Vibrio, Campylobacter & Helicobacter

Vibrio

- Vibrio spp. can be found mainly in marine environment
- Cause diseases in fish, shellfish, mammals
- >100 species within the genus
- Important spp

Vibrio cholerae

Vibrio parahaemolyticus

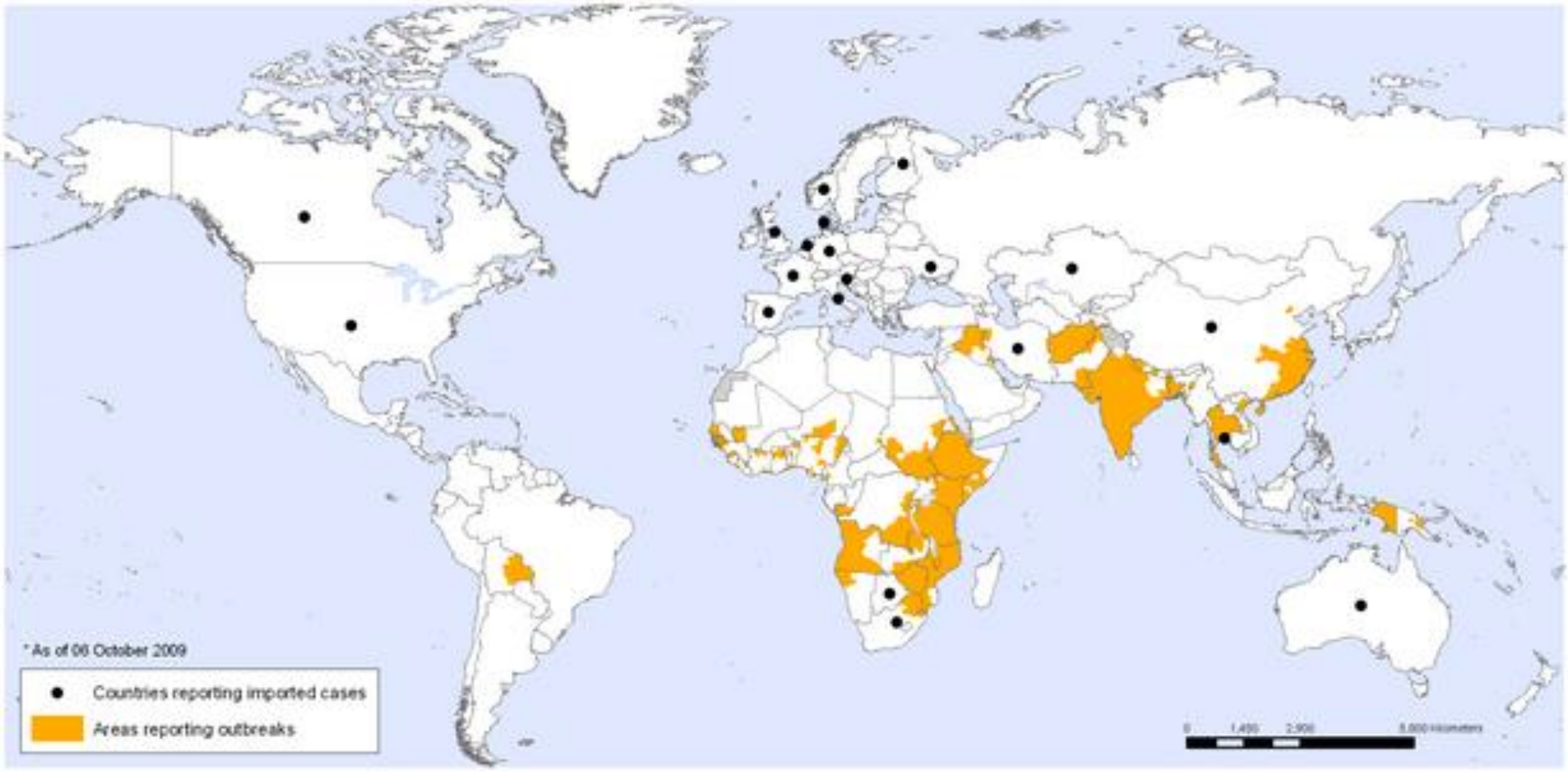
Vibrio vulnificus

Vibrio alginolyticus

Clinical significance

- ***Vibrio cholerae***
  - Cholera
    - Seven pandemics; serotypes **O1** and O139
- Non-O1/non-O139 serotypes of vibrio
  - sporadic cholera cases
  - extra intestinal infections
- *Vibrio parahaemolyticus*
  - gastroenteritis
- *Vibrio vulnificus &V. alginolyticus*
  - wound infections

Cholera, areas reporting outbreaks, 2007–2009*



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Data Source: World Health Organization
Map Production: Public Health Information and Geographic Information Systems (GIS)
World Health Organization

