Helicobacter pylori is the most common infection affecting humans. It affects about 50% of subjects living in developed countries by the age of 50 years. The epidemiology of this infection is different in India. Indians are infected more frequently and at an earlier age; most studies report a prevalence of 80% by the age of 20 years. This difference in prevalence is related to poor living conditions and hygiene, fecal contamination of drinking water and overcrowding.

Further, most reports (some of them not properly designed) suggest a high rate of reinfection following eradication in India. Therefore, in a large number of studies, attempts have been made to find out the possible reservoir(s) and route(s) of transmission of H. pylori. Though a majority of studies suggest a fecal-oral route of transmission, in some studies oro-oral transmission has been suggested. Early Indian studies suggested a high frequency of carriage of H. pylori in dental plaques in healthy Indian subjects, in contrast to the low rate of carriage reported from the West. In fact, Desai et al. reported that all 40 healthy volunteers studied tested positive for H. pylori in dental plaque. These studies gained importance as evidence in favor of oro-oral transmission of H. pylori, explaining the high frequency of infection and reinfection in India.

The study by Kumat et al. from Mumbai in this issue of the Journal is interesting as it shows a low frequency of H. pylori in dental plaque (rapid urease test positivity in 23.2%, culture positivity in none, and polymerase chain reaction [PCR] positivity in 4.4%). The results of this study are quite similar to those reported from the West.

One immediate lesson that comes to mind is that drawing a conclusion based on a single study or a few studies may sometimes be fallacious. Most studies from the West reported the rarity of H. pylori in dental plaque. This is in accordance with the known biological behavior of H. pylori, i.e., lack of growth in the absence of gastric mucosa. Therefore, it was important to look at the methodology used in the Indian studies, which reported a high rate of carriage of H. pylori in dental plaque. Most early workers used positive urease test as a criterion for the diagnosis of H. pylori infection in dental plaque. Though the sensitivity and specificity of this test have been validated for gastric H. pylori infection, they have not been evaluated in dental plaque. With the abundance of urease-producing organisms in dental plaque, a false-positive diagnosis of H. pylori infection is possible. Other bacteria which can give a positive urease reaction include Proteus mirabilis, Citrobacter freundii, nonpathogenic Neisseria, and Streptococcus faecalis. Though the existence of unknown inhibitors of PCR in dental plaque cannot be entirely excluded, as argued by Kumat et al., it seems less likely considering that from none of the samples with positive urease test could H. pylori be isolated in culture. This study thus argues against oro-gastric spread of H. pylori as an explanation for the increased prevalence and reinfection after eradication in Indian patients.

Another issue which has attracted the attention of many scientists is the relatively low frequency of peptic ulcer both in India and abroad despite a very high frequency of infection. In India, the point prevalence and estimated lifetime prevalence of peptic ulcer vary between 2.8% and 7.8% and 11-16%, respectively, despite a high prevalence of infection (80% in adulthood). Though a difference in host response cannot be excluded, heterogeneity in the H. pylori strain is a more likely reason. In 1988, Leunk et al. showed that some H. pylori strains produced a protein in culture supernatant which induced vacuolation in cultured epithelial cell lines in vitro. Subsequently, both the gene encoding for this protein and the protein have been characterized. Though all H. pylori possess the vacA gene, only 40% of strains are toxigenic. The gene encoding for the protein possesses three regions: signal sequence, mid region and C-terminal region. There are three allelic types for signal sequence, s1a, s1b and s2, and two for the mid region, m1 and m2. Strains with s1a m1 alleles are more toxigenic than those with s1b-m1 alleles; m2 alleles are least toxigenic. Cytotoxicity-associated gene A (cagA) is another pathogenicity determinant of H. pylori. cagA-positive strains are associated with higher levels of inflammation and pro-inflammatory cytokines (e.g., interleukin 8) expression. Strains with cagA usually have the vacA s1a or b genotype, and are therefore toxigenic; in contrast, strains without cagA usually have the vacA s2 genotype, and are therefore non-toxigenic. A large number of studies showed that cagA+ strains are more common in patients with peptic ulcer and gastric adenocarcinoma than in controls. One earlier study showed that these strains are more common in non-ulcer dyspepsia (NUD) than in asymptomatic subjects. The interrelation between these cytotoxicity determinants and host factors determines the outcome of infection. Further, all subjects infected with pathogenic strains do not develop disease; on the other hand, patients infected with H. pylori without any of these toxigenic factors have been reported to have peptic ulcer disease.

