Helicobacter pylori infection

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H. Pylori is a spiral-shaped G – rods, associated with gastritis, duodenal (peptic) ulcer , gastric ulcer, & gastric carcinoma.

H. Pylori has many characteristics in common with campylobacters. It is actively motile by multiple flagella at one pole.

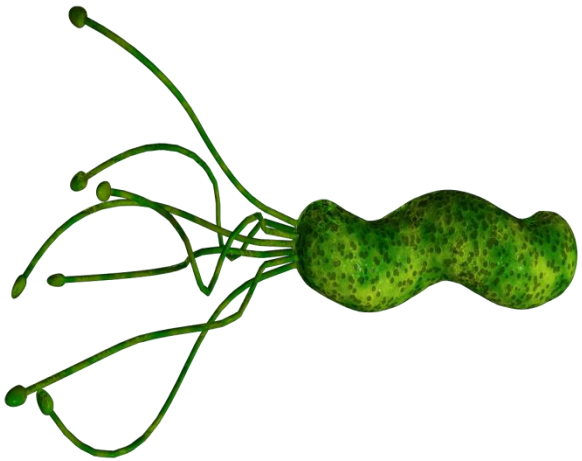
Culture & growth characteristics:

H. Pylori grow in 3-6 days when incubated at 37 ^{0}C in a microaerophilic condition, the medium for primary isolation is Skirrow's medium with vancomycin, polymyxin B & trimethoprim. The colonies are translucent & 1-2 mm in diameter***.***

Pathogenesis:

H. Pylori grows at a pH of 6-7 & would be killed at the PH within the gastric lumen. Gastric mucus is relatively impermeable to acid & has strong buffering capacity. On the lumen side of the mucus, the pH is low (1-2), while on the epithelial side is 7.4. ***H. Pylori*** is found deep in the mucous layer near the epithelial surface where physiologic pH is present. ***H. Pylori*** also produce a protease that modifies the gastric mucus & further reduce the gastric acid to diffuse through the mucus. ***H. Pylori*** produce potent urease activity which yield production of ammonia & further buffering of acid. ***H. Pylori*** is actively motile even in mucus & can be near the epithelial surface. ***H. Pylori*** overlies gastric-type, but not intestinal type epithelial cells.

In humans, ingestion of ***H. Pylori*** resulted in development of gastritis & hypochlohydria. There is a strong association between the presence of ***H. Pylori*** infection & duodenal ulceration. Antimicrobial therapy results in clearing of ***H. Pylori*** & improvement of gastritis & duodenal ulcer.



Helicobacter pylori infection

The mechanism by which *H. pylori* causes mucosal inflammation and damage involve both bacterial & host factors. The bacteria invade the epithelial cells to a limited degree. Toxins & LPS may damage the mucosal cells, & the ammonia produced by the urease activity may directly damage the cells also.

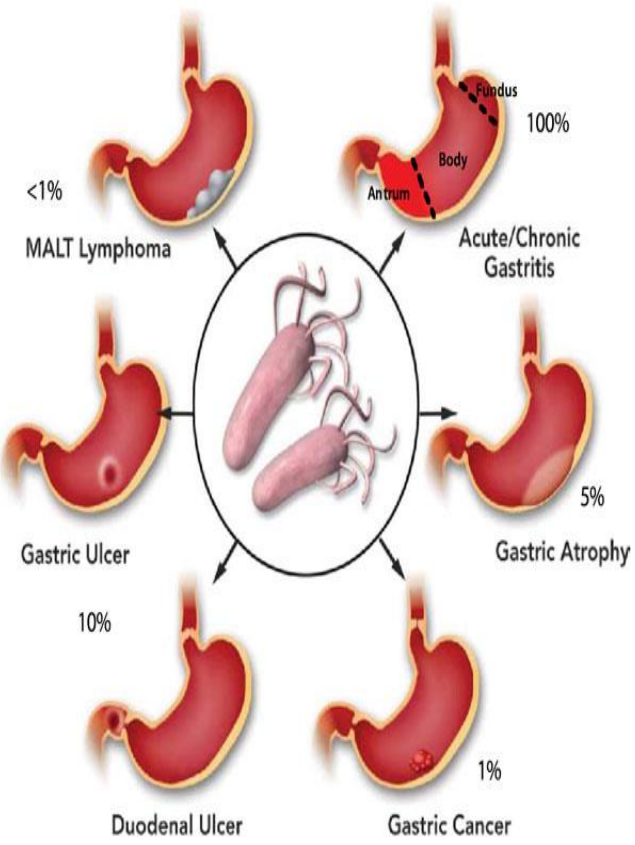
Clinical findings:

