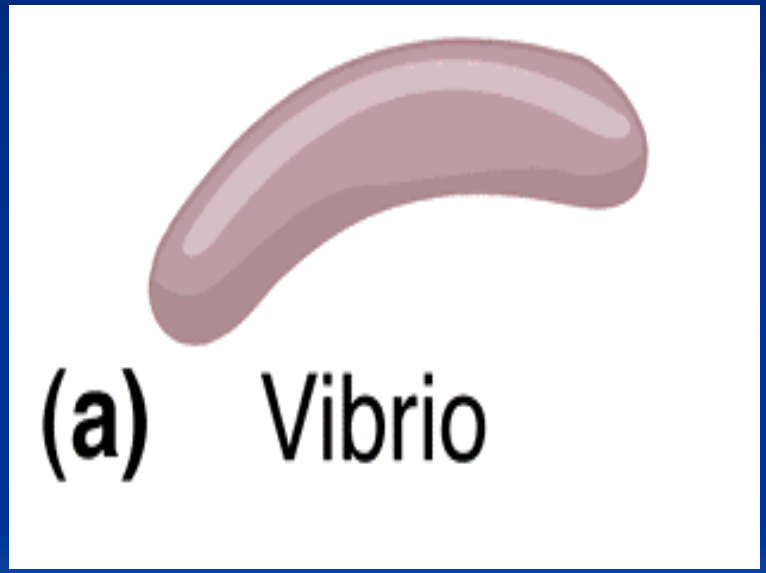
Curviform gram negative bacteria of medical importance

Three genera included

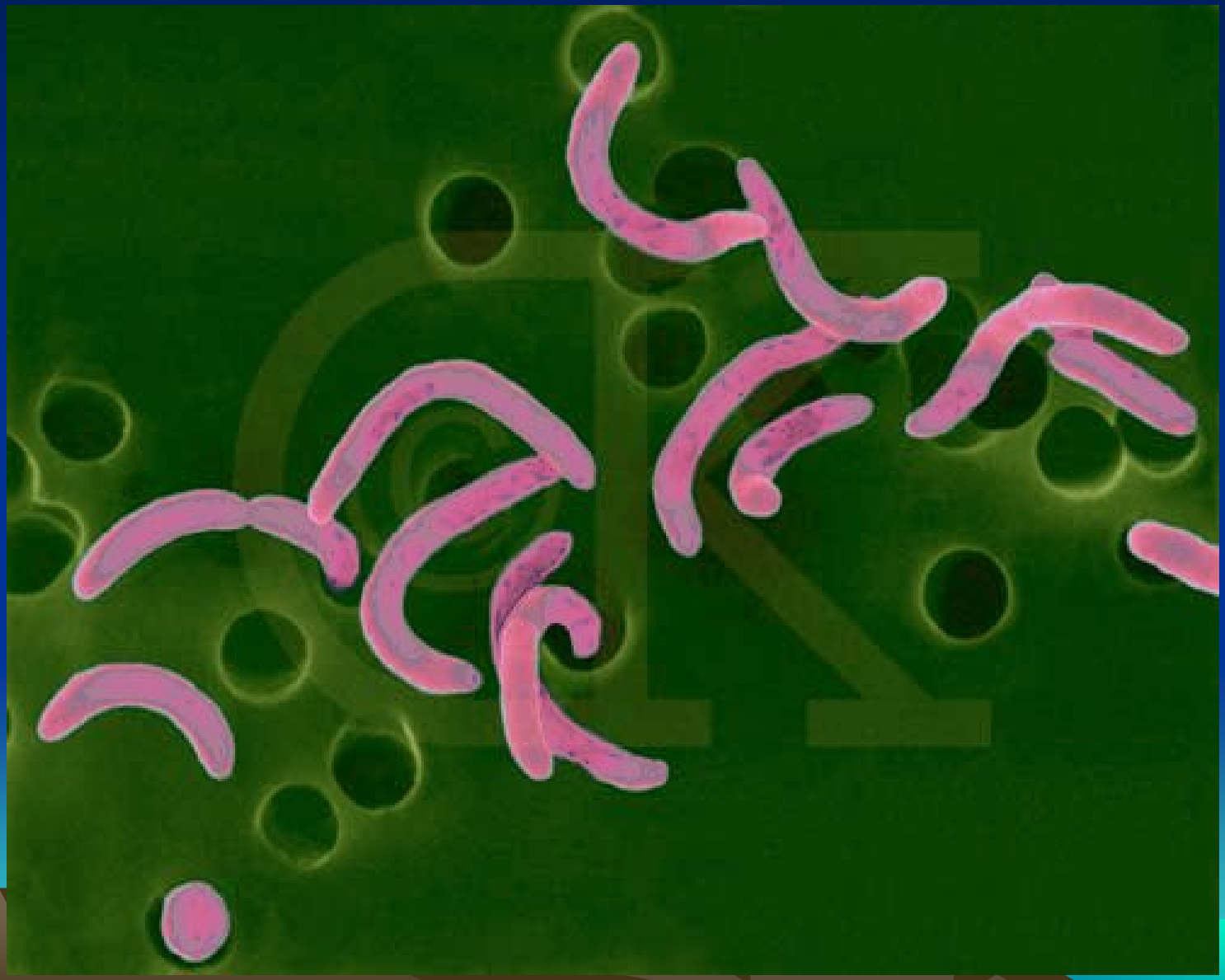
- Vibrio (F. vibrionaceae)
- Campylobacter (F.Spirillaceae)
- Spirillum (F.Spirillaceae)

Vibrios

- Gram negative rods
- comma shaped
- facultative anaerobes
- oxidase positive
- simple nutritional requirements
- readily cultivated

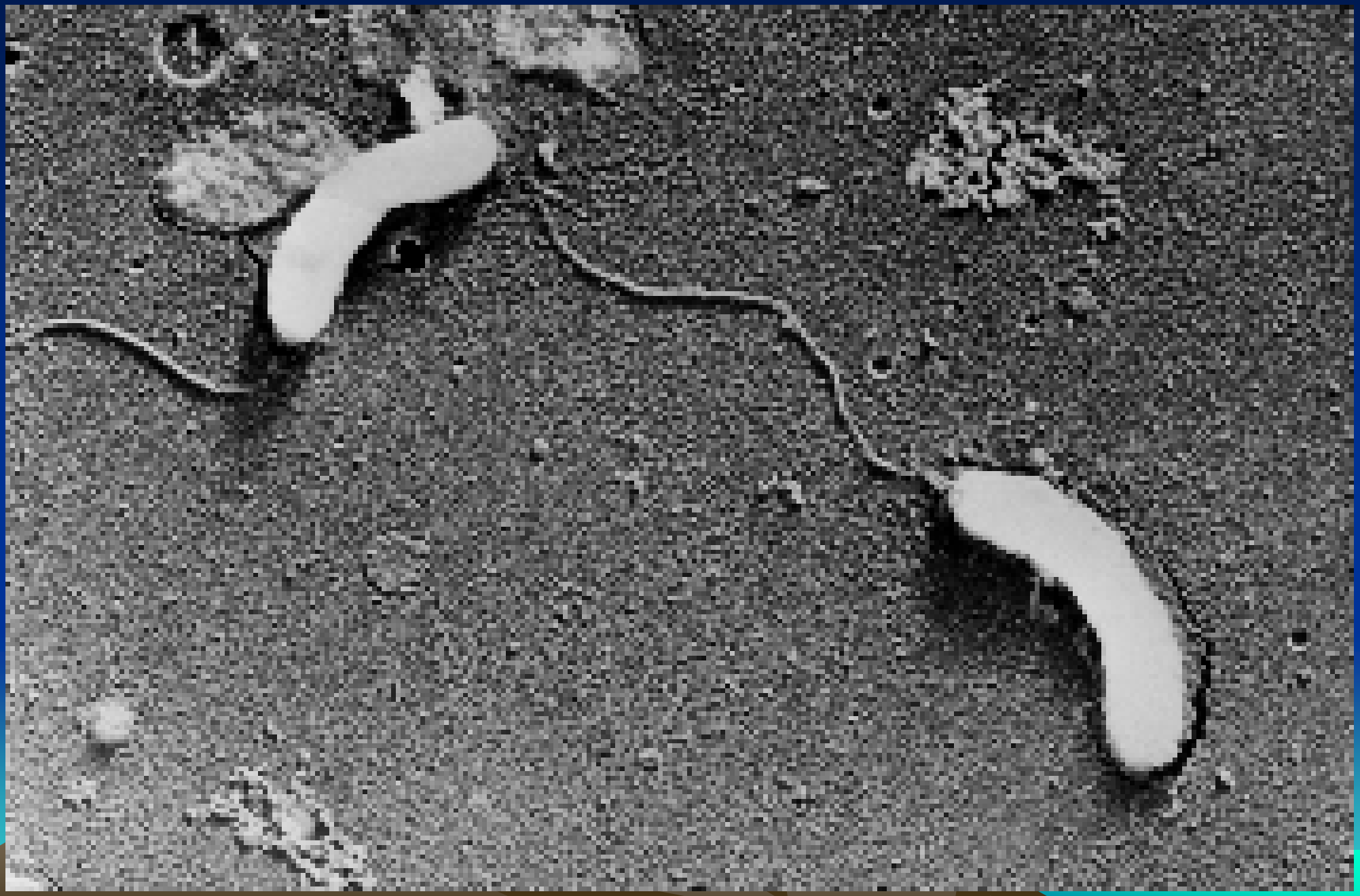


Vibrio cholerae



Vibrio cholera

- monotrichous., fast motile
- Cultivation on: blood agar, TCBS and MaCconky agar
- Oxydase positive
- Facultative anaerobic
- O and H antigen
- Membrane receptor antigens



Pathogenic strains:

- Sero group O1

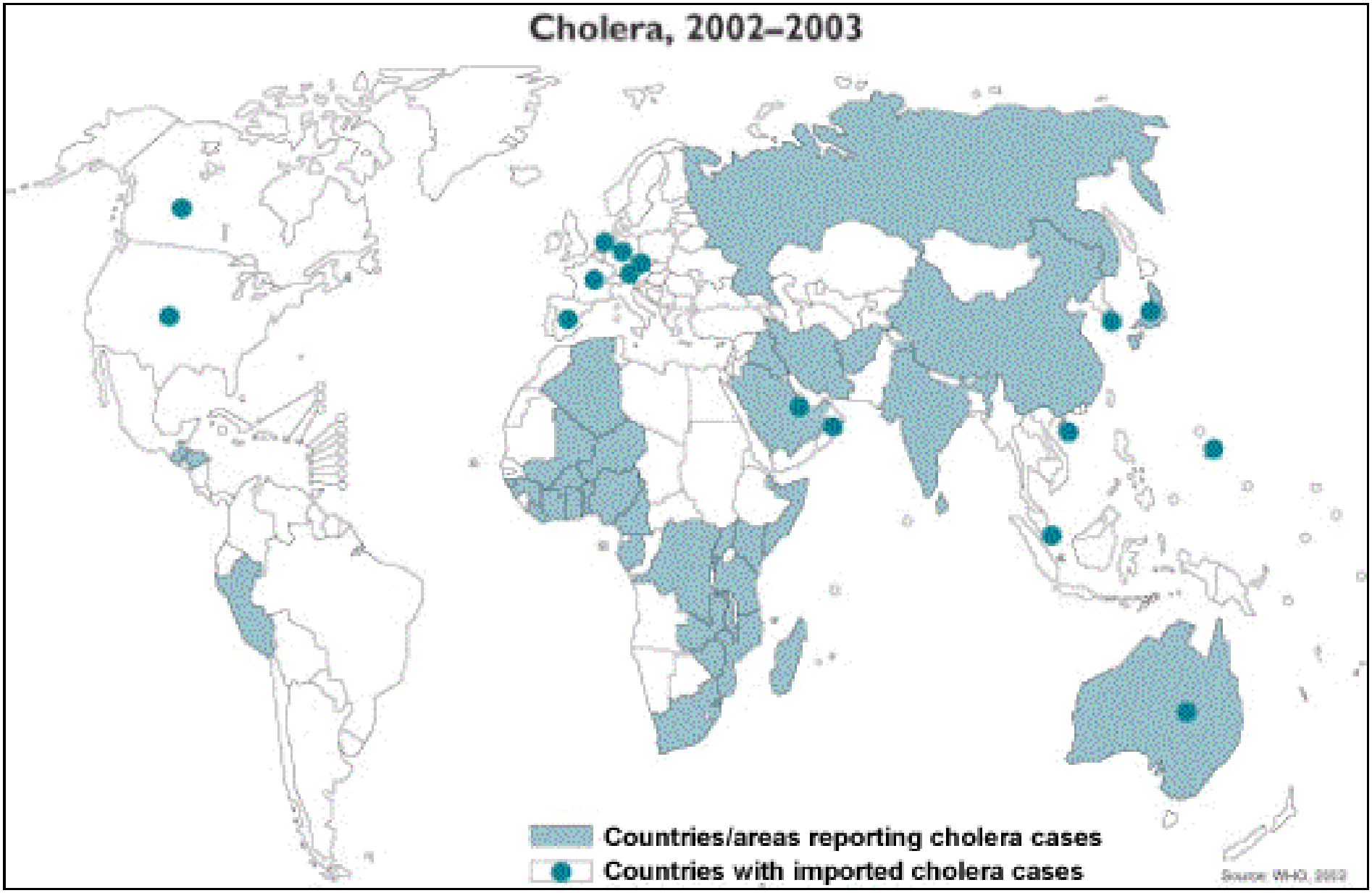
-biotype ElTor

-biotype classic

- Serogroup O139(Bangal strain)
- Non O1 vibrio cholera (sporadic)
- O2 &O138 Non Pathogen
- Vibrio parahaemolyticus (gastroenteritis)

Occurrence -cholera

- third world
- Sporadic cases in other countries
  - uncommon
    - * traveler
    - * ingestion of sea-food


Cholera, 2002–2003
Countries/areas reporting cholera cases
Countries with imported cholera cases
Source: WHO, 2003