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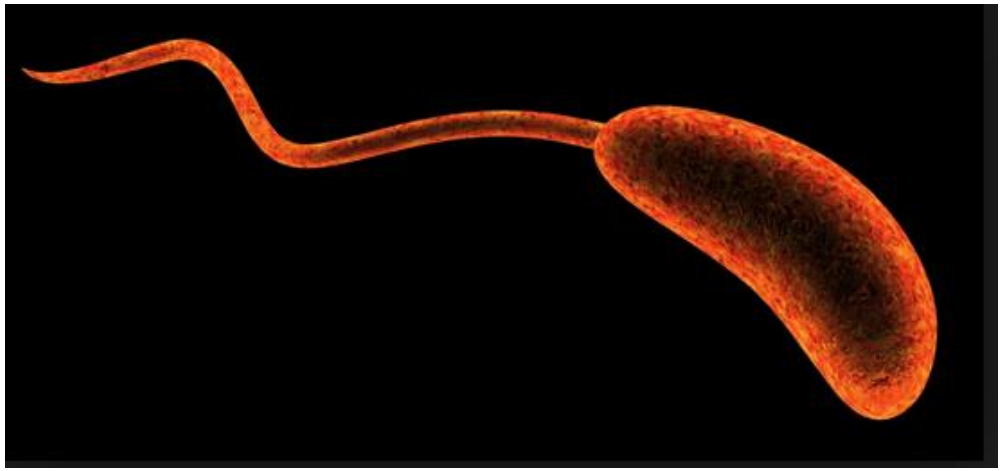
Features



- Short Gram (-) ve rods
- Curved
- Motile
- Has single polar flagellum
- Non-spore forming
- **Oxidative (+) ve**
- Aerobic/facultative anaerobic
- Halophilic and some are non - halophilic

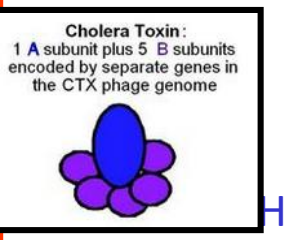
Pathogenesis

- IBP - few hrs to 5 days.
- **Large number of organisms** need for infection.
- Pathogenic only for humans.

Ingestion of organisms via contaminated water / food (from feces of patients/carriers)

Attach to microvilli of brush border epithelial cells on the mucosal surface of small intestine (Non-invasive)

