

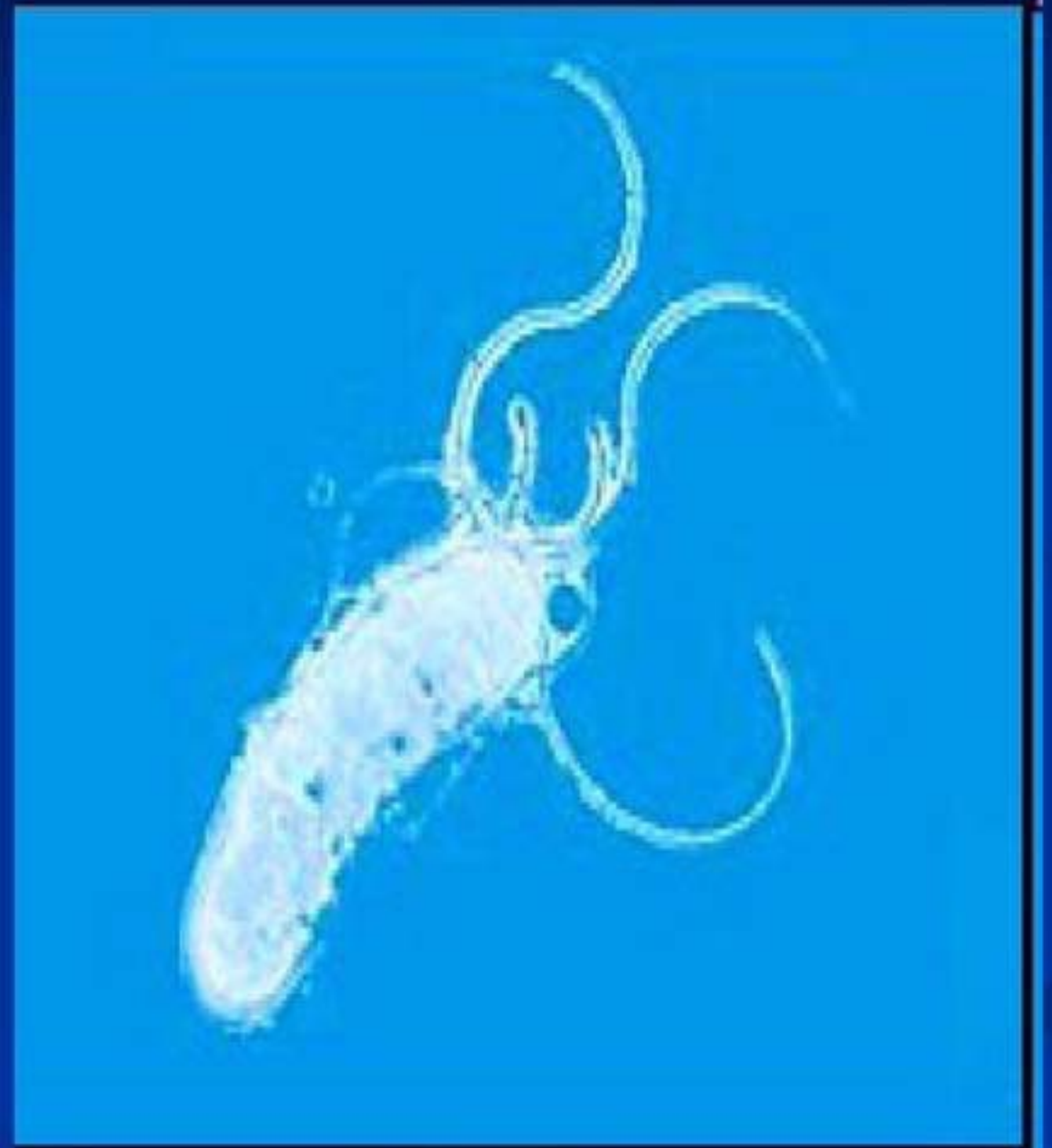
# Helicobacter pylori

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# Helicobacter pylori

- Helicobacter pylori is a spiral gram negative bacteria.
- It has a multiple polar flagella above the pole and motile

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# Culturing H.pylori

- H.pylori grows on Skirrow's medium with
  - 1 Vancomycin,
  - 2 Polymyxin
  - 3 TrimethoprimGrows in 3 -6 days at 37°C  
Colonies appear  
Translucent 1-2 mm in diameter  
Optimal growth occurs in  
Microaerophilic environment



