COMPARISON OF H. PYLORI FINDINGS FROM INFECTED PARENTS AND OFFSPRING USING DNA TYPING
Royal Victoria Hospital, Belfast. Departments of Microbiology and Pathology, The Queen's University of Belfast, Royal Belfast Hospital for Sick Children, and NCTC, Collindale, London

The source of infection and transmission mode of H. pylori is unclear. A high prevalence of infection within families has been reported using breath testing or serological techniques. The aim of this study was to analyse the strains of H. pylori present within a series of co-residing families. Blood and Biopsies from 4 infected children who had presented to a paediatric clinic with dyspepsia underwent endoscopy, gastric antral biopsy, and histology. Although the younger parents were asymptomatic at time of investigation, one male had reflux oesophagitis at endoscopy. Biopsies were cultured on Columbia-based blood agar under microaerophilic conditions for 4 days. Cultures from parents and children underwent DNA extraction and the electrophoretic patterns were compared after digestion with restriction endonucleases and the use of a binodiluted CO2a probe prepared from 16S and 23S RNA of H. pylori NCTC strain 11638. Seven parents were H. pylori positive by rapid urease test and histology, and cultures were obtained. DNA typing showed the same strain present in at least two members in 3 of the 4 families. The family 1, mother, father, son and father had the same strain; in family 2, father and son had the same strain while mother was culture negative; father and mother had the same strain in family 3; and all strains were unique in family 4. These findings suggest, for the first time, that there is intra-familial spread or a common source of infection within families.


Duodenal ulcer recurrence can be prevented by eradication of H. pylori: no evidence of infection at least one month after finishing treatment. The non-invasive 13C-urea breath test, free of sampling error and able to detect lower levels of active infection than endoscopic biopsy based methods or serology, is ideal for follow up after treatment. This study determines how soon H. pylori recurs after therapy thus providing a rational time for assessment of eradication.

Patients needing H. pylori eradication were invited to enter the study. Before starting treatment H. pylori status was assessed by antral histology (H&E, and Gimenez stains), culture (microaerophilic conditions for up to 10 days) and 13C-UBT (European standard protocol, positive result = excess 13CO2 > 5 per ml). The 13C-UBT was repeated immediately after finishing treatment and then at weekly intervals for 1 month (or until positive) and subsequently at 3, 6, and 12 months.

Forty-six patients (26 men, median age 45 y, range 19-67 y) with either active (n=10) or previous (n=36) DU were studied. All patients had a positive 13C-UBT (mean ±SEM excess 13CO2 excretion = 26.8 ± (12.4) per ml) and either positive histology (n=42), or culture (n=40) before starting treatment. H. pylori was cleared in all patients: 13C-UBT negative immediately after finishing treatment (double / triple therapy for 1 or 2 weeks). In 17/46 patients eradication therapy failed: H. pylori recurred in 13/17 without symptom recurrence, (mean ±SEM excess 13CO2 excretion = 12.4 ± (6.7) per ml) at median 8 days (range 6-18d). In all 29 patients in whom H. pylori was successfully eradicated at one month mean ±SEM excess 13CO2 excretion = 2.4 ± (0.9) per ml the breath test was negative at 2 weeks and thereafter (median t/b = 4.6 months, range 1.0 - 7.2).

These results show that the 13C-UBT can detect recurrent H. pylori within days of finishing anti-H. pylori therapy, and suggest that eradication can be accurately diagnosed 2 weeks after the end of treatment.

Dept Gastroenterology, Immunology and Clinical Medicine and Microbiology, St James's Hospital and Trinity College Dublin.