In this issue of the Journal, Kumar et al. report that the observed optical density for serum anti-Cag A anti-
body titre in patients with duodenal ulcer (DU) and non-ulcer dyspepsia (NUD) was higher than that observed in controls; there was no difference in titre between DU and NUD patients. The authors conclude that anti-Cag A antibodies do not discriminate DU from NUD patients in India. However, certain points need consideration before this conclusion can be accepted. The size of the sample does not appear large enough for an acceptable statistical power. Also, the authors have not mentioned their criteria for the diagnosis of NUD. Presumably, they excluded patients receiving antisecretory therapy; otherwise, a possibility of including DU as NUD remains. Finally, the high optical density in asymptomatic controls might suggest the presence of cagA-positive H. pylori in the stomach of some of these healthy subjects. This is not entirely unexpected considering the high prevalence of H. pylori infection in our adult population. 2

This study is a landmark in this issue; however, its clinical importance is limited. Presently, a majority of clinicians depend on endoscopic demonstration of gastrinocutaneous disease as proof of the pathogenic nature of H. pylori in a particular patient. A patient with peptic ulcer and H. pylori infection needs anti-H. pylori therapy. Every patient infected with pathogenic H. pylori does not develop disease. 23,28,29 Therefore, determination of pathogenic H. pylori by serum assay for anti-Cag A antibody will identify more patients infected with the pathogenic strain of the organism. But, unless studies show that treating these patients who harbor pathogenic H. pylori without peptic ulcer, gastric carcinoma or gastric lymphoma will benefit them, what is the use of identifying them by determination of anti-Cag A antibody status? One strong current viewpoint favors preventing gastric cancer by treating this subset of patients; unfortunately, we still do not have data-based evidence to support this stand.

D N Guha Mazumder, U C Ghoshal
Department of Gastroenterology, Institute of Postgraduate Medical Education and Research, 244, AJC Bose Road, Calcutta 700020

References
23. Abberton JC, Coo P, Peek RM, et al. Mosaicism in vacu-


Correspondence to: Prof Guha Mazumder

---

ISG NEWS

39th Annual Conference

This conference will be held at Pune, October 29-November 1, 1998. I am sure you have planned your journey to Pune. The scientific program is quite interesting and informative. Several national and international experts will be attending. For further information please write to Dr Vinay Thorat, Organizing Secretary, 3rd floor, 375 Narian Peth, Pune 411 030.

40th Annual Conference

This conference will held at Calcutta in November 1999, and the details will be published in forthcoming issues.

Awards

For the following awards, five complete sets of nominations/applications, properly tagged, should reach the Secretariat before July 15, 1999. Incomplete or late applications will not be considered. Details regarding these awards will be published in the January 1999 issue of the *Journal*.

* Parke-Davis Oration (2000)
* Knoll Oration (2000)
* Hoechst Om Prakash Memorial Award (1999)
* Olympus Mitra Endoscopy Award (1999)
* Dr P N Chuttani Oration (1999)

---

Membership dues

Ordinary members should send their annual dues to Dr Love Dalai, A-14 Silver Arch, Behind Town Hall, Ahmedabad 380 006. The revised fees are as follows:

- Ordinary member: Rs 500
- Life member: Rs 5000
- SAARC member: Rs 7500

State Chapters

Kindly inform the Secretariat the details of activities and names of office bearers of State Chapters of the ISG.

Y K Joshi
Hon Secretary, ISG
Department of Gastroenterology & Human Nutrition Unit
All India Institute of Medical Sciences
Ansari Nagar, New Delhi 110 029
Phones: (011) 659 4290, 659 4632, 686 4851 Extn 4290 (O)
696 8244 (R). Fax: (011) 686 2663
E-mail: ykjoshi@medinsternet.in (O)
smiti@giadl.vsnl.nct.in (R)

Indian Journal of Gastroenterology 1998 Vol 17 October - December 125