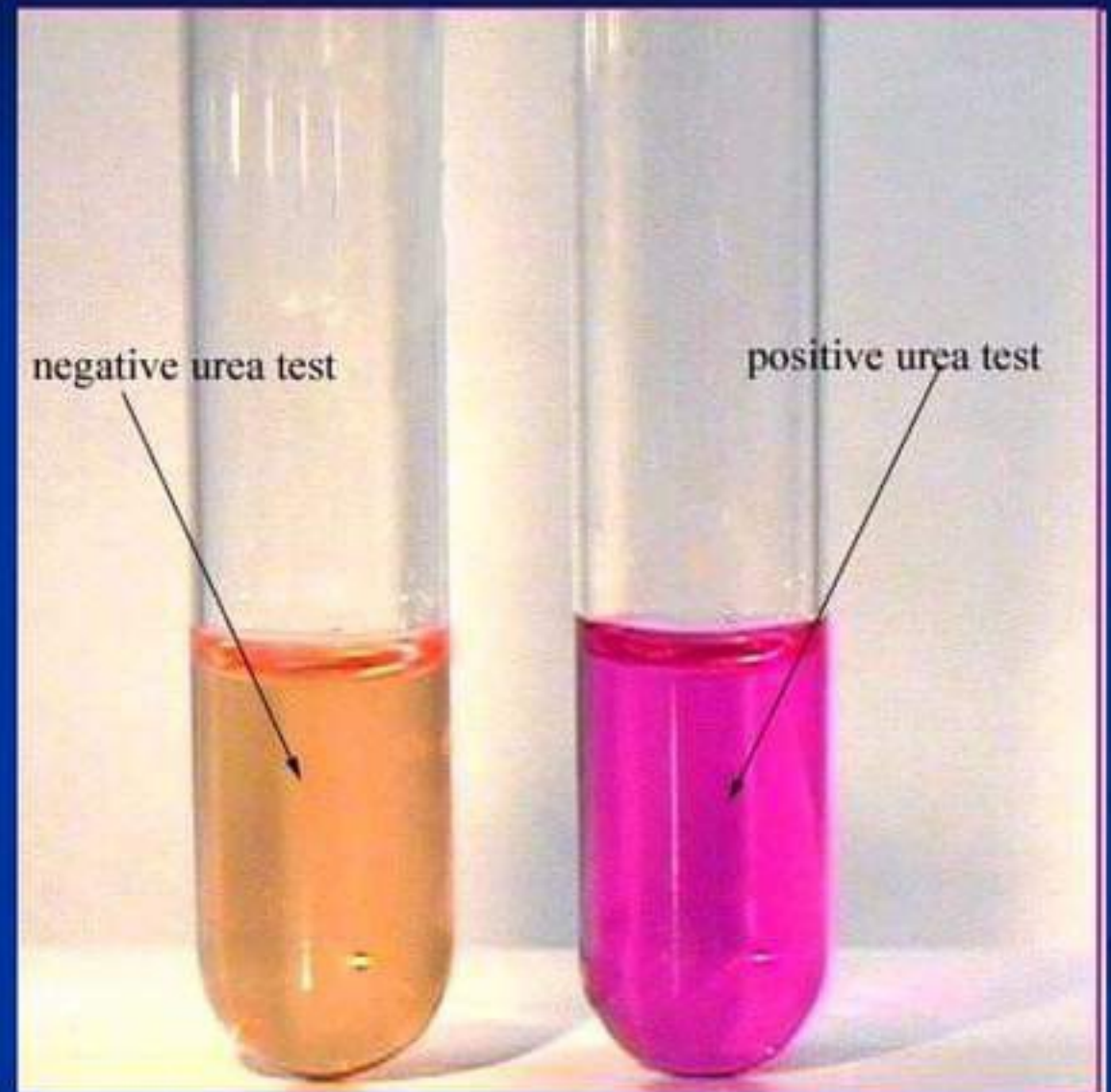


# *Biochemical Characters*

- Motile
- Catalase +
- Oxidase +

Strong producer of  
Urease

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# Pathology and Pathogenesis

- H.pylori is found in the deep mucus layer
- Grows optimally at pH 6.0 to 7.0
- But gastric mucosa has a strong buffering in spite of lower pH on the lumen side of stomach
- H.pylori also produces a protease that modifies the gastric mucus and further reduces the ability of acid through the mucus

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# Mechanisms in Pathogenicity

