LECTURE II

The causative agents of gastro-intestinal bacterial infections (genus of *Escherichia, Shigella, Salmonella, Vibrio, Campilobacter, Helicobacter*)

Lecture plan:

• Bacteria that cause gastrointestinal infections:

- Genus of *Escherichia*, morpho-biological characteristics of Escherichia, pathogenic factors, diseases they cause, antibiotic-resistant forms, microbiological diagnosis, specific treatment and prevention.

- Genus of *Shigella*, morpho-biological characteristics, pathogenic factors, diseases they cause, antibiotic-resistant forms, microbiological diagnosis, specific treatment and prevention.

- Classification of pathogenic bacteria belonging to the genus of *Salmonella*, morpho-biological characteristics, pathogenic factors, pathogenesis of diseases caused by these bacteria, the main clinical signs, microbiological diagnosis, specific principles of treatment and prevention. Broad-spectrum beta-lactamase-resistant bacteria.

• *Vibrios*. Classification. Cholera vibrio, morpho-biological characteristics. Biovar: classic plague vibrio and El-Tor. Pathogenesis of cholera, main clinical signs, microbiological diagnosis, specific principles of treatment and prevention

• *Campylobacteria*, their morpho-biological characteristics. Pathogenic factors. Diseases caused by. Pathogenesis, main clinical signs and microbiological diagnosis

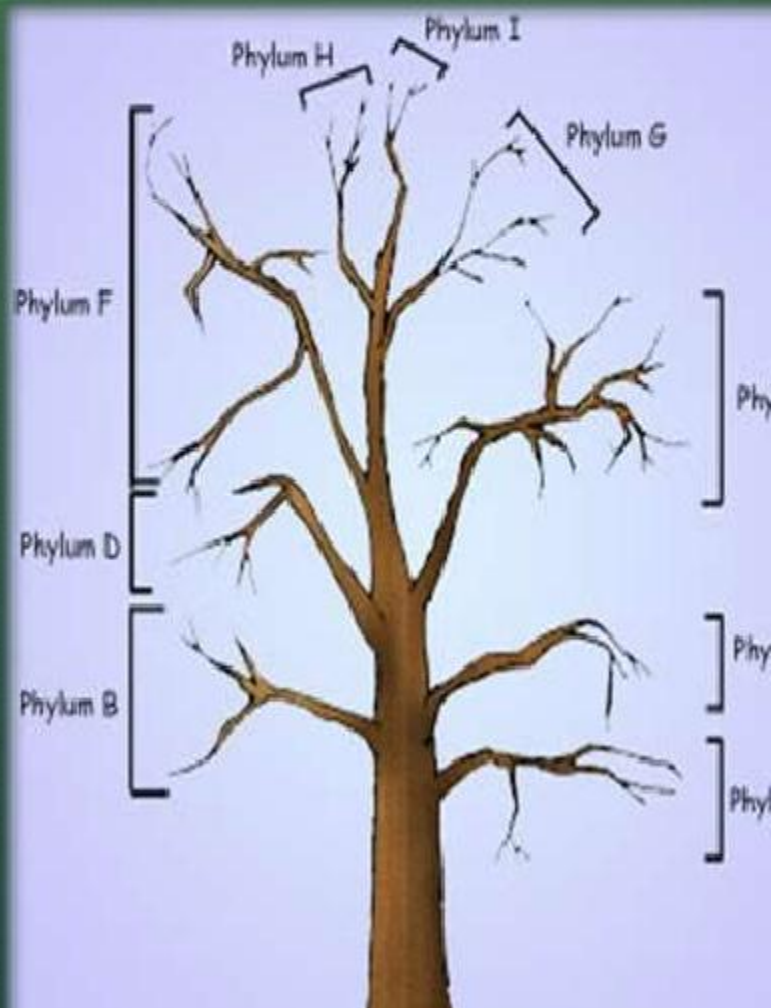
• *Helicobacter pylori*, their morpho-biological characteristics. Pathogenic factors. Diseases caused by. Pathogenesis, main clinical signs and microbiological diagnosis

Etiology of Acute Intestinal Diseases

- **bacterial agents**
  - *Escherichia coli*
  - *Shigella,*
  - *Salmonella,*
  - *Campylobacter jejuni*
  - *Yersinia enterocolitica.*
  - *Clostridium difficile*
  - *Vibrio cholerae*
- **enteroviruses (infectio enteroviralis)**
  - Rotavirus
  - Coxsackie viruses
  - ECHO (Enteric Cytopathogenic Human Orphan) viruses
  - Astrovirus
    Parvovirus
- **Parasites**
  - *Giardia lamblia*
  - *Cryptosporidium*

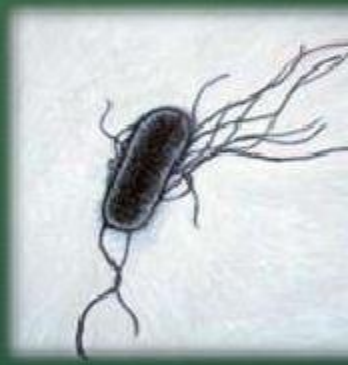
Classification

- Domain: Bacteria
- Kingdom: Bacteria
- Phylum: Proteobacteria
- Class: Gamma Proteobacteria
- Order: Enterobacteriales
- Family: Enterobacteriaceae
- Genus: Escherichia
- Species: Escherichia coli (E. coli)

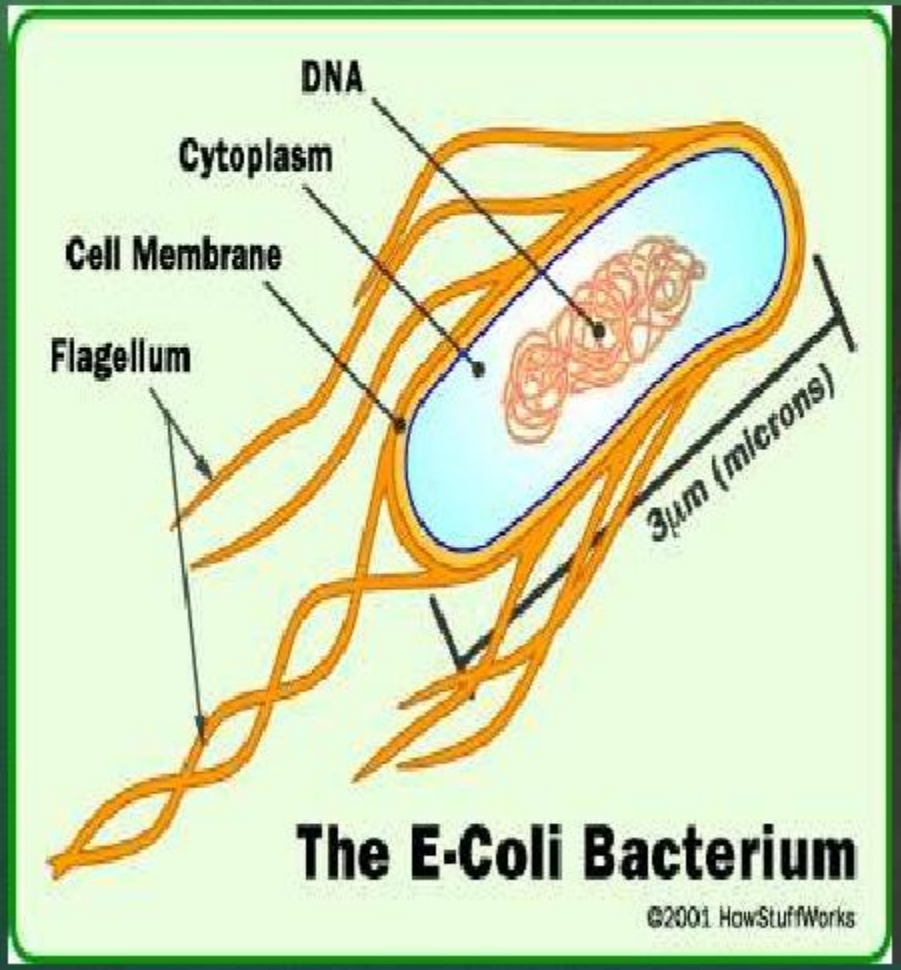


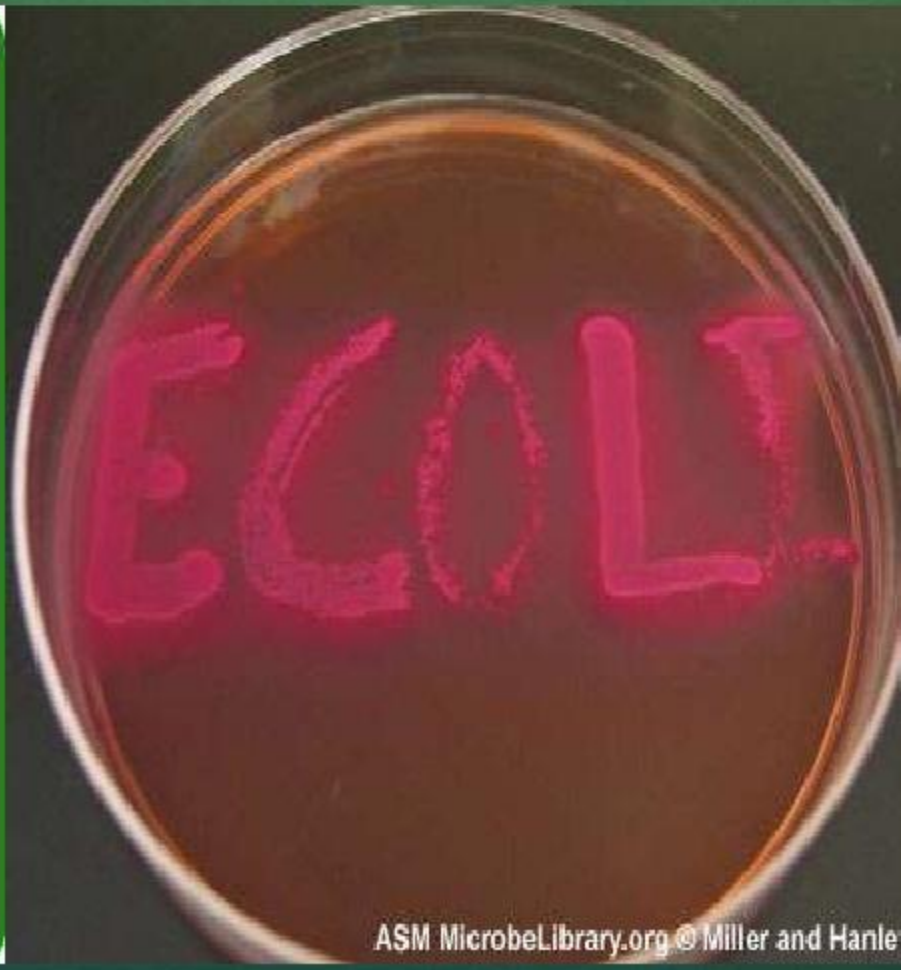
E.coli

- Morphology Gram - ve Straight rods,
- 1-3 X 0.4 -0.7 microns,
- Appear in singles or in pairs,
- Motile by peritrichate flagella.
- Very few strains non motile
- Not spore forming, Non acid fast.



E.coli


DNA
Cytoplasm
Cell Membrane
Flagellum
3µm (microns)
The E-Coli Bacterium
©2001 HowStuffWorks


E.COLI
ASM MicrobeLibrary.org © Miller and Hanle

Identification of *Enterobacteriaceae* Biochemical reactions

- Oxidase test
  - **All members of *Enterobacteriaceae* are oxidase negative**
  - ***Pseudomonas* is oxidase positive**
- O/F test
  - **All members of *Enterobacteriaceae* are O+/F+**
  - ***Pseudomonas* is O+/F-**
- Nitrate reductase
  - All members of Enterobacteriaceae are nitrate reductase positive
  - *Pseudomonas* is nitrate reductase negative

E.coli
Biochemical Characters,

Glucose,Lactose,Mannitol,Maltose

fermented. with A/G

I,M,Vi,C tests.

Indole +

Methyl Red +

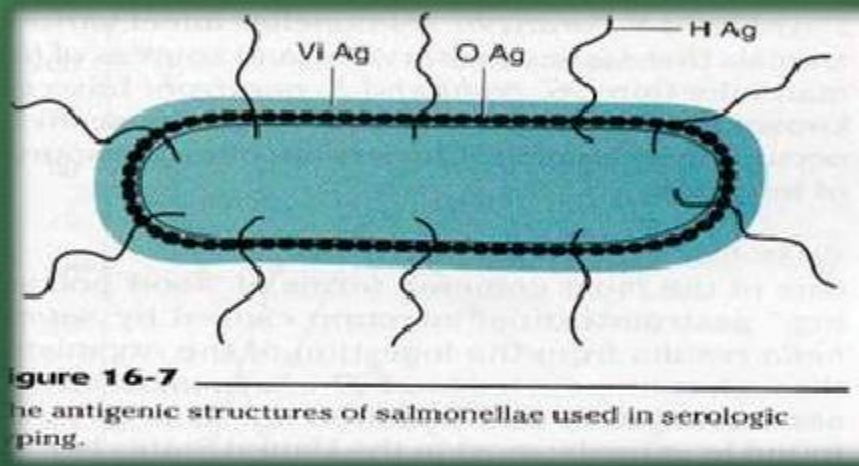
Voges Proskauer – ve I,M,Vi,C tests.

