GIT Module
Diarrhea Associated Pathogens
2025-2026

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Medical definition of diarrhea:

- Passage of ≥3 loose or watery stools per day (taking the shape of the container)
- Often also defined as stool weight >200–250 g/day in adults.

Causes

- Infection (viral, bacterial, parasites)
- Food poisoning
- Intolerance (e.g., lactose)
- Medications (antibiotics)
- Stress / GI disorders

See a doctor if

- 2–3 days
- Dehydration signs
- Blood/mucus in stool
- High fever
- Severe pain
- Child or elderly

Management

- Hydration
- Light diet
- Avoid fatty, spicy, dairy, caffeine, alcohol
- Rest

Bacterial infections of GIT

Classification of Pathogens Associated Diarrhea

Toxin mediated

S. aureus

C. botulinum

C. perfringens

B. cereus

Watery (secretory) diarrhea

V. cholera

ETEC

EPEC

Cell invasion

Shigella

Salmonella enteritidis

EHEC

EIEC

Antibiotic associated diarrhea

C. difficile

Cell invasion and bacteremia

C. Jejuni

Salmonella typhi

Gastritis and ulcers

H. pylori

Toxin mediated

S. aureus

- **Source: Human skin and nasal passages**
- Food is **handled by people** (spreading bacteria from hands, nose, cuts)
- **Preformed toxin in food**
- **Fast onset: 1–6 hours**
- **Toxin acts on vagal afferent nerves in gut→ stimulates vomiting center in brain (medulla)**
- **No fever**
- Foods: **salads, cream pastries**
- Toxin is **heat-stable** (reheating doesn't help)
- **Treatment: fluids only (no antibiotics)**

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Toxin mediated

C. perfringens

- **Toxin made in gut (after eating),** Spores survive cooking → germinate in intestine

- **Sourse: Soil + intestines of animals (especially poultry, beef, and pork)**
- **Watery diarrhea (main symptom):** Toxin binds intestinal epithelial cells → **disrupts cell membranes** → fluid leaks into gut)

- **Onset: 8–16 hours** (slower)
- Vomiting minimal or absent
- **No fever**
- Spores **survive cooking** → grow if food kept warm long time
- **Treatment: fluids only (no antibiotics)**

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Toxin mediated

Bacillus cereus

- Bacillus cereus: spores survive cooking
- **Classic: fried rice syndrome**
  - Bacillus cereus in cooked rice
  - Rice: cooked → left at room temp
  - Bacteria produce heat-stable toxin
  - Reheating/frying doesn't destroy toxin → illness

Toxin mediated

Bacillus cereus

Types on infections:

1- Emetic type (vomiting)

- You ingest **preformed toxin** in food
- Vagal stimulation
- Classic: **reheated rice**
- Rapid onset: **1–6 hours**
- Key idea: **toxin already made in food**

2- Diarrheal type

- Ingest spores (or bacteria)
- Germinate in intestine
- Produce toxin in gut (after ingestion)
- Onset: 8–16 hours
- Mechanism: toxin damages intestinal epithelium → ↑ secretion + fluid loss
- Result: watery diarrhea

- **"Preformed in food → Vomiting"**
- **"Produced in gut → Diarrhea"**

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Toxin mediated

C. botulinium

- Organism: Gram-positive, spore-forming, anaerobe
- Toxin blocks Acetylcholine release→ flaccid paralysis
- Descending paralysis (cranial nerves first)
- Infants: honey (spores) → floppy baby
- Adults: canned food (preformed toxin), through wounds
- No fever, normal sensation
- Tx: Supportive care + antitoxin ; airway priority