## 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

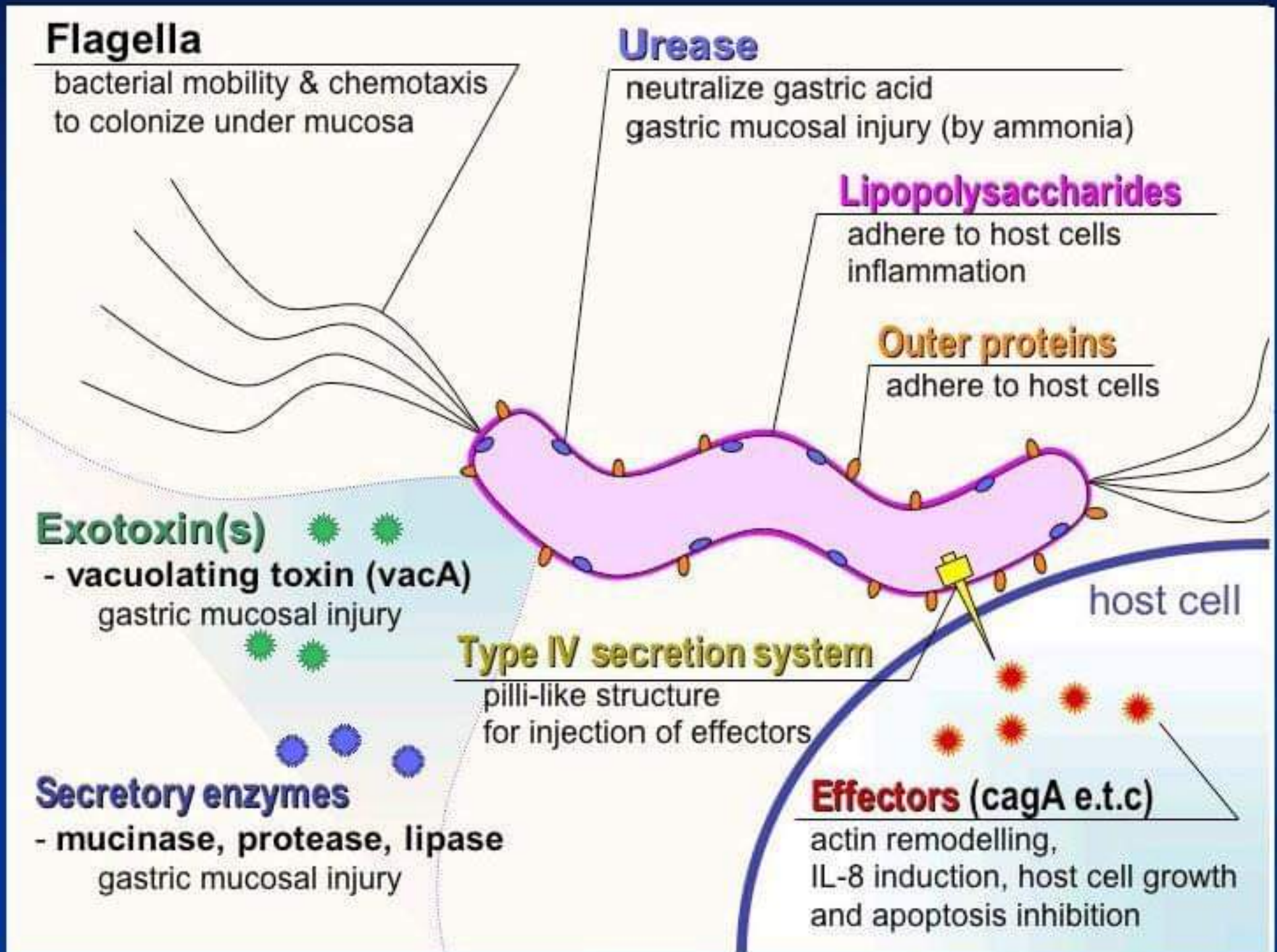
- **mucinase, protease, lipase**  
gastric mucosal injury

## 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



# Pathogenesis

- The potential character of H.pylori lie with production of potent Urease activity which yields production of Ammonia and further buffering acid.
- H.pylori is quite motile even in mucus finds its way to epithelial surface
- H.pylori overlies the gastric type but not intestinal epithelial cells.

