

CAMPYLOBACTER & HELIOCBACTER

Professor Doctor

Osama Nadhom Nijris

University of samarra, college of applied
science

CAMPYLOBACTER



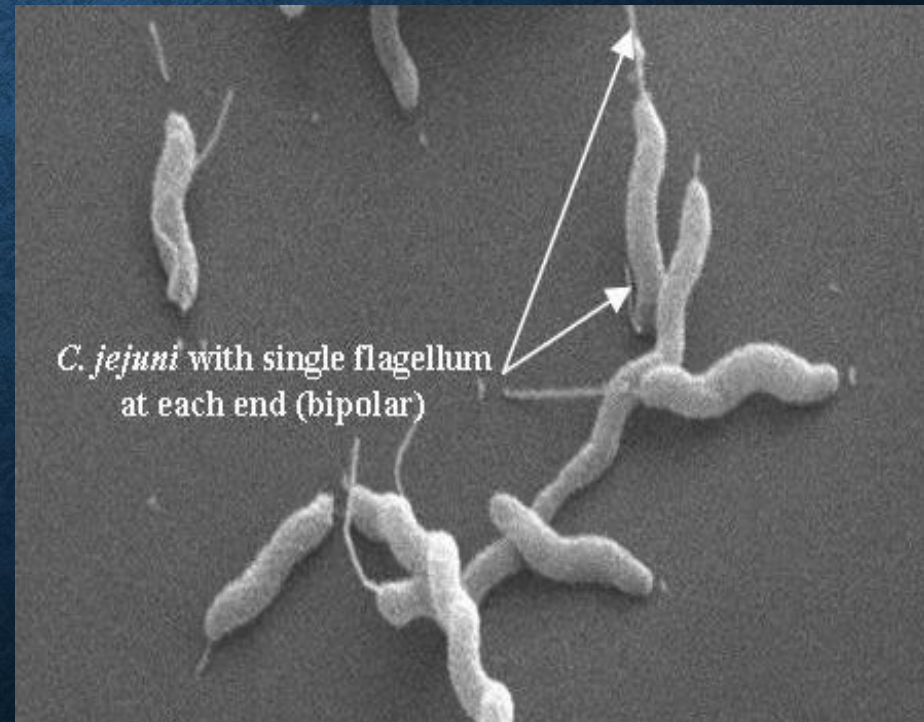
CAMPYLOBACTER SPECIES

- ❑ Campylobacter jejuni causes 95% of campylobacter enterocolitis especially in children.
- ❑ Campylobacter coli
- ❑ C. fetus, C. lari are rare causes of systemic infections such as bacteremia and meningitis.

Campylobacter jejuni

MORPHOLOGY

- ❑ Small Gram negative rods with comma or S or gull wing shapes.
- ❑ Motile with a single flagellum at one or both poles.
- ❑ Motility is darting with cork screw like movement.

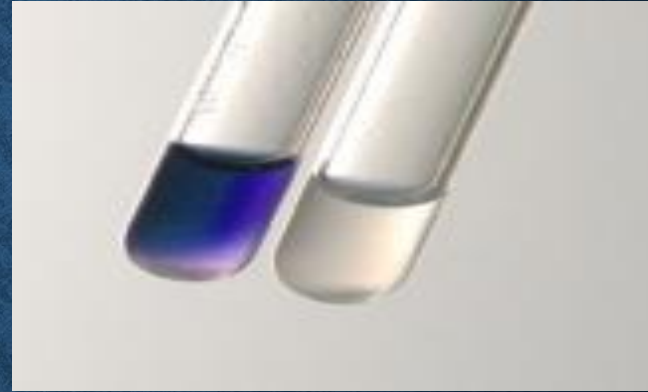
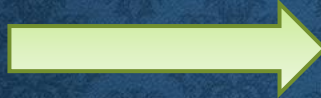


CULTURAL CHARACTERS

- ❑ Grows on Skirrow's medium.
- ❑ This medium is a selective medium used for campylobacter isolation from the stool as it contains vancomycin, polymyxin and trimethoprim contains lysed horse blood .
- ❑ Microaerophilic & capnophilic (grows best in presence of 5% oxygen and 10% CO₂).
- ❑ Grows best at 42 degree.
- ❑ Growth may take 2-5 days.
- ❑ Non-fermentative
- ❑ Can't grow under strictly aerobic conditions

BIOCHEMICAL REACTIONS

- ❑ Oxidase positive
- ❑ Catalase positive
- ❑ Hippurate positive
- ❑ Urease negative
- ❑ Non proteolytic
- ❑ Unable to attack carbohydrates
- ❑ Sensitive to nalidixic acid and erythromycin.