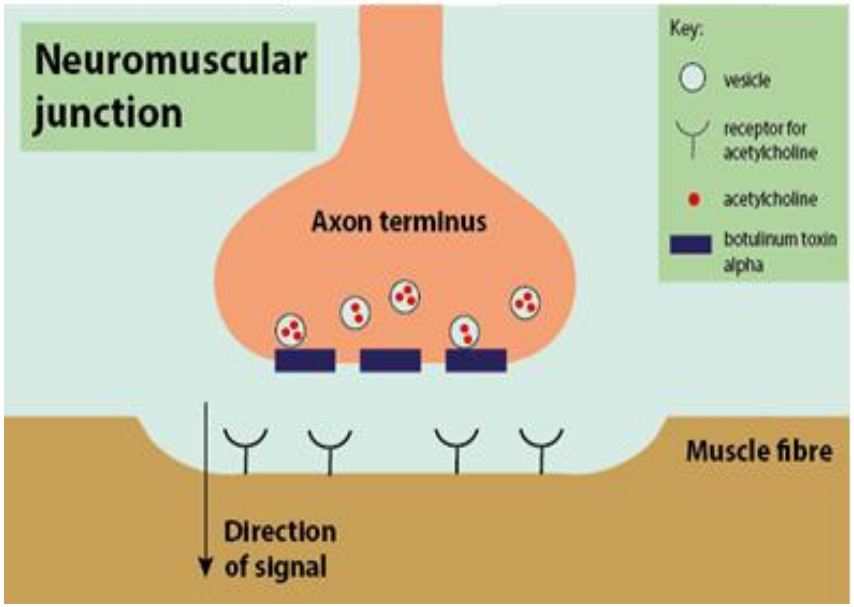
Toxin mediated

C. botulinium

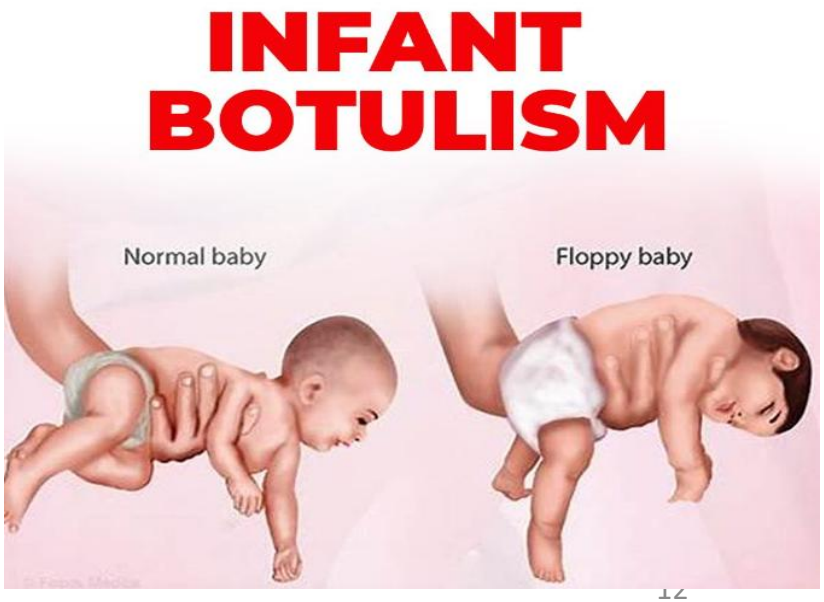
Foodborne: 12–36 hours (can be up to ~3 days) (preformed toxins)

Infant botulism: days–weeks (spore germination)

Wound botulism: 4–14 days



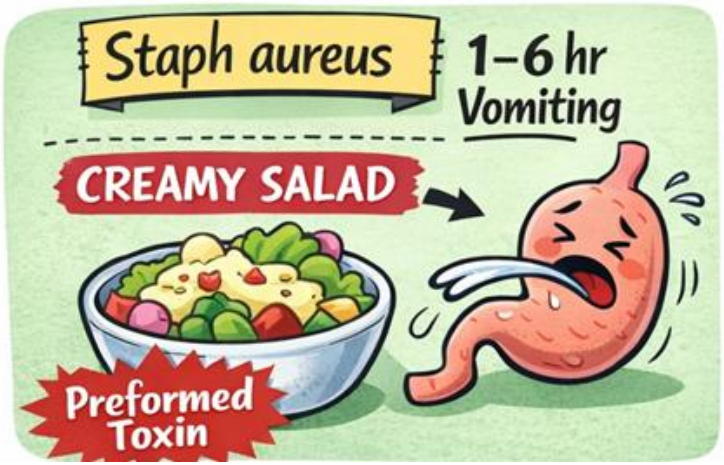
Mode of action

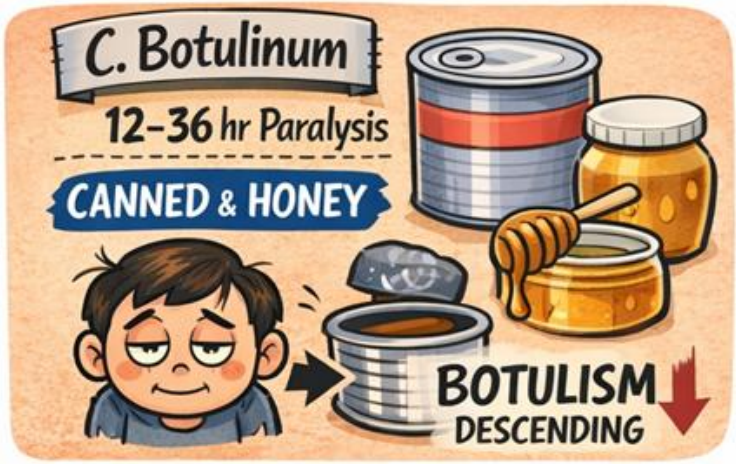


Toxin mediated pathogens

Bug	Onset	Main Symptom	Key Clue	Source/Food
Staphylococcus aureus	1–6 hr	Vomiting	Preformed toxin	Salads, cream (human handling)
Clostridium perfringens	8–16 hr	Diarrhea	Toxin made in gut	Meat, gravy (animal source)
Bacillus cereus	6–24 hr	Diarrhea	Spores → gut toxin	Rice, pasta, vegetables
Botulinium	12–36 hr	Paralysis	Toxin preformed	Canned food

Food Poisoning Bugs — Quick Review





USMLE- mini cases

Case 1

A 22-year-old eats potato salad at a picnic.
3 hours later → severe vomiting, no fever.
Diagnosis: Staphylococcus aureus

Case 2

A family eats meat and gravy left out overnight.
10 hours later → watery diarrhea, mild cramps, no vomiting.
Diagnosis: Clostridium perfringens

Case 3

A student eats reheated fried rice.
4 hours later → vomiting, no fever.
Diagnosis: Bacillus cereus (emetic type)

FAST Tricks

vomiting → Staph or Bacillus (rice?)
Diarrhea after meat → C. perfringens
After antibiotics → C. diff

Short Case

A 4-month-old infant is brought to the ER with **poor feeding, weak cry, and decreased movement**. The mother reports the baby has been **constipated for 3 days**. On exam, the infant has **floppy tone ("floppy baby")**, **ptosis**, and **poor suck reflex**. There is no fever.