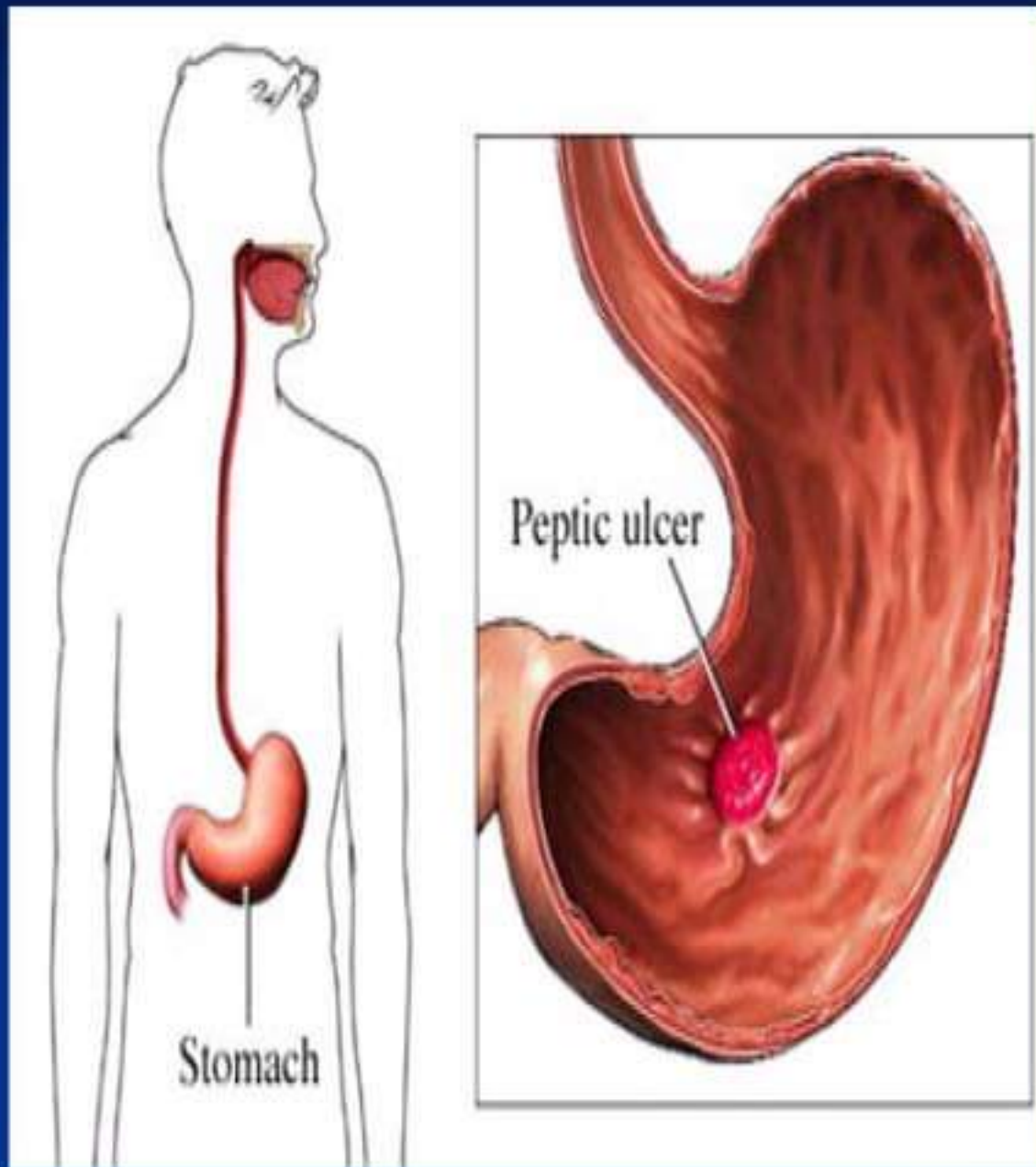


# Factors contributing to Peptic ulceration

- There is a strong association between presence of H.pylori infection and peptic ulceration
- Mucosal inflammation and damage involves both bacterial and host factors



# *H. Pylori causes Peptic ulcer in the Stomach*





# *Factors influencing Pathogenicity*

- Lipopolysaccharides - damage mucosal cells and Ammonia produced by Urease activity may directly damage cells.
- Gastritis – Chronic and active inflammation establishes Polymorphonuclear and Mononuclear cell infiltration within the Epithelial and Lamina propria
- Events lead to Destruction of epithelium is common.
- Glandular atrophy is common.



