HELICOBACTER PYLORI – NOBEL PRIZE 2005

Summary

The Nobel Prize in Physiology or Medicine for 2005 has been awarded jointly to Barry J. Marshall and J. Robin Warren for their discovery of "the bacterium *Helicobacter pylori* and its role in gastritis and peptic ulcer disease". This year's Nobel Winners made the remarkable and unexpected discovery that inflammation in the stomach (gastritis) as well as ulceration of the stomach or duodenum (peptic ulcer disease) is the result of an infection of the stomach caused by the bacterium *Helicobacter pylori*. Thanks to the pioneering discovery by Marshall and Warren, peptic ulcer disease are no longer a chronic, frequently disabling condition, but a disease that can be cured by a short regimen of antibiotics and acid secretion inhibitors. The discovery of *Helicobacter pylori* has also led to increased understanding of the connection between chronic infection, inflammation and cancer.

Helicobacter pylori, a gramnegative spiral-shaped bacterium, member of -Proteobacteria, colonizes the gastric mucosa of humans. It is now recognized that *H. pylori* infects about half of the world's population (87% of Polish population). Infection is typically contracted in early childhood, frequently by transmission from mother to child, and the bacteria may remain in the stomach for the rest of the person's life. *H. pylori* has been identified as the causative agent of chronic inflammation, chronic gastritis and peptic ulceration and is believed to be a risk factor for the development of mucosa-associated lymphoid tissue lymphoma and adenocarcinoma of the stomach. The World Health Organization has assigned *H. pylori* as class I carcinogens. Although more than 50% of the human population is infected with *H. pylori* only a subset develops the disease. The nature and severity of the disease depend on host characteristics, bacterial genotype and environmental factors.

The focus of this minireview is on three major virulence factors of *Helicobacter pylori*: vacuolating cytotoxin VacA, CagA — an effector molecule of the type four secretion system and urease. VacA and CagA have also immunomodulatory activities that enable *H. pylori* to establish a chronic infection. The molecular basis by which *H. pylori* triggers cell signaling cascades and promotes inflammation and epithelial cell proliferation is described as well. This minireview also highlights recent developments in the field of *H. pylori* diagnosis and vaccine construction.