Since its discovery in 1982\textsuperscript{1}, Helicobacter pylori has rapidly emerged as an important pathogen in gastric microbiology. It has led to a re-appraisal of our understanding regarding various gastroduodenal diseases and has therefore transformed their management. Formerly known as Campylobacter pylori, H. pylori is a gram negative microaerophilic organism. Colonization is common throughout the world and approximately half the world’s population is infected\textsuperscript{2}. Infection is usually acquired during childhood\textsuperscript{3}, probably through person-to-person transmission although no single vector has been identified\textsuperscript{4}. Incidence rates in both children (36% per year) and adults (3-10% per year) in developing countries are significantly higher than in developed countries (0.4% and 2.7% per year respectively)\textsuperscript{5}. These differences are attributed to the low socioeconomic status and low level of education in developing countries\textsuperscript{6}.

H. pylori is one of the most common bacterial infections worldwide\textsuperscript{7}, but in majority of the infected individuals it only causes a histological gastritis with no clinical manifestations\textsuperscript{8}. It can lead to chronic active type B gastritis in virtually all infected subjects\textsuperscript{9} and is associated with an increased risk of chronic atrophic gastritis\textsuperscript{10}. Infection has a four-fold likelihood of developing duodenal and gastric ulcerative complications\textsuperscript{11}. In a small fraction of individuals H. pylori infection leads to proliferation of lymphoid tissue which progresses to a mucosal associated lymphoid tissue (MALT) lymphoma\textsuperscript{12}. H. pylori infection also has a well-defined risk of causing the intestinal form of gastric adenocarcinoma\textsuperscript{13,14}. In this respect, H. pylori has been categorized as a group 1 carcinogen\textsuperscript{2}. Non-gastroduodenal disorders have also been preliminarily linked to its infection\textsuperscript{15}.

However, host susceptibility, environmental and bacterial factors also modulate the clinical outcome. This explains the discordance between the proportion of people infected and the smaller proportion which develops symptomatic disease. Host factors include age at infection\textsuperscript{16} blood group O\textsuperscript{17} acid secretory capacity\textsuperscript{18} and specific genetic differences\textsuperscript{19}. Environmental factors also play an important role\textsuperscript{20}. The bacterial related factors are attributed to the varying pathogeneity of the infecting strain\textsuperscript{21}. Consequently, various colonization, persistence and putative disease inducing factors have been identified\textsuperscript{22}. These include structural components (flagellae, adhesins), extra-cellular products (ureases, proteases), cytotoxin associated gene A (CagA) related factors, vacouling cytotoxins (VacA and hemolysins. These aid in colonizaiton, evasion of host response, cause direct damage to the host and through the activation of the immune response and its mediators. (IL-1,IL-8, TNF-alpha)\textsuperscript{23}. Therefore specific markers are associated with an increased risk of disease\textsuperscript{24}. Strains expressing the CagA-protein are at an increased risk of developing2 duodenal ulcers and gastric MALT-type lymphoma\textsuperscript{25}.

The two methods of diagnostic tests available for H. pylori detection are invasive and non-invasive tests. The biopsy based invasive tests include rapid urease testing, histology and culture. Non-invasive tests include various methods of antibody detection and the carbon labelled urea breath tests. The final choice of a test depends on the economic cost, clinical circumstances like recent antibacterial therapy and the accuracy of the modality\textsuperscript{26}.

Eradication of H. pylori has been recognized as the most cost- effective treatment in the management of peptic ulcer disease, its recurrence and bleeding\textsuperscript{27}. Modern therapy can achieve up to 80-95% eradication rates in compliant patients\textsuperscript{28}. Dual therapy with a proton pump inhibitor and either
amoxicillin or clarithromycin and bismuth based triple therapy were used earlier. The preferred newer triple combination therapy with a proton pump inhibitor and two antimicrobial agents has shown higher eradication rates, faster ulcer healing, prompt symptomatic relief with fewer side effects. Research for the development of a vaccine for both therapeutic and prophylactic purposes is underway. In circumspect, the considerable public health impact due to H. pylori infection underlines the need for increasing our understanding regarding the virulence, transmission, pathogenesis, treatment and eradication of Helicobacter pylori.

References