Filtration of emulsified stool may be done using 0.45 μm pore size filters that allow the small campylobacter to pass and exclude other organisms present in the stool.

This method is required for isolation of campylobacter other than *C. jejuni* that are sensitive to the antibiotics in Skirrow's medium.

Campylobacter

**Gull
wing**

**Capnophi
lic**

**Thermophil
ic**

**Microaeroph
ilic**

VIRULENCE FACTORS

- Enterotoxin
- Endotoxin
- Adhesions
- Intracellular survival
- Ability to penetrate cells

Clinical significance

- Gastroenteritis

Caused mainly by *C. jejuni* and *C. coli*

PATHOGENESIS

- ❑ Infection is acquired by ingestion of food or water contaminated with faces of domestic animals.
- ❑ The organism invades the epithelium of the lower small intestine and multiplies.
- ❑ Human to human transmission is less frequent.
- ❑ The disease is caused by tissue invasion + enterotoxin production.

CLINICAL PICTURE

- ❑ Enterocolitis begins as watery foul smelling diarrhea followed by bloody stools + fever + severe abdominal pain.
- ❑ Complications of *Campylobacter jejuni* infections two weeks later include:
 - 1) **Guillain-Barre syndrome which is autoimmune disease that attacks neurons.**
 - 2) **Reactive arthritis which is also autoimmune.**

LABORATORY DIAGNOSIS

- ❑ Specimen: stool
- ❑ Wet smears will show the characteristic motility.
- ❑ Gram stained smears show the characteristic morphology.
- ❑ Stool is cultured on Skirrow's medium and incubated at 42 degree at microaerophilic and capnophilic conditions.
- ❑ Identification of the growing colonies.
- ❑ Direct detection of the organism by ELISA or by PCR.

TREATMENT

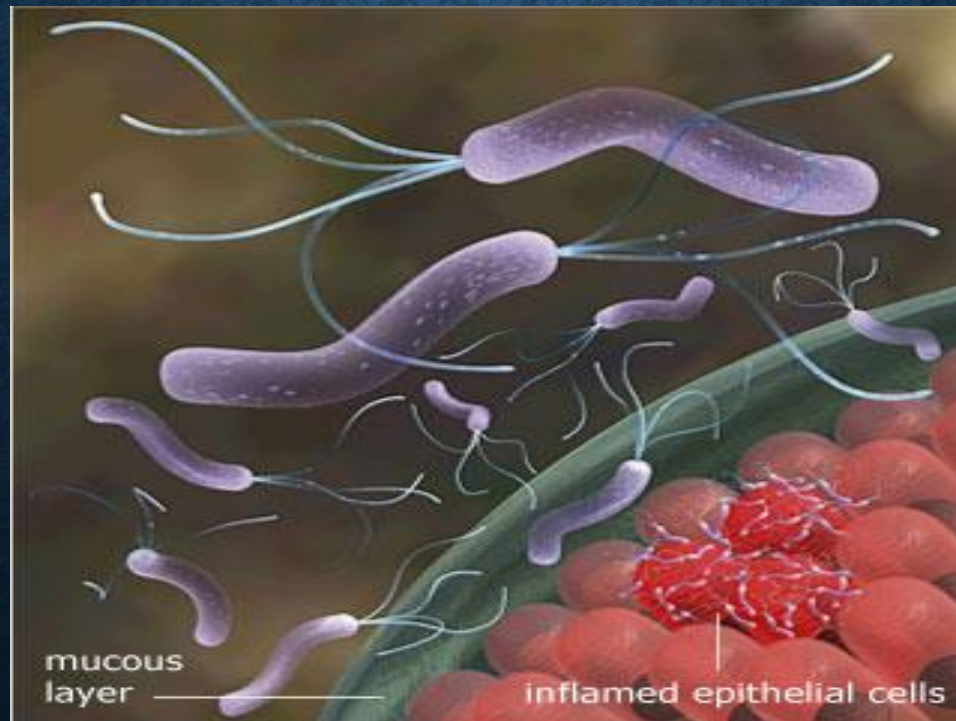
- ❑ Mainly fluid and electrolyte replacement.
- ❑ C. jejuni is sensitive to erythromycin and ciprofloxacin.

