63 men and 71 women, aged 18-75 years, were subjected to endoscopy for upper gastrointestinal symptoms. The presence of H.p. infection was estimated by CLO test and histological examination (modified Giemsa stain, H-E). All patients were assigned to three different triple-drug regimens of H.p. eradication by simple blind 'haphazard' randomization; amoxycillin 1gr bid, clarithromycin 500 mg bid and omeprazole 20 mg bid(group A: 40 patients) or lansoprazole 30 mg bid(group B:34 patients) or salt ranitidine-bismouth 400 mg bid(group C:29 patients)were given for ten days. The 1st follow up was done after 60-70 days.

Results: 103 patients were checked again up to now matched to age, sex, alcohol reception and smoking. H.p. eradication was achieved in group A in 31 pts (77,5%), in group B in 23 pts (67,6%) and in group C in 22 pts (75,8%). Side effects (epigastric pain, diarrhea, vomiting etc) were observed in 3 pts of group A, in 2 pts of group B and in 6 pts of group C.

In group A, 1 of the pt stopped therapy on the 9th day but H.p. was eradicated while the other 2 pts continued therapy. In group B, 1 of the pt stopped therapy on the 7th day but H.p. was eradicated while the other pt continued therapy. In group C, 2 of them continued therapy, 2 were given another regimen of H.p. eradication, 1 stopped therapy on the 7th day but H.p. was eradicated and 1 pt withdrew due to side effects.

Conclusions: From the forerunning results of our study, the giving of the three 'triple' regimens of H.pylori eradication is satisfactory in confronting the infection of H.p.. Perhaps, the side effects of group C in comparison with the other two groups constitute a cautions factor in providing the drug regimen.

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IS ACID SUPPRESSION EFFICACIOUS IN NON-ULCER DYSPEPSIA?

A double-blind, randomized, controlled trial with omeprazole

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Background

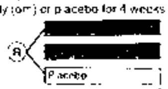
The efficacy of antisecretory therapy in non-ulcer dyspepsia (NUD) has been equivocal based on trials of H_2 receptor antagonists. The therapeutic place of proton pump inhibitors (PPIs) in NUD is unknown. While NUD subgroups have been proposed, their clinical relevance have been questioned. It has been theorised that *H. pylori* positive NUD patients may respond best to PPI's because of acid dysregulation.

Aim

To evaluate the efficacy of the PPI omeprazole in patients with NUD, in NUD subgroups and in *H. pylori* infected and uninfected cases.

Methods

The patients were recruited by family physicians (38) and gastroenterologists (101) and were treated with omeprazole 20 or 10 mg once daily (om) or placebo for 4 weeks.



	Visit 1 / Days 0	Visit 2 / Days 28±4
Symptoms	x	x
^{13}C Urea breath test (UBT)	x	
Diary card	x	x
Quality of Life	x	x

Study design

NUD patients between 18 and 80 years with at least a months history of dyspeptic symptoms were included. Persistent or recurrent epigastric pain/discomfort and normal findings at upper endoscopy were required. We excluded patients with a history of PUD or GERD or any esophageal or gastric surgery as well as patients with heartburn/regurgitation (without epigastric component) or IBS.

ASSESSMENTS

Epigastric pain/discomfort
Investigator's interview: 4 graded Likert scale (none, mild, moderate, severe). Recorded daily on diary cards (absent or present) during the whole study period. ***Treatment success (main efficacy variable) = no symptoms at 4 weeks*** during last 3 days.

Helicobacter pylori
At entry, a standardised UBT was obtained. The result of the test was kept blind throughout the study.

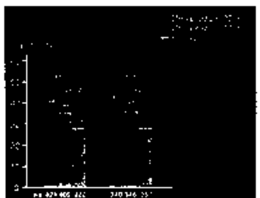
Dyspepsia subgroups
Patients ranked their most bothersome symptom from a list of specific dyspeptic symptoms. Dyspepsia subgroups were based on this evaluation.
- "Ulcer-like": epigastric pain
- "Dysmotility-like": bloating, belching, early satiety, postprandial fullness, nausea, flatus
- "Reflux-like": heartburn/regurgitation

Sufficient control of symptoms
Investigator's question at 4 weeks: "Does the study medication give sufficient control of your symptoms?" (no/yes)

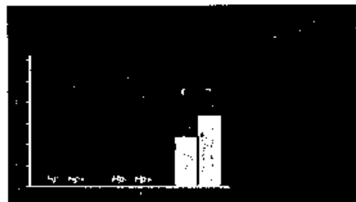
Quality of Life
Two validated questionnaires were used
- GSRS, the Gastrointestinal Symptom Rating Scale, measuring gastrointestinal symptoms on a 7-graded Likert scale
- PGWB, the Psychological General Well Being Index, measuring subjective well being or distress on a 6-graded Likert scale

Results

A total of 1248 patients (470 males/778 females) at 139 centres in 14 countries* were included in the intention to treat analysis (ITT). The per protocol analysis (PP) included 1043 patients. The three treatment groups were well balanced regarding baseline characteristics. Mean age was 43 years and 63% had been symptomatic for more than a year. A total of 41% of the patients were *H. pylori* positive by the UBT test.



Complete relief of epigastric pain/discomfort after 4 weeks of treatment



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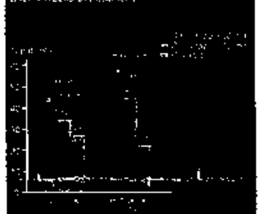
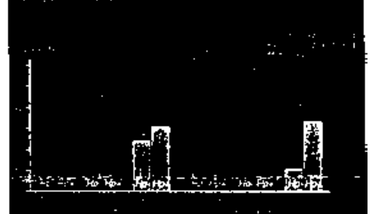


Complete relief of epigastric pain/discomfort after 4 weeks of treatment by dyspepsia subgroup, ITT



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In the group of patients with dysmotility dyspepsia no advantage in complete relief of epigastric pain/discomfort of omeprazole 20 mg and omeprazole 10 mg was seen compared to placebo (31%, 35% and 33% (NS)) respectively. Sufficient control of symptoms was reported by 51% on placebo compared with 61% on omeprazole 20 mg (p=0.006) and 59% on omeprazole 10 mg (p=0.02).

The total GSRS score was significantly improved in patients receiving omeprazole 20 mg (p=0.02) compared with placebo. The total PGWB score improved similarly in all groups (NS).

A logistic regression analysis indicated that recruitment in family practice (p=0.003), older patients (p=0.02), patients with a higher body mass index (p=0.02) and patients with reflux-like dyspepsia (p=0.07) were significantly more likely to respond to active treatment than placebo.

Omeprazole is superior to placebo in NUD patients at standard (20 mg) and low (10 mg) doses; there is a clinically significant effect in the subgroups ulcer- (omeprazole 20 mg) and reflux-like (omeprazole 10 mg and 20 mg) but not in dysmotility-like dyspepsia. The association between treatments effect and subgroups was not explained by differences in *H. pylori* status.

*The International Multicenter study group [illegible] Belgium: [illegible] Canada: [illegible] Denmark: [illegible] France: [illegible] Greece: [illegible] Hungary: [illegible] Ireland: [illegible] The Netherlands: [illegible] Poland: [illegible] Portugal: [illegible] UK: [illegible]