Citrate –ve

Urease not produced.

E.coli
Antigenic Structure

- Somatic 0 170
- Capsular K 100
- Flagella H 75
- Virulence factors



igure 16-7
he antigenic structures of salmonellae used in serologic
ping.

Surface Antigens Toxins

O Endotoxic activity

K protects against the phagocytosis

Fimbriae promote virulence (important in UTI)

Transmission

The way of transmission

- Contact
- Alimentary (by water, milk,
- food)

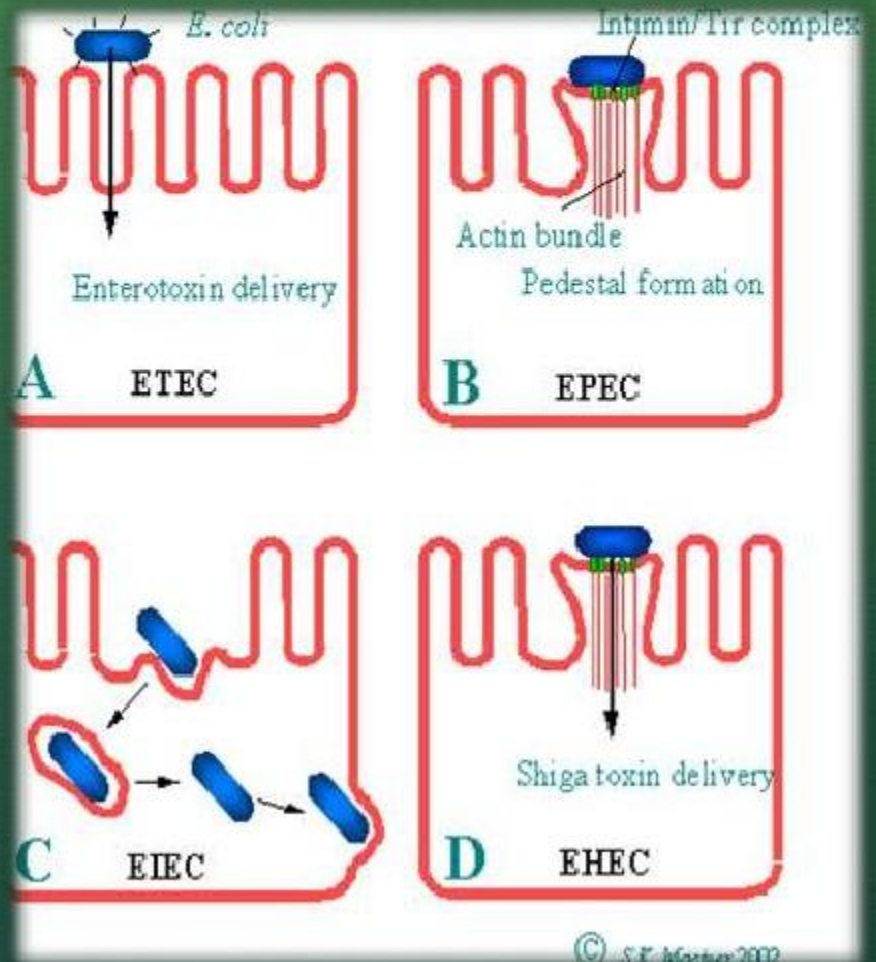
Toxins and E.coli

- E.coli produce Exotoxins
- Hemolysins, Enterotoxins causes Diarrheas,
- Important toxins produces.
- Heat labile HL Heat stable HS

Vero toxins VT Like Shigella toxins

Mechanism of action of Toxins

- Increased cAMP alters the activity of sodium and chloride transporters producing an ion imbalance that results in fluid transport into the bowel



Classification of E.coli

1. Enteropathogenic EPEC
2. Enterotoxigenic ETEC
3. Enteroinvasive EIEC
4. Enterohemorrhagic EHEC
5. Enteroaggresive EAEC

Enteropathogenic E.coli

- Causes diarrheal disease in children,
- EPEC O26/O11
- Produce Verocytotoxin
- Infantile enteritis, Involves upper part of Intestine
- Brush border of the intestine is lost
- Intimacin – EPEC adhesion factor.
- Frequent in summer months
- Poor hygiene predisposes.

Enterotoxigenic E.coli

- Causes travelers diarrhea
- Water contaminated with Human and Animal feces predisposes.
- Laboratory Diagnosis

Demonstration of Enterotoxin LT and ST

Tissue culture tests,

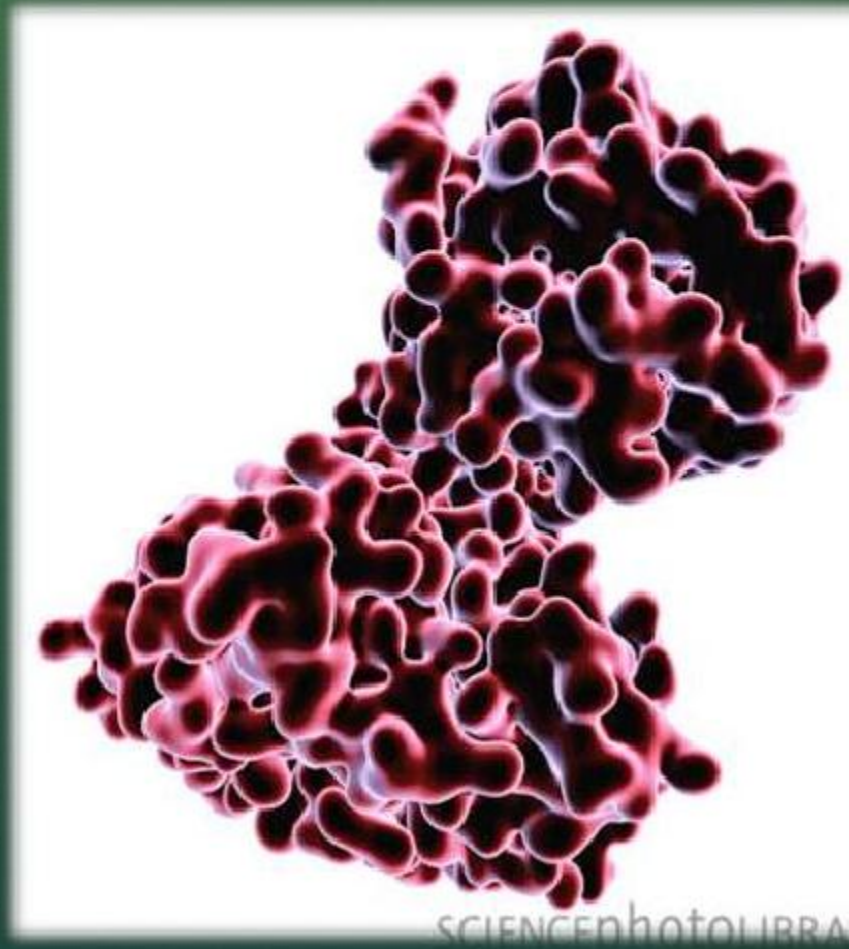
ELISA

Passive agglutination tests.

Animal experiments in Rabbit ileal loop test.

Entero invasive E.coli

- Some are non motile strains,
- Atypical resembles like Shigella.
- Clinically mild diarrhea
- Sereny test positive animal Rabbit.
- ELISA



EHEC (contd)

- Culture
- DNA detection methods.
- Cytotoxic effects on Vero cells.
- Detection with monovalent sera O157/H7

Enterohemorrhagic bacteria Escherichia coli (EHEC)

Most Escherichia coli (E.coli) strains are harmless.

But some, like enterohemorrhagic **E. coli (EHEC)**, are a hazard to human health and life.

Incubation period:

three to eight days

E. coli (EHEC), once in th human stomach, begins p ing toxins that cause serio illnesses

Symptoms caused by E. coli (EHEC)

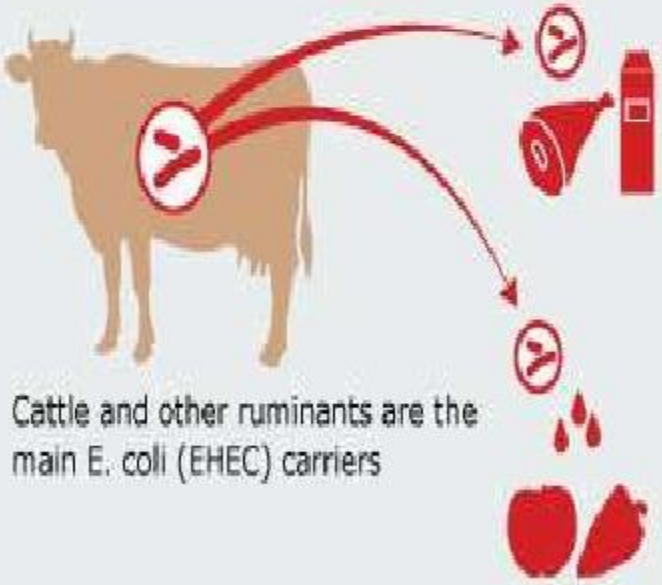
- Stomach muscle spasm
- Diarrhea (sometimes bl diarrhea)
- Fever
- Vomiting

Complications:

hemolytic uremic syndrom

Death rate: 3-5 %

INFECTION SOURCES



Cattle and other ruminants are the main E. coli (EHEC) carriers

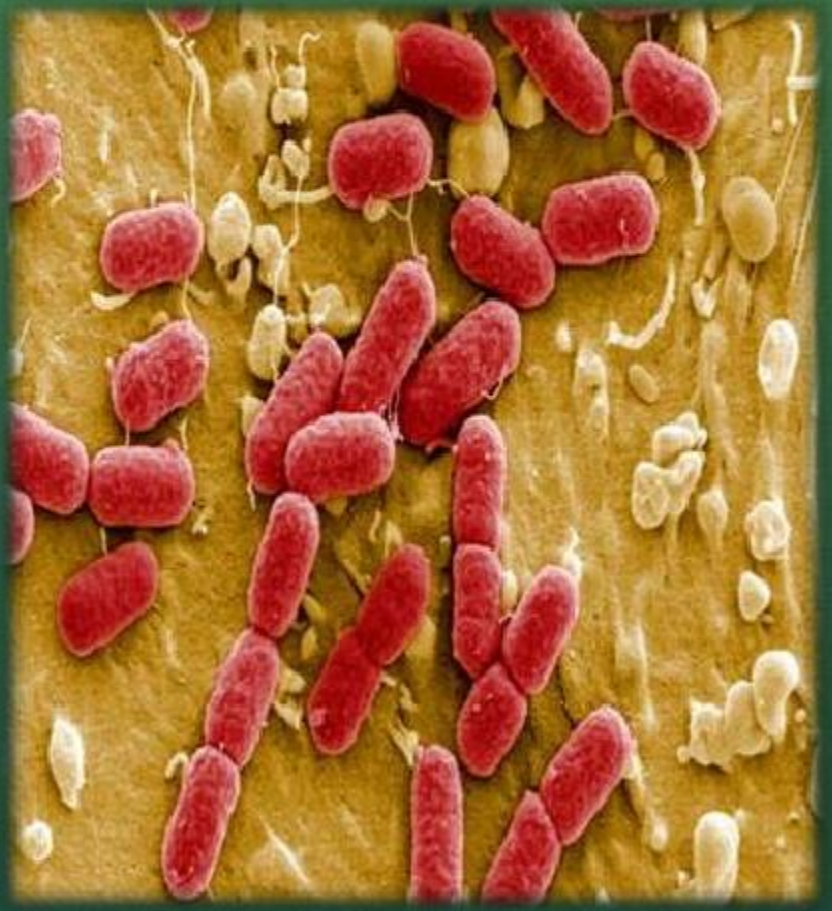
Uncooked meat and raw milk

The bacteria die when food is exposed to heat (70°C and higher)

Fruit and vegetables (droppings of sick animals find their way into water bodies that in turn feed the soil)

Enteroaggresive E.coli EAEC

- Can cause Diarrhea Detect by Culture methods
- Brick-like aggregates on cell surfaces
- Mucus biofilm inhibits fluid absorption
- Diarrhea
- Detection of Enterotoxin



Culturing for E.coli

- Mid stream sample/semi quantitative culturing (Kass et al) >_ 1.00,000/ml of urine. (significant Bacteriuria)
- Urine should not be kept in wards for > 2 hours and to be preserved at 4 c
- Culture by standard loop method.
- Fixed volume cultured on MacConkey agar Lactose fermenters I M Vi C
- Antibiotic sensitivity tested