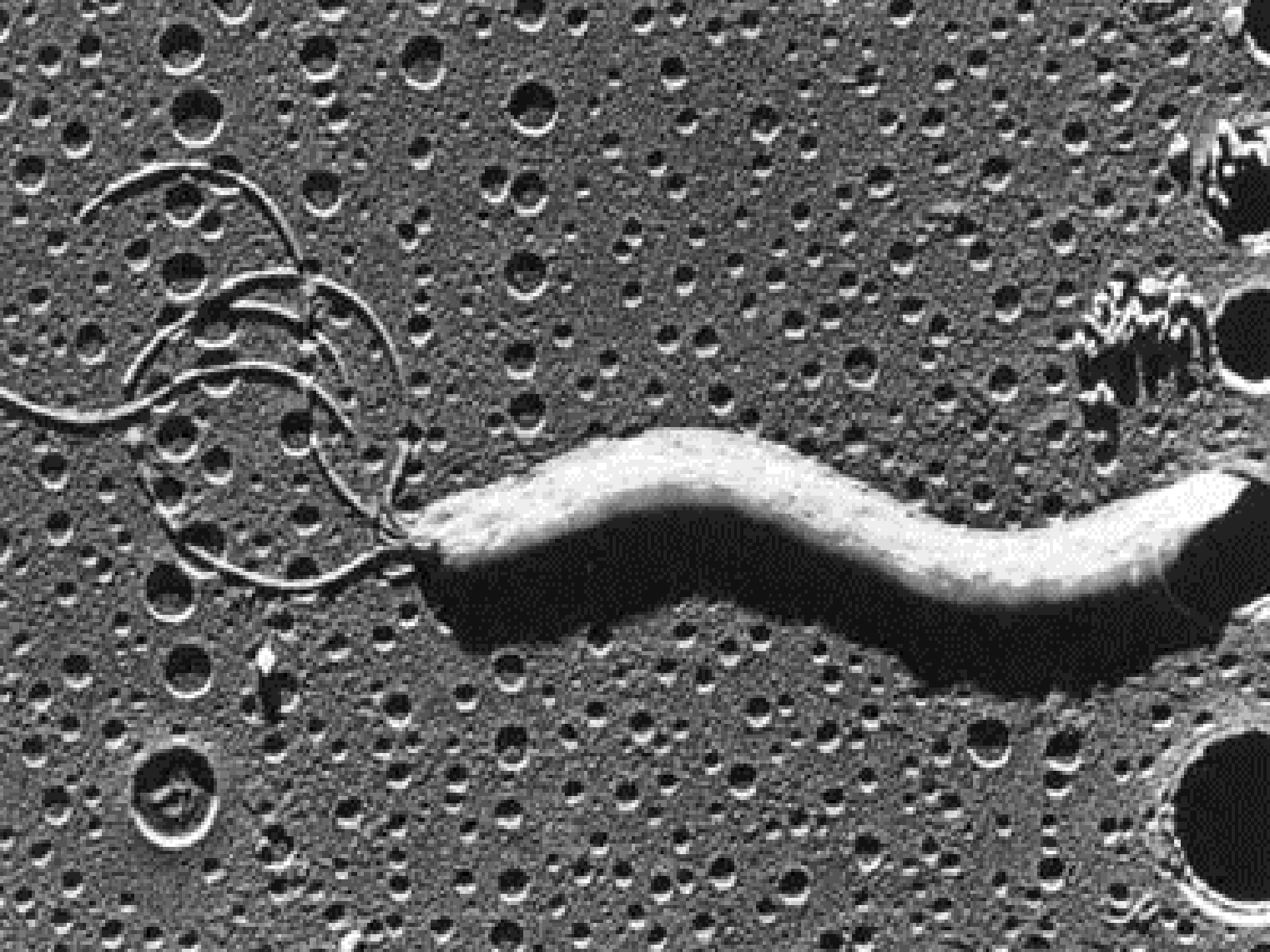
HELICOBACTER PYLORI



MORPHOLOGY

- ❑ It is similar to campylobacter in morphology but differs in:
 - Having multiple sheathed polar flagella.