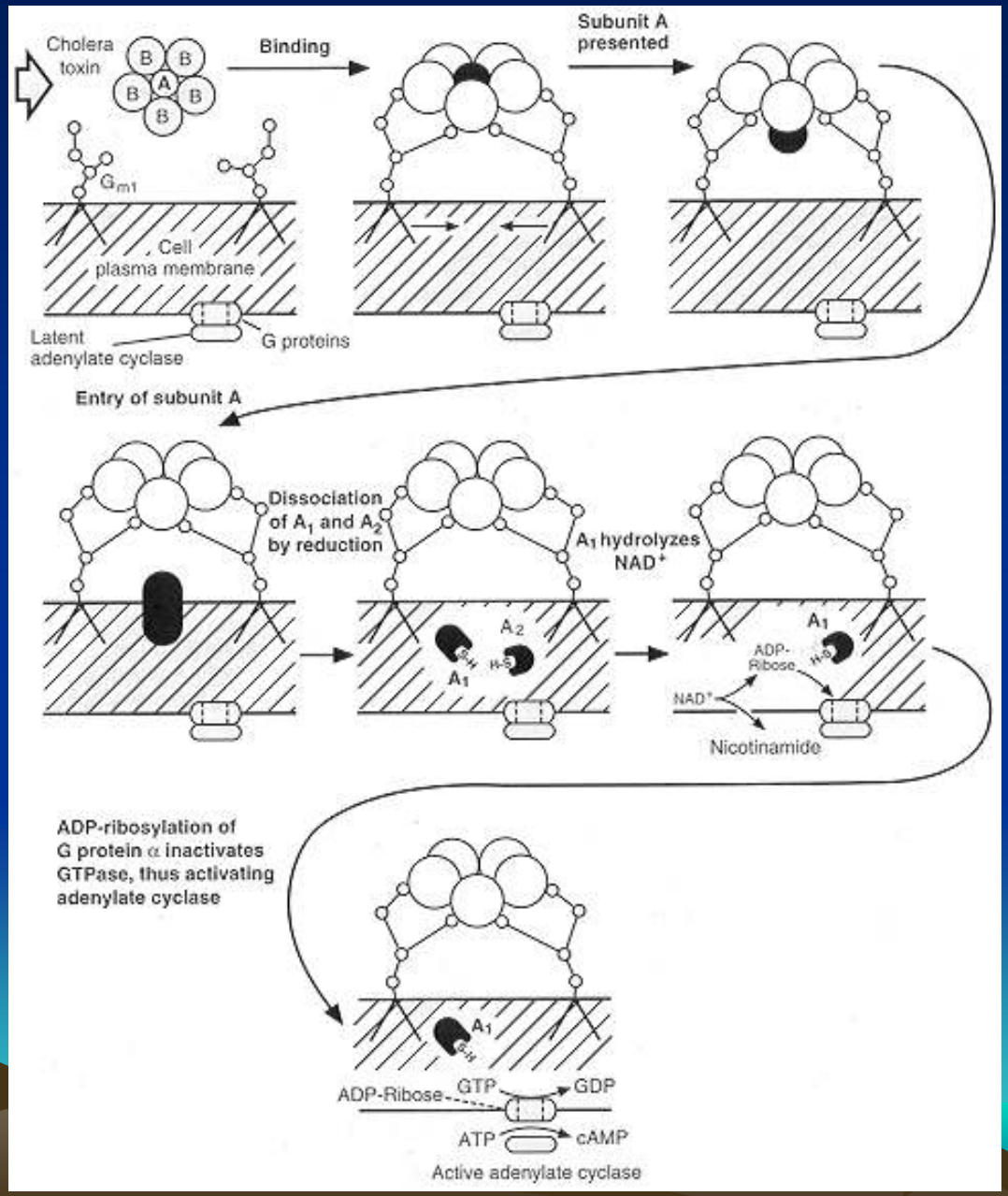
Transmission

- Contaminated water or food
- No animal or insect vectors reported

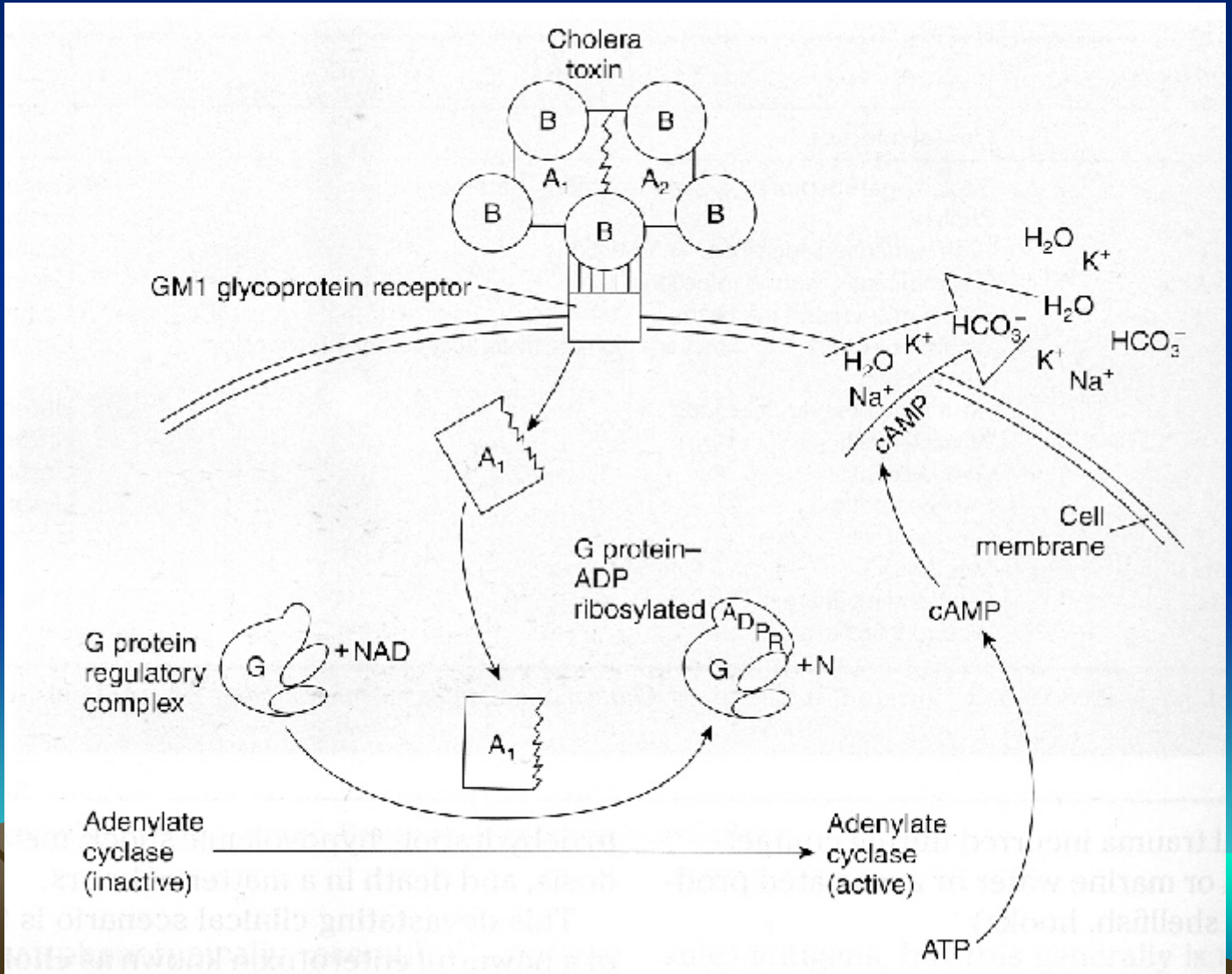
Pathogenesis

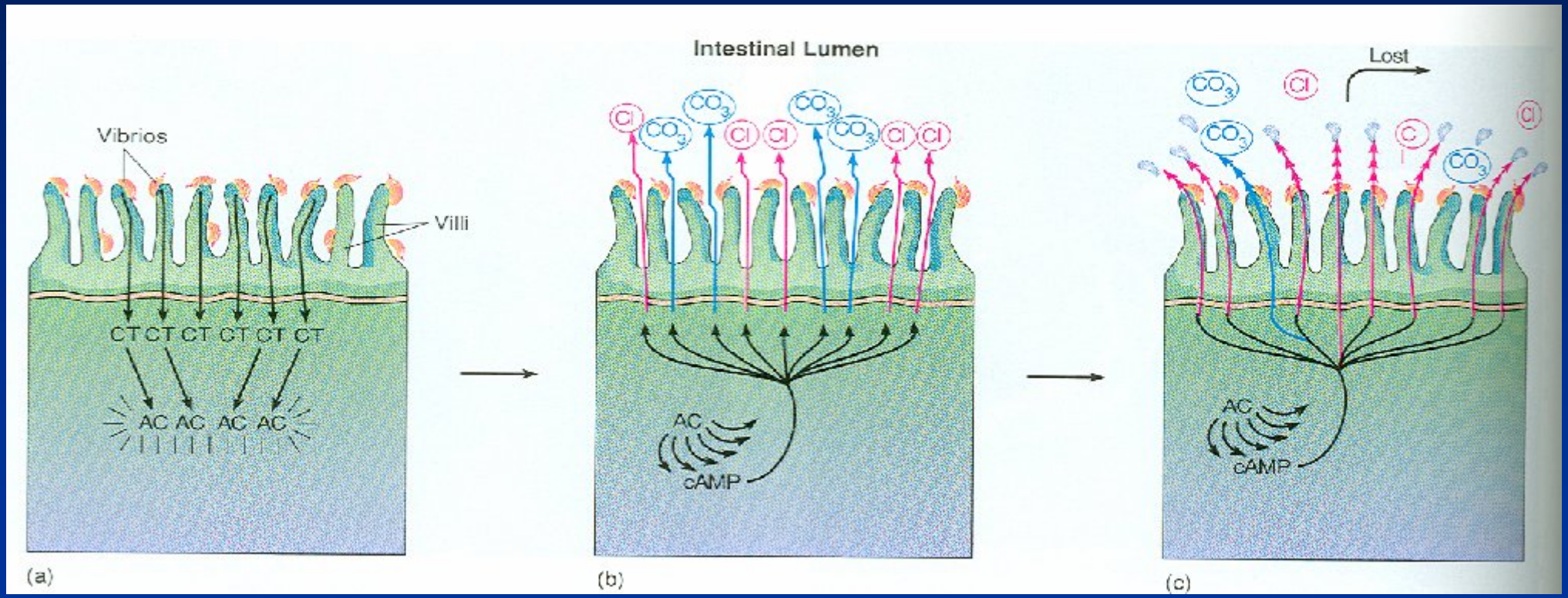
- ID 10^8
- Duodenum and jejunum
- CT (cholera toxin)
- Incubation period few hours to few days
- Fulminant with diarrhea ,vomiting , rice water stool,
- Loss of water up to 1 Lit/hour

Action of cholera toxin



Cholera toxin activity




Intestinal Lumen
Vibrios
Villi
CT CT CT CT CT CT
AC AC AC AC
cAMP
Lost
(a)
(b)
(c)

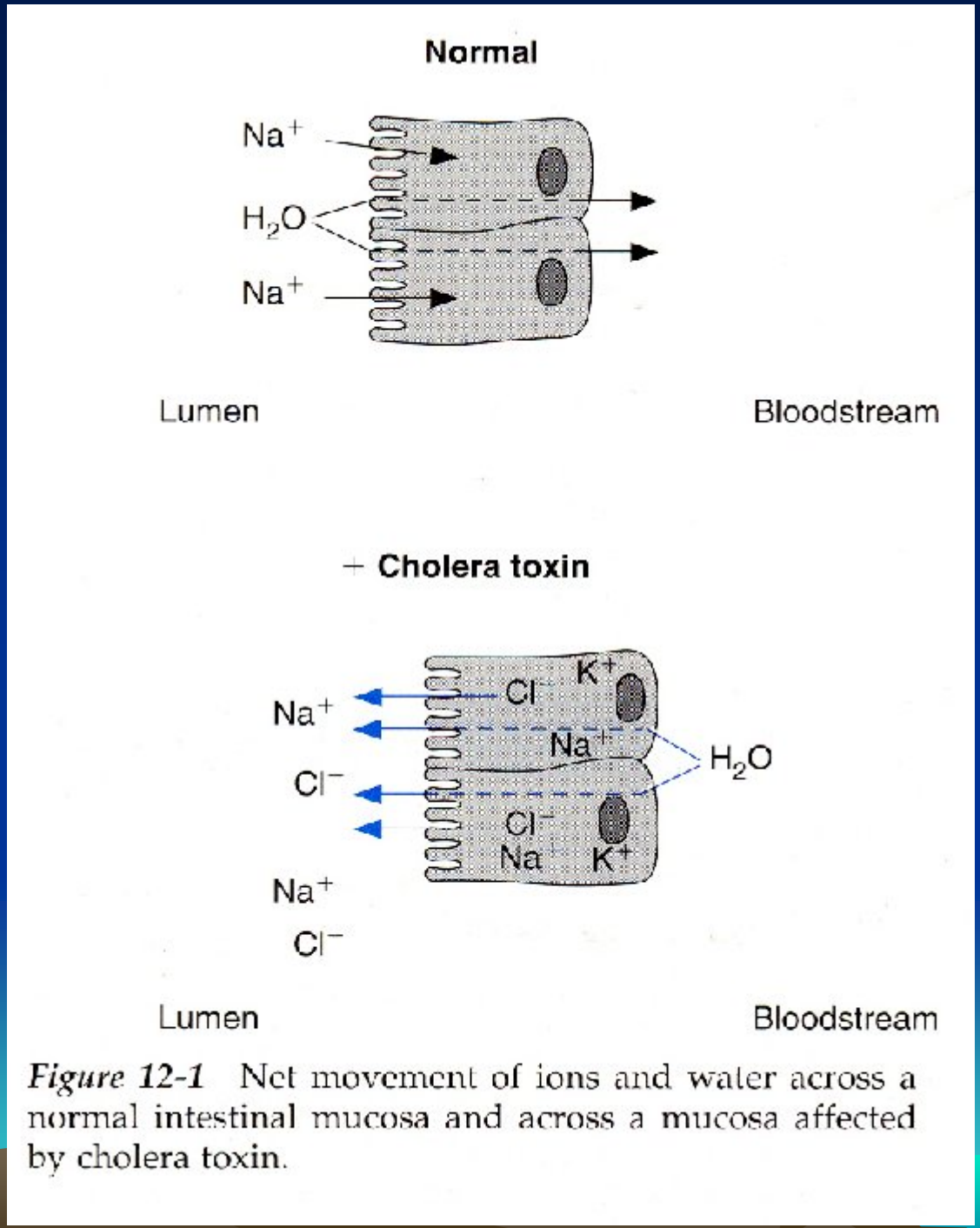
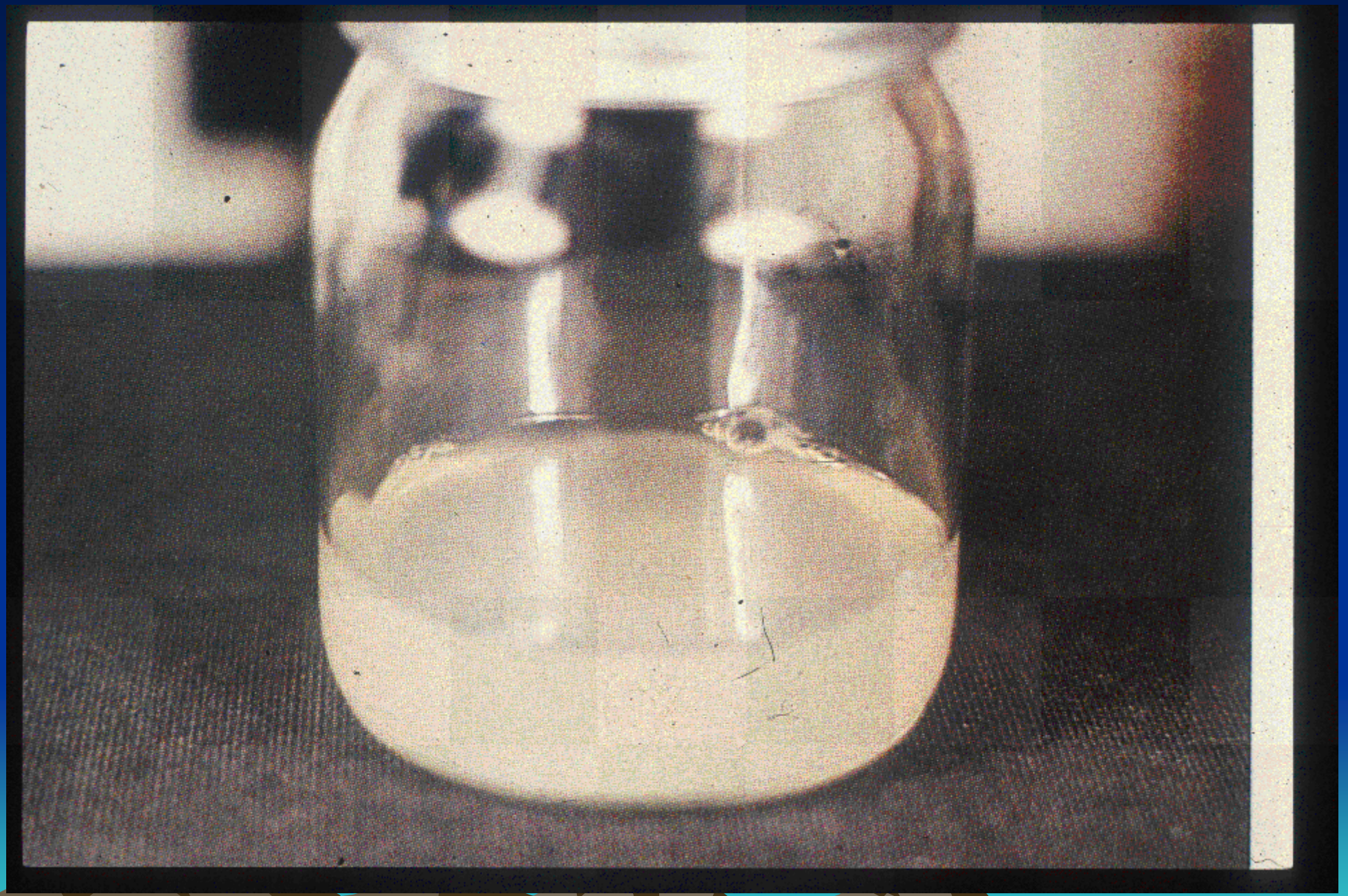