Gastroenteritis Caused by *Escherichia coli*

Organism	Site of Action	Disease	Pathogenesis	Diagnosis
ETEC	Small intestine	Traveler's diarrhea; infant diarrhea in developing countries; watery diarrhea, vomiting, cramps, nausea, low-grade fever	Plasmid-mediated, ST and LT enterotoxins that stimulate hypersecretion of fluids and electrolytes	Most U.S. outbreaks caused by ST-producing strains; commercial immunoassays available for detecting ST in clinical specimens and cultures; PCR assays used with clinical specimens
EPEC	Small intestine	Infant diarrhea in developing countries; watery diarrhea and vomiting, nonbloody stools; believed to be rare in United States	Plasmid-mediated A/E histopathology, with disruption of normal microvillus structure resulting in malabsorption and diarrhea	Characteristic adherence to HEp-2 or HeLa cells; probes and amplification assays developed for the plasmid-encoded bundle-forming pili and gene targets on the "locus of enterocyte effacement" pathogenicity island
EAEC	Small intestine	Infant diarrhea in developing and probably developed countries; traveler's diarrhea; persistent watery diarrhea with vomiting, dehydration, and low-grade fever	Plasmid-mediated aggregative adherence of rods ("stacked bricks") with shortening of microvilli, mononuclear infiltration, and hemorrhage; decreased fluid absorption	Characteristic adherence to HEp-2 cells; DNA probe and amplification assays developed for conserved plasmid
STEC	Large intestine	Initial watery diarrhea followed by grossly bloody diarrhea (hemorrhagic colitis) with abdominal cramps; little or no fever; may progress to hemolytic uremic syndrome	STEC evolved from EPEC; A/E lesions with destruction of intestinal microvilli, resulting in decreased absorption; pathology mediated by cytotoxic Shiga toxins (Stx1, Stx2), which disrupt protein synthesis	Screen for O157:H7 with sorbitol-MacConkey agar; confirm by serotyping; immunoassays (ELISA, latex agglutination) for detection of the Stx toxins in stool specimens and cultured bacteria; DNA amplification assays developed for Stx genes
EIEC	Large intestine	Rare in developing and developed countries; fever, cramping, watery diarrhea; may progress to dysentery with scant bloody stools	Plasmid-mediated invasion and destruction of epithelial cells lining colon	Sereny (guinea pig keratoconjunctivitis) test; plaque assay in HeLa cells; probes and amplification assays for genes regulating invasion (cannot discriminate between EIEC and *Shigella*)

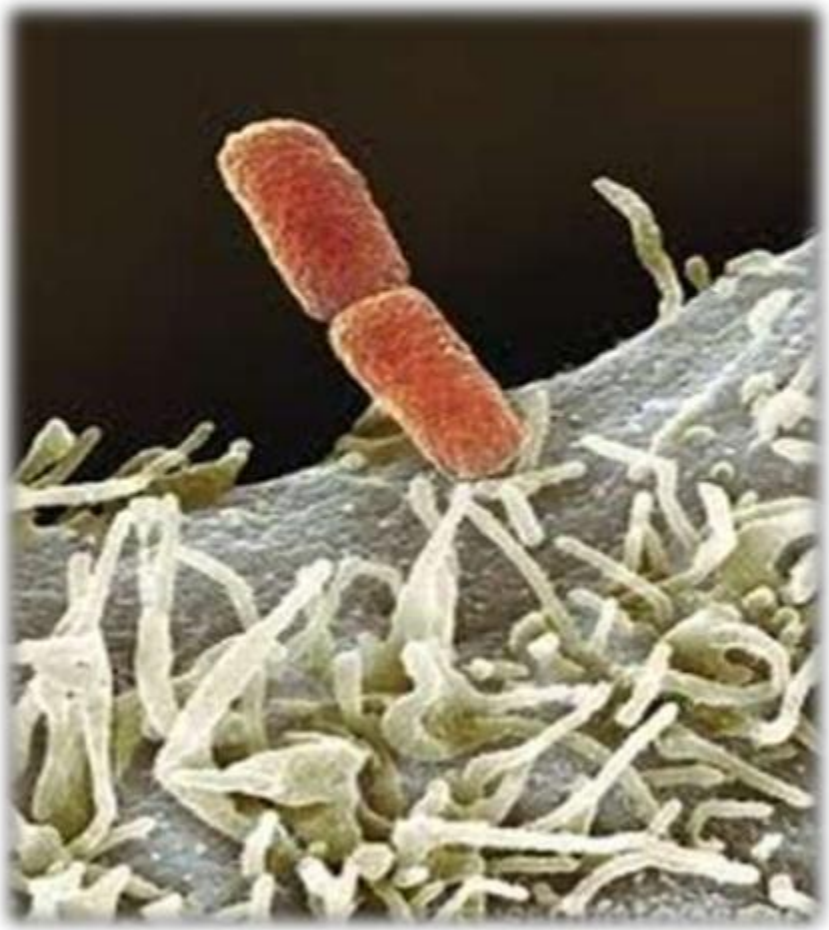
A/E, Attachment/effacement; *DNA*, deoxyribonucleic acid; *EAEC*, enteroaggregative *E. coli*; *EIEC*, enteroinvasive *E. coli*; *ELISA*, enzyme-linked immunosorbent assay; *EPEC*, enteropathogenic *E. coli*; *ETEC*, enterotoxigenic *E. coli*; *LT*, labile toxin; *PCR*, polymerase chain reaction; *ST*, stable toxin; *STEC*, Shiga toxin–producing *E. coli*.

Shigella a Highly Infectious Bacteria

- *Shigella* is one of the most infectious of bacteria and ingestion of as few as **100-200 organisms** will cause disease.
- Most individuals are infected with shigellae when they ingest food or water contaminated with human fecal material.
- Shigella can survive up to 30 days in milk, eggs, cheese or shrimps.

MORPHOLOGY AND STAINING:

- Short rods
- - Non-encapsulated
- - Non-motile
- - Non-spore former
- - Gram-negative



Classification of Shigella Infection

I. **Clinical Form**

- With dominance of toxicosis
- with dominance of local inflammation

II. **Severity** (mild, moderate and severe)

III. **Course**

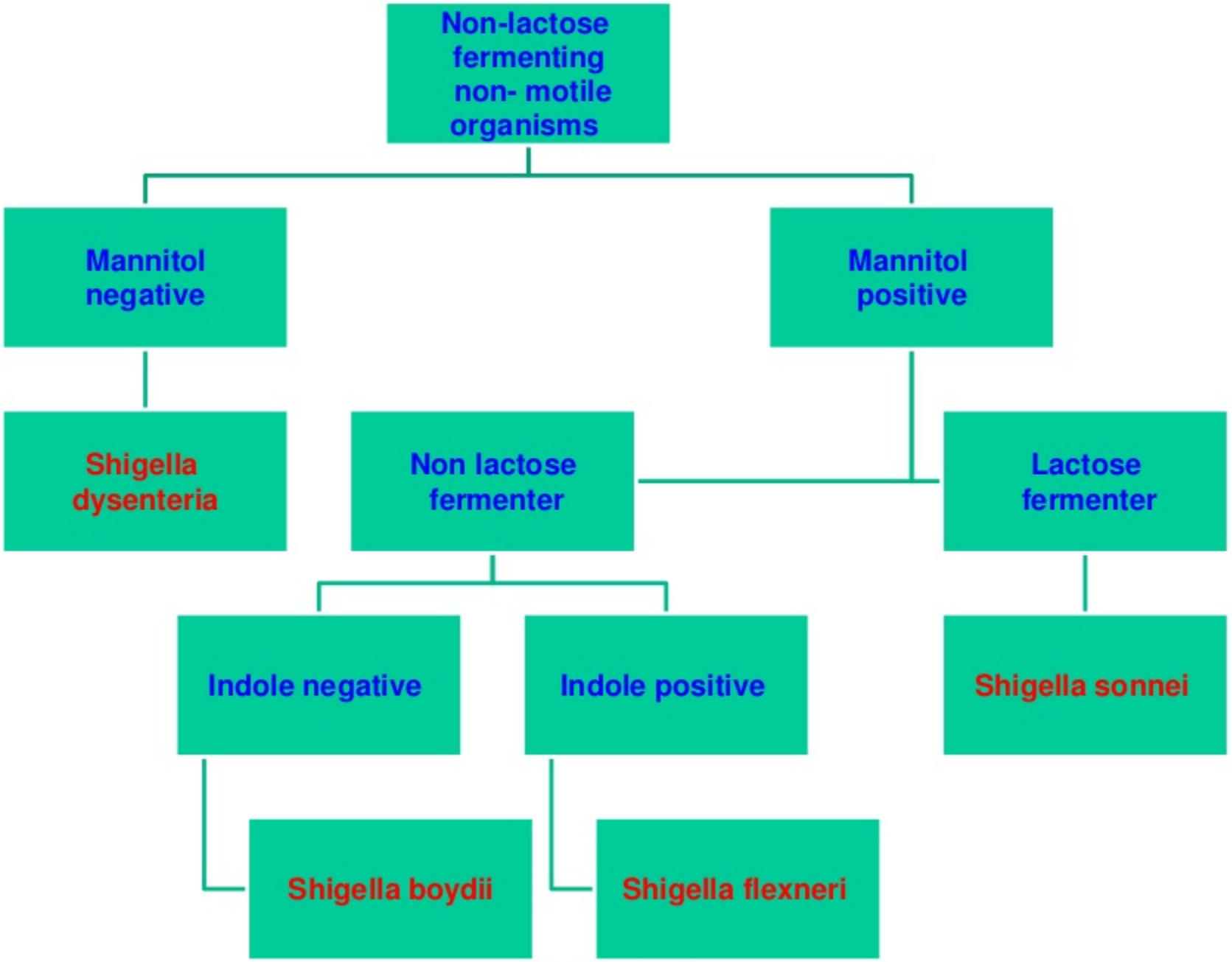
- acute (up to 1.5 mo)
- subacute (up to 3 mo)
- chronic (about 3 mo)
  - recurrent
  - constantly recurring

IV. **Complicated or uncomplicated**

V. **Bacterium carrying**

CLASSIFICATION
on Basis of Mannitol Fermentation:

- 1. Non-mannitol-fermenters
- *Shigella dysenteria*
- 2. Mannitol-fermenters
- *Shigella flexneri*
- *Shigella boydii*
- *Shigella sonnei*


Non-lactose fermenting non- motile organisms
Mannitol negative
Mannitol positive
Shigella dysenteria
Non lactose fermenter
Lactose fermenter
Indole negative
Indole positive
Shigella sonnei
Shigella boydii
Shigella flexneri

Transmission

Shigella is spread through **fecal-oral mechanism** of transmission.

The way of transmission

- Contact
- Alimentary
- Watery

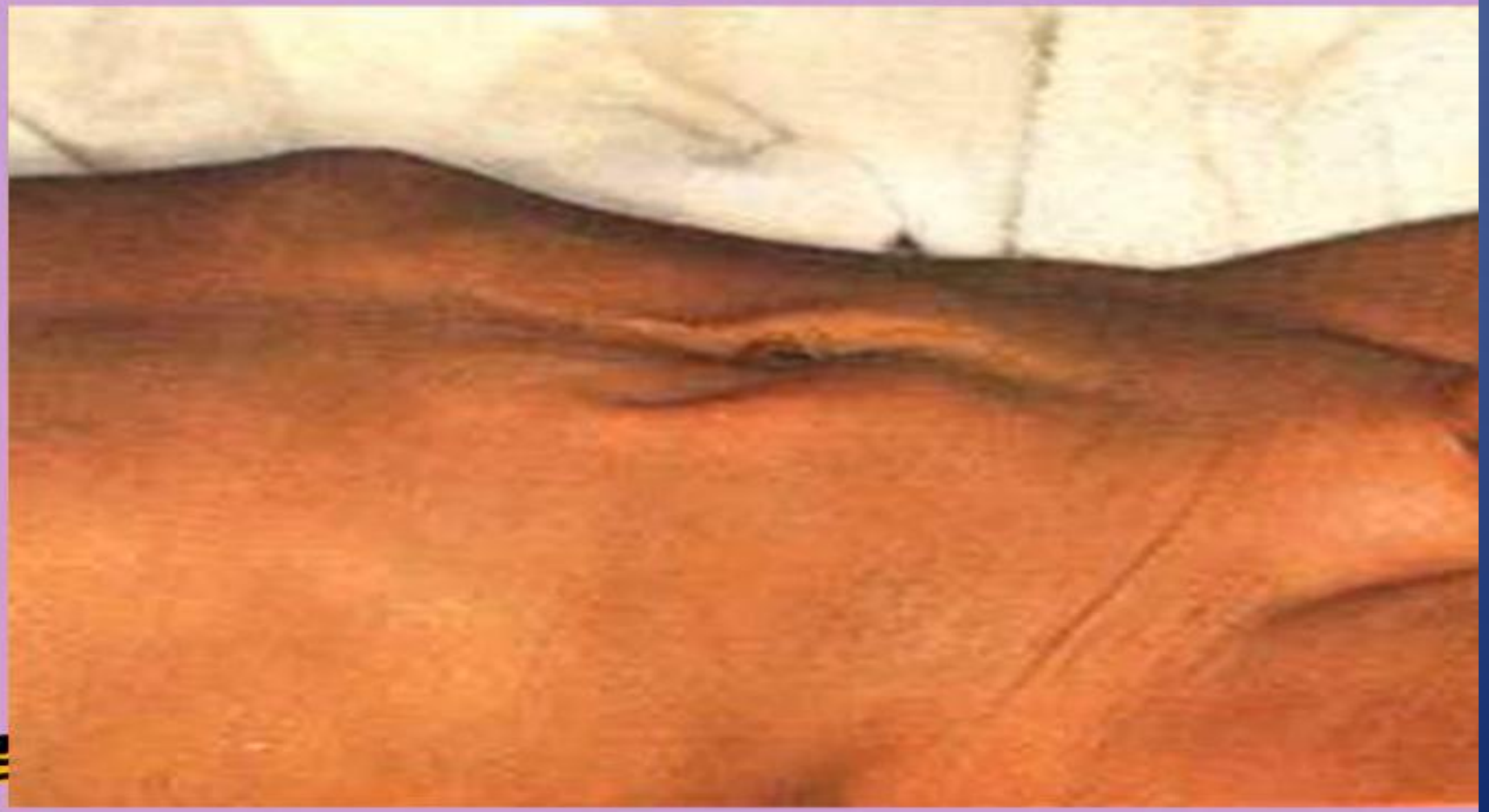
Pathogenesis & Immunity

- **Exotoxin (Shiga toxin)** is neurotoxic, cytotoxic, and enterotoxic, encoded by chromosomal genes,
- **Enterotoxic effect**: Shiga toxin adheres to small intestine receptors
- Blocks absorption (uptake) of electrolytes, glucose, and amino acids from the intestinal lumen