Organisms multiply & produce toxins



Enterotoxin
(**Cholera toxin**)
Have A & B sub units

B binds to GM1 ganglioside receptors

This facilitate entry of A
Result in **activation of adenylate cyclase**

Increase intracellular Cyclic AMP

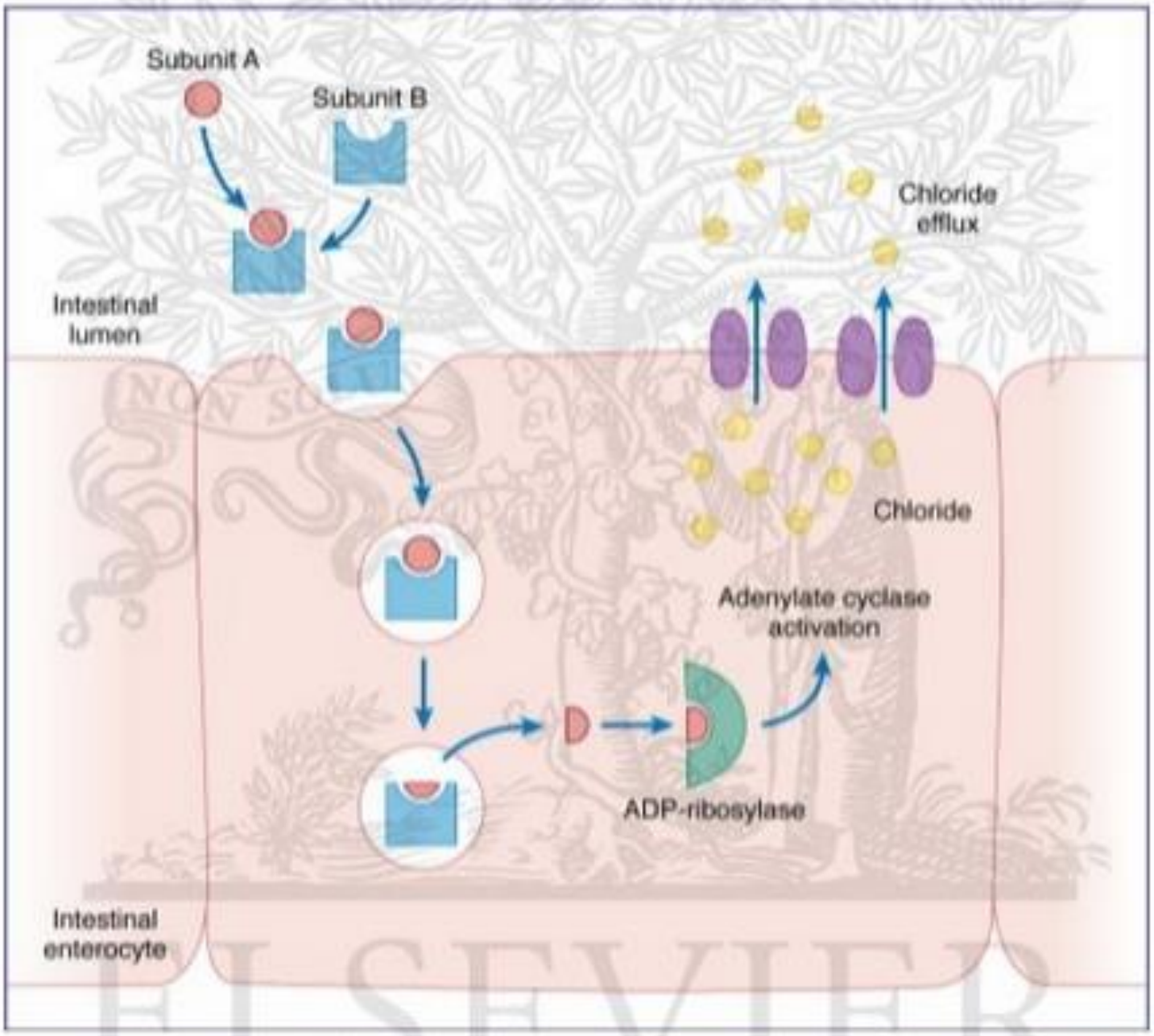
Zonula Occludens Toxins

modify intracellular tight junctions of epithelial cells

Increase Permeability

Hypersecretion of salt & water from GUT lumen.

Watery diarrhoae


Subunit A
Subunit B
Intestinal lumen
Chloride efflux
Chloride
Adenylate cyclase activation
ADP-ribosylase
Intestinal enterocyte

Clinical features

- LOA, abdominal discomfort
- Diarrhoae

-Range from asymptomatic/mild diarrhoae – life threatening

profuse watery diarrhoae leading to **circulatory collapse**

- Case fatality rate – 50% in untreated severe cases
- But less that 1% with proper & timely M_X
- Colorless stool with flecks of mucus

(Rice water stool)

- Effortless passing of large volumes of fluid (>1L/hr)
- Vomiting
- Muscle cramps

Lab diagnosis

Specimens

- **Stool**

  Collect directly to **screwed capped wide-mouthed** bottle

- **Rectal swabs**

  Specially from small children

Transport

- As soon as possible
- If delay >6 hrs, use a transport media

  e.g:

  Alkaline peptone water

Ix

Microscopy

- Gram stain - Gram (-) ve rods.
  - Motility testing

Culture

- On **alkaline peptone** water
- Subculture to **TCBS.** (Thiosulphate Citrate Bile Sucrose agar)
  - Typical **sucrose fermentation** indicate as yellow

Rx

- Maintain fluid & electrolyte balance with ORS/IV fluids.
- Antibiotics - decrease period of excretion of *V. cholerae*

  -Tetracycline

  -Chloramphenicol

  -Co-trimoxazole

Cholera vaccine

- Inactivated oral cholerae vaccines

Vibrio parahaemoliticus

- Halophilic
- Produce non-sucrose fermenting colonies in TCBS medium
- IBP-12-24 hours
- Produce enterotoxin similar to toxin of cholera /*E. coli*
- Cause **food poisoning** associated with seafood

Clinical features

- Explosive watery diarrhoea

  May have blood in stool

- Cramping abdominal pain

- Fever, chills, headache

Campylobacter

Campylobacter

- A cause of acute infective diarrhoae

Features

-Gram (-) ve

-Has single flagellum at one or both poles.

-Arrange in S or M shapes.

-Rapidly motile.

Campylobacter

- Culture -Microaerophilic

-Non-spore forming

-Grow on selective media

-Colonies are,

Colourless or gray

Typically flat

Diffuse

Important spp

Organism	Reservoir	Human disease
C. jejuni	Birds	Diarrhoae
C. coli	Pigs	Diarrhoae
C. fetus	Cattle/sheep	Septicemia in immuno-compromised patients.

Pathogenesis.

Ingestion of organisms via contaminated food/water.

↓

Colonized in jejunum / ileum / colon / rectum.

↓

Organisms multiply & **invade** the epithelium.

↓

Inflammation & ulceration.

↓

Occasionally invade the blood stream.