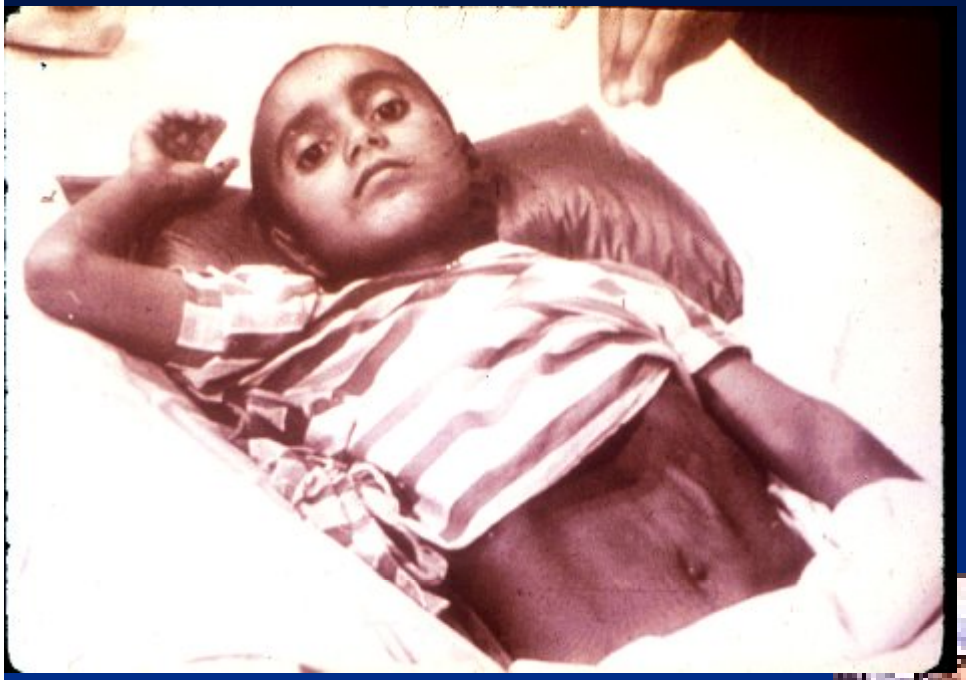
Figure 12-1 Net movement of ions and water across a normal intestinal mucosa and across a mucosa affected by cholera toxin.

Clinical signs

- Acidosis
- Hypotension
- tachycarida
- Sunken eyes
- Convulsions, fever ,coma
- Mortality rate up to 55% in 48 hours

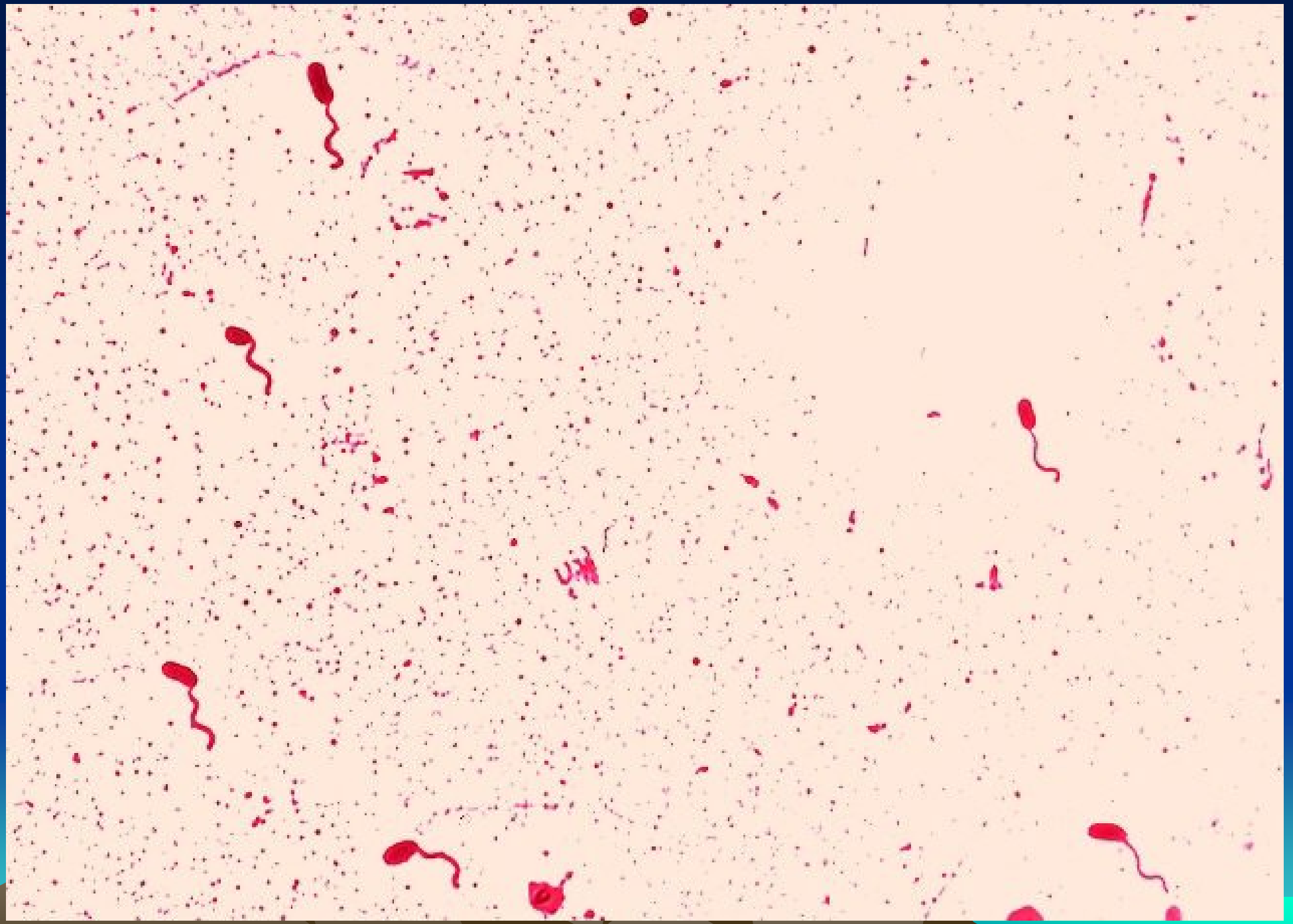

要免霍亂
快打防疫針
不要喝生水
潔清要物食
一圖防預病染傳定法





Diagnosis

- Clinical signs
- Stool culture and vomits culture
- TCBS



V. cholera on TCBS

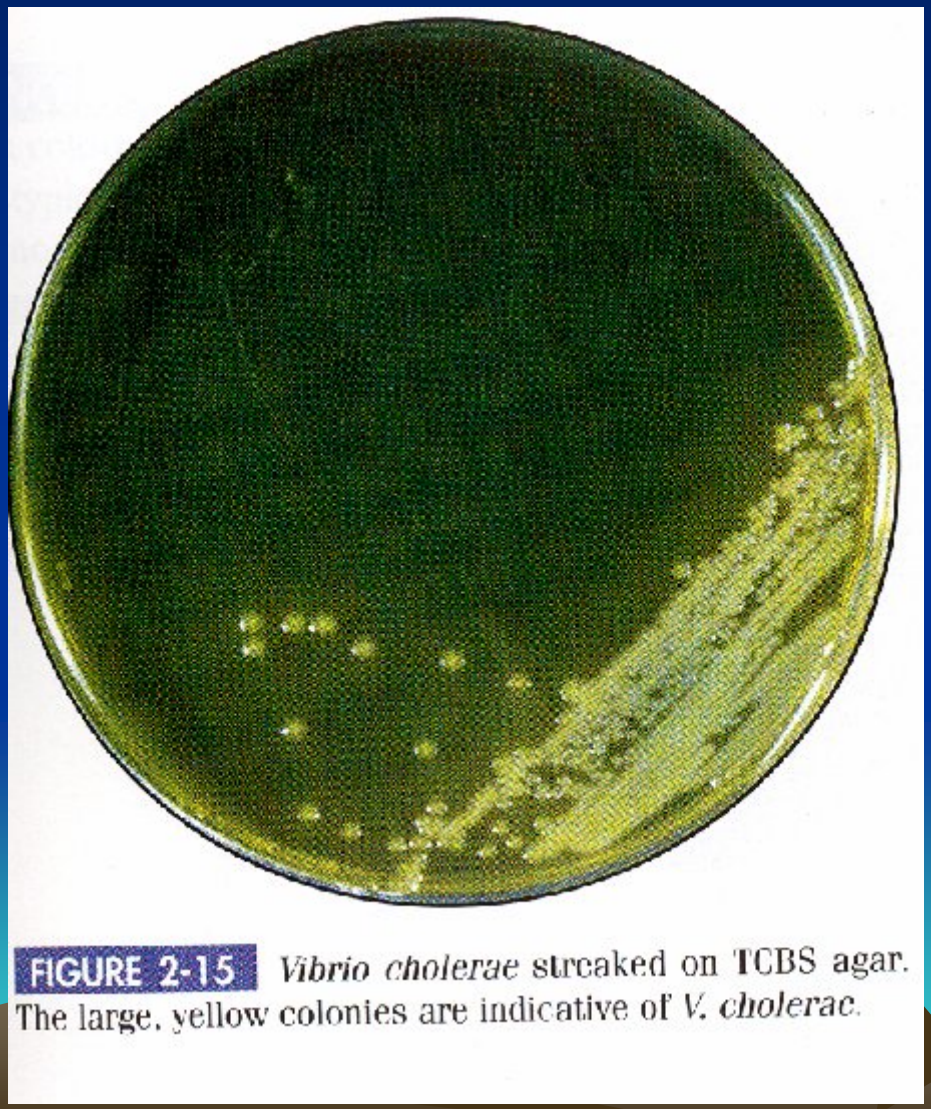


FIGURE 2-15 *Vibrio cholerae* streaked on TCBS agar. The large, yellow colonies are indicative of *V. cholerae*.

Treatment

- 2 part serum physiologic and one part sodium bicarbonate continuously through IV perfusion.
- Nacl---------- 4g/lit

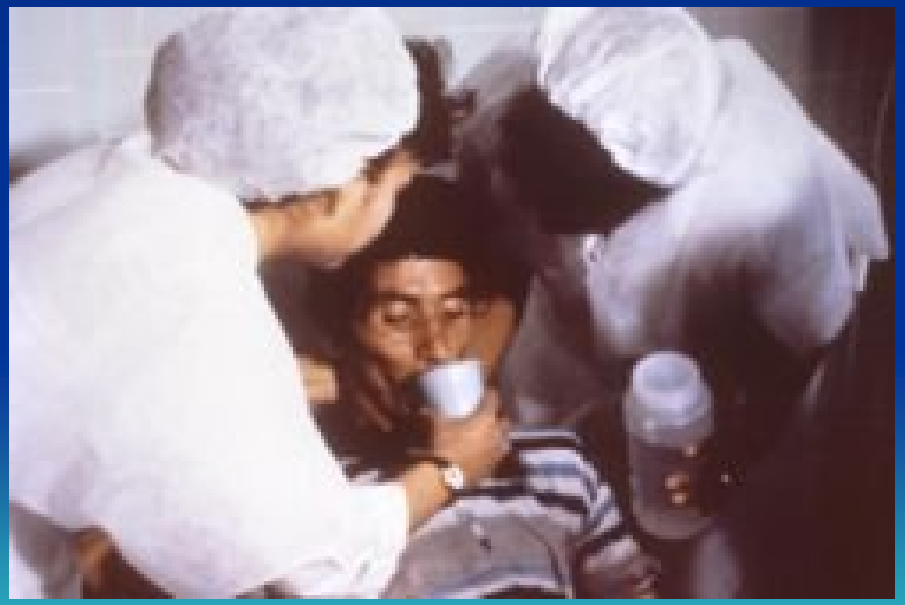
  $NaHCO_3$----------4g/lit

  KCl ----------1-2g/lit

  Glucose----------21g/lit

Treatment

- Tetracycline 0.5g every 6 hours
- Doxycyline 200mg once
- Ciprofloxacine, Cotrimoxazol,Ampicilline, Chloramphenicol.