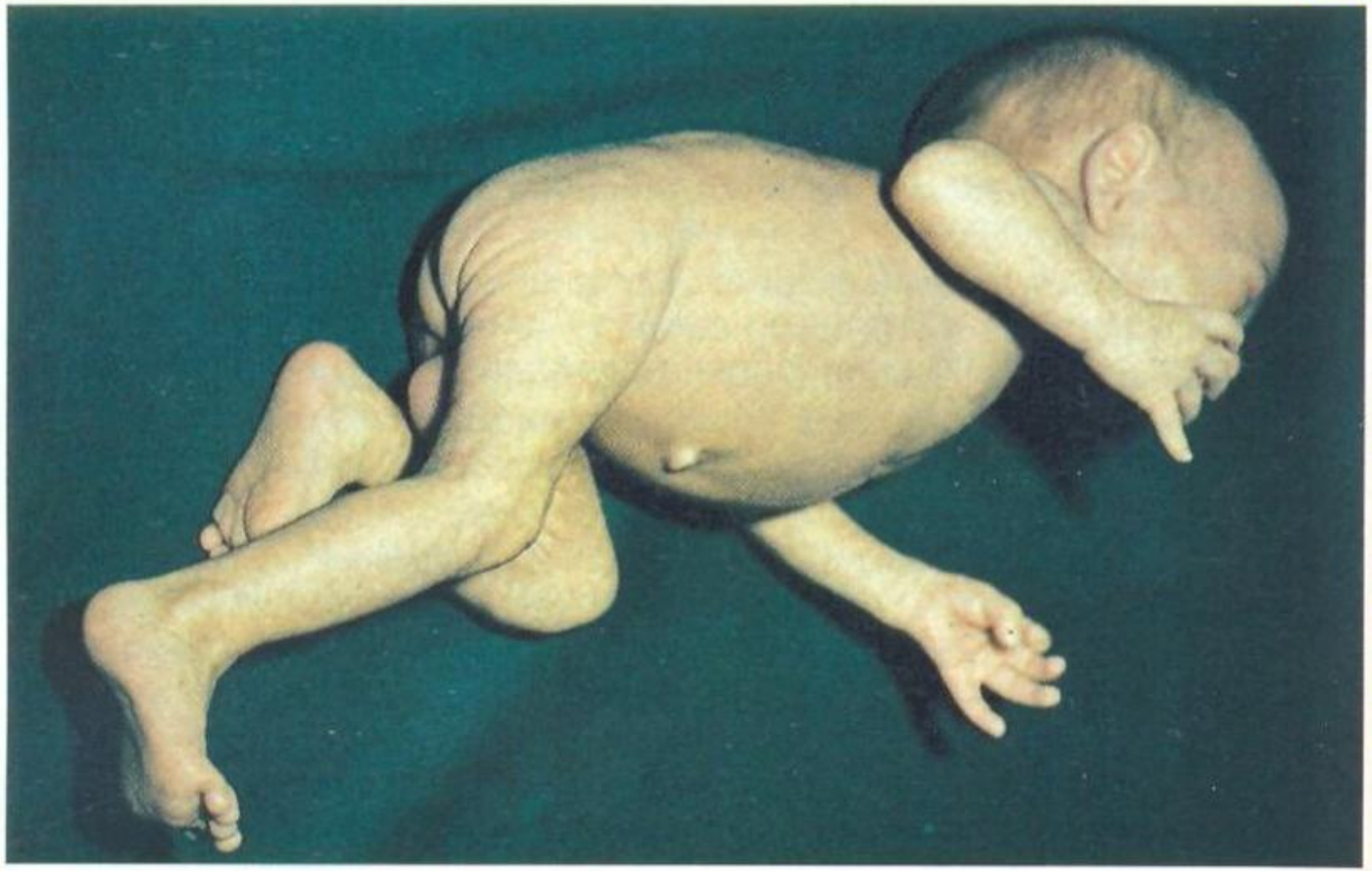
With dominance of local inflammation

- Sudden onset of high-grade fever
- abdominal cramping
- abdominal pain,
- tenesmus,
- and large-volume watery diarrhea →
- fecal incontinence, and small-volume mucoid diarrhea with frank blood

Sunken abdomen, dehydration

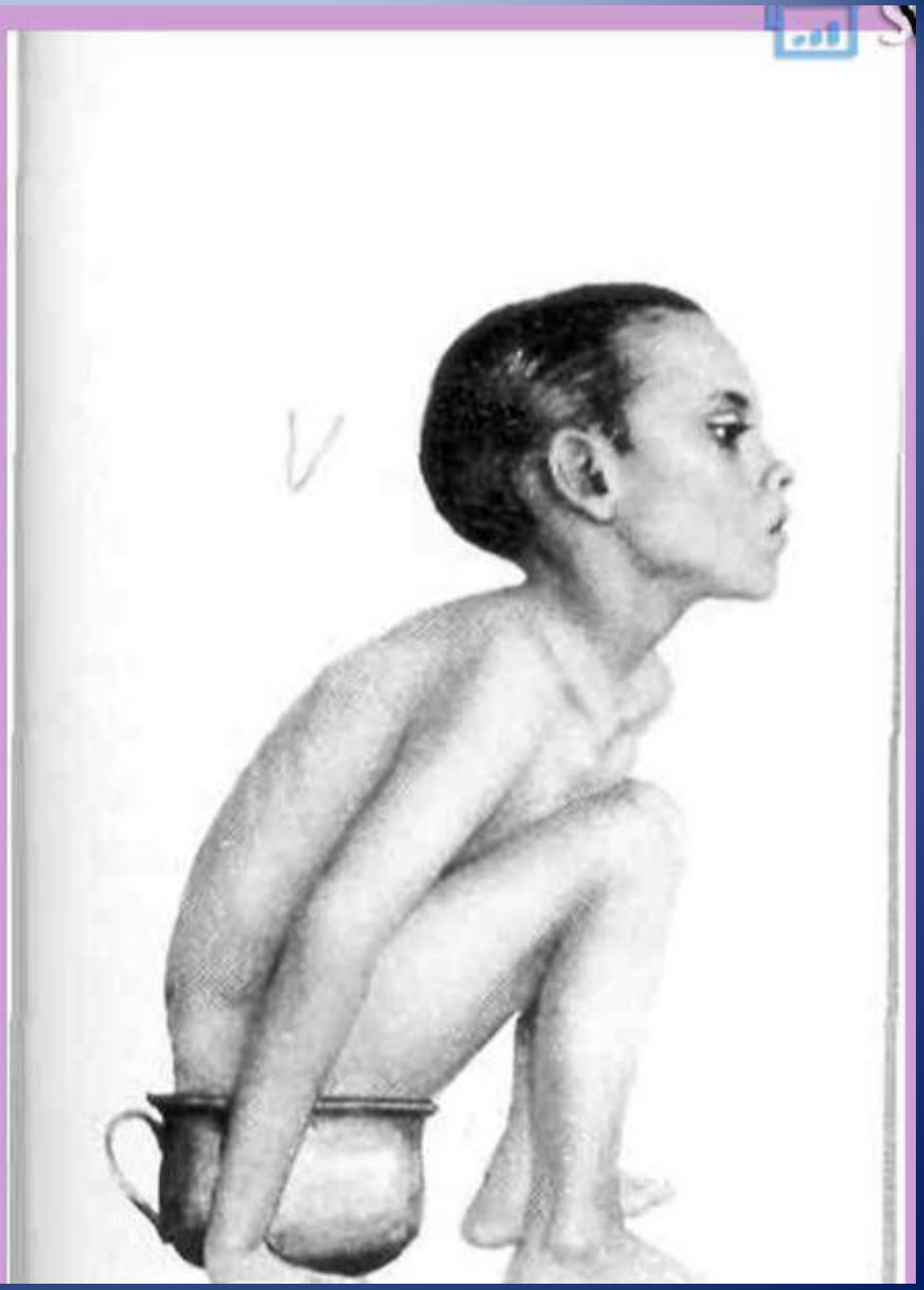


Toxicosis, marble skin



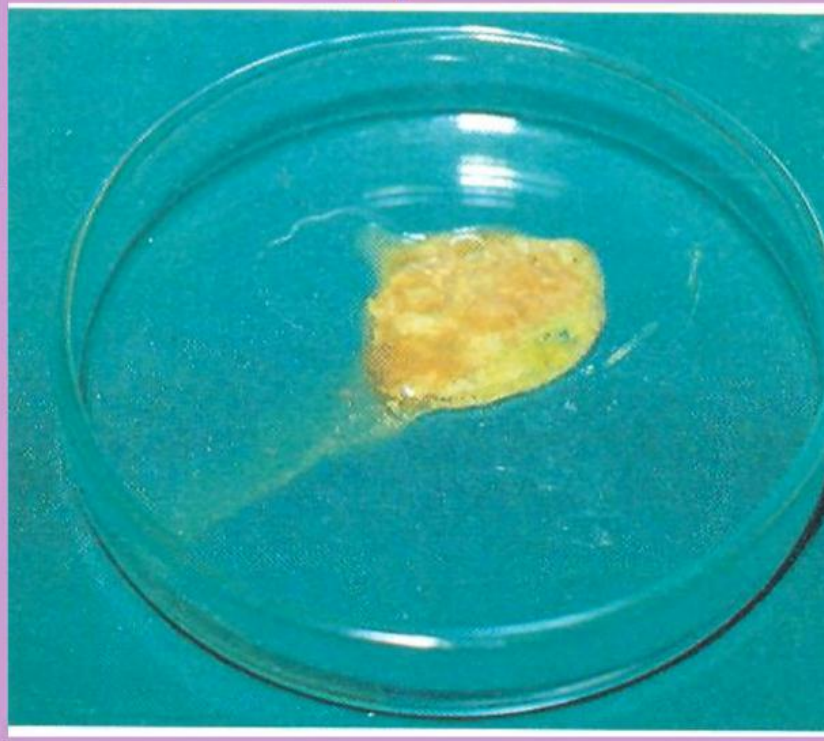
Shigella Infection

false urge to defecate

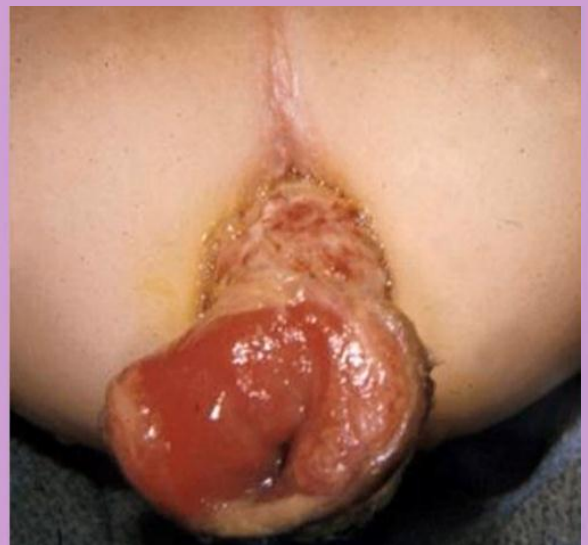


Stools with greenish and mucous

Rectal spit



Rectal prolapse



Characteristics of Shiga Toxin

- Enterotoxic, neurotoxic and cytotoxic
- Encoded by chromosomal genes
- Two domain (A-5B) structure
- Similar to the Shiga-like toxin of enterohemorrhagic *E. coli* (EHEC)
  - NOTE: except that Shiga-like toxin is encoded by lysogenic bacteriophage

