

***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:**

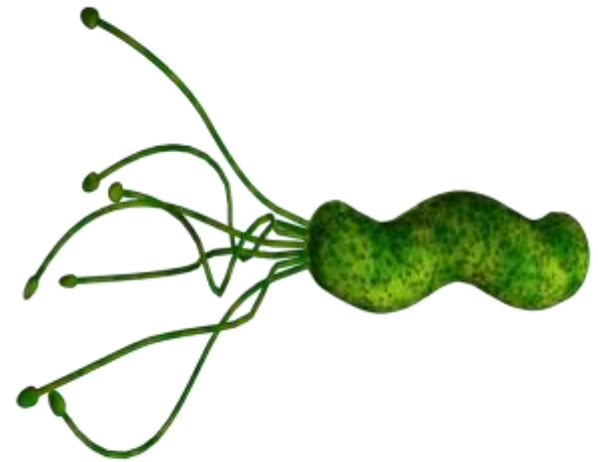
***H. Pylori*** grow in 3-6 days when incubated at 37 °C in a microaerophilic condition, the medium for primary isolation is Skirrow's medium with vancomycin, polymyxin B & trimethoprim. The colonies are translucent & 1-2 mm in diameter.

### **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:

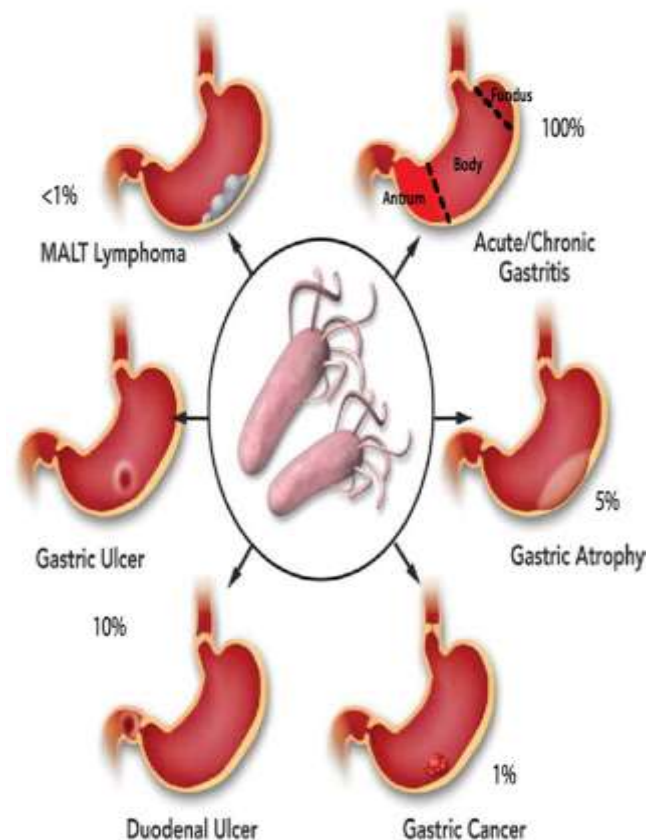
Acute infection can yield an upper gastrointestinal illness with nausea & pain. Vomiting & fever may be present also. The acute symptoms may last for less than 1 week or 2 weeks. Once colonized, *H. pylori* infection persists for years or even lifetime. About 90% of patients with duodenal ulcer & 50-80% 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 demonstrates gastritis & Giemsa or special silver stains can show the curved or spiral bacteria.