# *Clinical Manifestations*

- Acute infection

Upper Gastrointestinal illness

Nausea

Pain

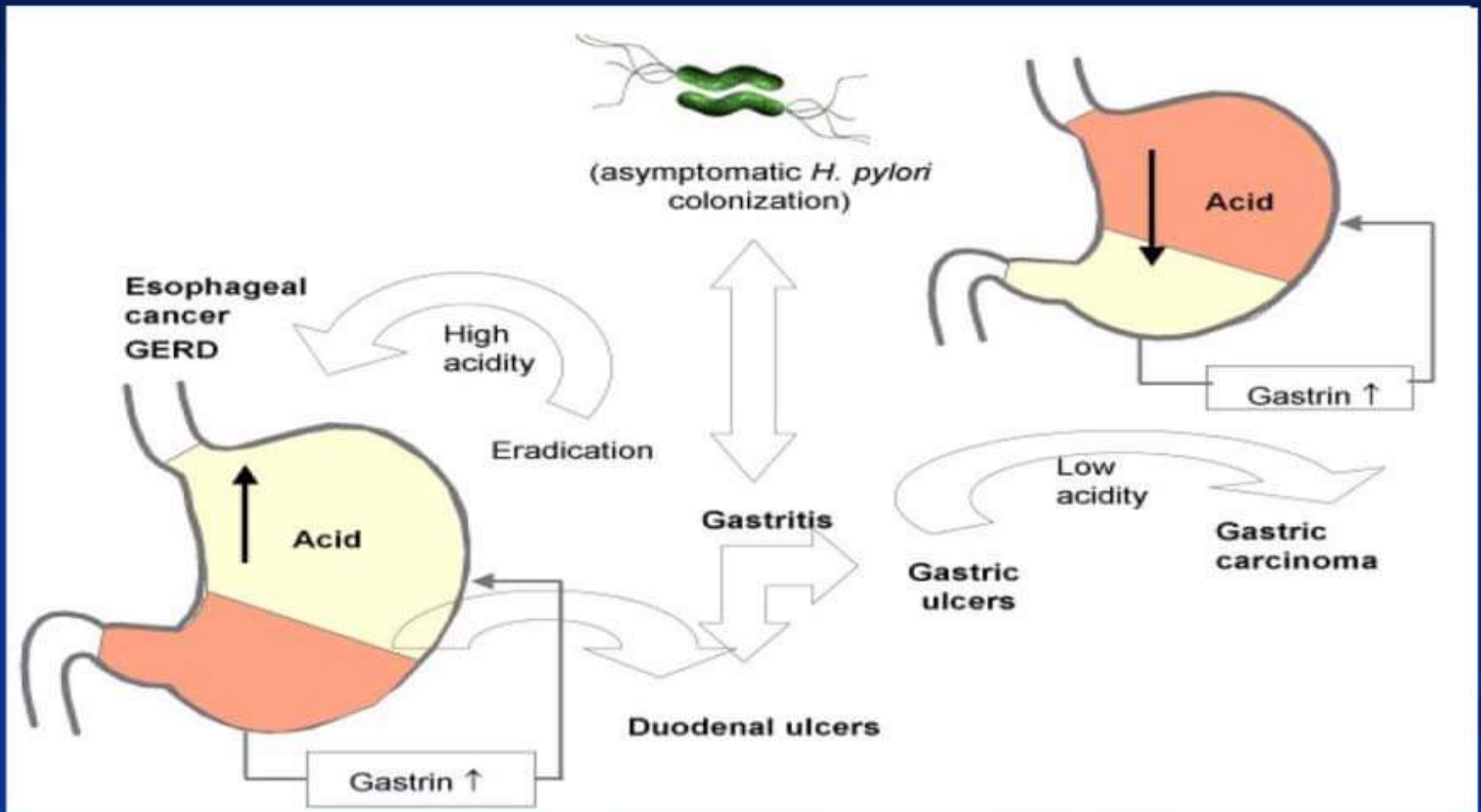
Fever – very occasionally

Acute symptoms lasts for < 1 week,

May extend upto 2 weeks