Lab Studies:

- **The white blood cell count** is often within reference range, with a high percentage of bands. Occasionally, leukopenia or leukemoid reactions may be detected.
- **If HUS**, anemia and thrombocytopenia occur.
- **Stool examination**
- Increasing of red blood sells and leukocytes
- **Stool culture**
- Specimens should be plated lightly onto MacConkey, xylose-lysine-deoxycholate, or eosin-methylene blue agars.
- **Serological test** in dynamics with fourfold title increasing in 10-14 days

Growth on Selective Medium

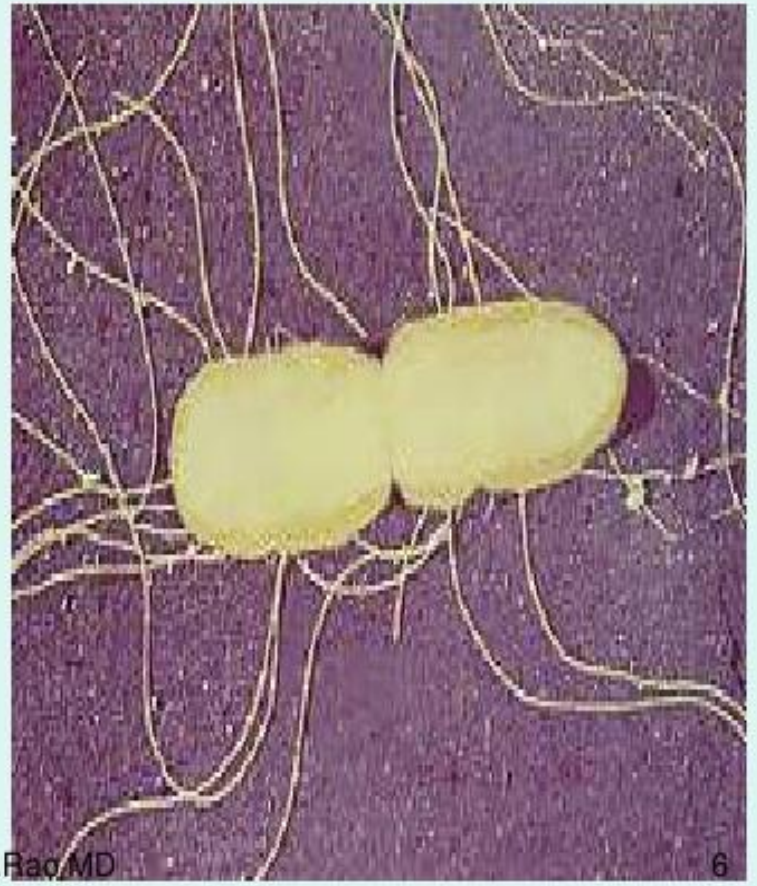
- In XLD they appear pinkish to reddish colonies while in Heaktoen Enteric Agar (HEA), they give green to blue green colonies.


Shigella on EMB
Shigella on MacConkey
Shigella on ENDO
Shigella on Hektoen
Shigella on SS agar

SALMONELLA

Morphology of Salmonella

- Gram negative bacilli
- 1-3 / 0.5 microns,
- Motile by peritrichous flagella



Cultural Characters

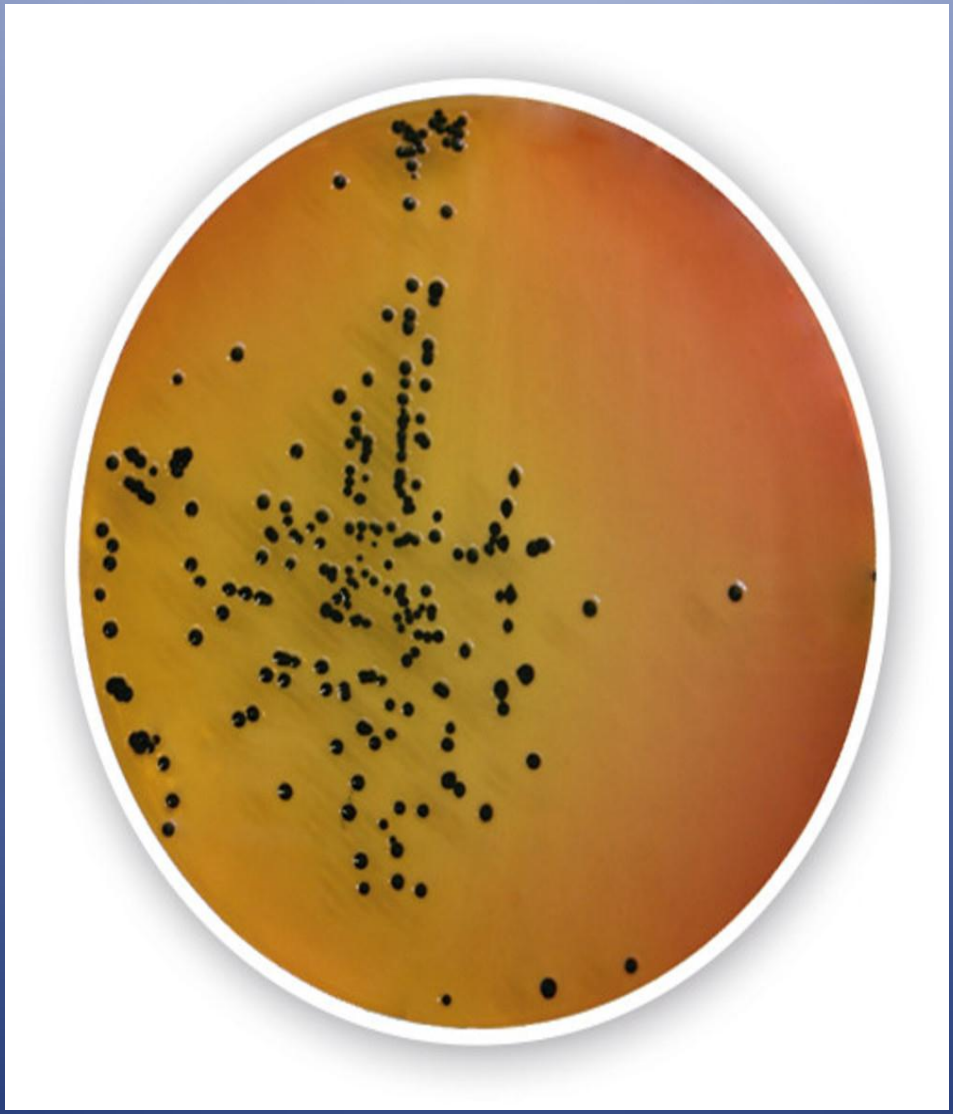
- Aerobic / Facultatively anaerobic
- Grows on simple media - Nutrient agar,
- Temp 15 - 41°c / 37° c
- Colonies appear as large 2 -3 mm, circular, low convex,
- On MacConkey medium appear

  Colorless (NLF)

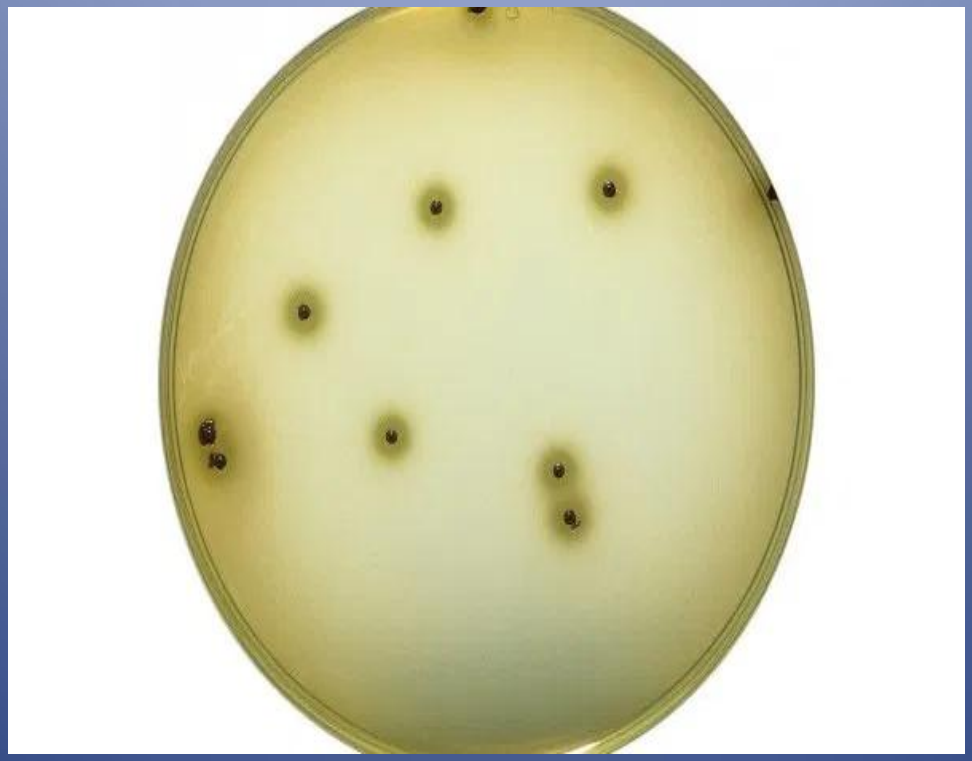
Selective Medium - Wilson Blair Bismuth sulphide medium. Produce Jet black colonies

H_2S produced by Salmonella typhi

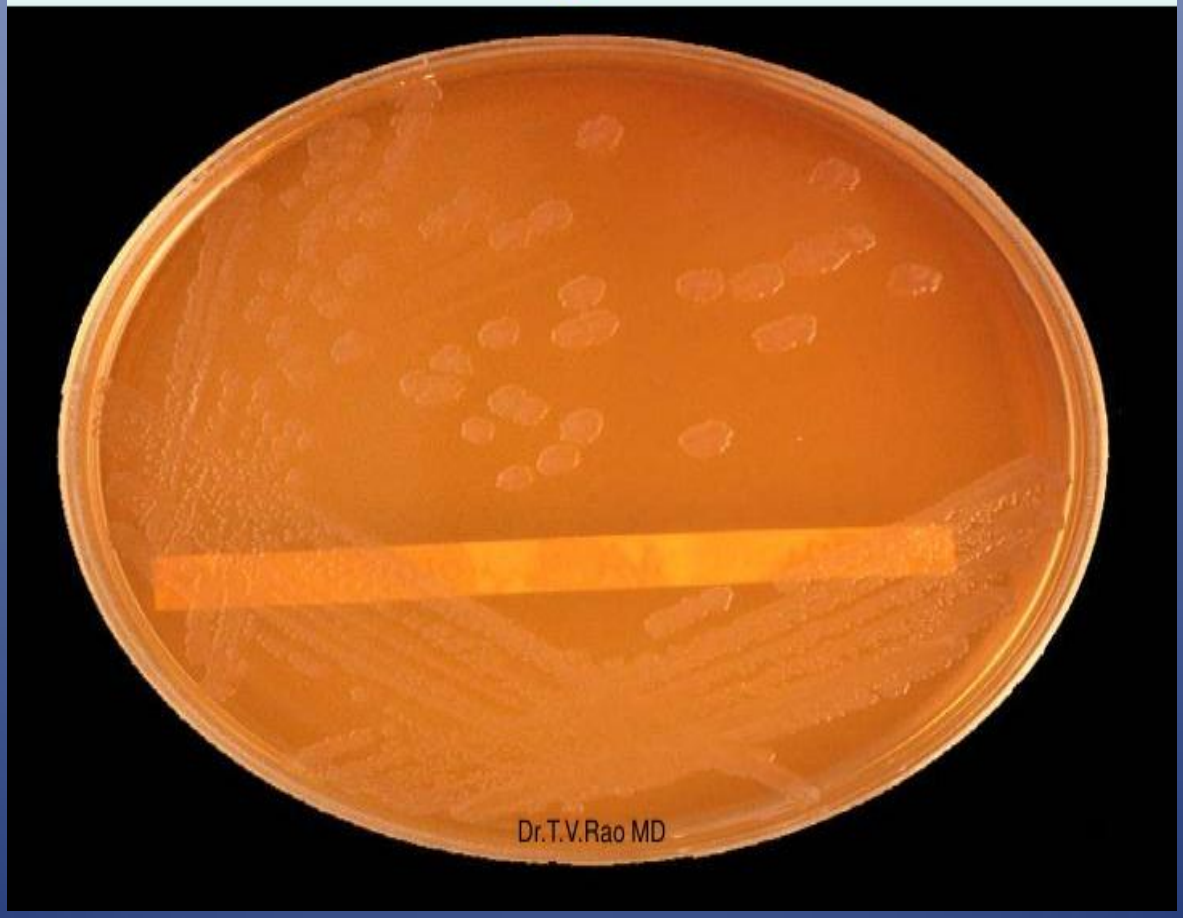
Salmonella spp.
SS (salmonella-shigella) agar