CULTURAL CHARACTERS

❑ Similar to campylobacter but
grows at 37 degree.

BIOCHEMICAL REACTIONS

□ Similar to campylobacter but
helicobacter is urease positive.

Helicobacter

**Helical
(spiral)**

**Capnophi
lic**

**Urease
positive**

**Microaeroph
ilic**

VIRULENCE FACTORS

- **Adhesions**
- **Vacuolating Cytotoxin (VacA)** - forms a pore in host cell membranes and induces apoptosis
- **Neutrophil-Activating protein (NAP)** - activates neutrophils and mast cells that damage local tissues
- **Endotoxin**
- **Urease** – facilitates survival in the stomach by raising the pH, provides access to nitrogenous nutrients needed by the bacteria for growth,
- **Flagella** – allow bacteria to penetrate through gastric mucous
- **Collagenase/Mucinase** –degrades gastric collagen and mucous, exposing gastric epithelium to gastric acid
- **CagA** – is injected into host epithelial cells where it activates host signal transduction pathways that can stimulate growth→ cancer?

PATHOGENESIS

- ❑ Transmission occurs by feco-oral route.
- ❑ By its motility, *H. pylori* penetrates the mucous layer.
- ❑ Then, it attaches deeply in the gastric mucosa by adhesins away from the gastric acidity.
- ❑ *H. pylori* produces urease enzyme which splits urea into ammonia.
- ❑ The production of ammonia + inflammation damage the gastric mucosa.
- ❑ This results in **acute gastritis** and **gastric & duodenal ulcers**.
- ❑ The ammonia produced neutralizes gastric acidity allowing the organism to survive leading to **chronic gastritis**, gastric atrophy and **gastric carcinoma**.
- ❑ Some strains produce other virulence factors that help in damaging the mucosa such as the vacuolating cytotoxin (VacA) and a cytotoxin associated protein (CagA).