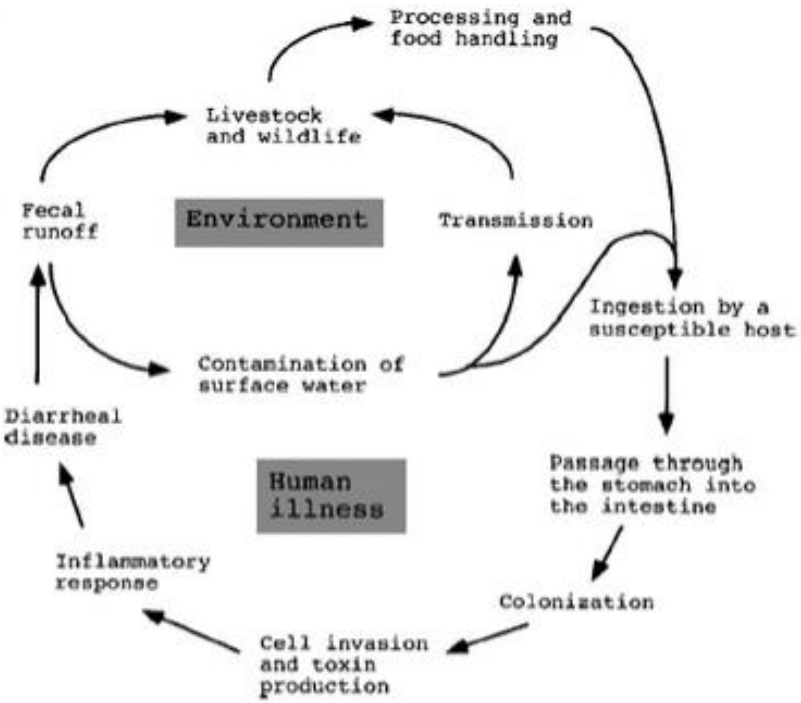


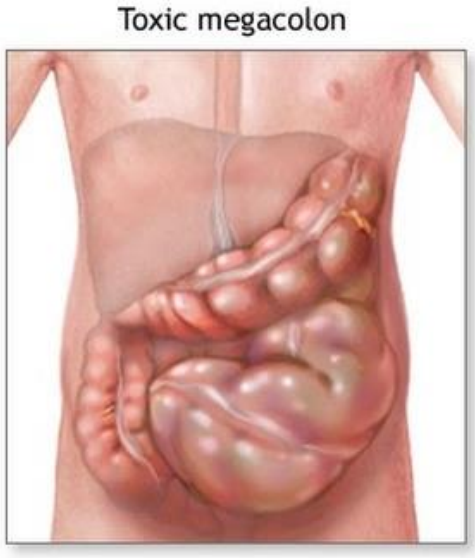
Figure 1. *C. jejuni* infections are commonly acquired by handling and consuming undercooked chicken, and drinking unpasteurized milk and polluted water. Human illness with *C. jejuni* ranges from mild to severe diarrheal disease, the latter of which is characterized by the presence of blood and leukocytes in stool specimens.

Clinical features.

- IBP- 1-7 days
- Fever, malaise, headache
- Abdominal pain
- Profuse, **bloody diarrhoae**

Complications

- Intestinal haemorrage
- Toxic megacolon
- HUS
- Reactive arthritis
- Peripheral neuropathy



Lab diagnosis.

- Specimen – stool
- **Ix**

Microscopy

Gram stain

- S or M shaped rods.

Motility

Culture

- At **42 ^{0}c** in **microaerophilic** conditions.

Serology

-Useful in patients with

Arthritis

Peripheral neuropathy

-Can use

CFT

ELISA

Rx

- Supportive measures - rehydration
- Antibiotics – only for severe & complicated infections
- Erythromycin
  - effective
  - start early stage
  - reduces fecal shedding of organisms
- Ciprofloxacin
  - for resistant cases

Prevention

- Prevention of infection in chicken
- Proper purification of water
- Pasteurization of milk
- HE

Helicobacter

Association of peptic ulcers & *Helicobacter pylori*

- *H. pylori* was discovered in 1982 by Marshall and Warren

- **Major contributor to peptic ulcer disease**

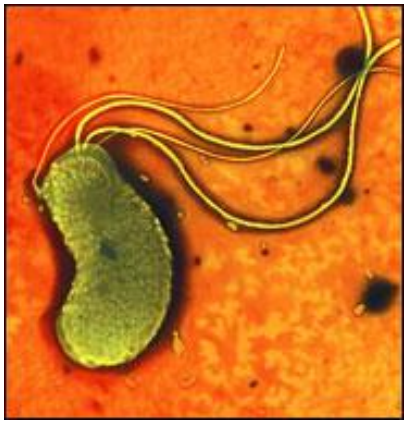
- 90% duodenal ulcers & 50 - 80% benign gastric ulcers are associated with ***H. pylori***