**Infection last for years, decades or even lifetime**

# Consequences of *H. pylori* Infection

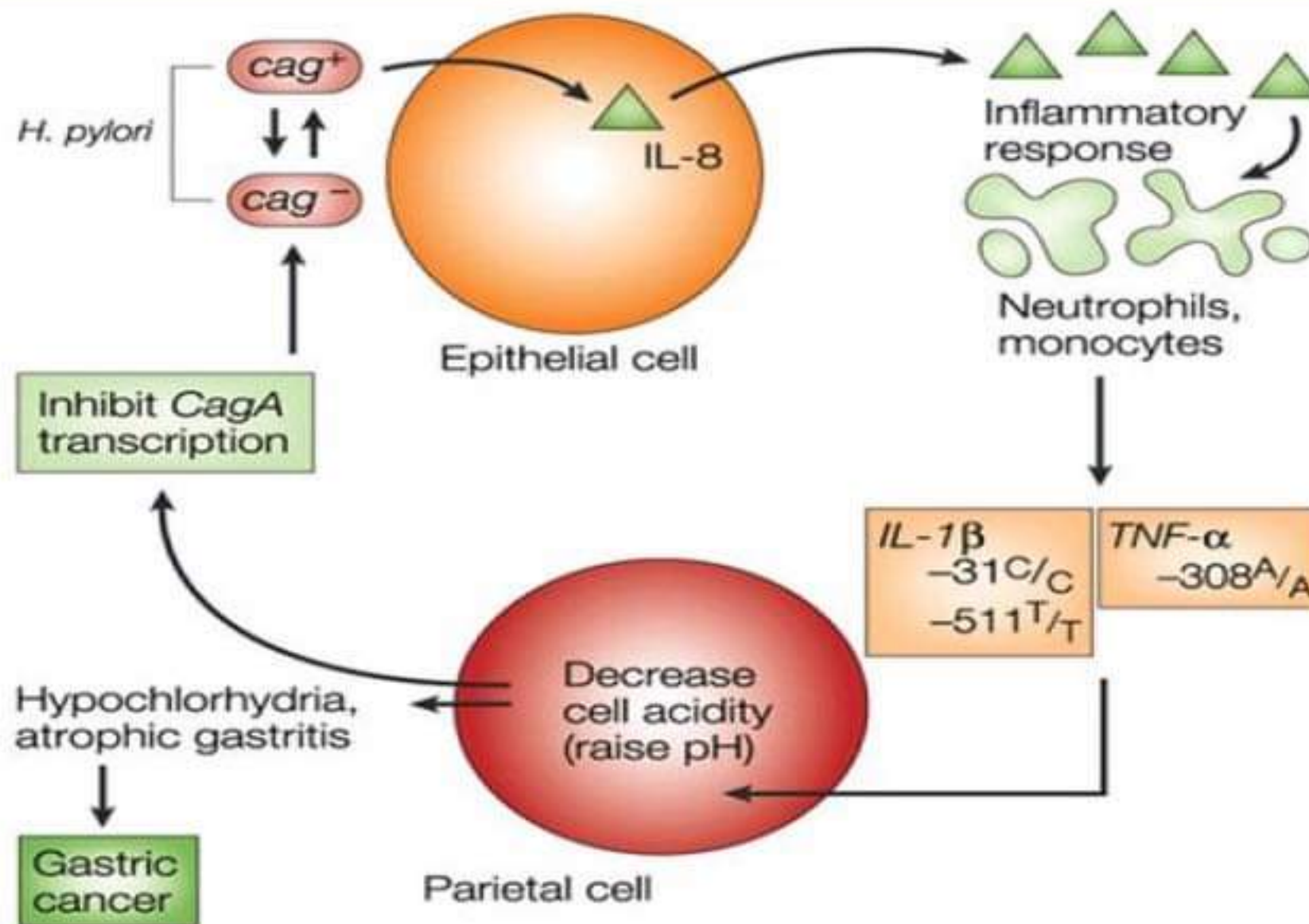




# Association of Duodenal and Gastric ulcers in H.pylori

- About 90 % of patients with Duodenal ulcer, and 50- 80 % of