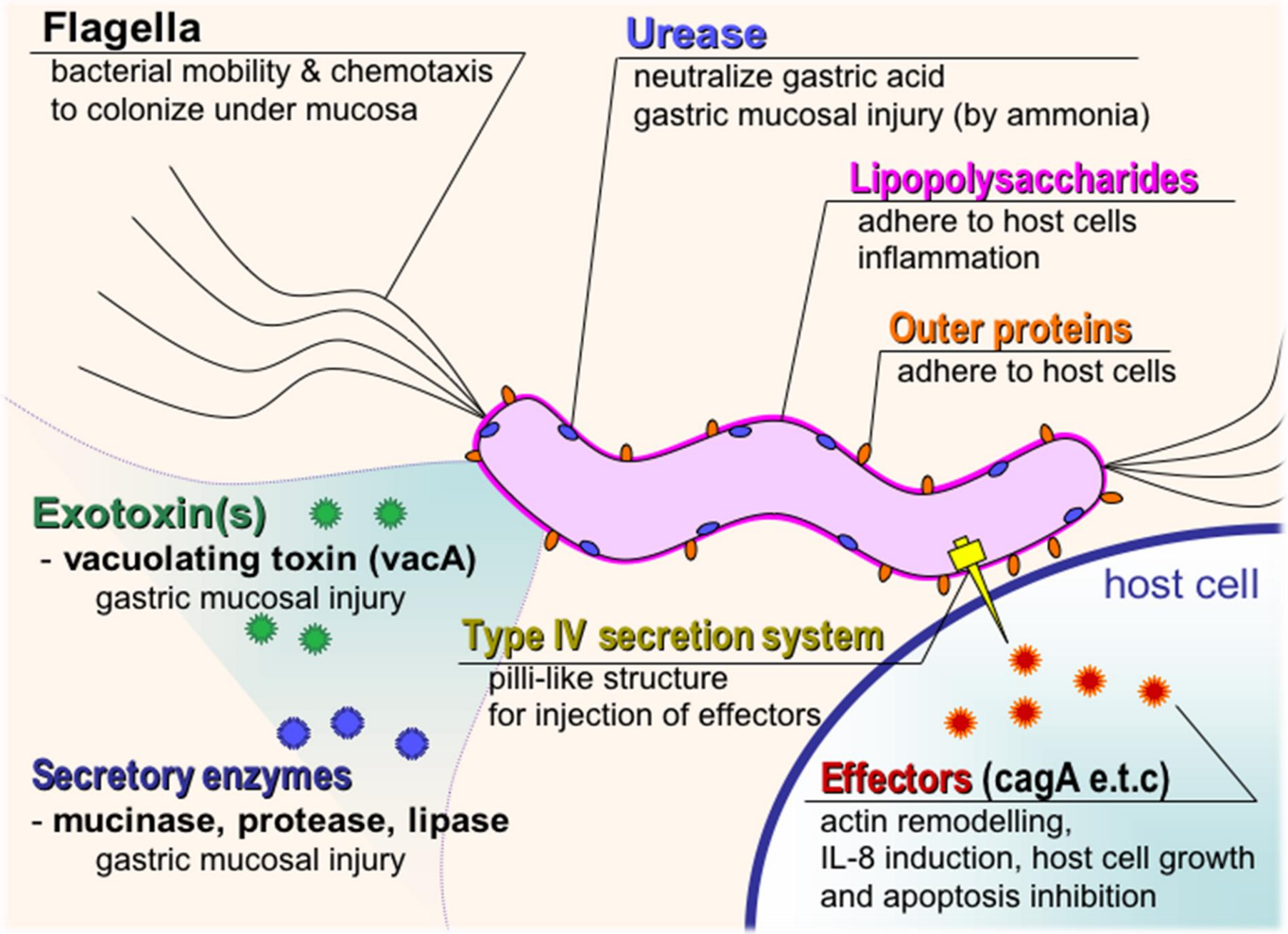
Acute infection can yield an upper gastrointestinal illness with nausea & pain. Vomiting & fever may be present also. The acute symptoms may lasts for less than 1 week or 2 weeks. Once colonized, *H. pylori* infection persist for years or even lifetime. About 90% of patients with duodenal ulcer & 50-80% of those with gastric ulcer have *H. pylori* infection. *H. pylori* may have a role in gastric carcinoma & lymphoma.