H. pylori in Sri Lanka

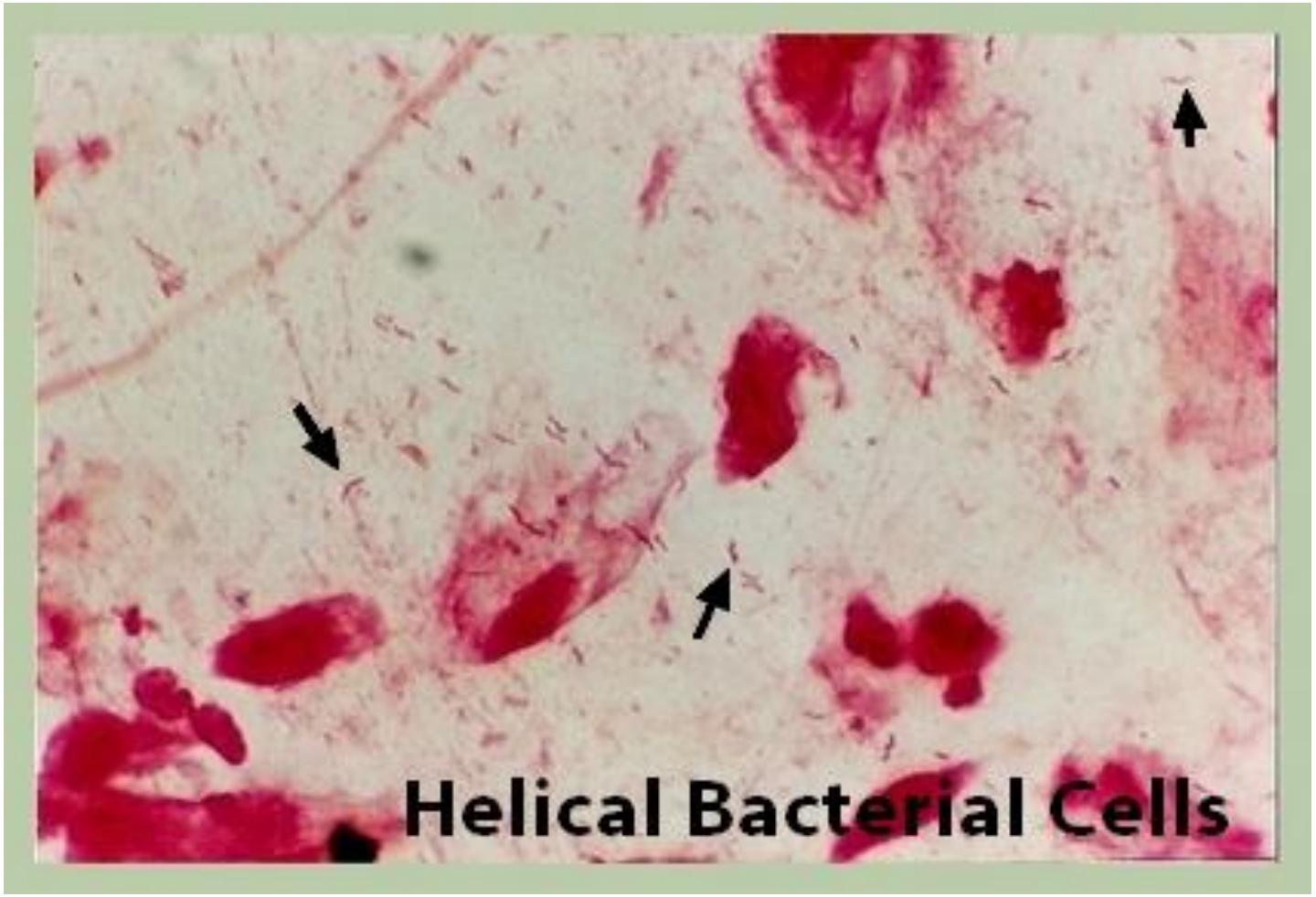
- First reported in Sri Lanka in 1992 in **67%** of adult patients with **duodenitis** and 8**%** of adults with **non-ulcer dyspepsia**

  Fernando D. *Cey Med J* **1992**; **37**(1): 15-7.

H. pylori



- Small Gram-negative bacilli
- Comma / “s” shaped
- Highly motile
- Grows in **microaerophilic conditions** with high humidity
- Has a **strong urease activity**
- Catalase and **oxidase positive**
- Found in the gastric mucous layer or adherent to the epithelial lining of the stomach, duodenum , oesophagus


Helical Bacterial Cells

Transmission

- Possible environmental reservoirs include contaminated water sources

- spread person-to-person through fecal-oral routes

- Iatrogenic spread through contaminated endoscopes has been documented

Pathogenesis

- Gastric juice is a potent combination of dietary enzymes and hydrochloric acid

Pathogenesis

- Once inside the mucosa,

Organism breaks down urea (urea hydrolysis)

↓

produce bicarbonate and ammonia

↓

neutralize stomach acid

↓

create a protective cloud

↓

H. pylori grows well in a reduced oxygen environment (microaerophilic)

↓

Survive and proliferate in the gastric mucosa

In addition,

- Adhesins - assist in attachment

- Spiral shape & rapid movement of the organism resist the peristalsis of the digestive tract

H. pylori weakens the protective mucous coating of the stomach and duodenum

↓

Allows acid that is normally in the stomach to get through to the lining beneath

↓

Both the acid and the bacteria irritate the lining and cause ulcers

Pathogenesis ---

▶ Immune response to organism results in inflammation of the stomach lining causes,

Accumulation white blood cells

WBC not penetrate the mucous lining

↓

remain in the area

↓

die & spill superoxide radicals

↓

Further damage the mucosa

↓

Nutrients released from dying cells facilitate the growth of bacteria

↓

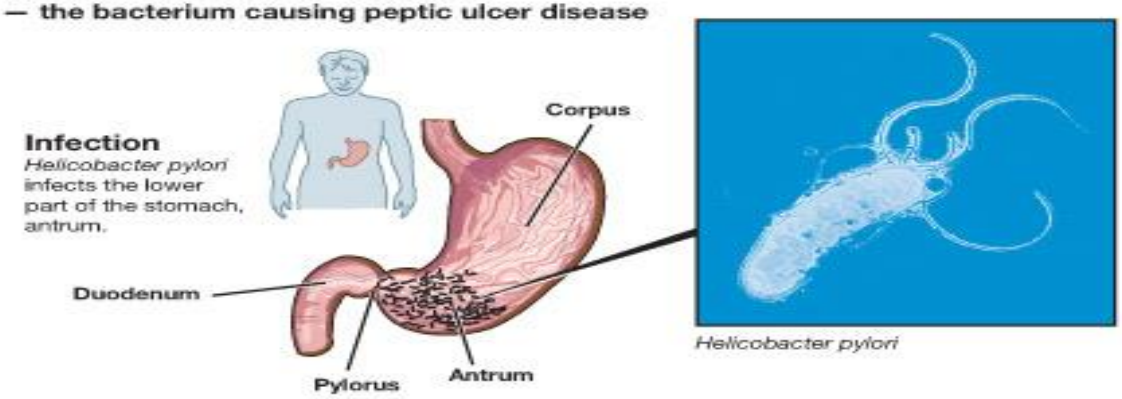
Gastritis (chronic active, chronic persistent, atrophic gastritis)