Question

Most likely mechanism of disease?

A. Toxin blocks 60S ribosome
B. Toxin blocks acetylcholine release
C. Toxin increases cAMP in intestinal cells
D. Autoimmune destruction of postsynaptic ACh receptors
E. Demyelination of peripheral nerves

Case

A 22-year-old student develops **sudden nausea and severe vomiting** about **3 hours** after eating cream-filled pastries at a party. Several others have similar symptoms. No fever. Diarrhea is mild.

Question

Most likely cause?

A. Clostridium perfringens
B. Staphylococcus aureus
C. Salmonella
D. Clostridium botulinum
E. Bacillus cereus

Answer: B. Staph aureus

Key Clues

- **Rapid onset (1–6 hrs)**
- **Preformed toxin**
- Prominent **vomiting**
- Dairy/pastries

Case

A 19-year-old eats **reheated fried rice**. Within **4 hours**, she develops **intense vomiting** without fever.

Answer: *Bacillus cereus* (emetic type)

Why:

- Reheated rice (classic!)
- Rapid onset
- Vomiting
  → Preformed toxin

Case

Several people at a **wedding buffet** develop **watery diarrhea 12 hours later** after eating **meat dishes**. Minimal vomiting.

Answer: ***Clostridium perfringens***

Why:

- 8–16 hrs
- Meat, large batch food
- Diarrhea > vomiting
  → Toxin produced in gut

Mnemonic (How to remember)

Master Mnemonic: "Some Bad Clams Cook Beef"

S → ***S****taphylococcus aureus*

B → ***B****acillus cereus*

C → ***C****lostridium botulinum*

C → ***C****lostridium perfringens*

"Some Bad" → FAST vomiting (preformed toxins)

S*taphylococcus aureus*

B*acillus cereus* (emetic)

1–6 hours=Vomiting dominant

"Clams Cook Beef" → SLOW toxins (after ingestion)

C*lostridium botulinum* → **Neurotoxin (descending paralysis)**

C*lostridium perfringens* → **Watery diarrhea**

Longer incubation

Cell invasion

Shigella

Salmonella enteritidis

EHEC

EIEC

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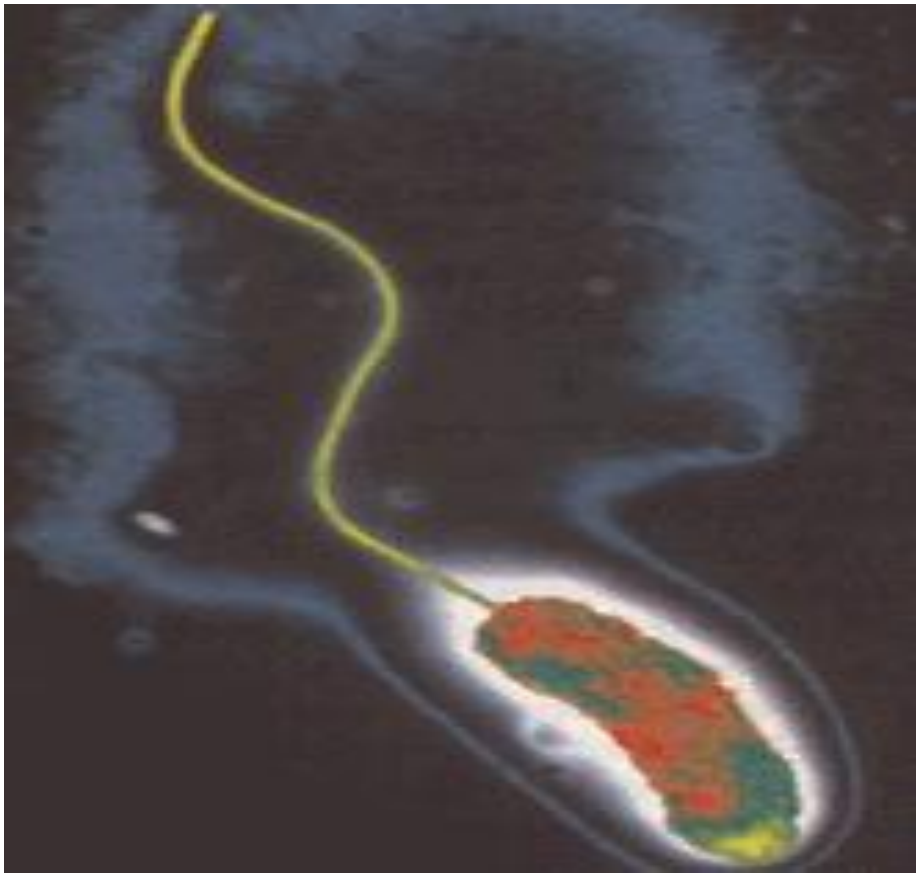
Vibrio Cholera