Prevention

- Personal and environmental hygiene
- Boiling of water and adequately cooked foods.
- Vaccine of cholera contains inactive or dead bacteria (phenol or 65^0C for 1 hour)
- Immunity for six months

Cholera -therapy

- massive secretion of ions/water into gut lumen
- dehydration and death
- therapy
  - fluid replacement
  - antibiotic therapy
- vaccination
  - partially effective
  - not generally used
  - international travelers

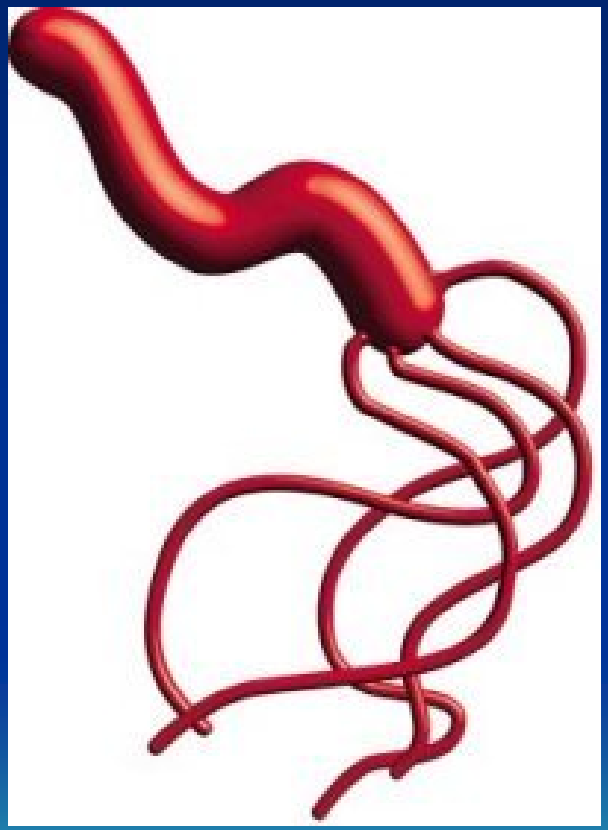
Vibrio parahemolyticus

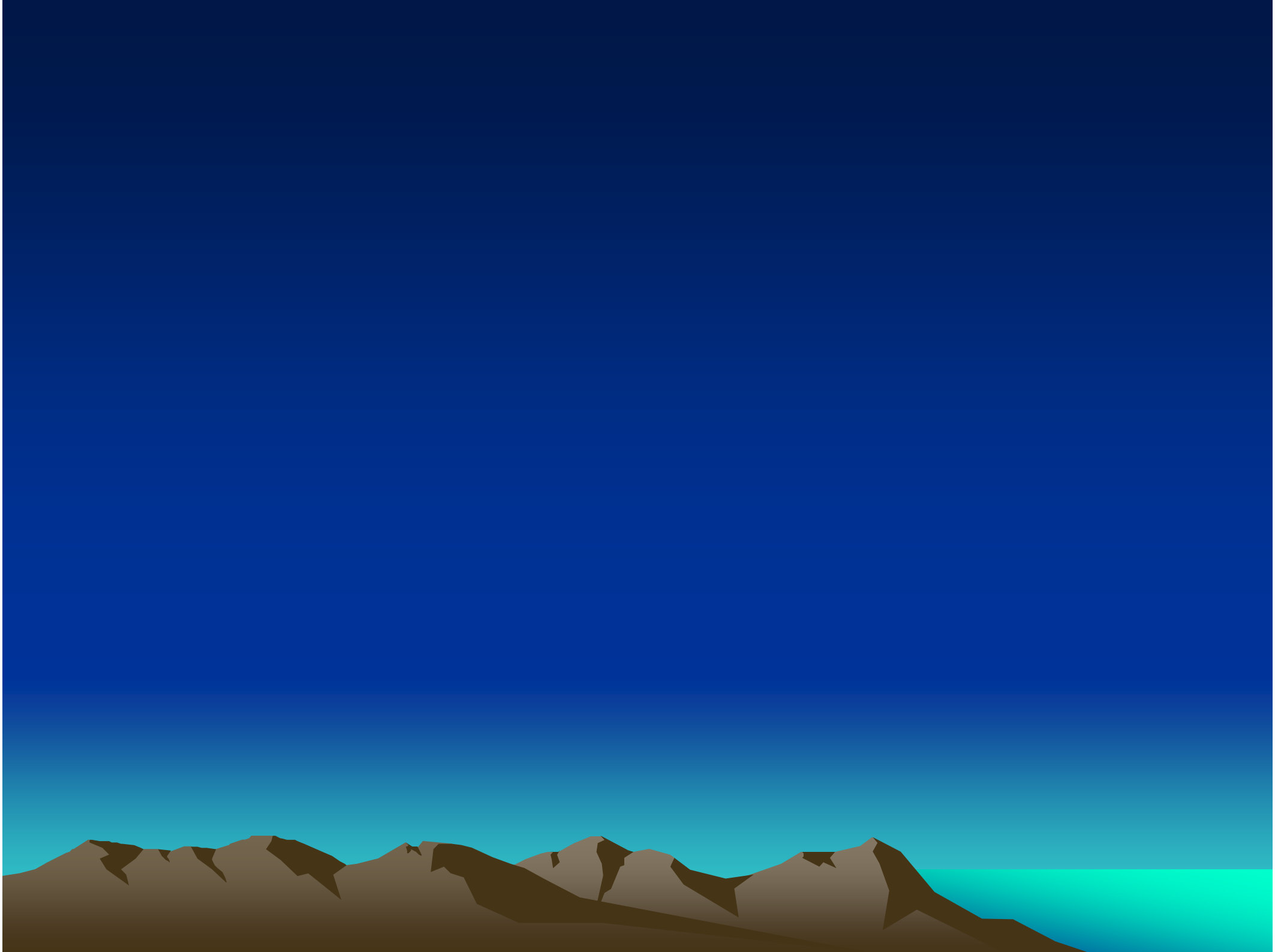
- Similar to V.cholera but can differentiate by culture in 10% NaCl .
- Transmission through seafoods.
- Mild to moderate infections
- Usually gastroenteritis

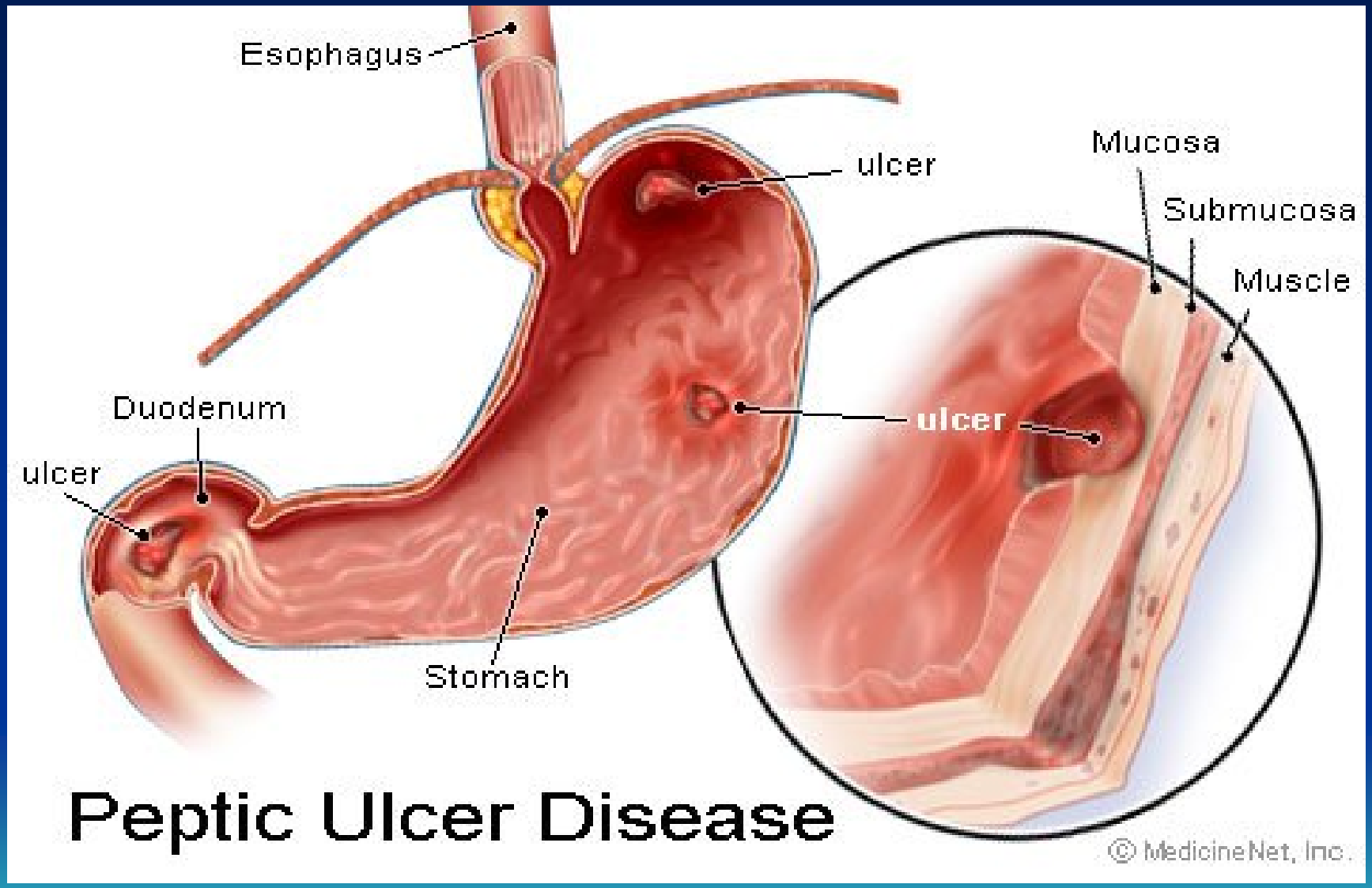
Helicobacter pylori

morphology

- Spiral shaped and multipollar flagella
- Corckscrew motivation
- Micreaerophile
- Producing Urease

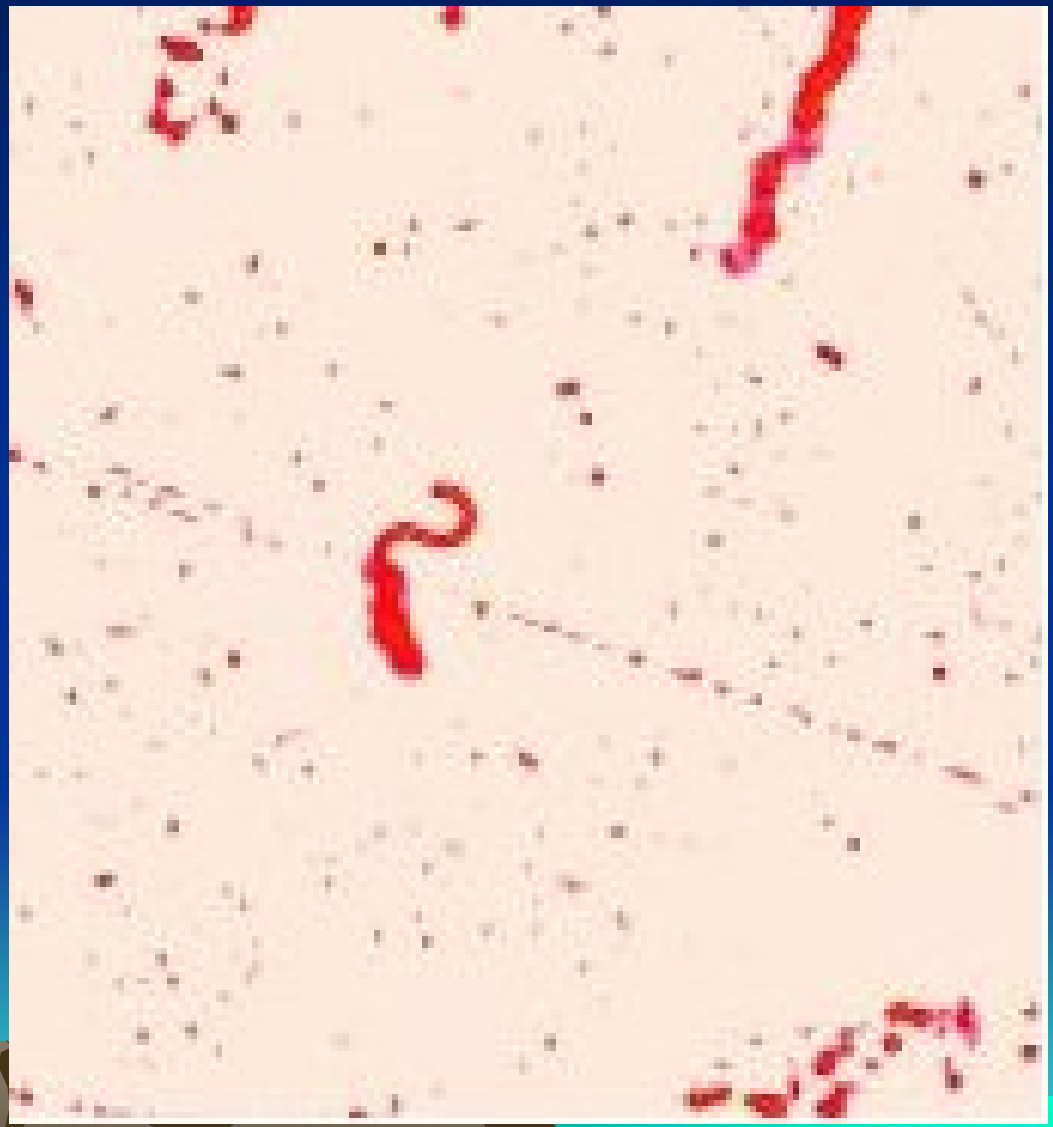





Esophagus
ulcer
Mucosa
Submucosa
Muscle
Duodenum
ulcer
ulcer
Stomach
Peptic Ulcer Disease
© MedicineNet, Inc.