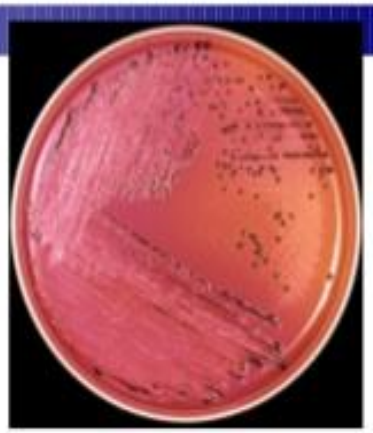
Salmonella spp.
Bismuth Sulfite agar



Salmonella on Mac Conkey's agar



Cultural character



- Grow on ordinary culture media
- In MacConkey agar and DCA: Small, circular, translucent, nonlactose fermenting colonies.
- In Wilson and Blair Bismuth sulfite medium: Black colonies with metallic sheen due to production of H_2S
- Selenite F and tetrathionate broth (enrichment media for stool specimen culture)

- Salmonella can cause

3. Enteric fever
4. Gastroenteritis
5. Septicemia with or without focal suppuration
6. Carrier state

Pathogenicity

- Enteric Fever-
2. Typhoid fever- S. typhi
3. Paratyphoid fever – S.paratyphi A,B,C

- Salmonella typhi—typhoid fever
- Eberth-Gaffky bacillus/Eberthella typhi

- Practically salmonella has been divided into

6. The enteric fever group-typhoid,paratyphoid bacilli
7. The food poisoning group

Clinical Manifestations

- Typhoidal salmonella – Enteric fever
- Non typhoidal salmonella – Gastroenteritis

- Bacteremia
- Osteomyelitis
- Localised infections
- Carriers

Clinical Diseases Induced by Salmonellae

	Enteric Fevers	Septicemias	Enterocolitis
Incubation period	7–20 days	Variable	8–48 hours
Onset	Insidious	Abrupt	Abrupt
Fever	Gradual; then high plateau with "typhoidal" state	Rapid rise; then spiking "septic" temperature	Usually low
Duration of disease	Several weeks	Variable	2–5 days
Gastrointestinal symptoms	Often early constipation; later, bloody diarrhea	Often none	Nausea, vomiting, diarrhea at onset
Blood culture results	Positive in first to second weeks of disease	Positive during high fever	Negative
Stool culture results	Positive from second week on; negative earlier in disease	Infrequently positive	Positive soon after onset

Ideal specimen

First week	Blood (culture)
Second week	Serum (Antibodies)
Third week	Stool
Fourth week	Urine

Chance of isolation

Specimens	First week	Third week
Blood	50 to 80%	30%
Feces	40 to 50%	80%
Urine	-	25%

Blood culture

- Volume of blood :

  10 to 15 ml from adults and adolescents , 2 to 4 ml in children
- Ratio of blood to bile broth: 1:10
- Or add saponin to BHI broth with 0.05% SPS
- Inoculate the blood immediately
- Transport immediately, never store under 15degC
- Incubate as soon as possible

Diagnosis of Enteric Fever

Widal test

- Serum agglutinins raise abruptly during the 2nd or 3rd week
- The Widal test detects antibodies against O and H antigens
- Two serum specimens obtained at intervals of 7 - 10 days to read the raise of antibodies.
- Serial dilutions on unknown sera are tested against the antigens for respective Salmonella
- False positives and False negative limits the utility of the test
- The interpretative criteria when single serum specimens are tested vary
- Cross reactions limits the specificity

Salmonellosis

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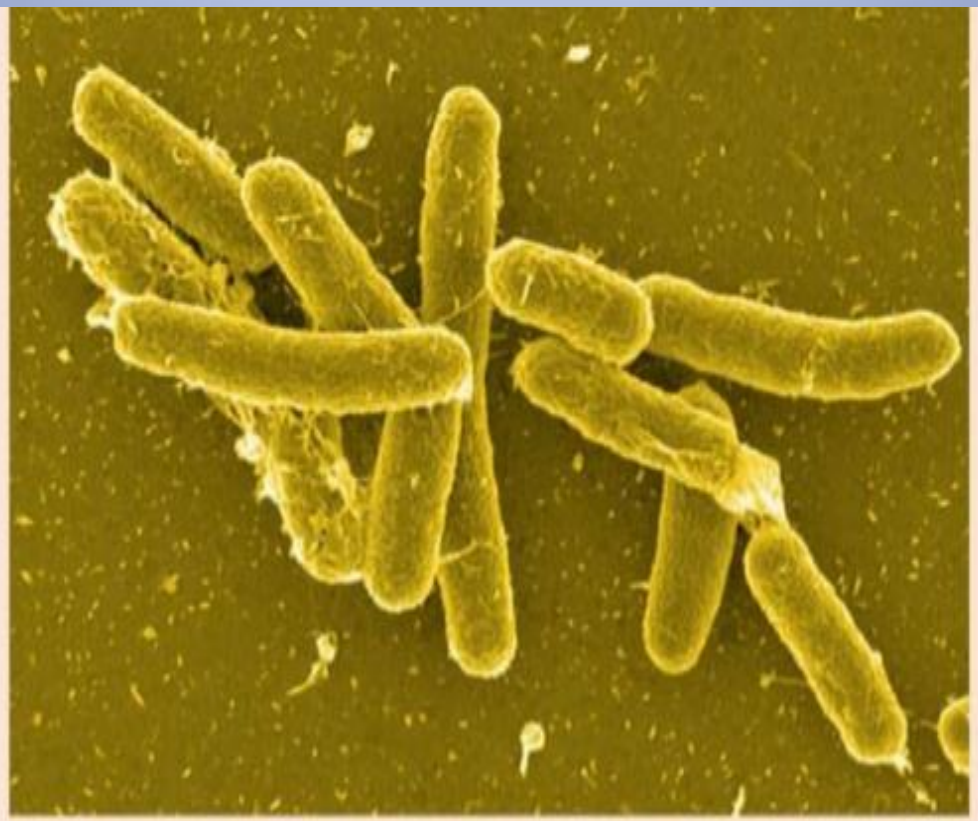


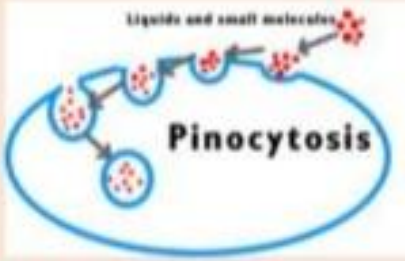
- Gram Negative Bacteria
- Species:
  - S. Bongori
  - S. Enterica
  - Six sub species
- More than 2500 Serovars/Serotypes
- Many are zoonotic

Human Transmission

- Fecal-oral: direct or indirect
- Commonly contaminated items
  - Meat, eggs, water
- Fecal material from:
  - Reptiles
  - Chicks
  - Ducklings
  - Livestock, dogs, cats, adult poultry

Pathophysiology

- Bacteria colonizes in the ilium – the end part of small intestine
- They invade intestinal epithelium
- They expand within the epithelium and lymphoid cavities
- The invasion happens when organisms start to ruffle
- This way they stimulate the pinocytosis



- They multiply & spread through mesenteric lymph nodes
- After reaching intestines they create an acute inflammation

CLINICAL PRESENTATION

Incubation period: 6-72 hours

but illness usually occurs within 12-36 hours after exposure

Symptoms:

- acute diarrhea
- abdominal pain
- fever
- Vomiting
- Headache, fever, chills, myalgia
- Severe dehydration: infants, elderly

- The illness usually lasts 4-7 days, and most people recover without treatment.

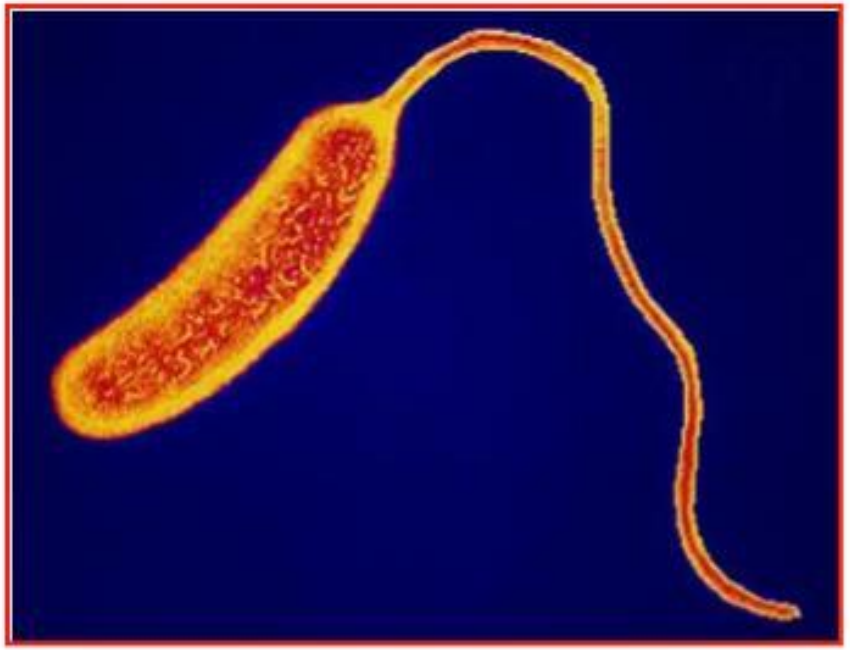
- Rates of invasive infections and death are generally higher among infants, older adults, and people with immunosuppressive conditions (including HIV), hemoglobinopathies, and malignant neoplasms.

DIAGNOSIS

- Diagnosis is based on isolation of Salmonella organisms
- About 90% of isolates are obtained from routine stool culture, but isolates are also obtained from blood, urine, and material from sites of infection
- Isolates of salmonellae are needed for serotyping and antimicrobial susceptibility testing
- PCR

Vibrio Cholerae

- Gram negative
- Facultative anaerobe
- Curved-shaped rod
- One polar flagellum
- Housed by zooplankton in both fresh and salt water
- Found in water contaminated with fecal matter
- Cholera Toxin

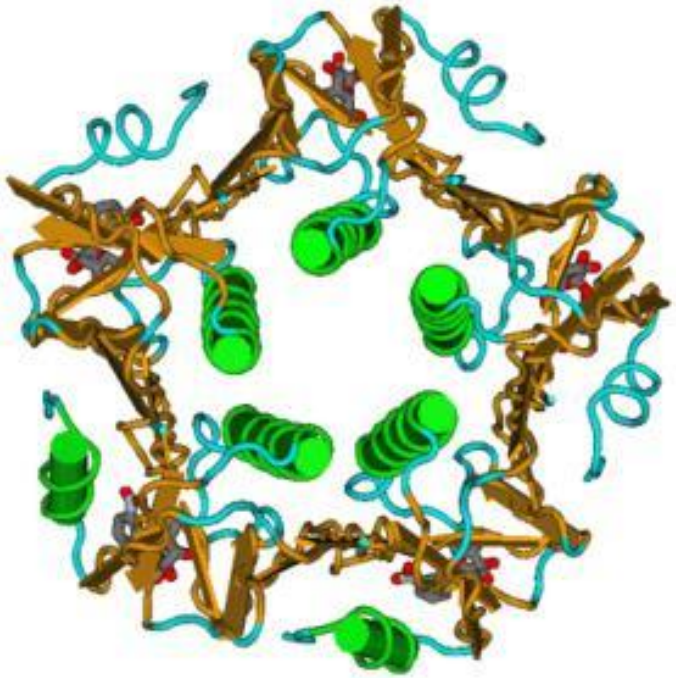


VIBRIO CHOLERAE

Trigger Words
Serogroup O1, cholera, cholera toxin, shellfish, gastroenteritis

Biology and Virulence
- Curved gram-negative rods
- Fermentative, facultative anaerobic; require salt for growth
-Strains subdivided into more than 200 serogroups (O–cell wall antigens)
-*V. cholerae* serogroup O1 is further subdivided into serotypes (Inaba,Ogawa, and Hikojima) and biotypes (Classical and El Tor)
-Disease mediated by cholera toxin (complex A-B toxin) and toxin coregulated pilus

Cholera Toxin



- A-B Exotoxin
- Causes Cholera in humans
- Produced by the CTXf bacteriophage
- A subunit (activating)
  - A1 domain: enzymatic active site
  - A2 domain: a-helical tail
- B subunit (binding)
  - Pentameric ring around central pore

Bio Chemical Reactions

	V.cholrae(Classical)	V.cholrae (El Tor)
Hemolysis	-ve	+ve
Voges -proskauer test	-ve	+ve
Polymyxin sensitivity	+ve	-ve
Group IV phage Susceptibility	+ve	-ve
Chick erythrocyte Agglutination	-ve	+ve