Watery (secretory) diarrhea

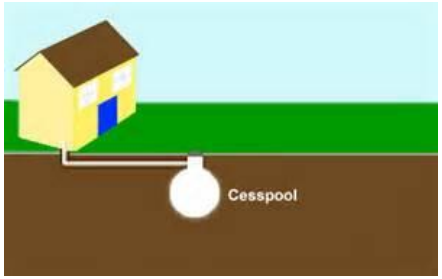
Vibrio Cholera

Basics

- **Oxidase positive**
- **Alkaline-loving** (grows in high pH)
- Found in **contaminated water / seafood**

Pathogenesis:

- Colonizes **small intestine (non-invasive)**
- Produces **cholera toxin (AB toxin)**:
  - **A subunit** → activates **adenylate cyclase**
  - ↑ **cAMP**
  - massive **Cl^- + H_2O secretion**
- Result: **profuse watery diarrhea**

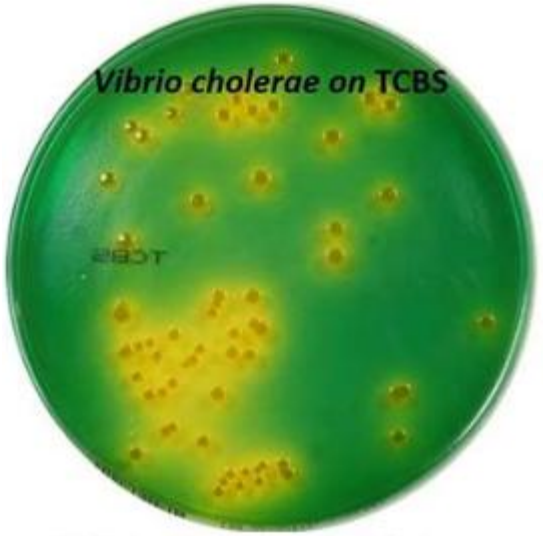


Watery (secretory) diarrhea

Vibrio Cholera

Lab / Diagnosis

- Stool: **no RBCs/WBCs**
- Culture on **TCBS agar** (yellow colonies)
- Clinical diagnosis in outbreaks



Vibrio cholerae on TCBS Agar

Treatment:

- **Immediate rehydration (oral or IV Ringer lactate)**
- **Doxycycline / azithromycin → ↓ duration**
- **Zinc in children**

Watery (secretory) diarrhea

Vibrio Cholera

Clinically

- **Rice-water diarrhea** (watery + mucus/cells)
- Nausea, vomiting, muscle cramps
- **Severe dehydration** → dry mouth, thirst, ↓ BP, arrhythmia
- Signs: **sunken eyes, poor skin turgor, ↓/no urine**

Complications

- Hypovolemic shock
- Metabolic acidosis (HCO_3^- loss)
- Hypokalemia

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Enteropathogenic vs. Enterotoxigenic E. coli

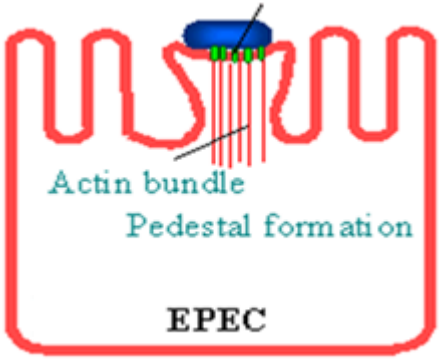
EPEC vs. ETEC

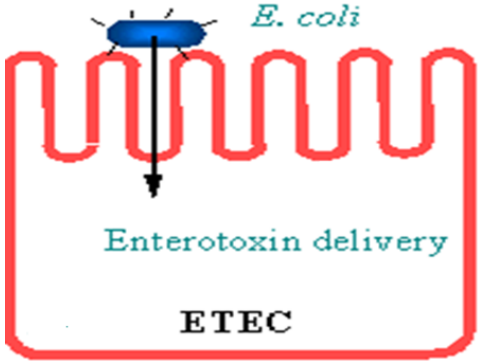
Feature	EPEC	ETEC
Mechanism	Attaching & effacing → destroys microvilli	Toxins (LT, ST) → ↑ cAMP / cGMP
Effect	↓ absorption	↑ secretion
Diarrhea	Watery (no toxin)	Watery (secretory)
Fever	Usually mild/absent	Absent
Population	Infants	Travelers ("traveler's diarrhea")
Invasion	No	No

pathogenic=

pediatrics

Attachment and eliminating villi





*Entero**T**oxigenic*

= **T**ravelers

Cholera vs. EPEC vs. ETEC

Feature	Cholera	ETEC	EPEC
Stool	Rice-water	Watery	Watery
Population	Endemic areas	Travelers	Infants
Mechanism	↑cAMP (CFTR)	↑cAMP & ↑cGMP	↓ absorption
Severity	Severe dehydration	Mild	Chronic

Case

A 24-year-old medical student returns from a trip to rural India with **profuse watery diarrhea**, abdominal cramps, and no fever. Symptoms started 2 days after eating street food. No blood or mucus is present.