## *Helicobacter pylori* infection



## 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

## Secretory enzymes

- **mucinase, protease, lipase**  
gastric mucosal injury

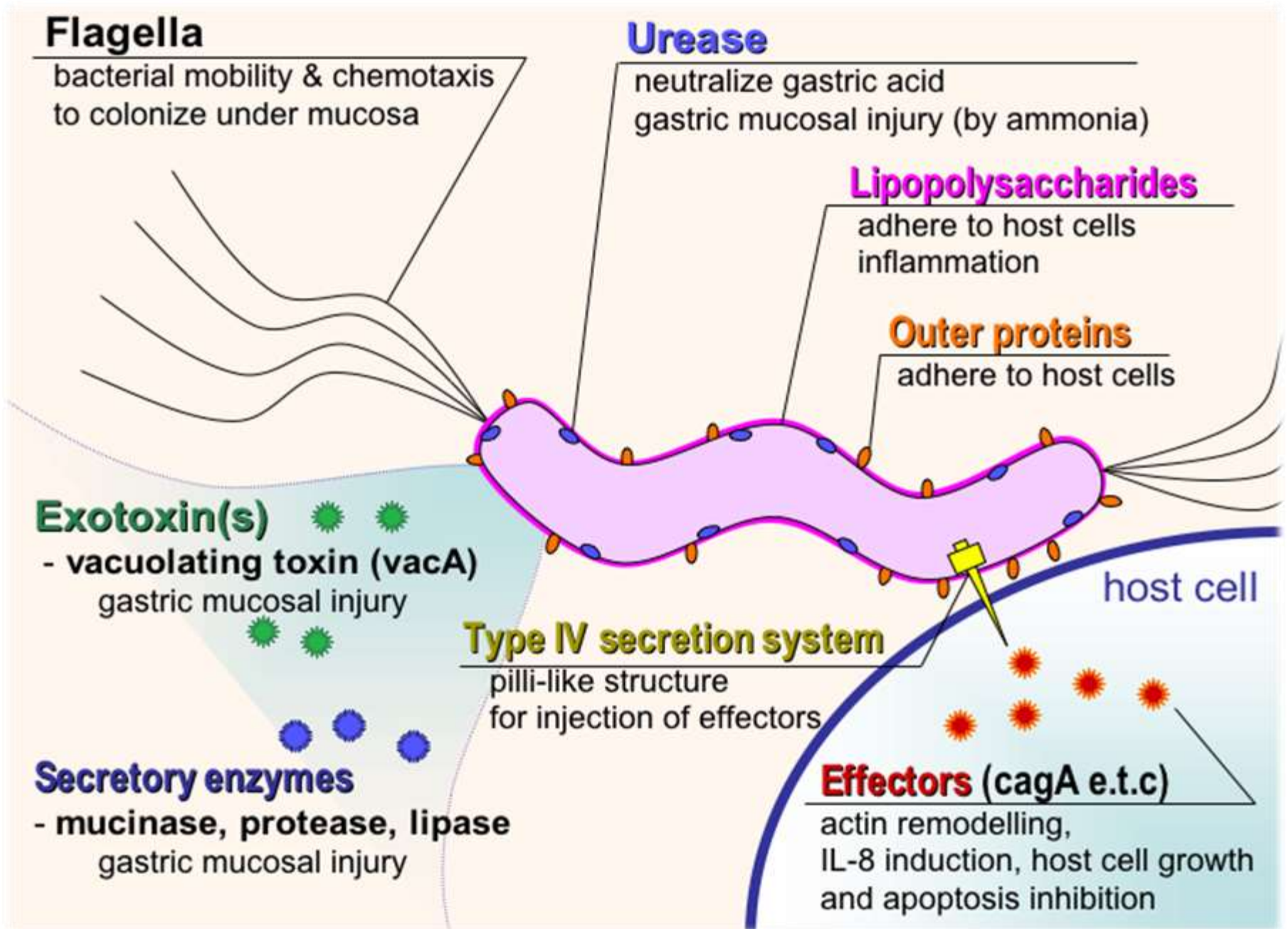
## Type IV secretion system

pilli-like structure  
for injection of effectors

## Effectors (cagA e.t.c)

actin remodelling,  
IL-8 induction, host cell growth  
and apoptosis inhibition

host cell

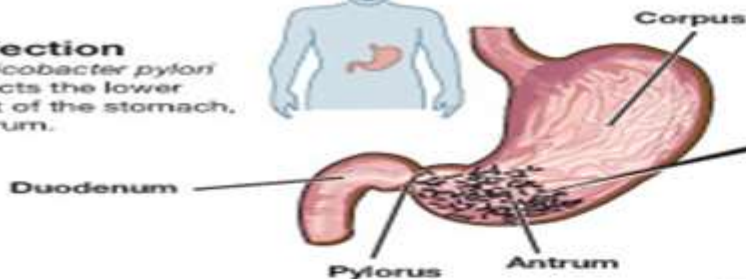


# Helicobacter pylori

— the bacterium causing peptic ulcer disease

## Infection

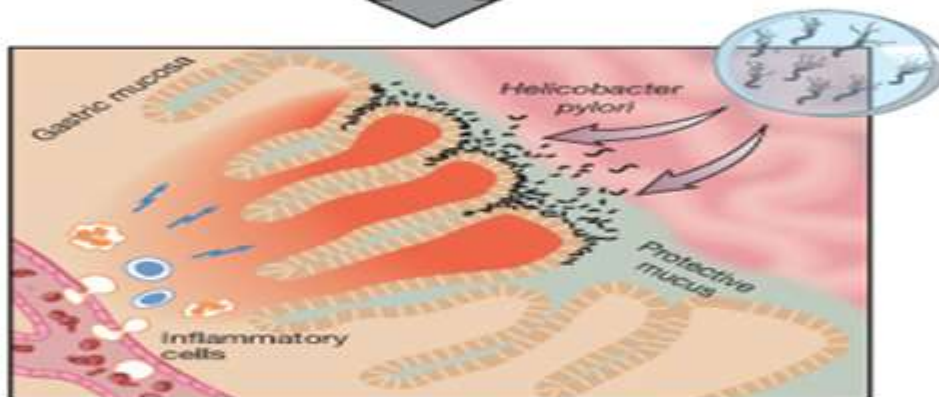
*Helicobacter pylori* infects the lower part of the stomach, antrum.



*Helicobacter pylori*

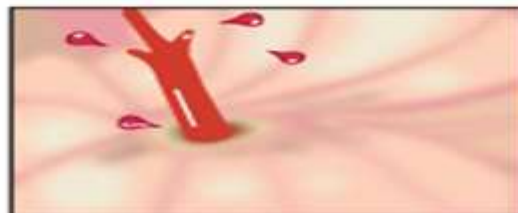
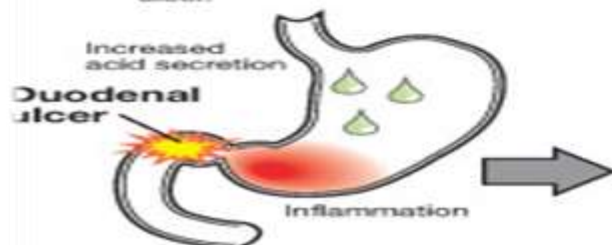
## Inflammation

*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.





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.

## Helicobacter pylori infection

