1-INTRODUCTION

In the early 1900s, descriptions of postmortem examinations of the human stomach included the presence of then-unknown spirochete. However, it was not until 1983 that the organism was isolated and cultured by Warren and Marshall at The Royal Perth Hospital in Western Australia. In the ensuing years, it was found in most patients with peptic ulcer disease and eventually was classified as Campylobacter Pylori. In 1989, the organism was assigned its own genus and became known as Helicobacter Pylori. Since then, the understanding of the role of *H. Pylori* in the pathophysiology and treatment of peptic ulcer disease has become one of the most important development in medicine in the 20th century[(1)].

It is estimated that half of the worlds population is infected with *H. Pylori*[(2)]. However, incidence and prevalence vary widely depending on the state of economic development[(3)]. Average rates of infection are 40% to 50% in developed countries and more than 90% in developing countries. Factors associated with an increased prevalence include low socioeconomic status, poor sanitary condition and overcrowding. Overall, the incidence and prevalence of *H. Pylori* infection have been decreasing in industrialized nations over recent generations. This decrease may be linked t improved social conditions in the population as a whole[(1)].

Helicobacter Pylori, which is responsible for the most common infection worldwide, has been implicated in several gastrointestinal

diseases, such as peptic ulcer disease, non-ulcer dyspepsia, gastroesophageal reflux disease, gastric adenocarcinoma, colorectal carcinoma, mucosa associated lymphoid tissue (MALT) lymphoma, and hepatic carcinoma[1].

The exact mechanism by which *H.Pylori* exerts its pathogenic effects on gastric mucosa is unknown, but several explanations exist. Certain bacterial factors, including those related to cytotoxin-associated gene A (CagA), as well as other inducers of cytokines, can cause both direct and indirect cellular damage and inflammation[4].

A variety of highly sensitive and specific tests are available to diagnose *H. Pylori* infection. However, testing should be performed only if treatment is intended. Asymptomatic persons should not be tested unless there is endoscopic or radiographic documentation of peptic ulcer disease [1].

In recent years, myriad treatment regimens have been proposed for the eradication of *H.Pylori*. Success of antimicrobial regimens for *H. Pylori* eradication depends on patient compliance and lack of antimicrobial resistance. Thus, complicated regimens or those associated with side effects, or both may result in noncompliance and failure to eradicate *H.Pylori*. Theoretically, therapies have improved such that regimens should demonstrate 85% to 90% efficacy [1].