Question

Most likely organism?

A. Escherichia coli (ETEC)
B. Shigella
C. Salmonella
D. Vibrio cholerae
E. Entamoeba histolytica

Answer: A. ETEC

Parasitic Infections of GIT (B. coli, G. lamblia)

Parasites

Protozoa

Helminths

Single celled organisms

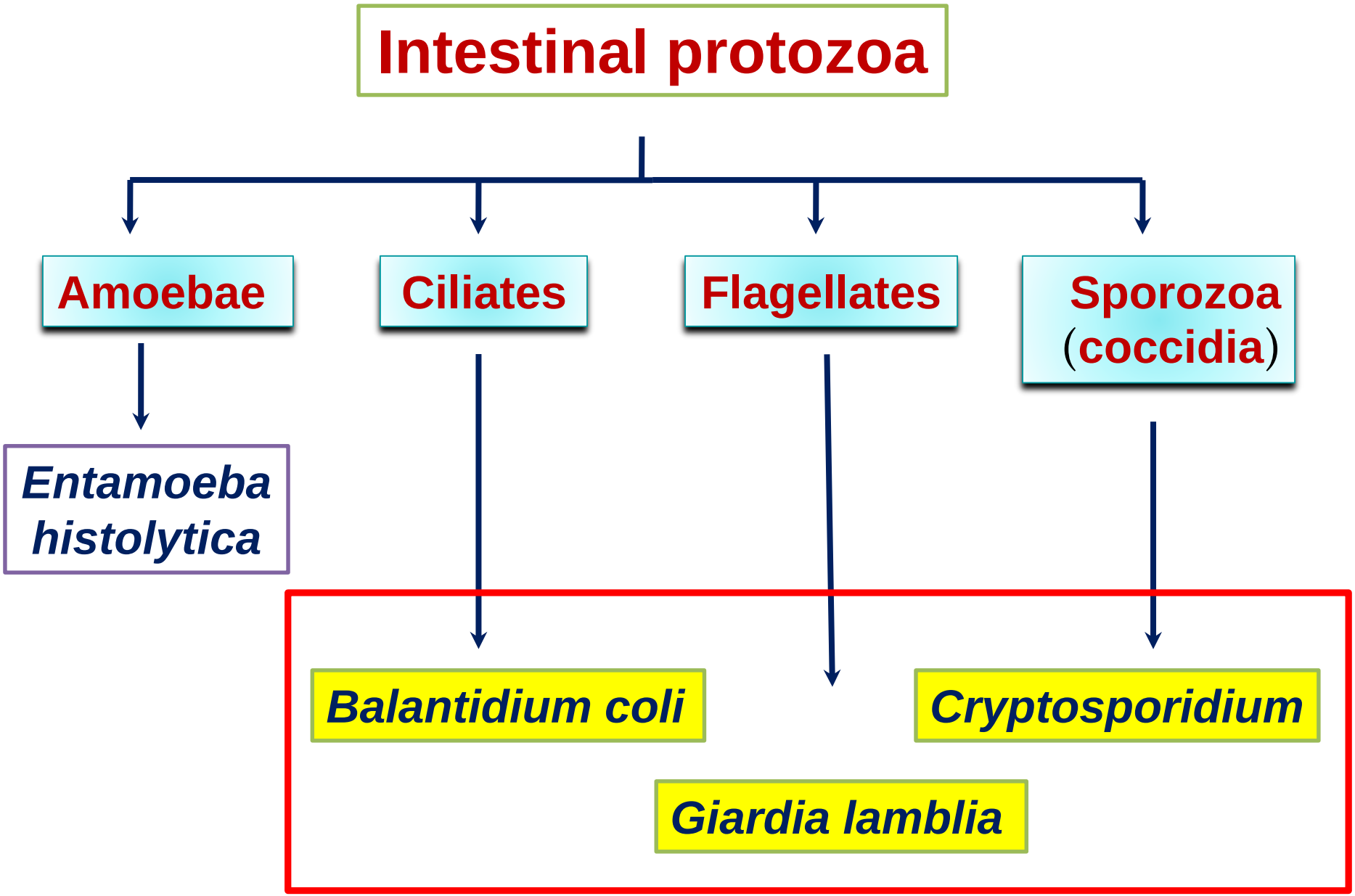
Classification based on motility

Pseudopodia → Amoebae

Flagellates → Flagella

Ciliates → Cilia

Sporozoa (**coccidia**) → Non-motile
Obligatory intracellular


Intestinal protozoa
Amoebae
Ciliates
Flagellates
Sporozoa (coccidia)
Entamoeba histolytica
Balantidium coli
Giardia lamblia
Cryptosporidium