The Medically Important Vibrios

Organism	Human Disease
Vibrio cholerae serogroups O1 and O139	Epidemic and pandemic cholera
Vibrio cholerae serogroups non-O1/non-O139	Cholera-like diarrhea; mild diarrhea; rarely, extraintestinal infection
Vibrio parahaemolyticus	Gastroenteritis, wound infections, septicemia
Vibrio vulnificus	Gastroenteritis, wound infections, septicemia

Virulence Factors of *Vibrio* Species

Species	Virulence Factor	Biological Effect
Vibrio cholerae	Cholera toxin	Hypersecretion of electrolytes and water
	Toxin coregulated pilus	Surface binding site receptor for bacteriophage CTXΦ; mediates bacterial adherence to intestinal mucosal cells
	Chemotaxis protein	Adhesin factor
	Accessory cholera enterotoxin	Increases intestinal fluid secretion
	Zonula occludens toxin	Increases intestinal permeability
	Neuraminidase	Modifies cell surface to increase GM_1 binding sites for cholera toxin
V. parahaemolyticus	Kanagawa hemolysin	Enterotoxin that induces chloride ion secretion (watery diarrhea)
V. vulnificus	Polysaccharide capsule	Antiphagocytic
	Cytolysins, proteases, collagenase	Mediates tissue destruction

Vibrio Species Most Commonly Associated with Human Disease

Species	Source of Infection	Clinical Disease
Vibrio cholerae	Water, food	Gastroenteritis, bacteremia
V. parahaemolyticus	Shellfish, seawater	Gastroenteritis, wound infection, bacteremia
V. vulnificus	Shellfish, seawater	Bacteremia, wound infection

Epidemiology

- Serotype O1 is responsible for major pandemics (worldwide epidemics), with significant mortality i n developing countries; O139 can cause similar diseases

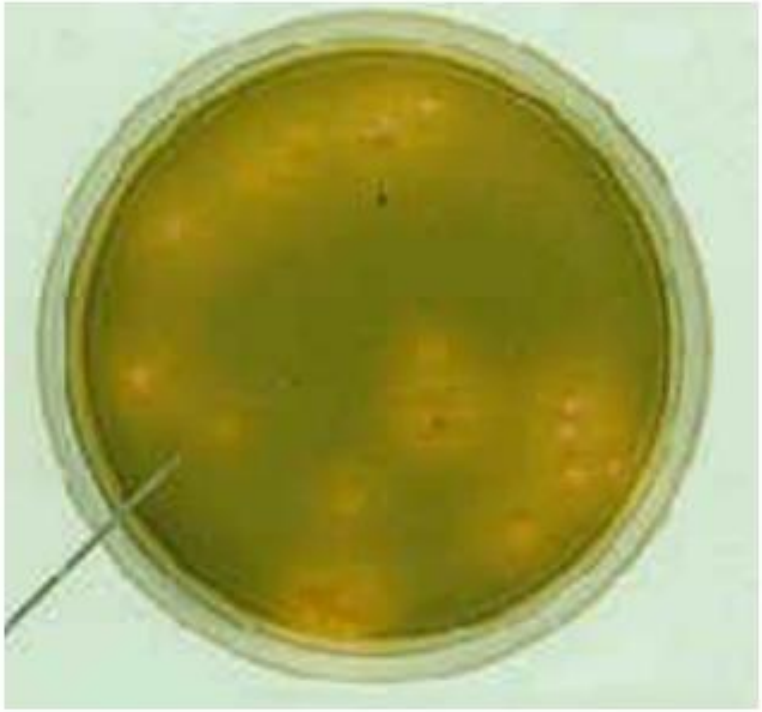
-Organism found in estuarine and marine environments worldwide (including along the coast of the United States); associated with chitinous shellfish
-Organism can multiply freely in water
- Bacterial levels in contaminated waters increase during the warm months
-Most commonly spread by consumption of freshly contaminated water
-Direct person-to-person spread is rare because the infectious dose is high; the infectious dose is high because most organisms are killed by stomach acids

Diagnosis

- Stool culture: Toxigenic *Vibrio cholerae* O1
- Use Cary Blair Transport media if available
  - Viable for many days at room temperature
- Use TCBS media for culture
- Use *V. cholerae* serogroup O1 antisera
- Confirm presence of cholera toxin
- Cholera Rapid Test Dipsticks

How is the Infection Diagnosed?

- Isolated from stool or vomit
- Cholera antibodies in the blood
- Fecal leukocyte stain
- Stool culture
- Thiosulfate Citrate Bile Salts Sucrose (TCBS) agar

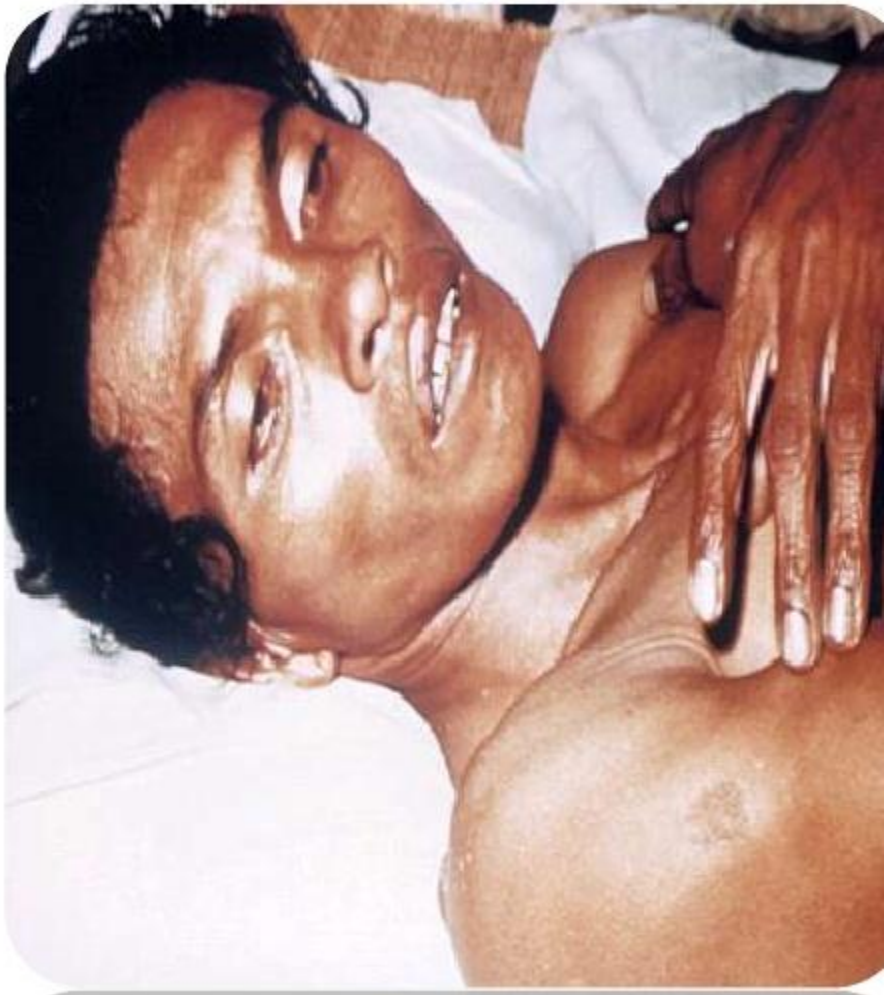


Selective Medium - TCBS

- V.cholrae grows well on Thiosulphate citrate bile sucrose (TCBS) agar, on which it produces yellow colonies that are readily visible against the dark green background of the agar.

Clinical manifestations

- Diarrhea occurs as much as 20 – 30 Liters/Day fluids are lost.
- Results in dehydration
- Shock
- Acidosis
- Can lead to death.
- About 60% of infections are caused with classic V.cholrae and are asymptomatic, about 75% of infections are caused by



CAMPYLOBACTER

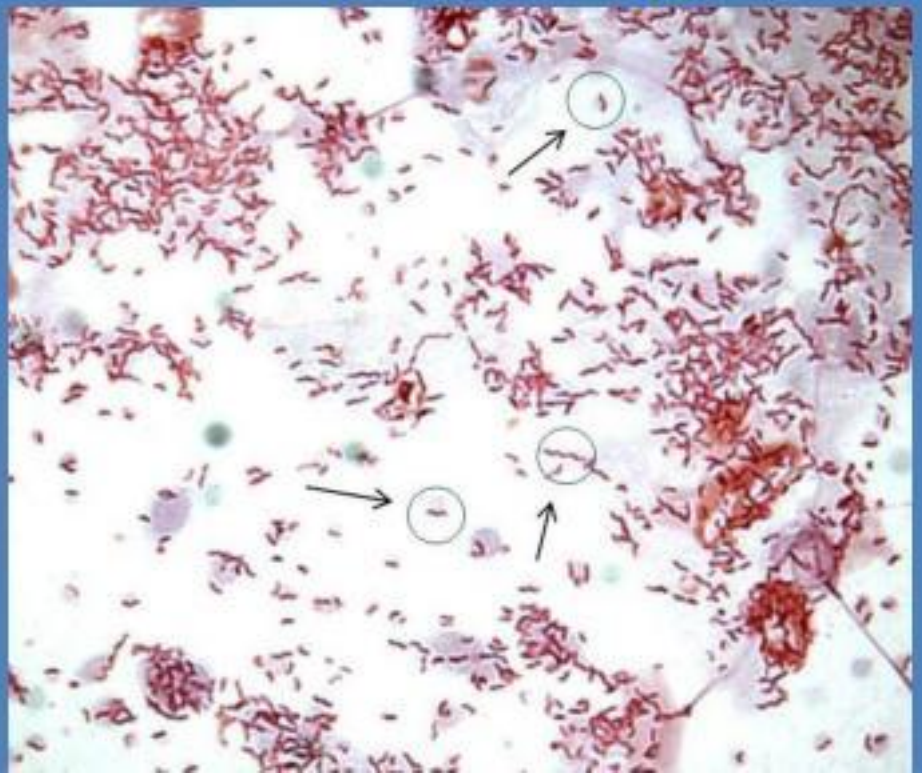
Campylobacter species

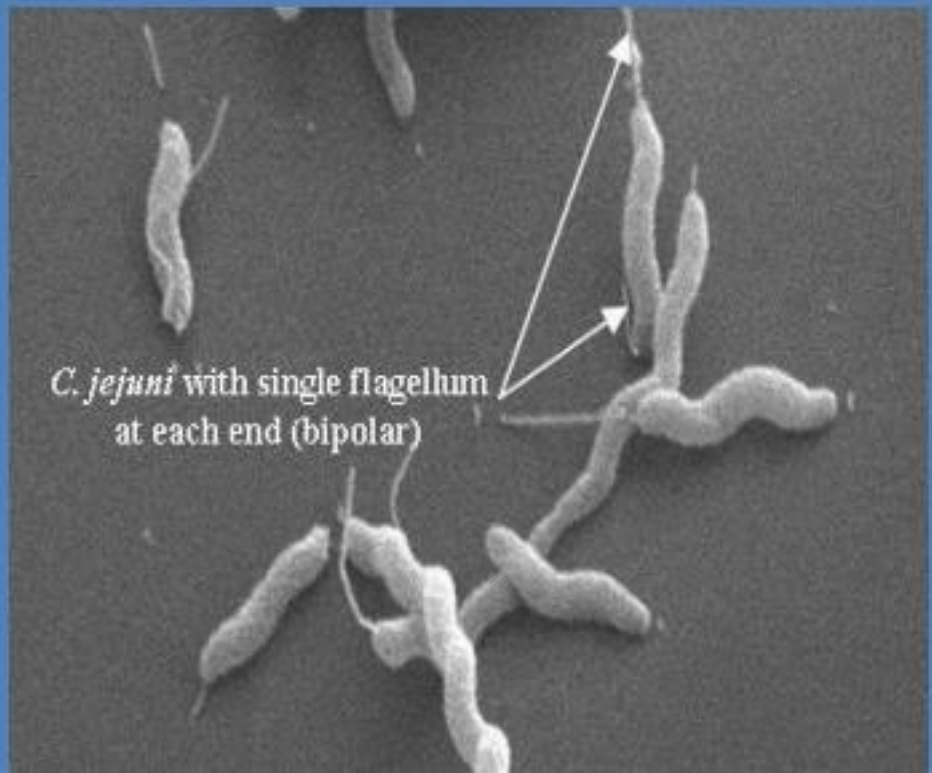
- **Campylobacter jejuni** causes 95% of campylobacter enterocolitis especially in children.
- **Campylobacter coli**
- **C. fetus**, **C. lari** are rare causes of systemic infections such as bacteremia and meningitis.

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.
- **Microaerophilic & capnophilic** (grows best in presence of 5% oxygen and 10% CO_2).
- Grows best at **42** degree.
- Growth may take 2-5 days.