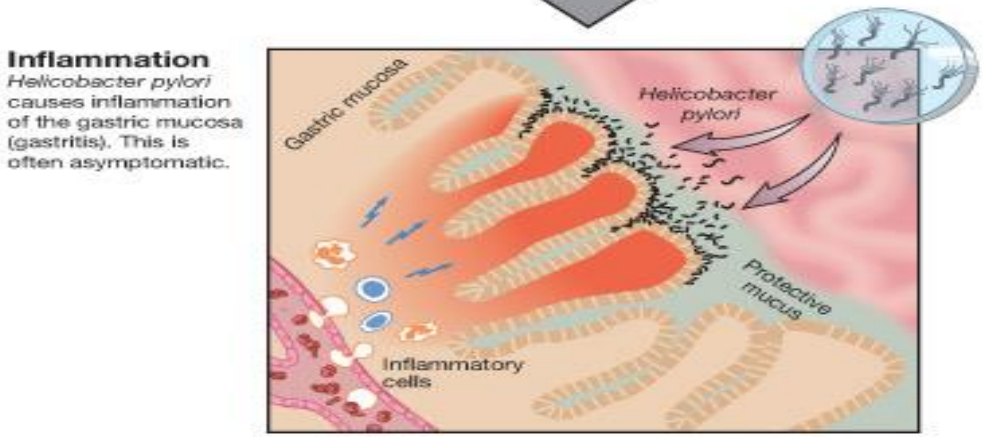
↓

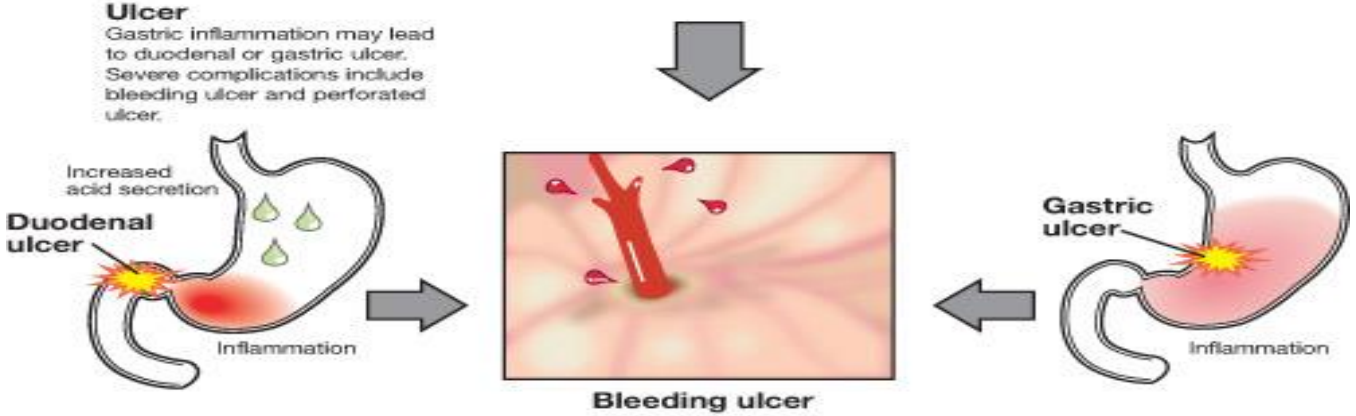
Progresses to peptic ulcer

Helicobacter pylori

— the bacterium causing peptic ulcer disease

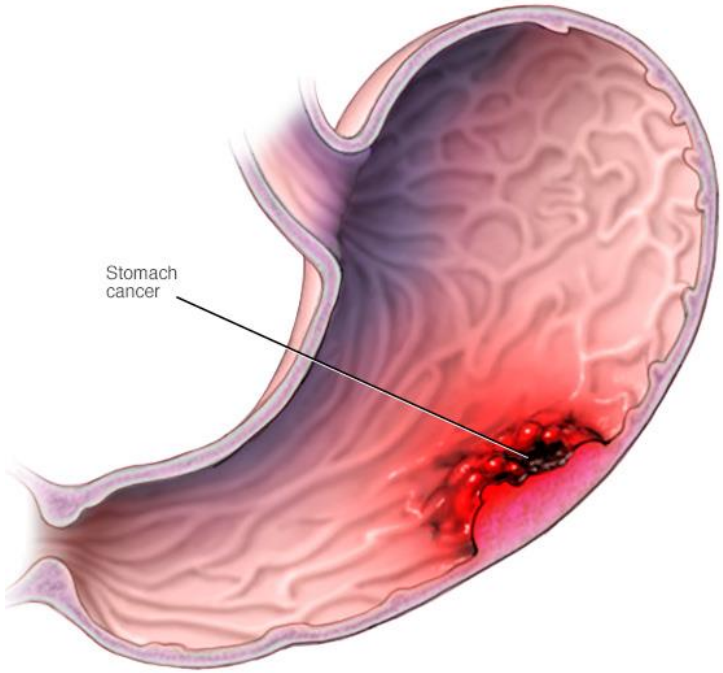






Increase risk of developing

- Gastric cancer (second most common cancer worldwide)



- Mucosal-Associated-Lymphoid-Type (MALT) lymphoma

Diagnosis

Detection of *H. pylori* infection is essential in all patients with peptic ulcers.