Balantidium coli
The largest protozoa

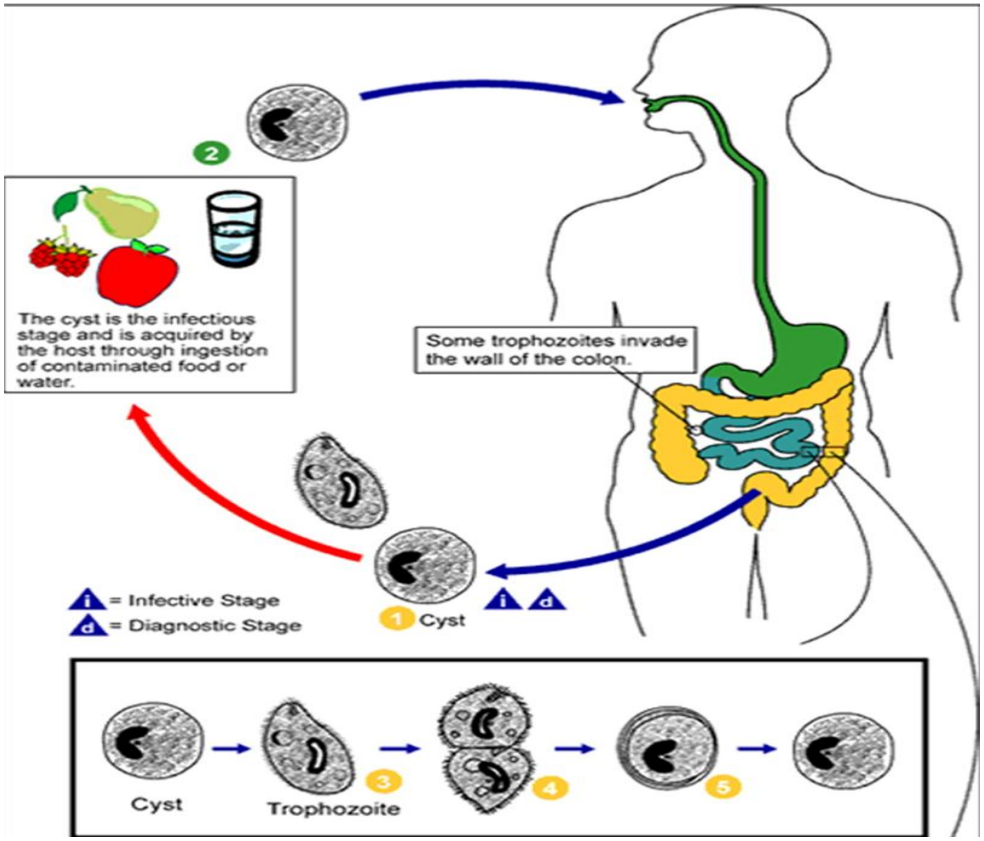
ONLY ciliated protozoan infecting humans

- Exists as:
  - **Trophozoite** (ciliated, motile)
  - **Cyst** (infective form)

Source / Transmission

- **Pig reservoir** (major clue)
- Fecal-oral transmission via:
  - Contaminated **water/food**
  - Food-handlers

Clues: *Farmer + pigs + diarrhea*



Balantidium coli **life cycle**

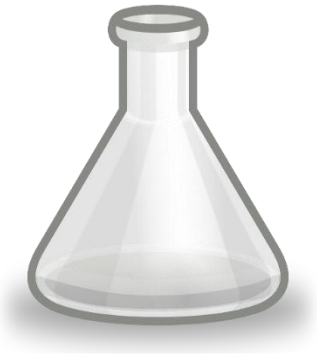
Balantidium coli
The largest protozoa

Pathogenesis

- Invades **colon mucosa**
- Causes **ulceration** → similar to Amoebic dysentery

Clinical Presentation

- **Bloody diarrhea** (can mimic amoebiasis)
- Abdominal pain
- Tenesmus
- Sometimes **asymptomatic**



Laboratory diagnosis

a) Direct

- Foul **bloody + mucoid diarrhea**
- **Stool exam (key)**
  - Trophozoites → loose stool
  - Cysts → formed stool
  - **Cilia (diagnostic!)**
- **Sigmoidoscopy** → ulcers ± trophozoites
- **Barium enema** → ulcers / stricture

b) Indirect

-Serological tests: CFT, IHAT, IFAT, ELISA and GDPT (gel-diffusion precipitin test).

