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Helicobacter pylori is a microaerophilic Gram negative bacterium colonizes human gastric mucosa and is one of the most common bacterial infections worldwide ***(Thomas and Pertolani, 1994).*** It was found to infect all age groups and to be associated with antral gastritis, peptic ulcer disease, and possibly gastric ulcers and carcinomas ***(Brooks et al., 1998).*** Although spiral organisms have been seen in the stomach of humans and animals since 1893, it was not until 1983 that ***Warren and Marshall*** associated these S-shaped spiral bacilli with chronic gastritis. The organism was first called campylobacter pyloridis because of its structural similarity to the campylobacter species, later it was named C. pylori ***(Coghlan et al., 1987)*** only in 1989 C. pylori acquired its recognition as a new genus and was named Helicobacter pylori. The new name reflects the helical appearance of this organism in vitro as well as the most common isolation place, the pylorus of the stomach ***(Goodwin et al., 1989).***

Although the pathogenesis of H. pylori related gastroduodenal diseases is not well understood, there are several potential virulence factors of H. pylori that may contribute to the exacerbation of mucosal damage ***(Maeda et al., 1998).*** One of these factors which is known to differ among H. pylori strains is the expression of an 87 KDa vaculating cytotoxin which is toxic to epithelial cells in vitro ***(Cover et al., 1993).***

The majority of strains isolated from human ulcer biopsies produce this important protein, the cytotoxin that causes vaculisation (VacA) ***(Covacci et al., 1993).***

Although intracellular vaculation has been observed by electron microscopy in gastric epithelial cells from patients with chronic gastritis, the mechanism by which this damage is produced is still unclear ***(Covacci et al., 1993).***

Dent et al. (1988) reported that urease production by H. pylori is an important virulence factor for the development of gastritis.

Thomas et al. (1990) also suggested that the enzyme urease which allowed the bacteria to survive in the stomach was also acting as injurious factor either directly or through the ammonia that was produced from urea. Ammonia impaired mitochondrial and cell respiration.

Robert 1997 reported that the H. pylori produces urease enzyme, an enzyme that broke down urea to ammonia and carbon dioxide. Thus providing a protective alkaline environment for the organism to live and colonize gastric mucosa.