Biochemical reactions

- Oxidase positive
- Catalase positive
- Hippurate positive
- Urease negative
- Non proteolytic
- Unable to attack carbohydrates
- Sensitive to nalidix 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

Capnophilic

Microaerophilic

Thermophilic

Clinical picture

- Enterocolitis begins as watery foul smelling diarrhea followed by bloody stools + fever + severe abdominal pain.
- Complications of Camylobacter jejuni infections two weeks later include:

1) **Guillian-Barre syndrome which is autoimmune disease that attacks neurons.**
2) **Reactive arthritis which is also autoimmune.**

Common *Campylobacter* Species Associated with Human Disease

Species	Common Reservoir Hosts	Human Disease
Campylobacter jejuni	**Poultry**, cattle, sheep	**Gastroenteritis**, extraintestinal infections, **Guillain-Barré syndrome, reactive arthritis**
C. coli	**Pigs**, poultry, sheep, birds	**Gastroenteritis**, extraintestinal infections
C. fetus	**Cattle, sheep**	**Vascular infections** (e.g., septicemia, septic thrombophlebitis, endocarditis), meningoencephalitis, gastroenteritis
C. upsaliensis	**Dogs**, cats	**Gastroenteritis**, extraintestinal infections, Guillain-Barré syndrome

Bold type signifies the most common hosts and diseases.

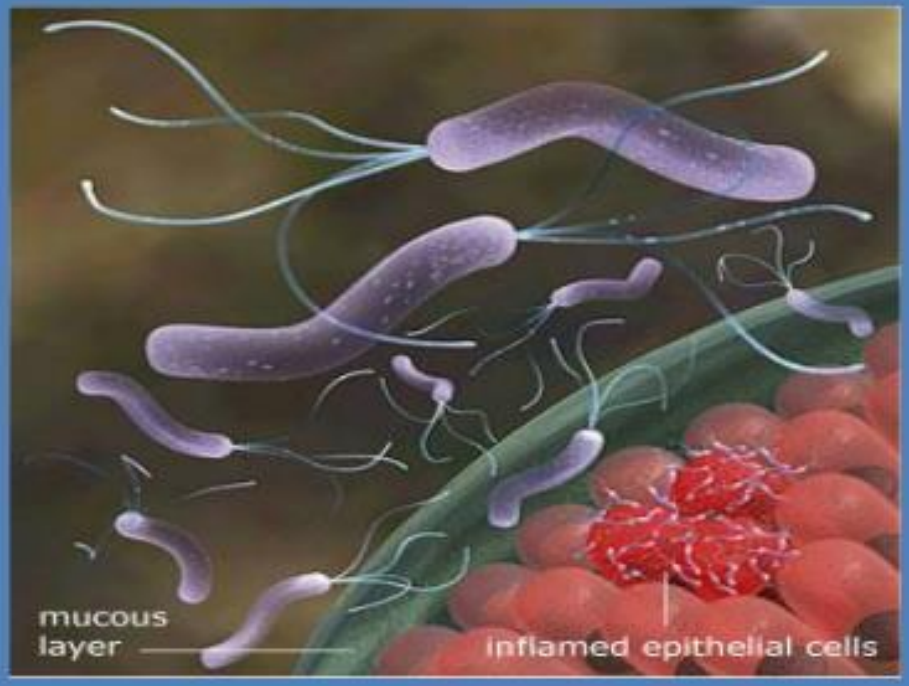
Laboratory diagnosis

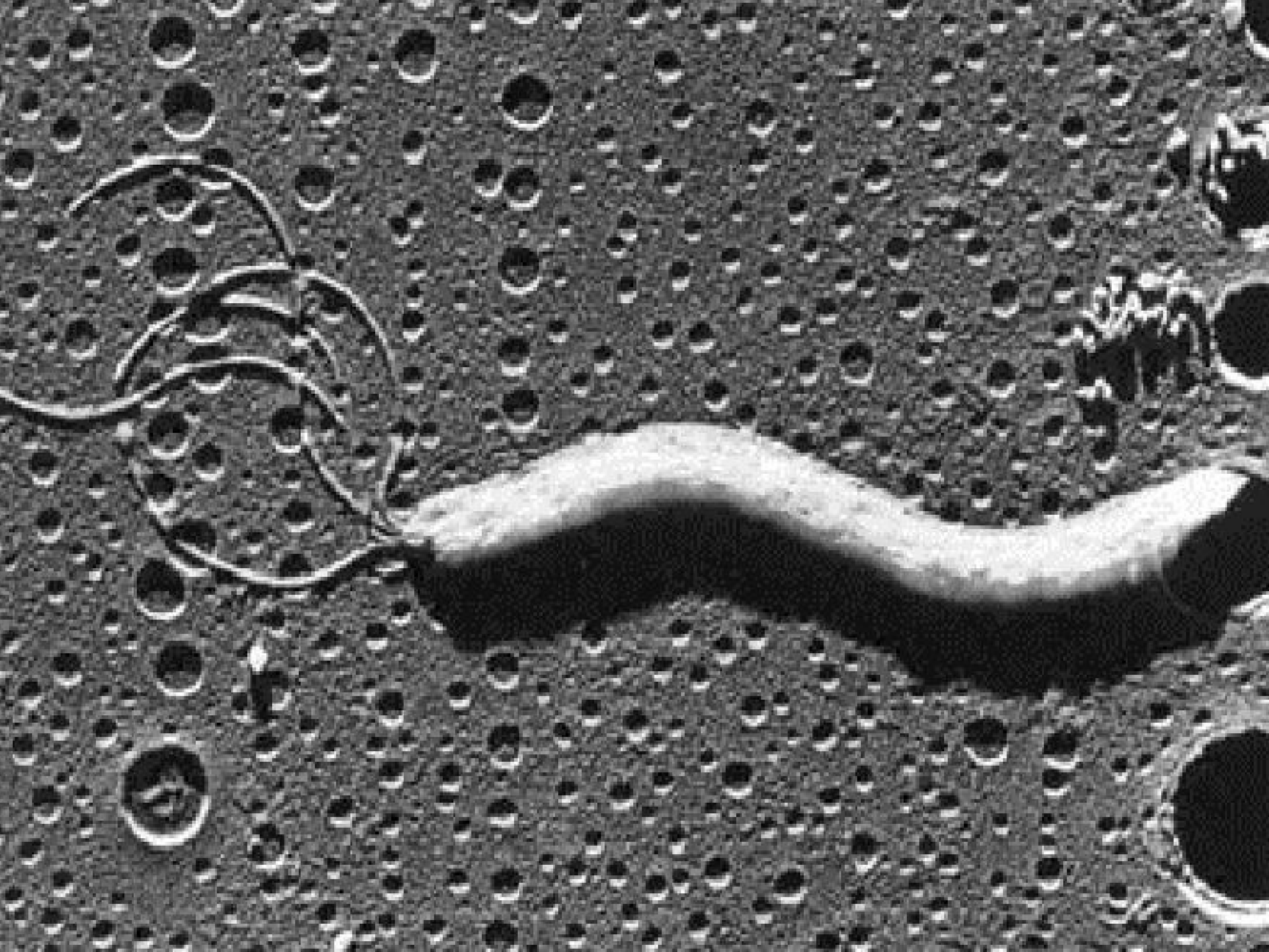
- Specimen: stool
- Wet smears will show the characteristic motility.
- Gram stained smears show the characteristic morphology.
- Stool is cultured on Skirrow's medium and incubated at 42 degree at microaerophilic and capnophilic conditions.
- Identification of the growing colonies.
- Direct detection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

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)

Capnophilic

Urease positive

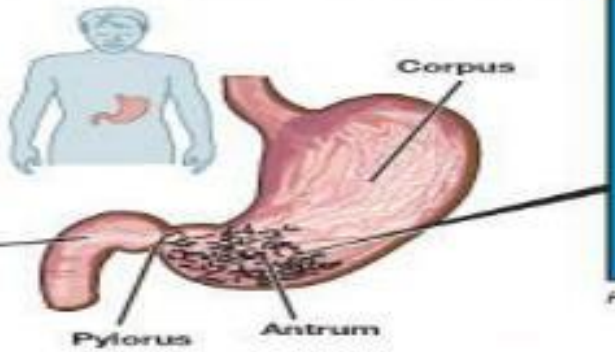
Microaerophilic

Helicobacter pylori

— the bacterium causing peptic ulcer disease

Infection

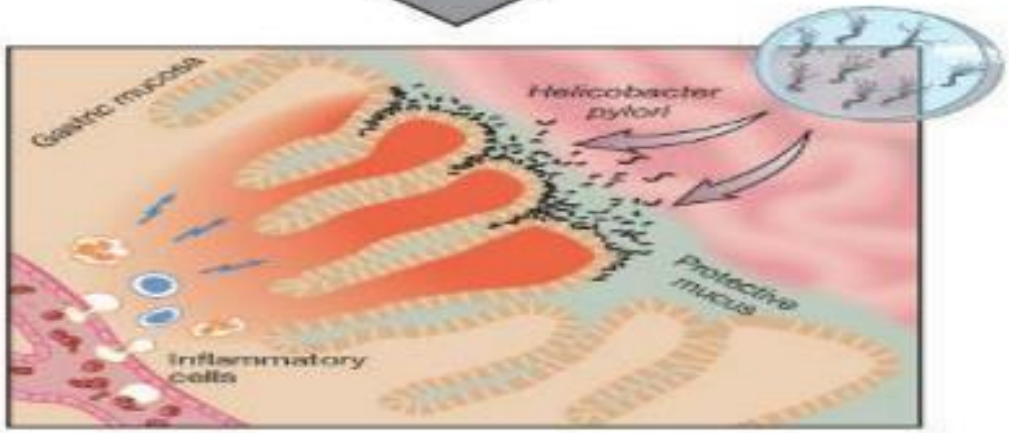
Helicobacter pylori infects the lower part of the stomach, antrum.



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

Helicobacter Species Associated with Human Disease

Species	Common Reservoir Hosts	Human Disease
Helicobacter pylori	**Humans**, primates, pigs	**Gastritis, peptic ulcers, gastric adenocarcinoma,** mucosa-associated lymphoid tissue B-cell lymphomas
H. cinaedi	**Humans,** hamster	**Gastroenteritis,** septicemia, proctocolitis
H. fennelliae	**Humans**	**Gastroenteritis,** septicemia, proctocolitis

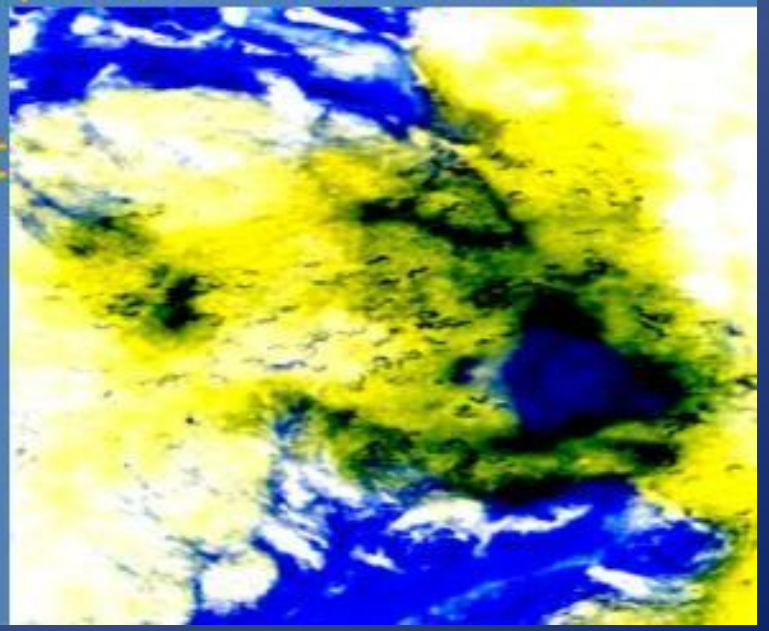
Bold type signifies the most common hosts and diseases.

Laboratory diagnosis

- **Invasive methods:**

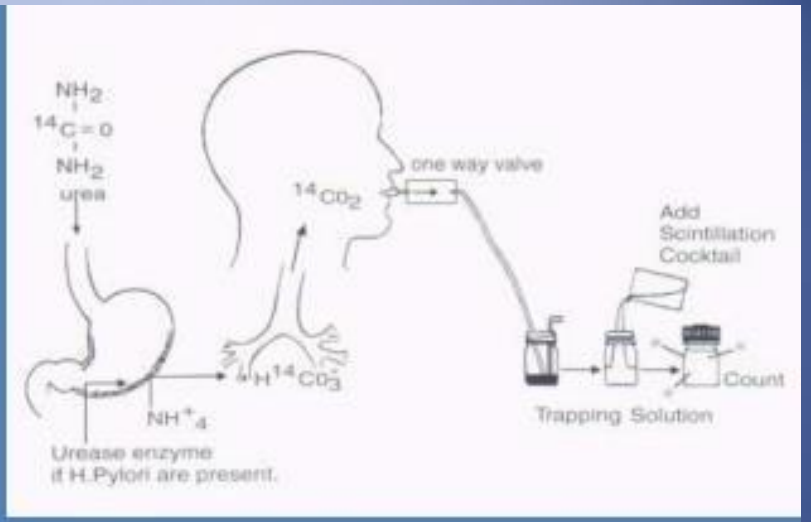
Gastric biopsy specimens

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 CO2 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.