Hint: **Pig + Cilia = Balantidium coli"**

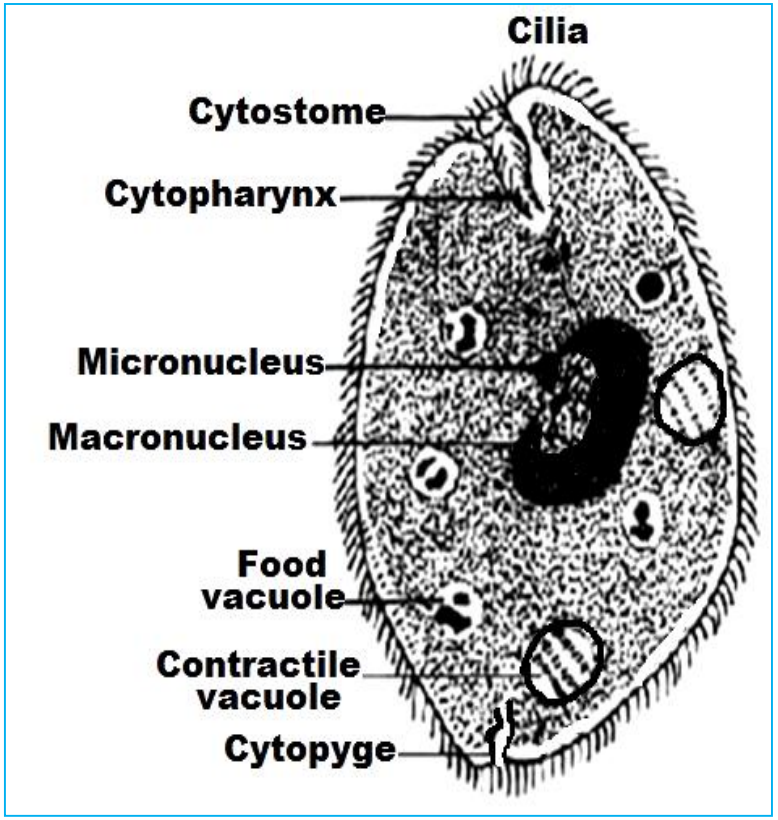
+

Treatment

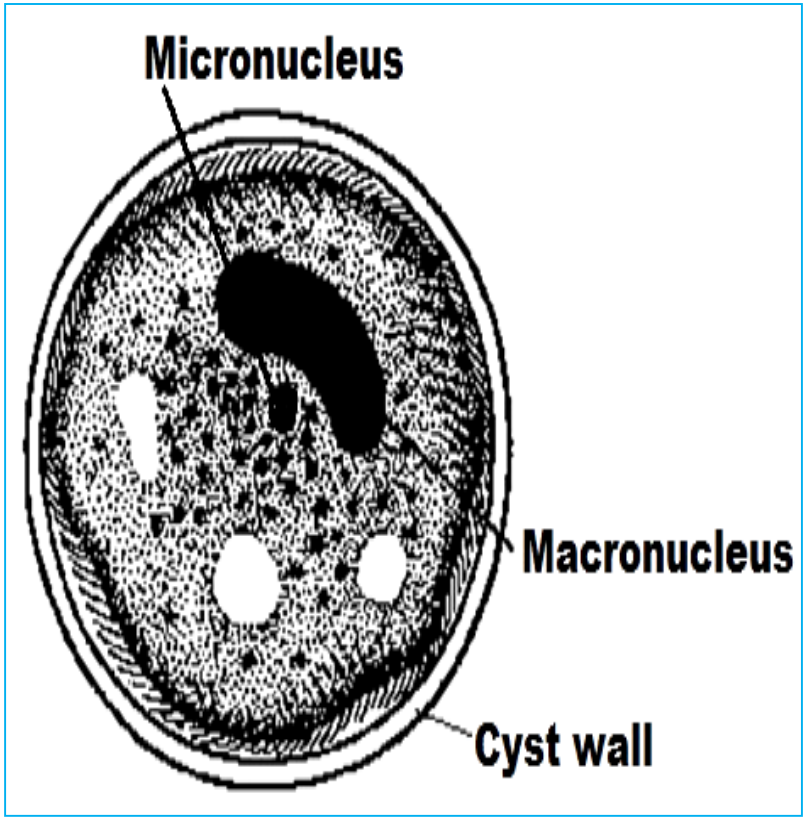
- **Tetracycline** (drug of choice)
- Alternatives:
  - Metronidazole

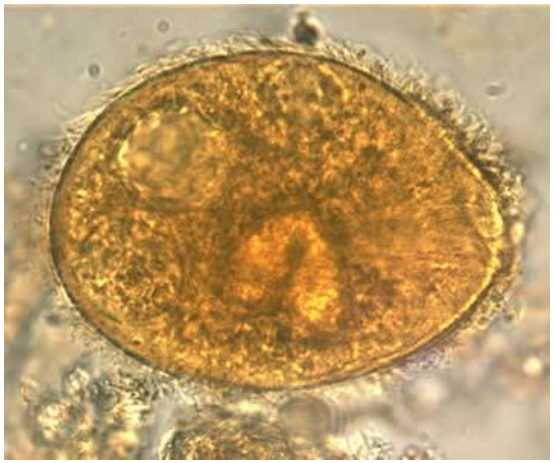
Morphological characters

1- Trophozoite stage

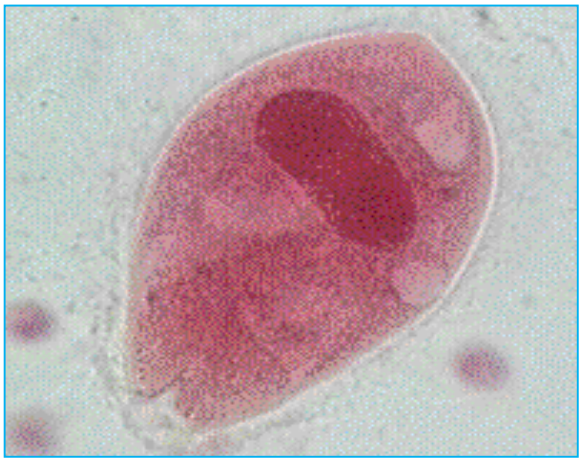


2- Cyst (I.S)