Biochemical characteristics

- Oxydase positive
- Catalse negative
- H2S positive



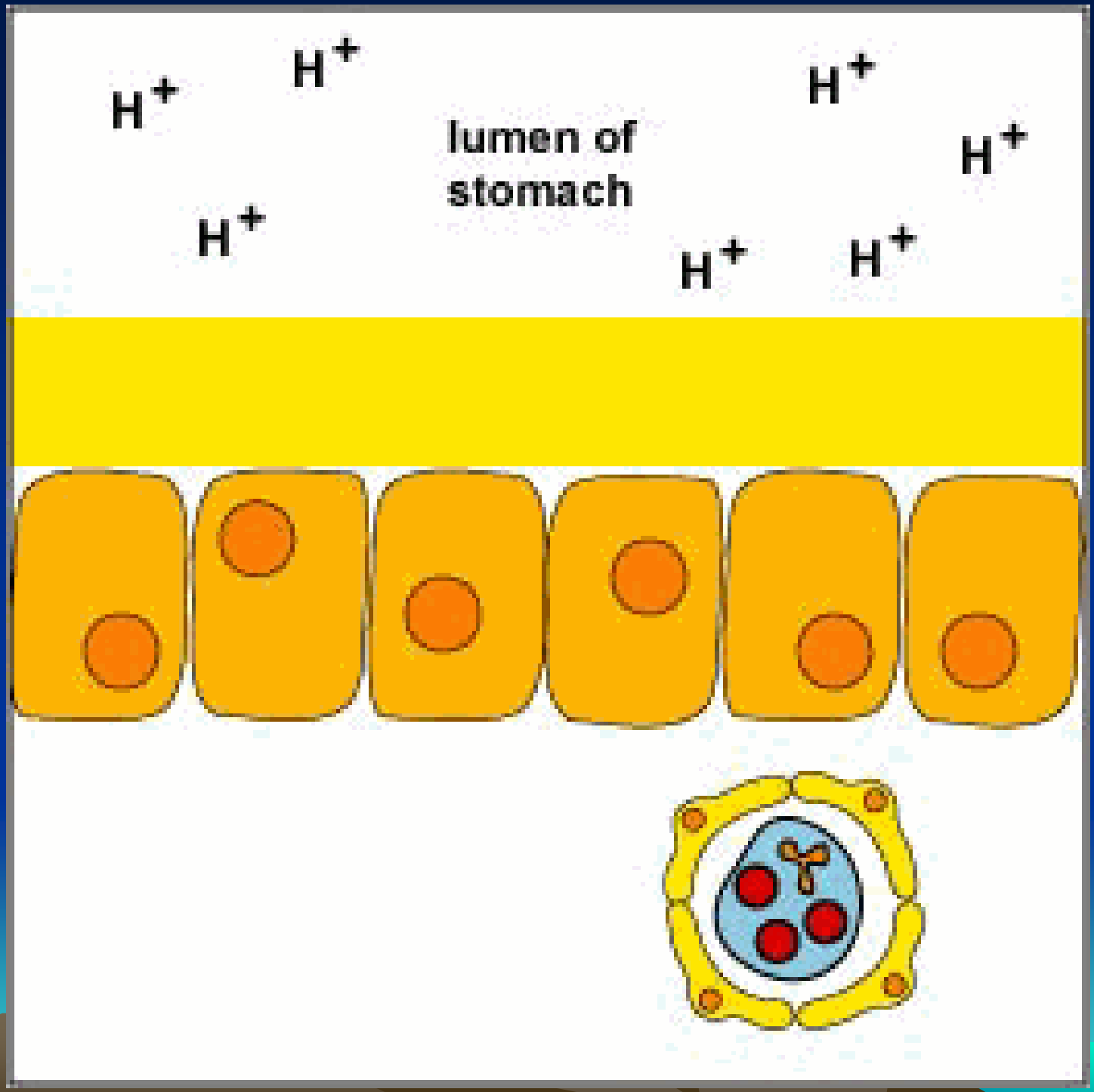
Short history

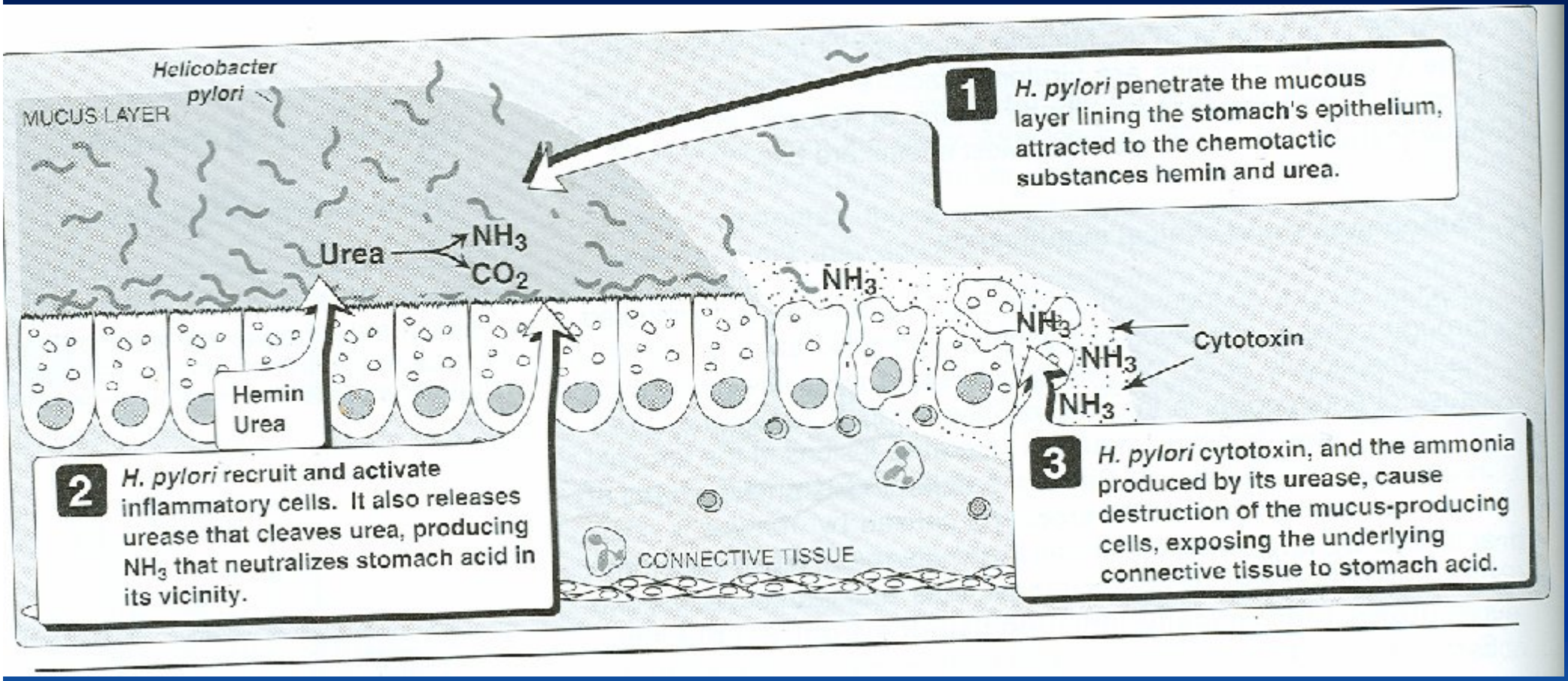
- 1875 German scientists
- 1893 Giulio Bizzozero
- 1899 Prof. Walery Jaworski
- 1979 Robin warren and Barry Marshall
- 1994 National institute of health (USA)

2005 Nobel prize

Pathogenesis

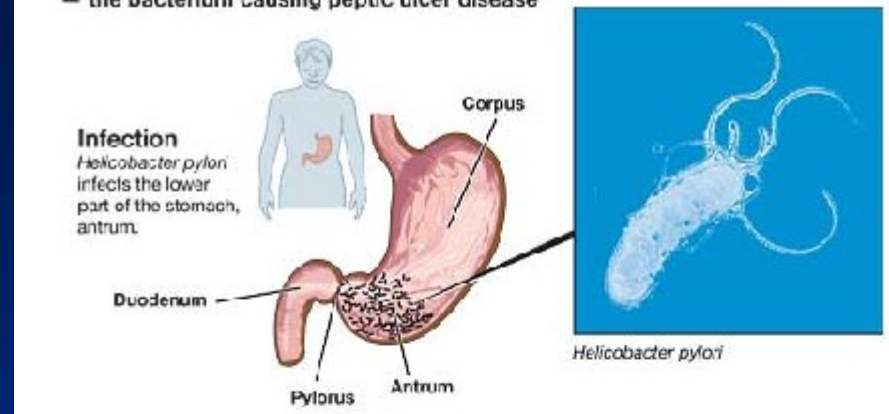
- Optimum PH 6-7
- Lumen of stomach is with PH of 1-2
- Live in deeper parts under lumen where PH is 7.4
- Urease reduce the acidity of stomach
- Cytotoxine

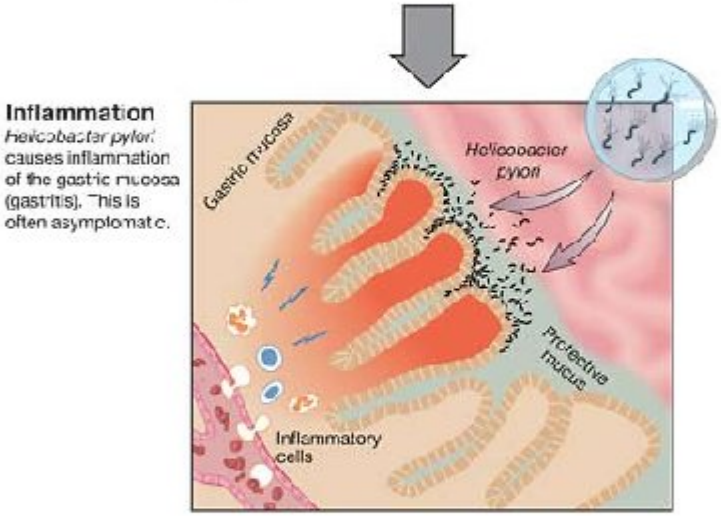

H+
H+
H+
H+
H+
H+
H+
lumen of
stomach

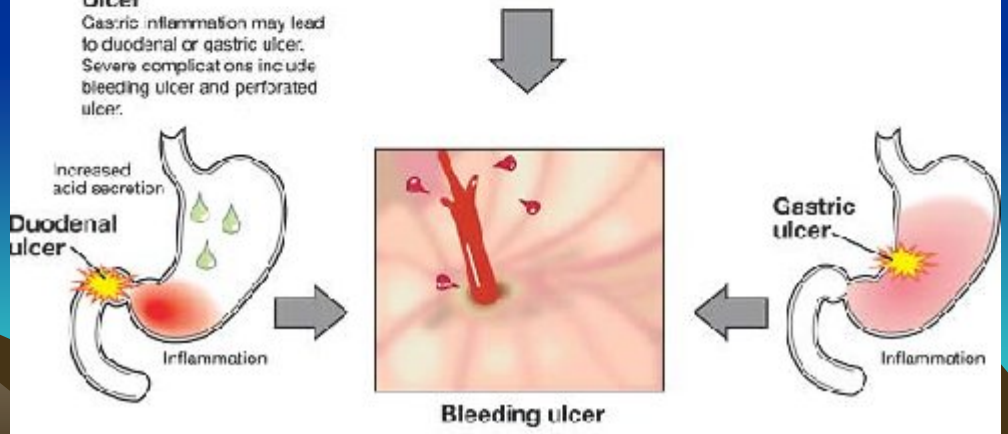

Helicobacter pylori
MUCUS LAYER
1 H. pylori penetrate the mucous layer lining the stomach's epithelium, attracted to the chemotactic substances hemin and urea.
Urea
NH3
CO2
NH3
NH3
NH3
NH3
Cytotoxin
Hemin
Urea
2 H. pylori recruit and activate inflammatory cells. It also releases urease that cleaves urea, producing NH3 that neutralizes stomach acid in its vicinity.
CONNECTIVE TISSUE
3 H. pylori cytotoxin, and the ammonia produced by its urease, cause destruction of the mucus-producing cells, exposing the underlying connective tissue to stomach acid.

Helicobacter pylori

— the bacterium causing peptic ulcer disease





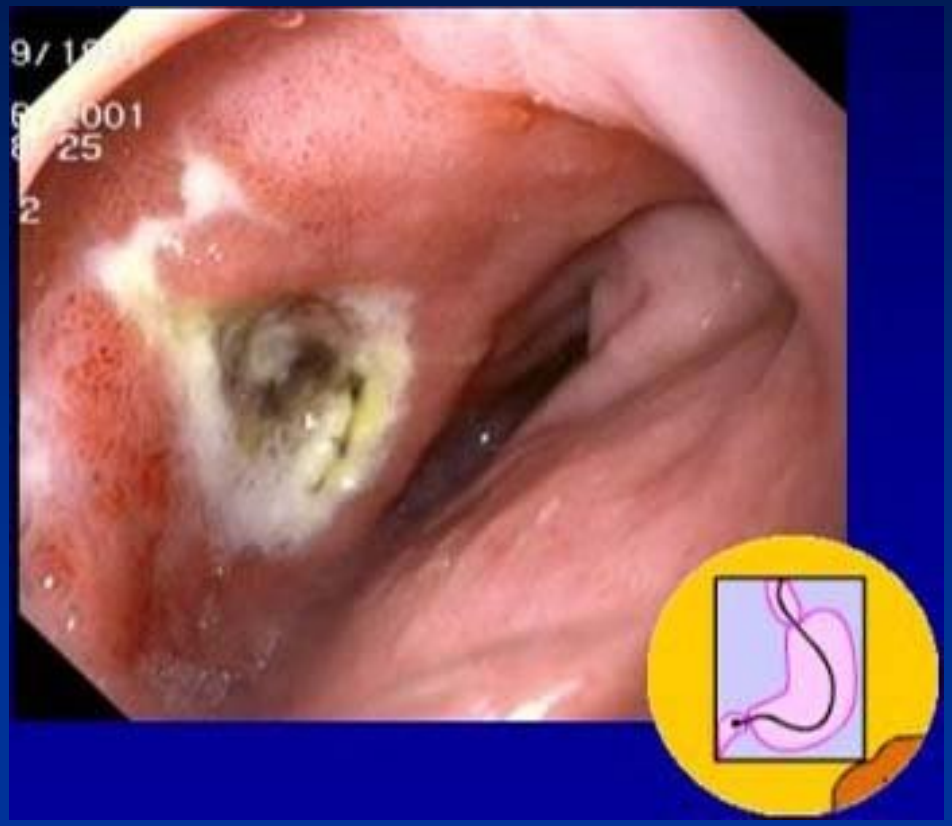


- Acute gastritis with epigastric disturbance and diarrhea for one week
- In 95% cases of gastric ulcers and duodenal ulcers isolated