Helicobacter pylori (HP) is capable of colonizing the gastric mucosa, despite a humoral response, in 30-60% of healthy adults. In this study we have examined the cellular response to HP by measuring in vitro proliferative response of peripheral blood mononuclear cells (PBMC) to purified HP antigens, purified protein derivative (PPD), whole cell inactivated E. coli antigens and phytohaumagglutinin (PHA) in 37 dyspeptic patients undergoing upper gastrointestinal endoscopy. A standard five day thymidine incorporation assay was used to test the response of HP antigen preparation (as determined in preliminary experiments) of 3ug/ml was utilized. HP status was determined by culture, histology and the rapid urease test on antral biopsy. Patients with HP colonisation (HP+) had significantly lower proliferative responses relative to HP negative (HP-) individuals [10±0.55 (n=21) vs 396±278 (n=16)] cpm 3H-Tdr incorporation, x SEM, p<.0.1]. There was no significant difference in proliferative responses to PPD [477±163 vs 810±2466, x SEM, p ns] or to PHA [104±215.6 vs 95±0.525] in patients with HP colonisation (as determined in preliminary experiments) of 3ug/ml was utilized. HP status was determined by culture, histology and the rapid urease test on antral biopsy.

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**H. Pylori Lectins Detected by Neoglycoconjugates.**
Medical Research Centre, Nottingham City Hospital, United Kingdom.

Gastric epithelial surface glycoconjugates were found previously to bind Peanut agglutinin (PNA, gal-Nac-HSA binding), Vicia villosa agglutinin (VVA, galNac-HSA binding) Ulex europaeus I (fucose binding), Arthroplax simplicifolia I (galactose binding) and concanavalin A (mannose binding) lectins. We have used 5 neoglycoconjugates (NG), each bearing one lecthir-mannosyl phosphatase, to detect H. pylori surface lectins in this study. The NG gal-Nac-HSA-Human serum albumin (HSA) and galNac-HSA were conjugated to biotin and then purified by gel filtration. They were subsequently confirmed to bind PNA and VVA by ELISA. Gold labelled Fucose-Bovine serum albumin (BSA), galNac-BSA and mannose-BSA were obtained commercially. These 5 NG were used in the ELISA. H. pylori surface proteins coated micro-ELISA wells were incubated with 100 ng (20 μg/ml) conjugated NG. The unbound NG were then washed off and the bound biotin-labelled NG were identified by using avidin-biotin complex/peroxidase and O-phenylenediamine diimine in citric acid buffer containing H₂O₂. The bound gold-labelled NG were identified by silver enhancement controls. The controls were performed by replacing the NG by unconjugated HSA or BSA followed by washings and then further incubation with an HSA-labelled anti-HSA or anti-BSA antibody. The peroxidase activity was similarly identified as before.

**Prevalence of Helicobacter pylori (H. Pylori) Infection Amongst Attendees at the Autumn BSG Meeting 1991: “Bare Handed” Endoscopy a Risk Factor for HP Acquisition.**
S. Pugh.
Department of Gastroenterology, University Hospital of Wales, Cardiff.

Diagnostic testing for Hp infection by ¹³C breath test was offered by a pharmaceutical company at the Autumn 1991 meeting of the BSG. 144 attendants were tested and 115 returned an anonymously completed questionnaire giving details of their test result, age, sex, professional status, endoscopy service, use of gloves and past medical history of dyspepsia and proven upper GI disease. Respondents were 63 doctors (61 male, (mean age ± SD) 43.0 ± 8.9), 37 nurses (all female, 44.2 ± 8.6) and 15 non-clinical respondents (7 male, 42.4 ± 12.3). Positive tests for Hp infection were found in 25 of 63 doctors (13 of 27 nurses and only 5 non-clinical respondents (P=0.05 cf doctors and nurses, Fisher's exact test). Life table analysis did not show a significant increase in probability of infection with age, length of clinical or endoscopic service. However, Endoscopy Unit staff who always wore gloves had a lower rate of infection (8 Hp +ve of 34) than did staff who had at some time worked “bare-handed” (27 Hp +ve of 57) P<0.05 (Chi square test). Overall, Hp +ve respondents were more likely to suffer from dyspepsia (21 of 39) than Hp -ve respondents (21 of 76) P<0.01 (Chi square test). Three Hp +ve respondents and one Hp -ve respondent had a past medical history of DU (NS, Fisher’s exact test).

In this small survey both doctors and nurses have a greater prevalence of Hp infection than the non-clinical group. Acquisition is probably early in the career as prevalence does not increase with length of service. Always wearing gloves is associated with a lower rate of infection. Infection with Hp is associated with a greater rate of dyspepsia but not in proven cases of ulcer disease, but the survey may have been too small to show this.