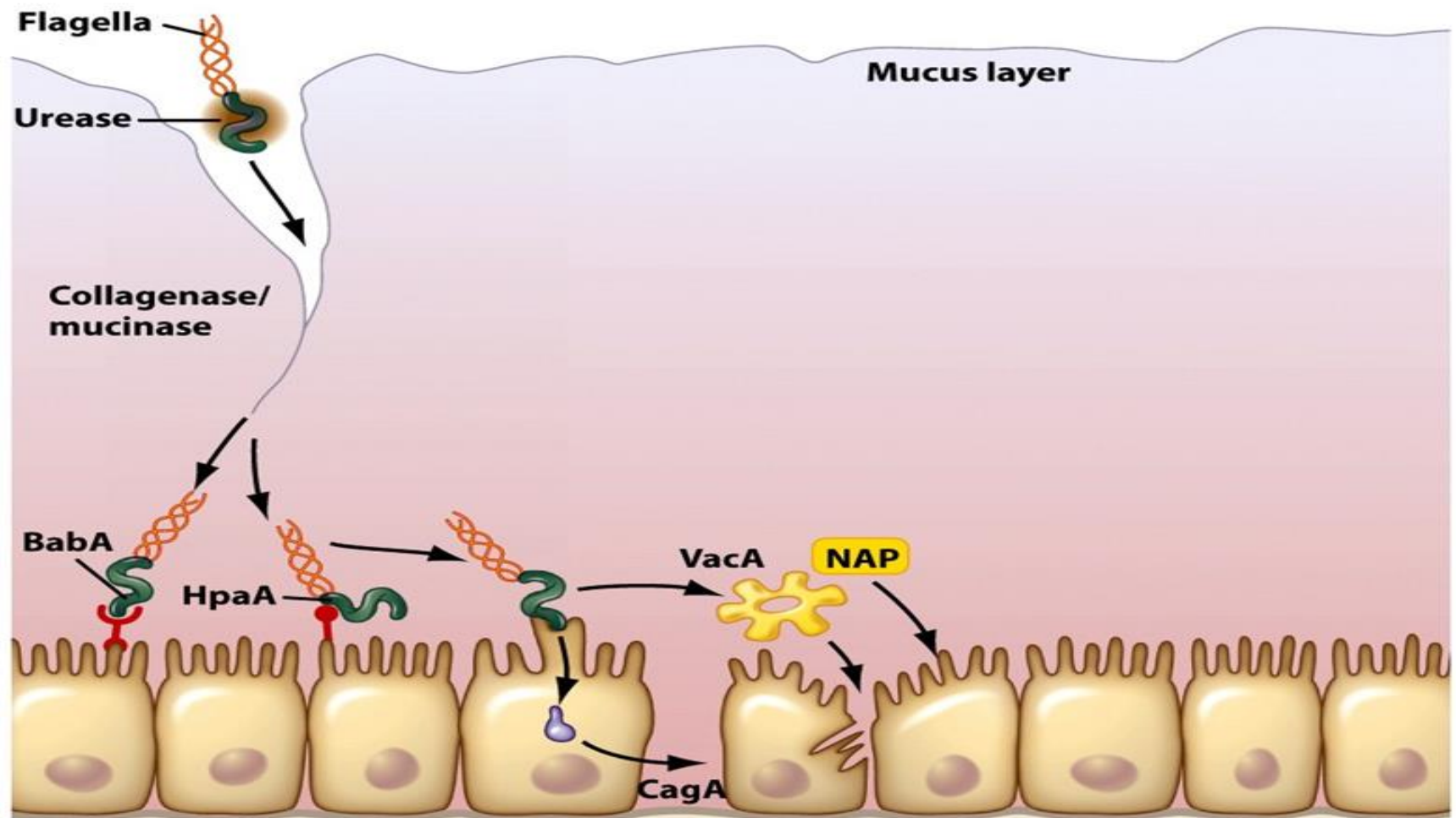


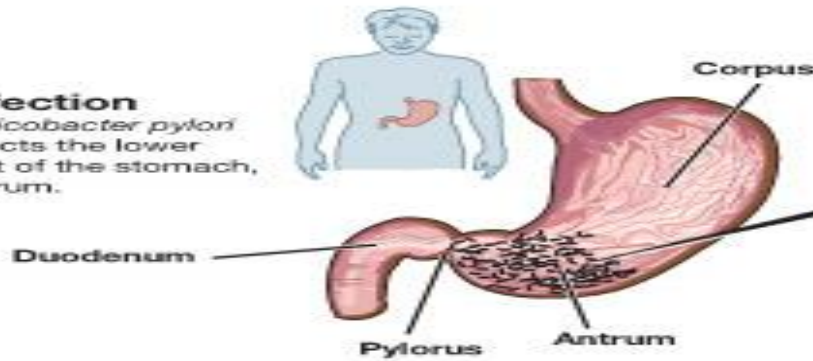
Figure 25.33 Microbiology: An Evolving Science
© 2009 W. W. Norton & Company, Inc.

Helicobacter pylori

— the bacterium causing peptic ulcer disease

Infection

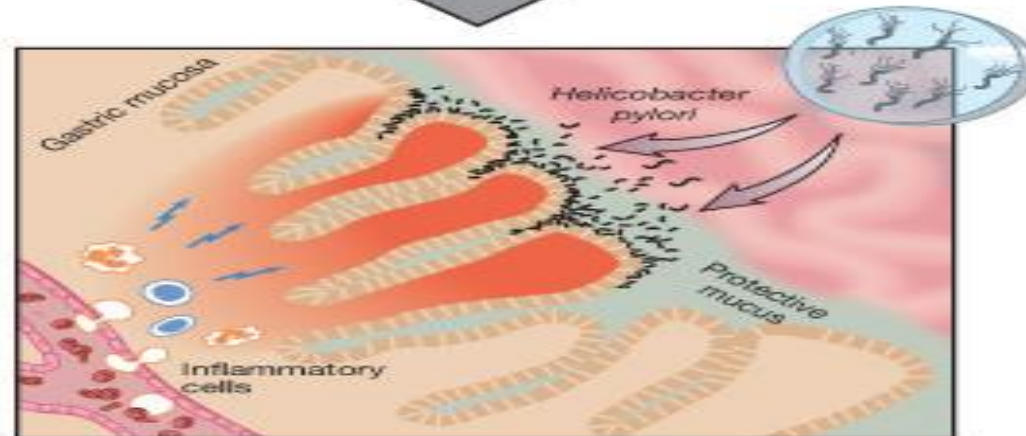
Helicobacter pylori infects the lower part of the stomach, antrum.