gastric ulcers are associated with H.pylori infection.
- H.pylori may have greater role in Gastric carcinoma and Lymphomas

# Mechanism of Cancer in *H. pylori*

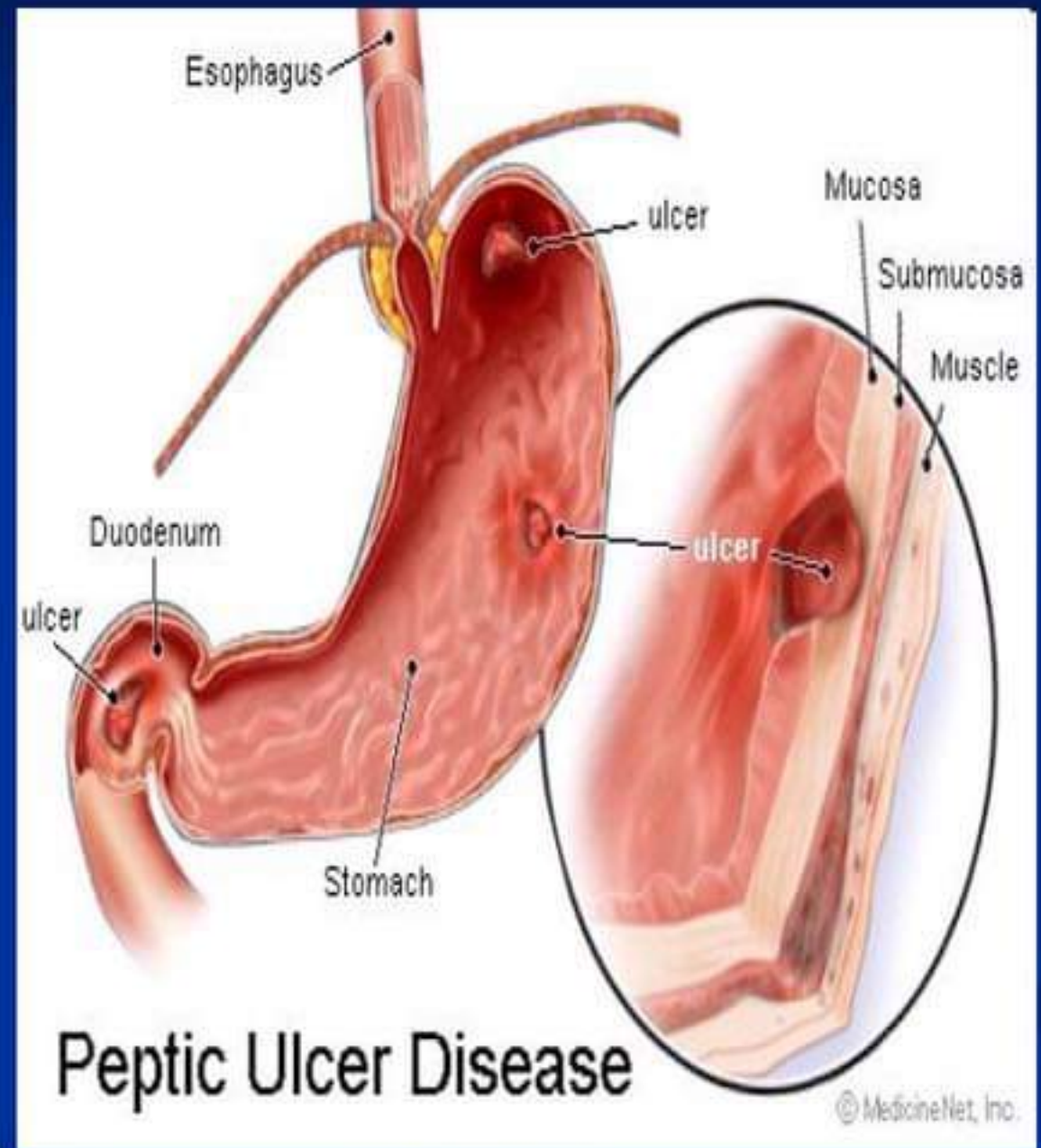
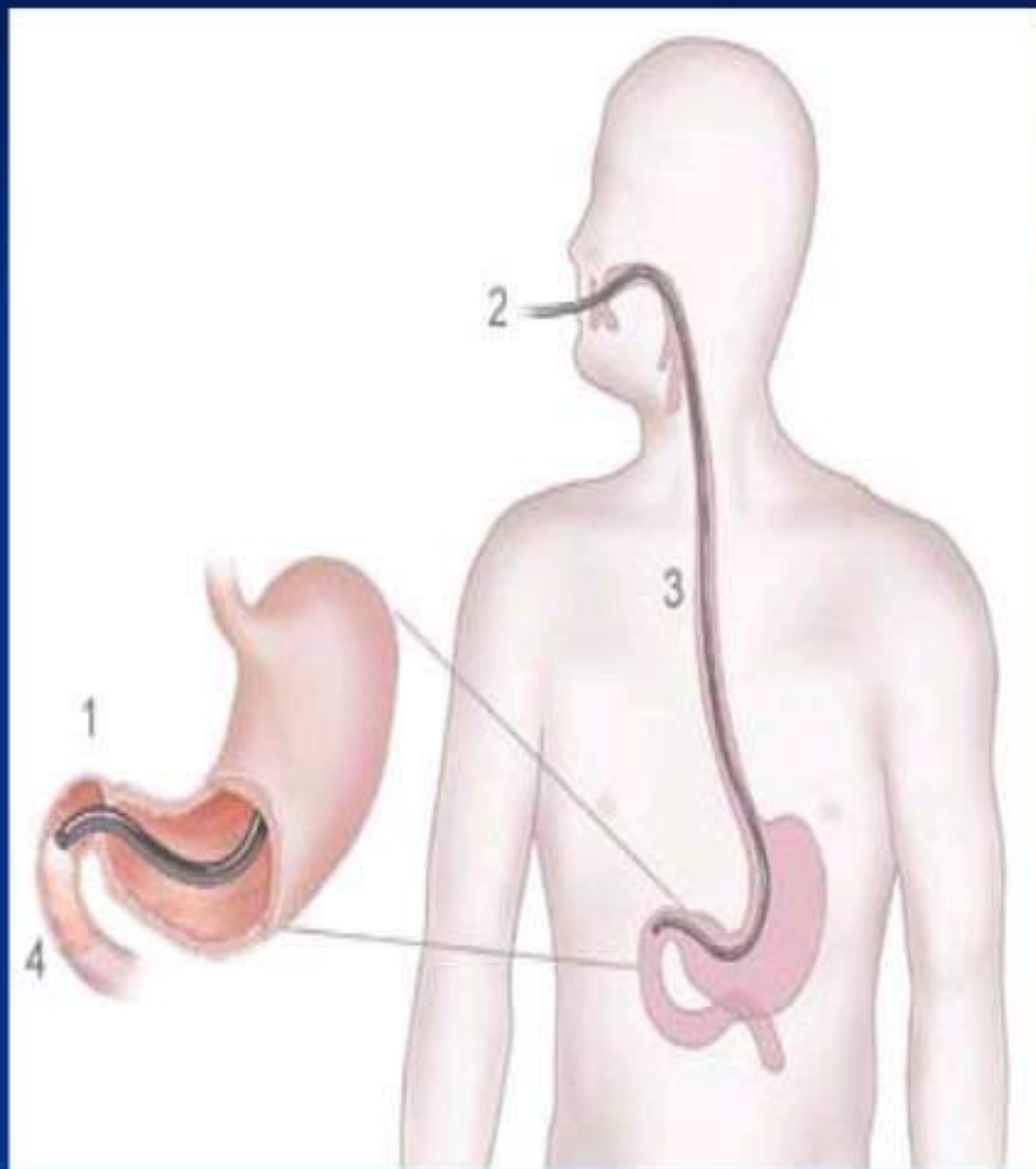