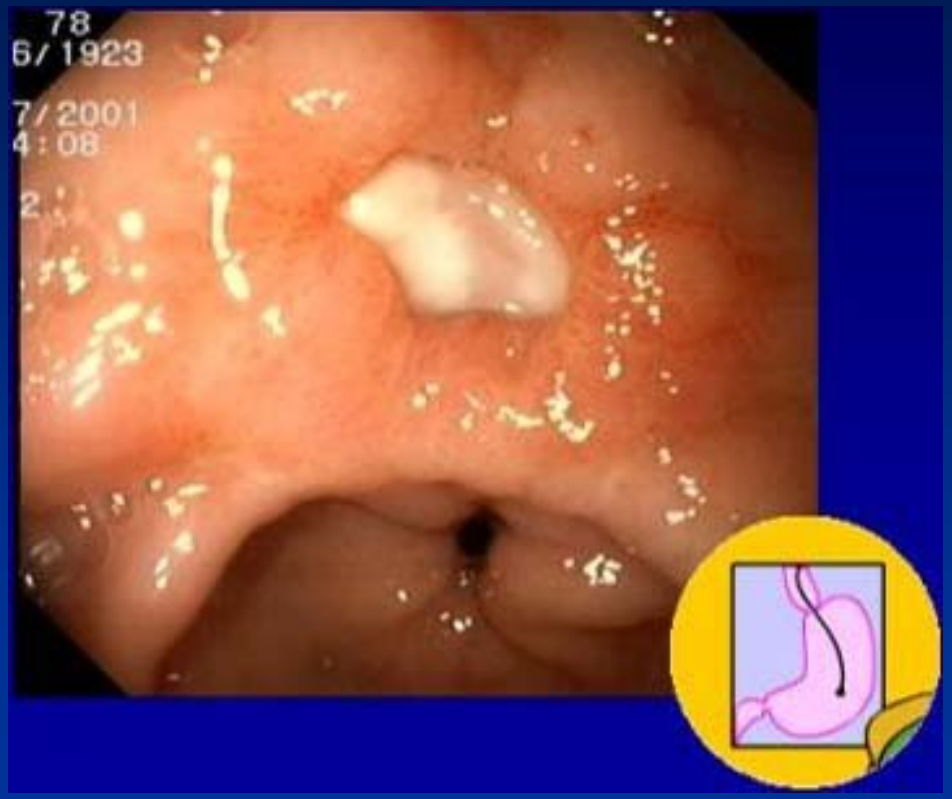
- Due to destruction of epithelium and atrophy of secretory glands is a main factor in gastric carcinoma

Helicobacter pylori

- stomach mucosa
- ulcers



A duodenal ulcer caused by H.pylori

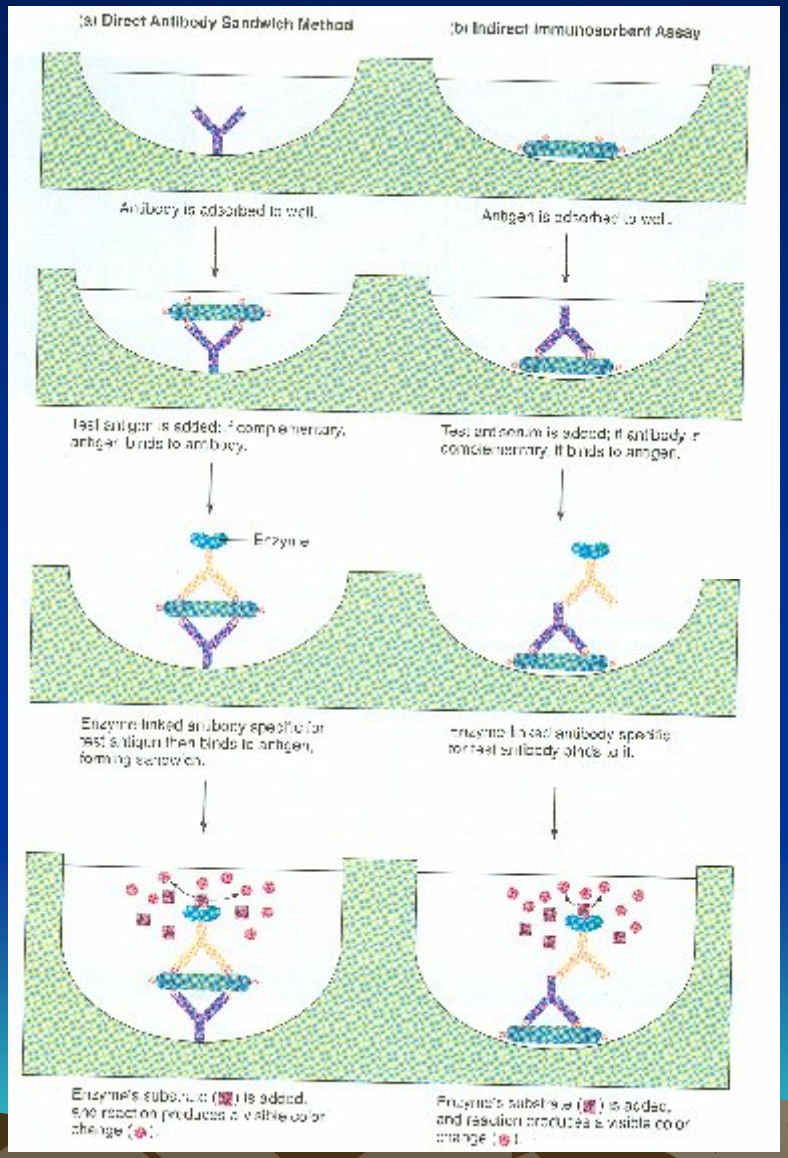


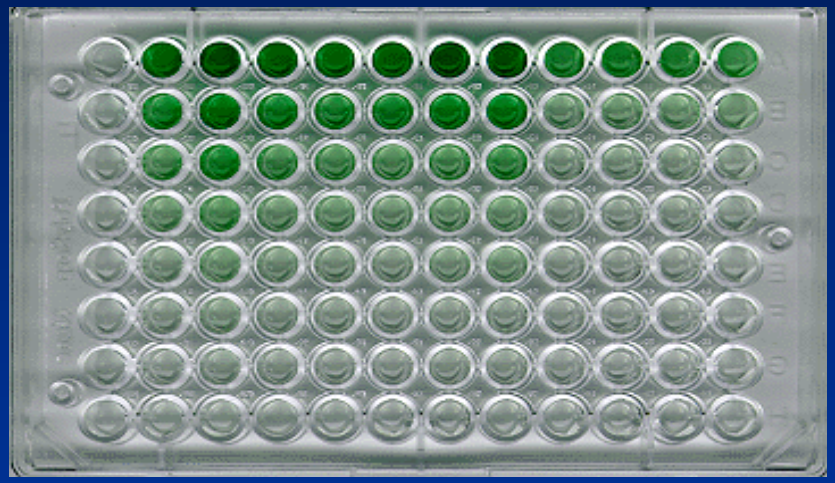
A gastric ulcer caused by H.pylori

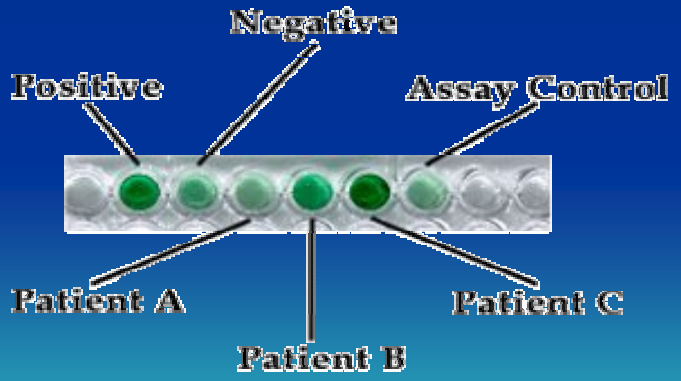
Urease

- Important in neutralizing stomach acid

ELISA


(a) Direct Antibody Sandwich Method
(b) Indirect Immunosorbent Assay
Antibody is adsorbed to well.
Antigen is adsorbed to well.
Test antigen is added; if complementary, antigen binds to antibody.
Test antiserum is added; if antibody is complementary, it binds to antigen.
Enzyme
Enzyme-linked antibody specific for test antigen then binds to antigen, forming sandwich.
Enzyme-linked antibody specific for test antibody binds to it.
Enzyme's substrate is added. Reaction produces a visible color change.
Enzyme's substrate is added, and reaction produces a visible color change.




