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Molecular Pathogenesis of *Helicobacter pylori* Infection

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There are three central questions in regards to *H. pylori* infection.

First, why are *H. pylori* infections prevalent in Korea?

Second, how does *H. pylori* infection lead to peptic ulcers, especially duodenal ulcers?

Third, how could *H. pylori* infection lead to stomach cancers?

Answers will be presented as follows.

Deficiency of anti-oxidants, especially vitamin C due to socio-psychological stress predispose people vulnerable to *H. pylori* infection in children.

H. pylori infection does aggravate the vitamin C deficiency in the gastric mucosa. Urease of *H. pylori* does inactivate the sensory function of antral mucosa to pH of the gastric lumen. This induces hypersecretion of acid-pepsin and gastric metaplasia in the duodenal mucosa. Gastric metaplasia provides ecological niche favorable to *H. pylori* colonization. *H. pylori* does invade the epithelial cells up to lamina propria. This induces persistent inflammation in the gastric mucosa. *H. pylori* has many antigens cross-reactive to gastric tissue as well as other human tissues. Therefore, *H. pylori* infection induces auto-immune hypersensitivity. The chronic inflammation in the gastric mucosa is the ultimate source of carcinogen, oxygen-free radicals.

How can we stop the chain of *H. pylori* infection?

Vaccine development and chemotherapy are not practical at this moment, and there are limited options available for the study of *H. pylori* with conventional bacteriological methods. Genomic and proteomic analyses at this laboratory up to now will be presented.

REFERENCES

1) Monograph of Gyeongsang National University College of Medicine, BK (Brain Korea) 21 *Helicobacter pylori* Research Center (1988-2000).