# Laboratory Diagnosis

- Specimens for histopathology – Gastric biopsy specimens can be used for Histological examination
- Specimens obtained after Gastroscope, Biopsy, routine stains will demonstrate Gastritis and special stains show curved spiral organisms
- Specimens collected in sterile saline mixed are used for culturing

# Endoscopy – Gastric Biopsy





# Culturing for H.pylori

- Culturing of H.pylori needs specific conditions
- Media
  - Skirrow's Medium
    - with
      - Vancomycin
      - Polymyxin B
      - Trimethoprim
  - Chocolate Medium with
    - Vancomycin, Nalidixic acid
    - Amphotericin





# Serology

- The detection of Antibodies in active infection is useful
- But the tests are limited utility as antibodies persist even after *H. pylori* infection is eradicated.
- Several commercial kits are available, but lacks the role in identifying acute infections.

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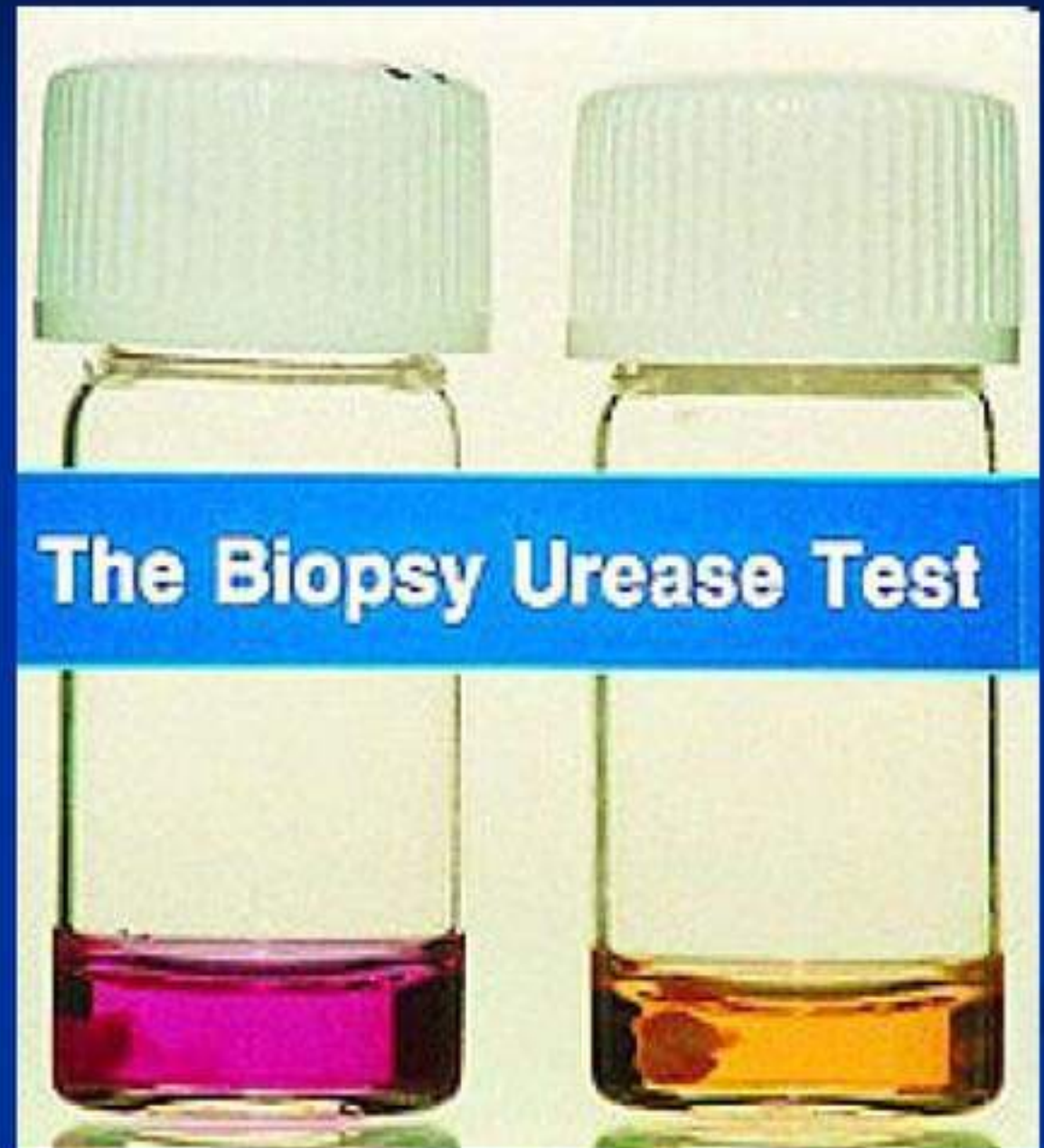




# Special Tests for H.pylori

- Rapid tests for detection of Urease activity are widely used in presumptive identification of Gastric Biopsy specimens.
- Gastric Biopsy can be placed into urea containing medium with color indicator.
- **If H.pylori is present the Urease rapidly splits urea and resulting shift in pH yields a color change in the medium**

Medical Information  
AND MCQs By Dr NMN





# *Urea Breath Test*

- *H. pylori* infection can be detected in the exhaled breath using this special test. This test is positive only if the person has a current infection. Sensitivity and specificity of this test ranges from 94-98%.





# Urea Breath Test

- In this test  $^{13}\text{C}$  or  $^{14}\text{C}$  labelled urea is ingested by patients
- **If *H.pylori* is present the urease activity generates labelled  $\text{CO}_2$  that can be detected in the patients exhaled breath**

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# Antigen Detection Test in Stool

- Detection of H.pylori antigen in stool is appropriate test in patients with H.pylori infection
- **Absence of antigen indicates cure of Infection after Chemotherapy.**



# Treatment

- Triple therapy has prompt response, contain a combination of following drugs
    - 1 Metronidazole
    - 2 Bismuth subsalicylate or Bismuth sub citrate
    - 3 Amoxicillin or Tetracycles
- administered upto 14 days
- Eradicates H.pylori
- In 70 – 95 % of patients
- Acid suppressing agent is supporting

# Other Drug Combination

## ■ Other alternatives

Proton pump inhibitor directly inhibit

H.pylori

Combined with

Amoxicillin

Clarithromycin or Amoxicillin

And Metronidazole



*Barry J. Marshall and J. Robin Warren have been awarded the 2005 Nobel Prize in medicine*