Negative
Positive
Assay Control
Patient A
Patient C
Patient B

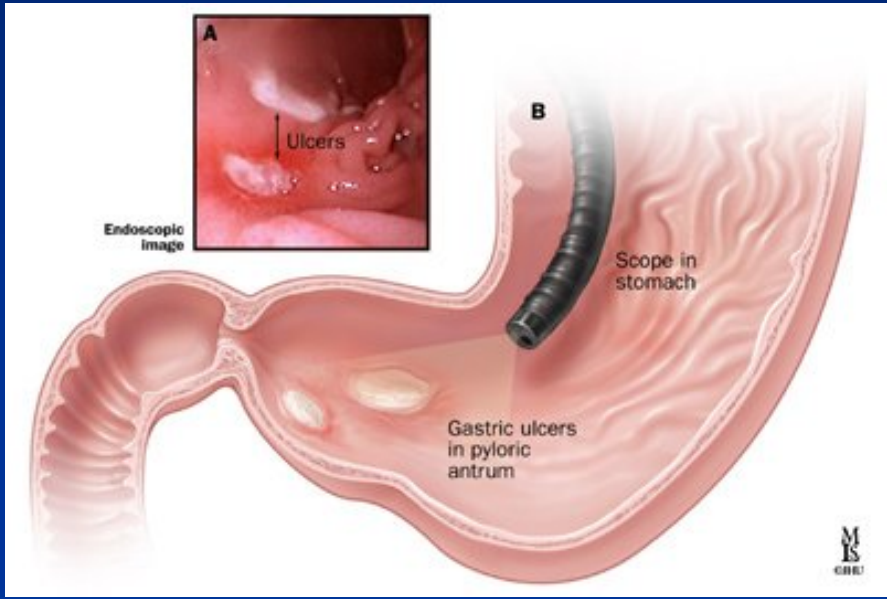

H. pylori C T
H. pylori C T

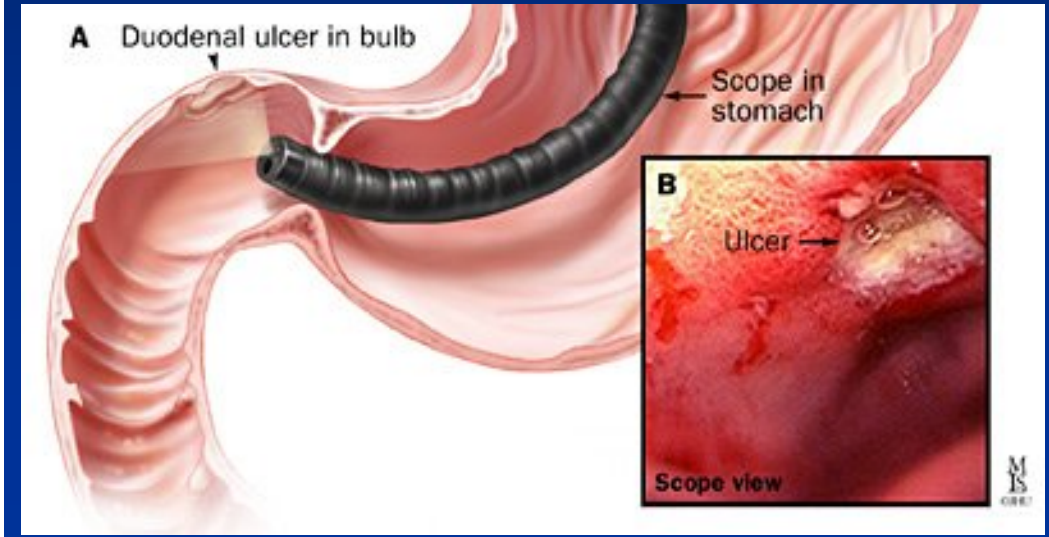
Diagnosis -*Helicobacter*

- Culture

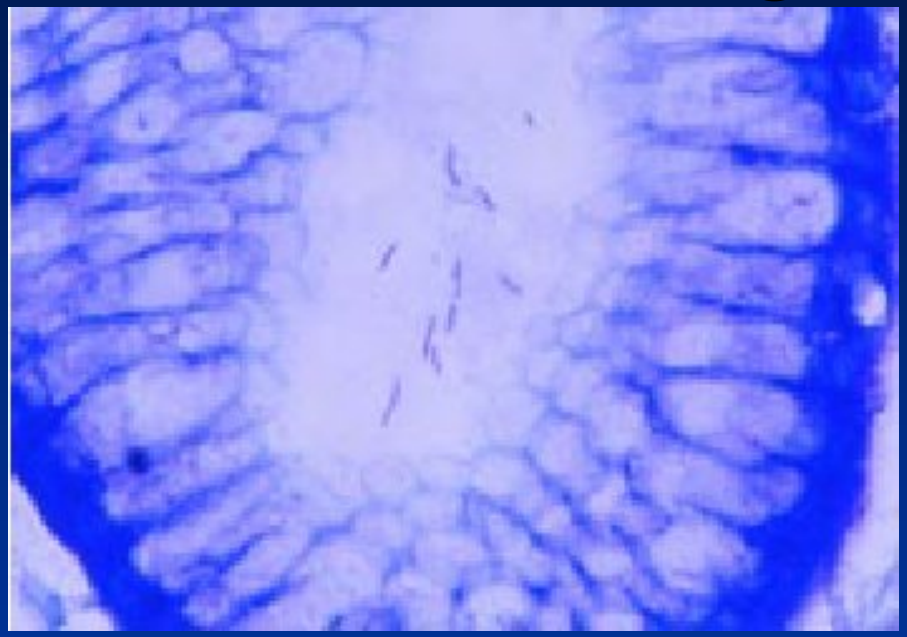
- Urease test

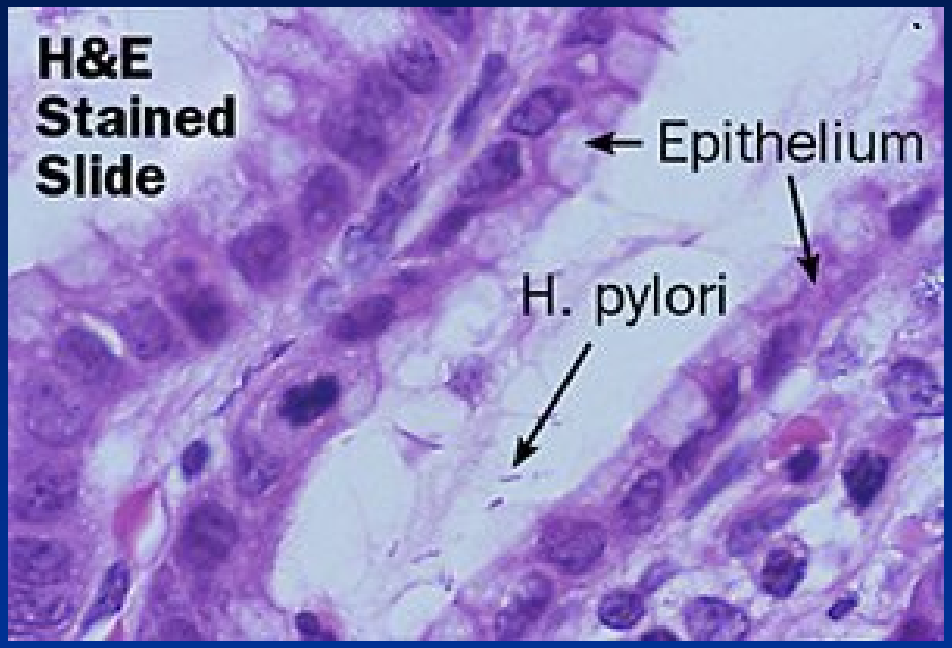
Endoscopy

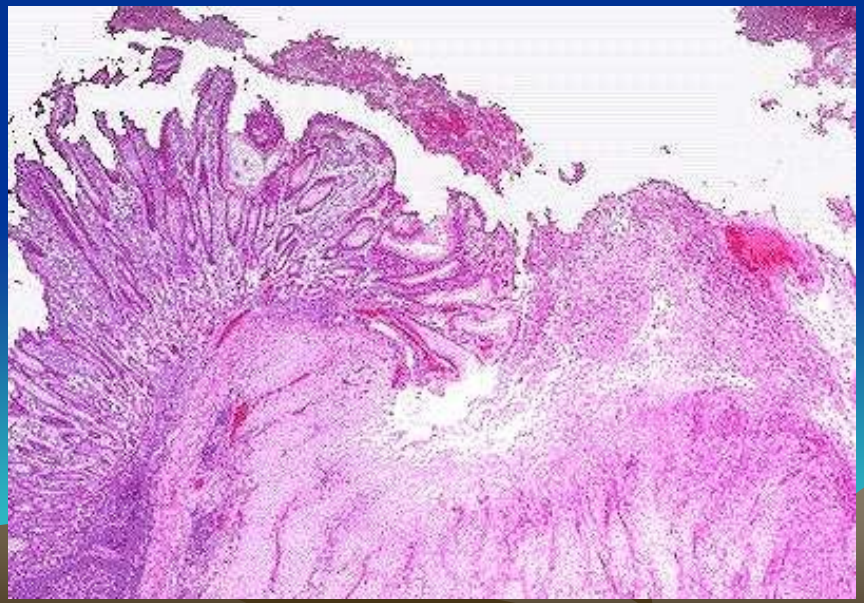

A
Ulcers
Endoscopic image
B
Scope in stomach
Gastric ulcers in pyloric antrum


A Duodenal ulcer in bulb
Scope in stomach
B
Ulcer
Scope view

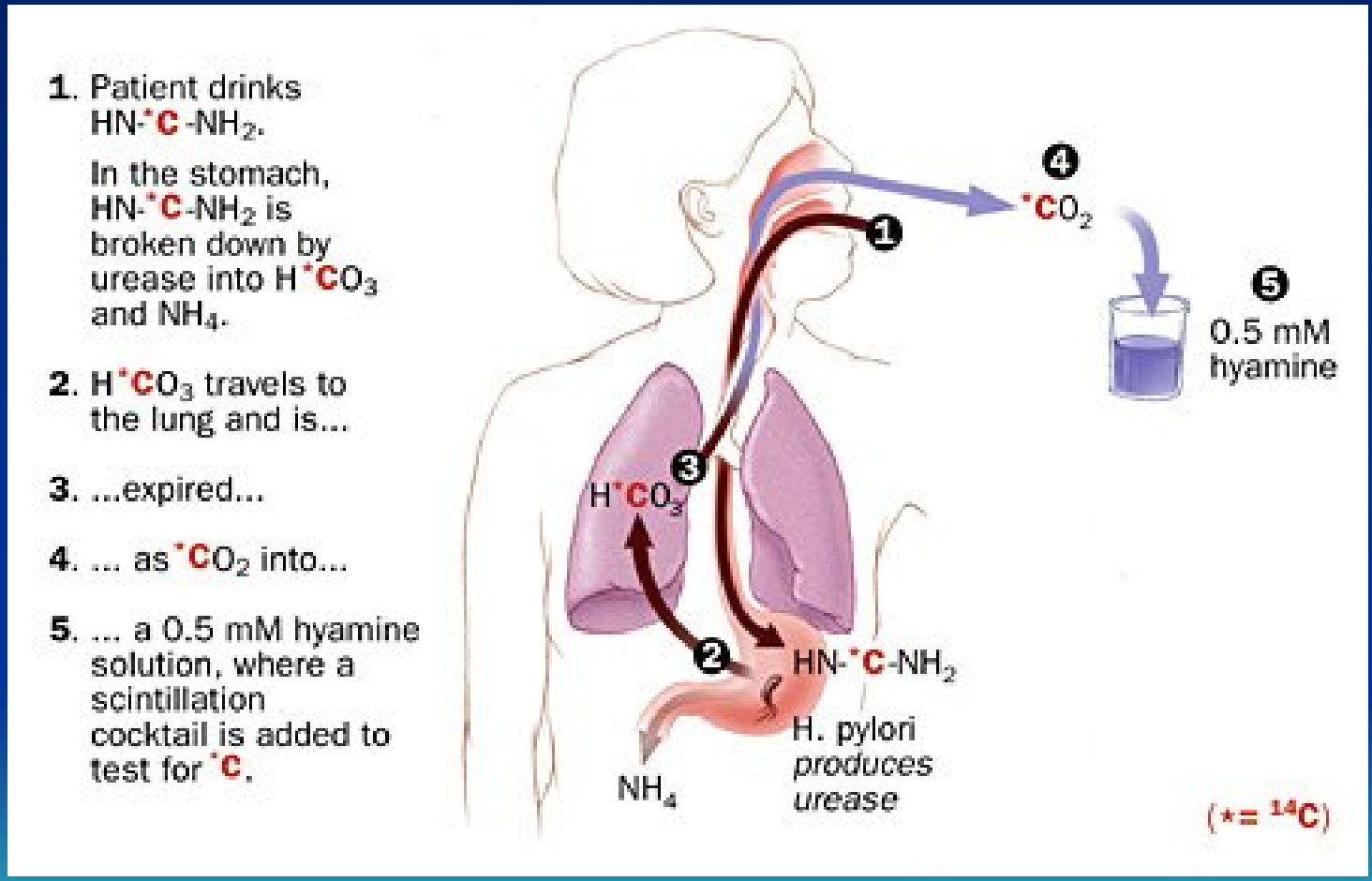
Histological examination







Urea breath test



Therapy -Helicobacter

- Antibiotics
  - cures ulcers

Therapy

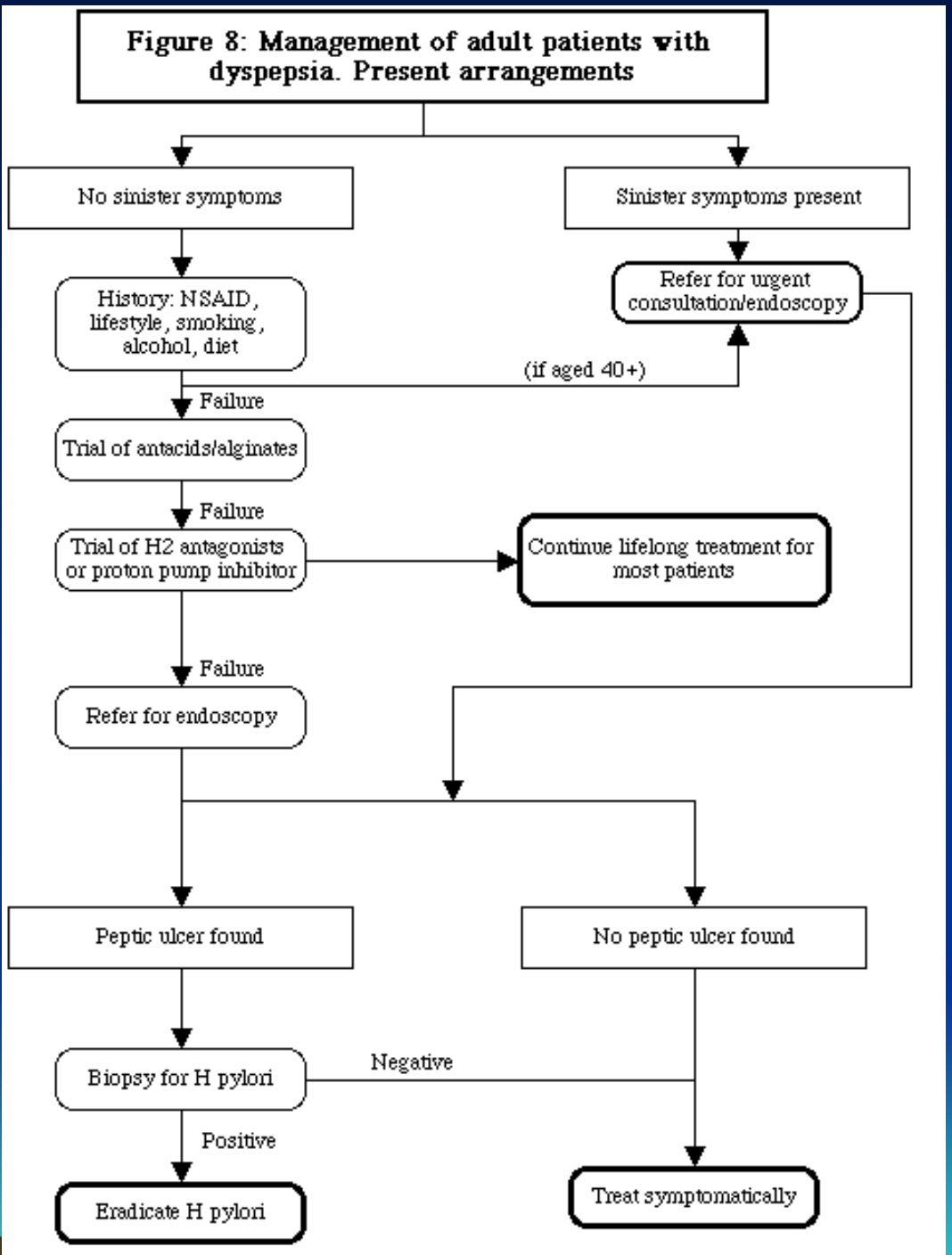
- severe diarrhea
  - fluid replacement essential

Treatment

- Triplet therapy
- Amoxicillin+ Clarithromycin+PPI

 - metronidiazol with Bismuth subcitrate,bismuth subsalycylate & Ampicillin or Tetracycline for 14 days.

Figure 8: Management of adult patients with dyspepsia. Present arrangements



This schematic represents the way in which patients with dyspepsia are likely to be treated in most present circumstances. It is not intended to be a treatment regimen.

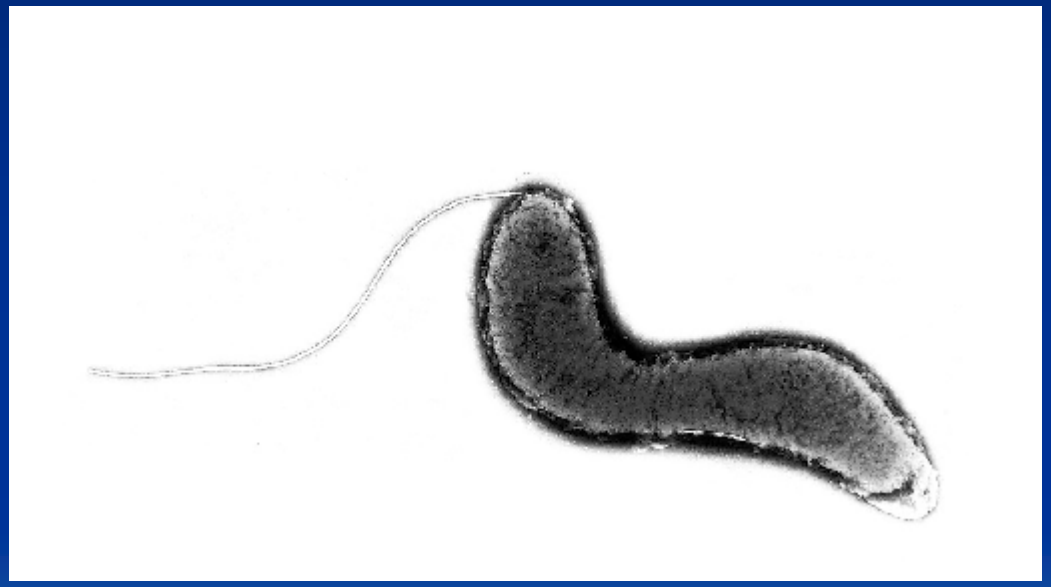
Campylobacter

General characteristics

- Monopolar or bipolar flagella
- S shaped In morphology
- Habitat of GI,GUT & mouth of humans and animals

Campylobacter jejuni,C.coli

- Morphology
- Gram negativ
- Curved,spiral or S shaped
- Micraerophile
- nonfermentative