Lab. Diagnosis:

Specimens: gastric biopsy can be used for histological examination or can be minced in saline & used for culture. Serum for demonstration of Abs.

Smear: The diagnosis of gastritis & *H. pylori* infection can be made histologically. A gastroscopy procedure with biopsy is required. Routine stain demonstrate gastritis & Giemsa or special silver stains can show the curved or spiral bacteria.



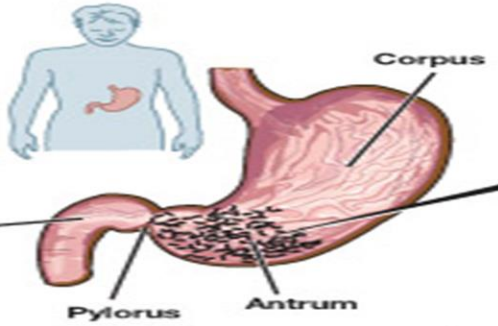

Flagella
bacterial mobility & chemotaxis
to colonize under mucosa
Urease
neutralize gastric acid
gastric mucosal injury (by ammonia)
Lipopolysaccharides
adhere to host cells
inflammation
Outer proteins
adhere to host cells
Exotoxin(s)
- vacuolating toxin (vacA)
gastric mucosal injury
Type IV secretion system
pilli-like structure
for injection of effectors
host cell
Secretory enzymes
- mucinase, protease, lipase
gastric mucosal injury
Effectors (cagA e.t.c)
actin remodelling,
IL-8 induction, host cell growth
and apoptosis inhibition

Helicobacter pylori

— the bacterium causing peptic ulcer disease

Infection

Helicobacter pylori infects the lower part of the stomach, antrum.



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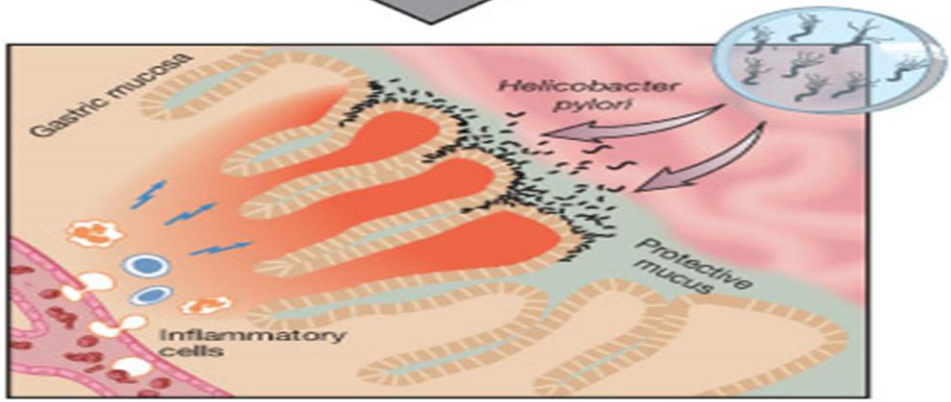
Inflammation

Helicobacter pylori causes inflammation of the gastric mucosa (gastritis). This is often asymptomatic.



Ulcer

Gastric inflammation may lead to duodenal or gastric ulcer. Severe complications include bleeding ulcer and perforated ulcer.

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Culture: on selective media & incubation conditions.

Serology: serum Abs specific for *H. pylori* infection may persist even if the infection is eradicated, so serology is of limited value in the diagnosis.

Other tests:

Tests to detect urease activity are widely used for presumptive identification of *H. pylori* infection in gastric biopsy. Detection of *H. pylori* Ag in stool is used to monitor treated patients with known infection by *H. pylori*.

Immunity:

Patients infected by *H. pylori* develop IgM Ab response, followed by IgG & IgA , & these persist both systemically & at the mucosa in high titers in chronically infected patients.

Epidemiology:

H. Pylori is present on the gastric mucosa of less than 20% of persons under age 30, & increases to 40-60% in those age 60 years. Including those who are asymptomatic. In developing countries, the prevalence of infection may be 80% or higher in adults. Person to person transmission of *H. pylori* is likely because intrafamilial clustering of infection occurs.