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.



Bleeding ulcer

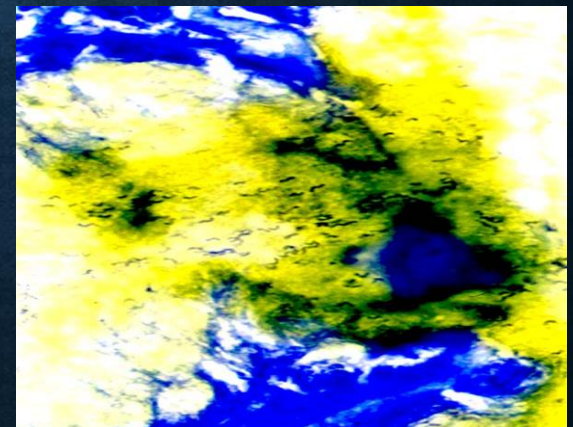
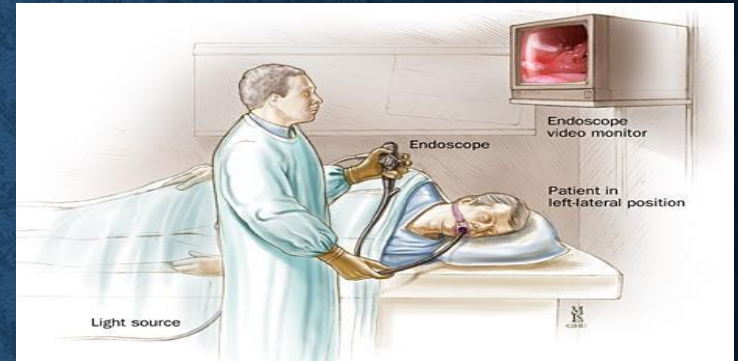


LABORATORY DIAGNOSIS

❑ Invasive methods:

Gastric biopsy specimens

- 1) Smears stained with Gram and special stains will show the spiral or curved organism.
- 2) Culture as in campylobacter but incubated at 37 degree for 7 days in a humid atmosphere.

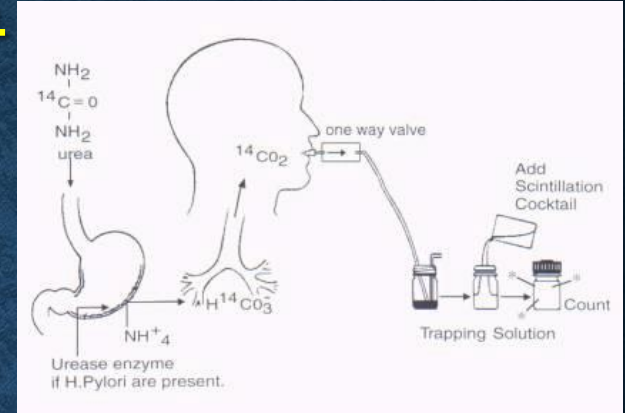


3) Rapid urease test in which gastric biopsy is placed on a medium containing urea with pH indicator. If *H. pylori* is present, the urease enzyme splits urea and results in shift of pH leading to color change.



□ Non-invasive methods:

Urea breath test



- A capsule of ^{14}C labeled urea is ingested by the patient. If the organism is present, the urease activity generates radiolabeled CO_2 that could be detected in the patient's breath.

Sandwich ELISA

- For detection of *H. pylori* antigen in the stool.

PCR

- For detection of the bacterial genes in the gastric juice, gastric biopsy or faeces.

Serological diagnosis

- For detection of *H. pylori* antibodies



TREATMENT

□ Triple therapy, one week course of clarithromycin + amoxicillin or metronidazole + omeprazole results in eradication of *H. pylori* in 90% of patients.