- Invasive - Endoscopy and biopsy
  - * Histopathology
  - * Culture
  - * Rapid urease test

Tiny gastric biopsy removed from the forceps. The biopsy can be tested for urease enzyme to detect the HP

Rapid urease test

- Endoscope diagnostic test of choice
  - Presence of *H. pylori* in gastric mucosal biopsy specimens is detected by testing for the bacterial product urease.
- kits are commercially available (ie, **CLOtest**, Hp-fast, Pyloritek)
- Each contain a combination of a urea substrate and a pH sensitive indicator
  - If *H. pylori* is present, bacterial urease converts urea into ammonia
- Changes pH and produces a color change

CLO test

- The CLOtest® is a test kit produced to detect presence of *H. pylori* .

- A piece of gastric mucosa is placed in a small well.

- The presence of urease is a surrogate measure of the presence of the organism and results in a colour change from yellow to magenta

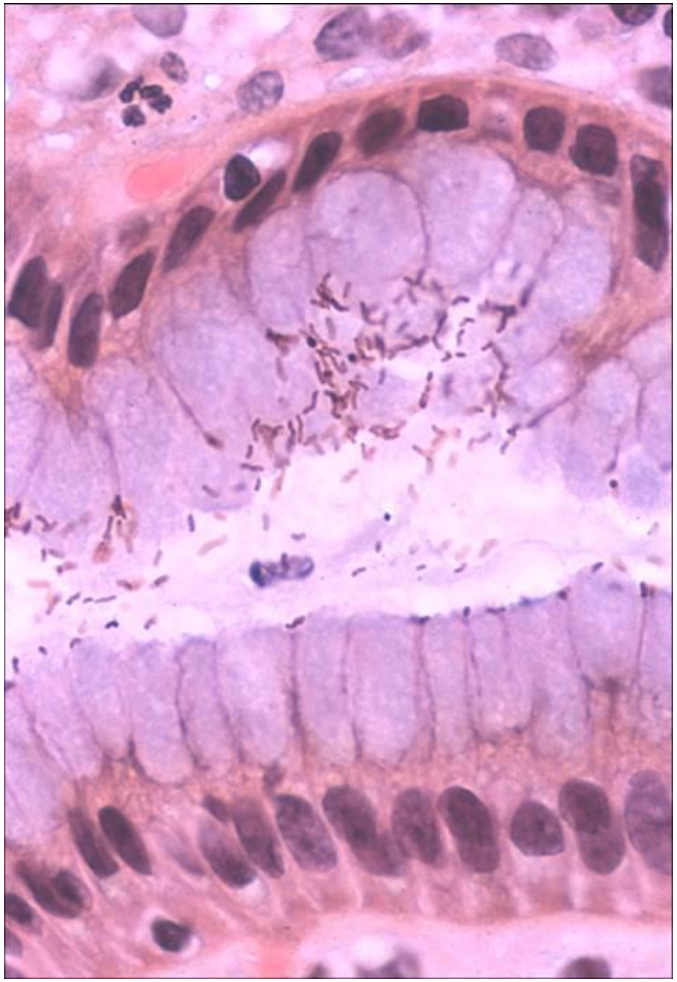
(CLO = Campylobacter like organism)


CLOtest*
Rapid Urease Test
= Positive
= Negative
imberly-Cla
CLOtest*
apid Urease Test
= Positive
= Negative
imberly-Clar

Histopathology

- Considered to establish a diagnosis of *H. pylori* infection, if the rapid urease test result is negative and a high suspicion for *H. pylori* persists

- Histology sections are stained with Gram stain /Giemsa / IF



Molecular biology tests

- PCR
- DNA probes

Culture of biopsy specimens for *H. pylori*

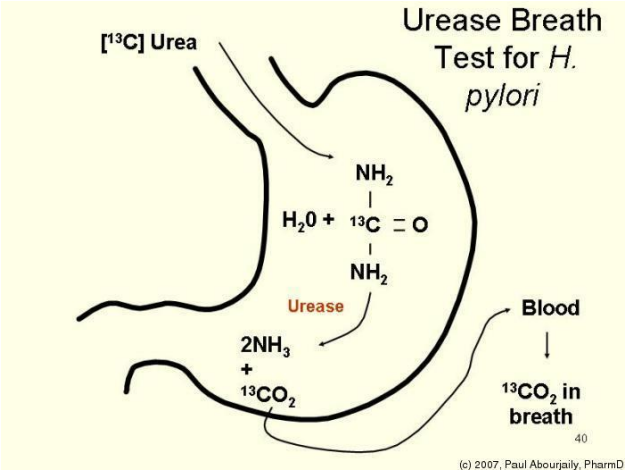
- Confirmatory
- Need when antimicrobial susceptibility testing is desired
- Requires an experienced laboratory
- Not available routinely for clinical use