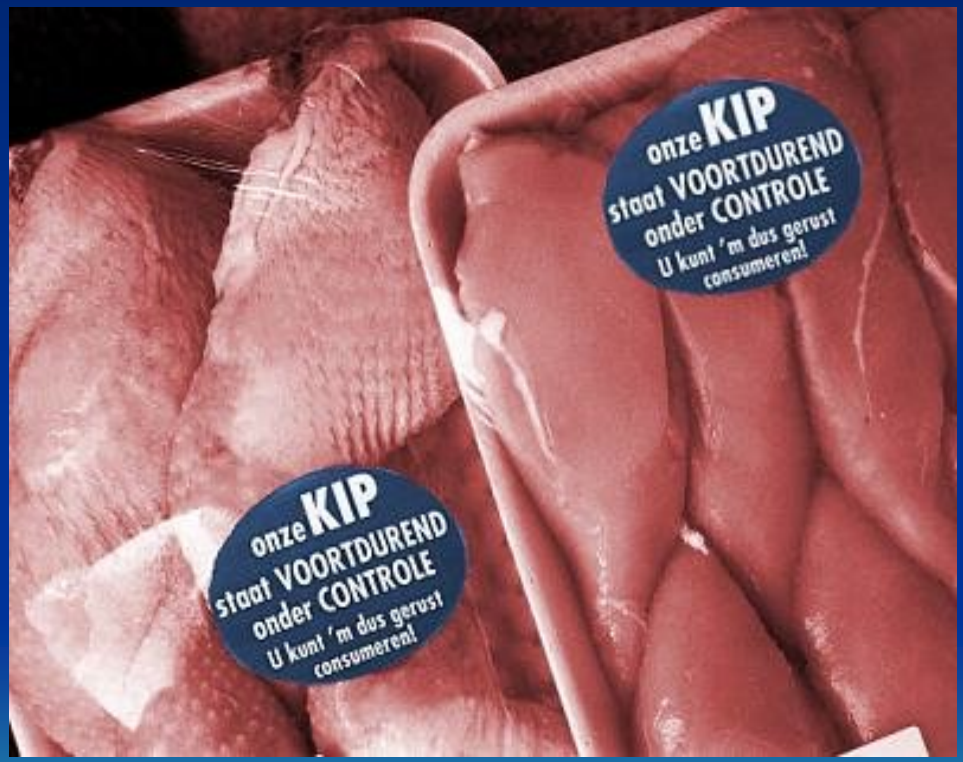


Biochemical characteristics

- Oxydase positive
- Catalase positive
- H_2S positive

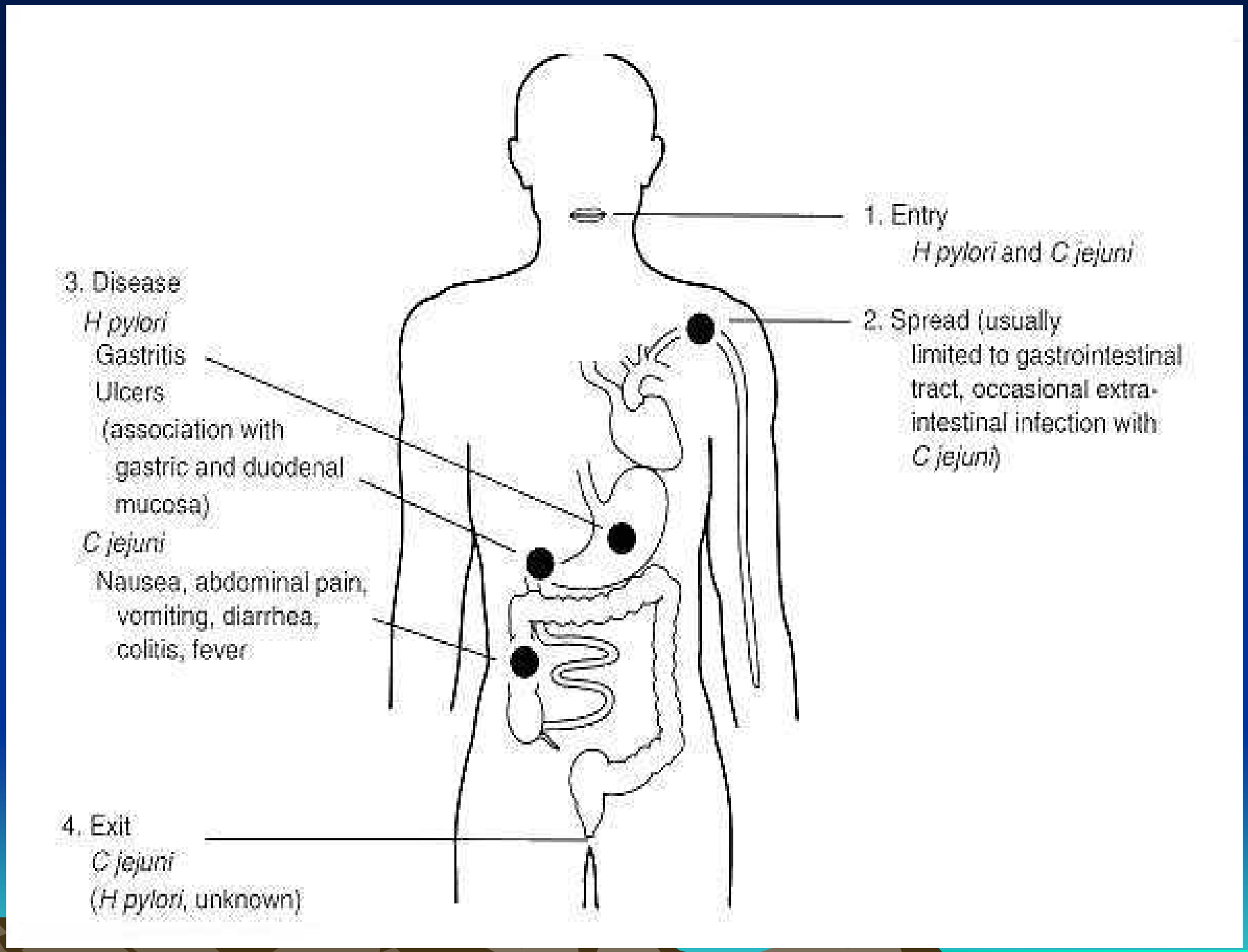
Pathogenesis

- Fecal-oral transmission
- Infected meats(poultry) or water



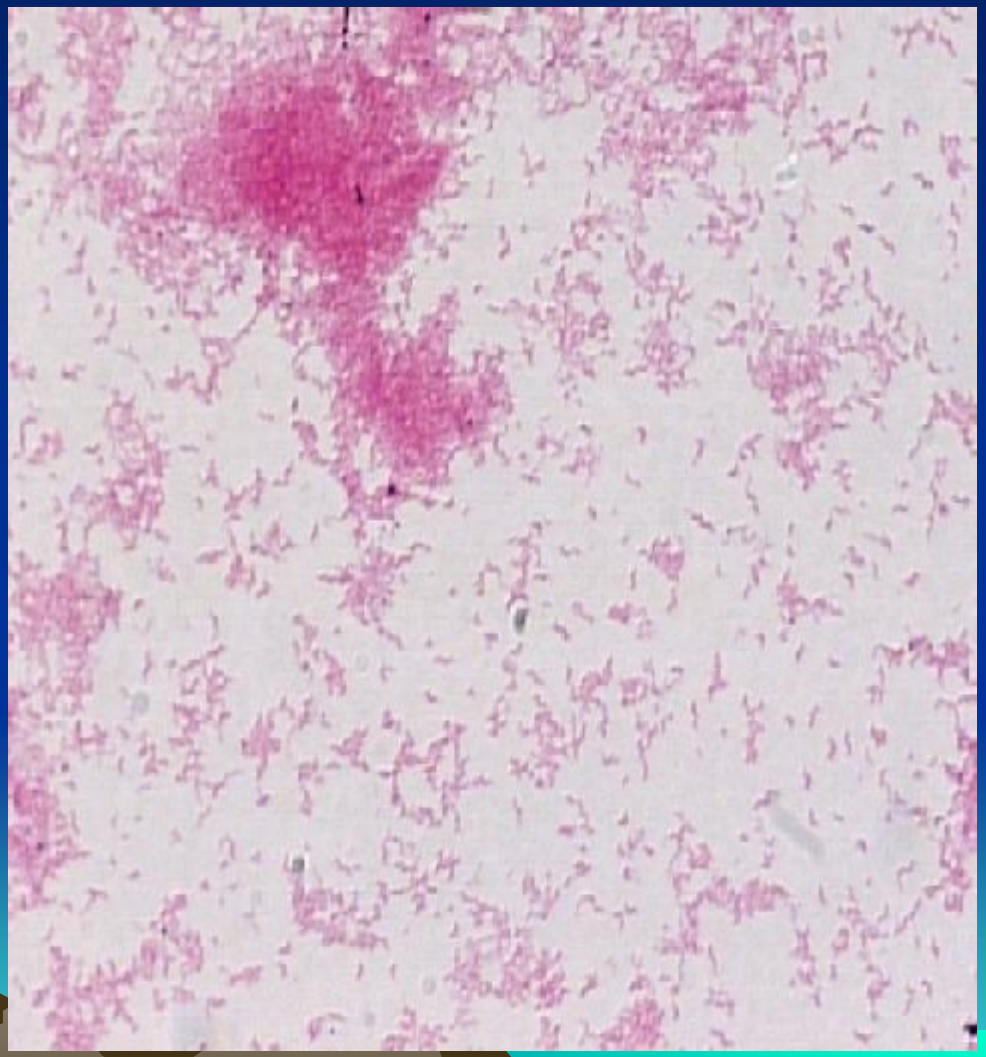
- Causes ulcers in illeum,jejunum and colon

- Acute enteritis;
- fever,headache,muscular pains,abdominal tensions ,bloody or without blood.
- Traveler’s diarrhea and pseudoappendicitis

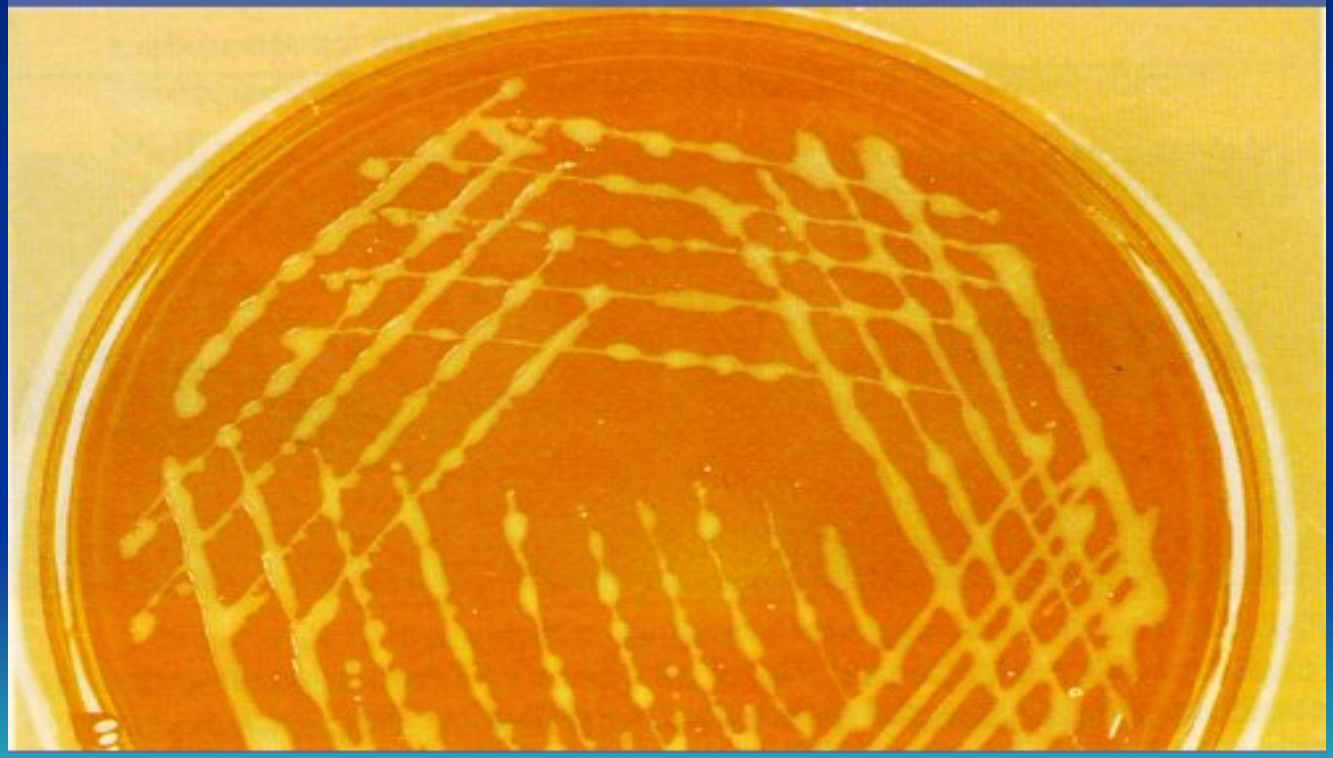

1. Entry
H pylori and C jejuni
2. Spread (usually limited to gastrointestinal tract, occasional extra-intestinal infection with C jejuni)
3. Disease
H pylori
Gastritis
Ulcers
(association with gastric and duodenal mucosa)
C jejuni
Nausea, abdominal pain, vomiting, diarrhea, colitis, fever
4. Exit
C jejuni
(H pylori, unknown)

Diagnosis

- Motility in stool sample
- Direct staining
- Culture on special media such as SKIRROW, that contains polymyxine,vancomycine and trimethoprim



Growth of *Campylobacter jejuni* on special medium, incubated in a microaerophilic environment.



Rapid diagnostic test

Treatment

- Electrolytes
- Ciprofloxacin
- Erythromycin for 14 days

Campylobacter fetus

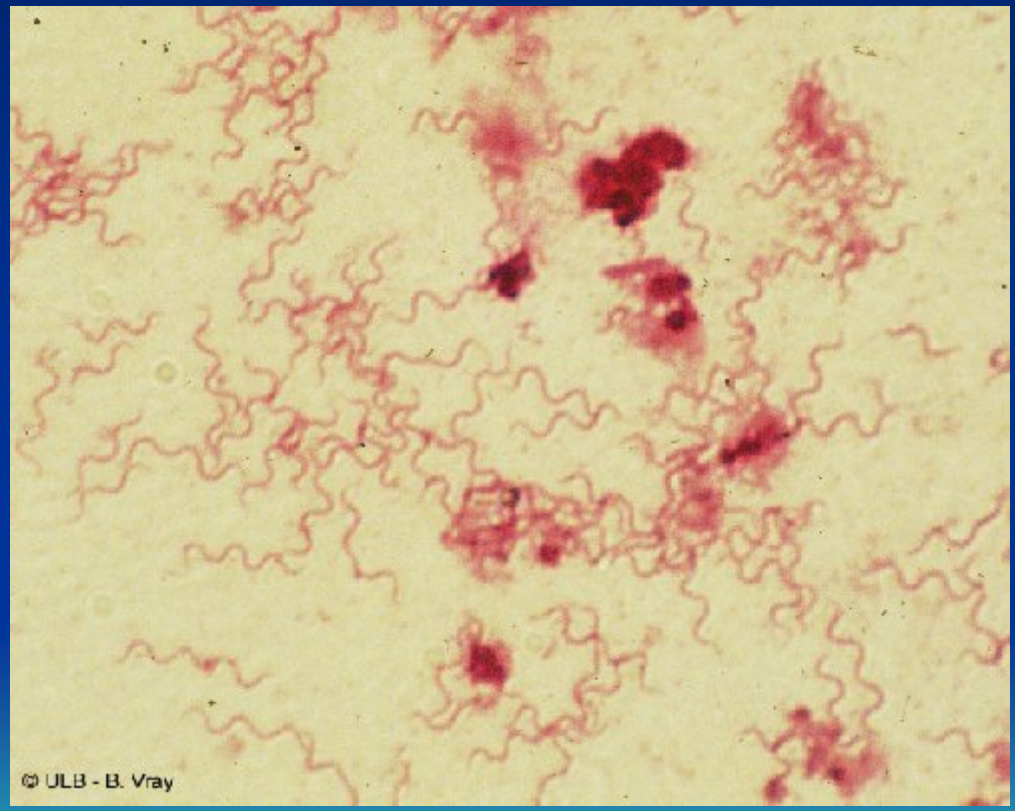
- Abortion in cows,goats and sheeps

Prevention

- Adequate hygiene
- Safe water
- Milk pasteurization
- Adequate heating of poultry

Spirillum minus

- Causes Rat bite fever (sodoku)
- Spiral and nonflexible



distribution

- Rural areas of Japan and far East
- Rat is primary host

-Septicemia

-Eye infections

-Lung infections

Transmission

- Through bites of rat
- Tow weeks of incubation period
- Local inflammation at bitten site and edema, Purplish
- Local lymhpadenopathy with fever,malaise,headache, slowly decreasing.

Diagnosis

- Dark field microscopy
- Not cultivable on artificial media
- Treatment
  - Penicillin
  - Streptomycin
  - Tetracyclin