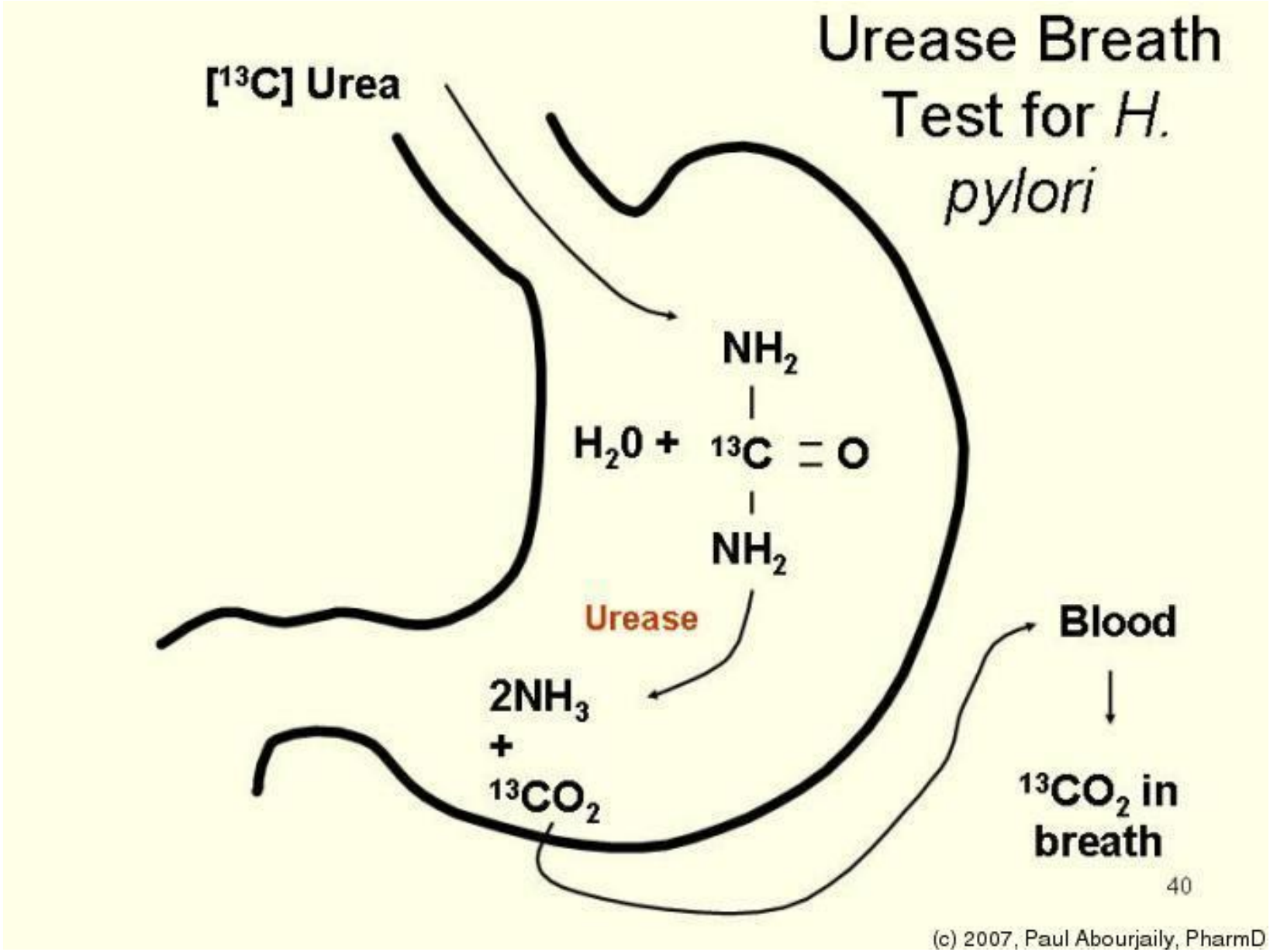
Non invasive tests

- Urease breath test
- Serology
- *H. pylori* Stool Ag test

Urease breath test

- Detect active *H. pylori* infection by testing for the enzymatic activity of bacterial urease
- Patient is given a solution containing urea attached to carbon 13 (^{13}C) or carbon 14 (^{14}C) to drink after fast
- Urease produced by *H. pylori* metabolizes the urea
- Release labeled CO_2 into stomach
- Absorbed into the bloodstream
- Diffused into the lungs, and exhaled
- Measured as CO_2 when the patient breath into a test apparatus





Serology

- High titer of IgG in colonized persons
- IgM present in recent colonization or recurrence

H. pylori Stool Ag test

- A test may be used to detect *H. pylori* Ag in the patient‘s stool (HpSA test).

Treatment

- Appropriate antibiotic regimens can successfully eradicate the infection in most patients and lowers ulcer recurrence

- Cure rates range from 70-90%

- Consists of a combination of 2-3 antibiotics

- A longer duration of treatment (14 d) - more effective