B. coli **trophozoite**



B. coli **trophozoite**

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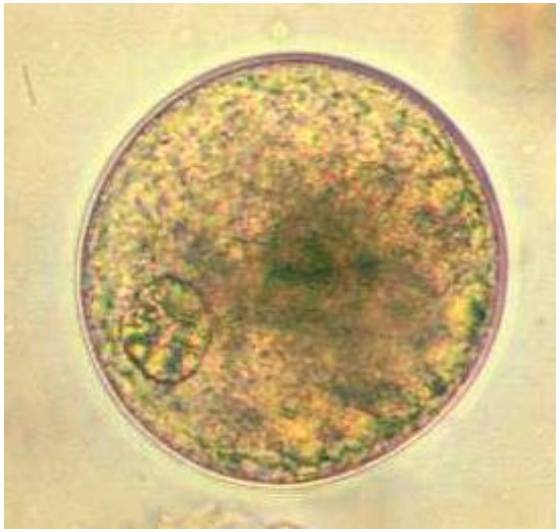


B. Coli **cyst**

Giardia lamblia

· **Distribution:** Cosmopolitan; more common in **tropical & subtropical regions**

· **Habitat: Small intestine (duodenum crypts)** → may also involve **bile duct & gallbladder**

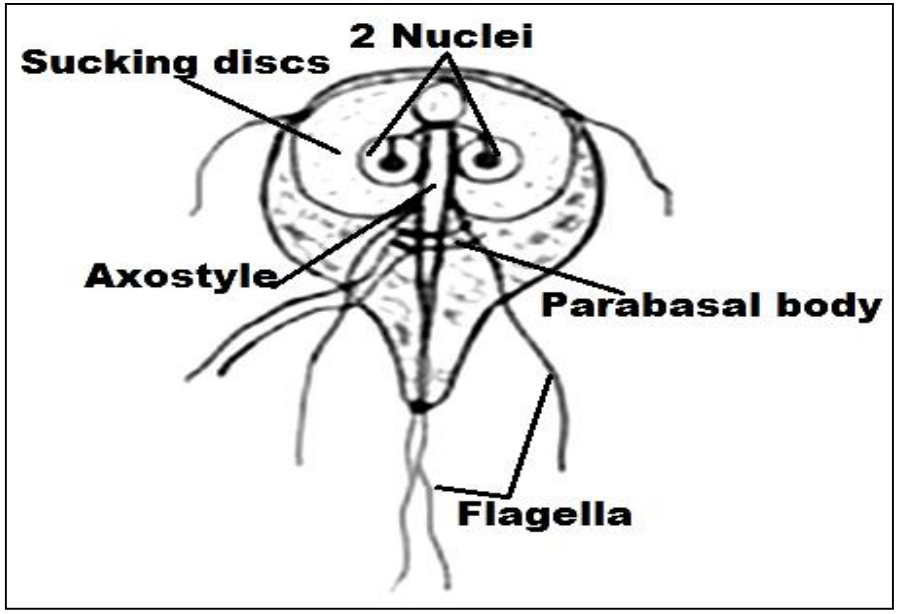
· **Definitive host: Humans**

· **Type: Opportunistic protozoan parasite** (flagellated)

· Key idea: intestinal + possible biliary involvement in chronic cases

Morphological characters

1-Trophozoite stage



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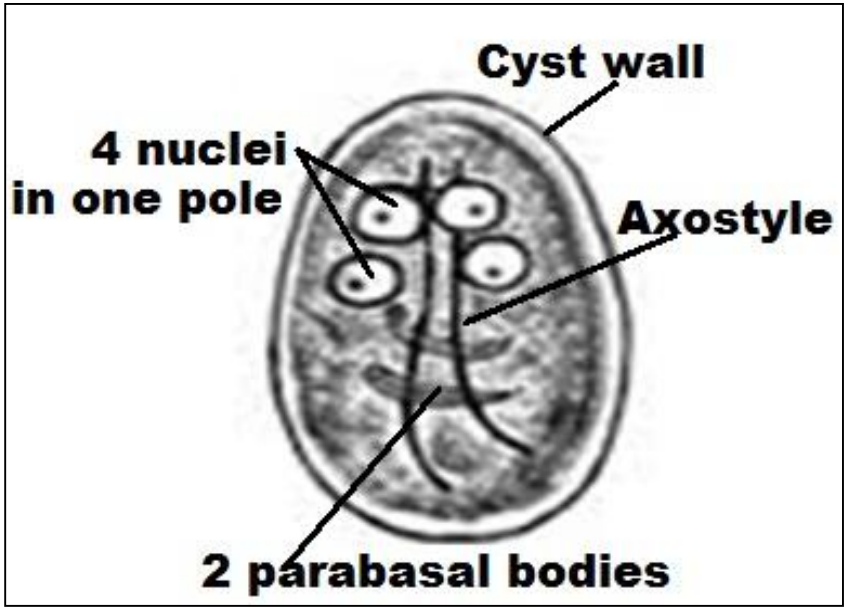
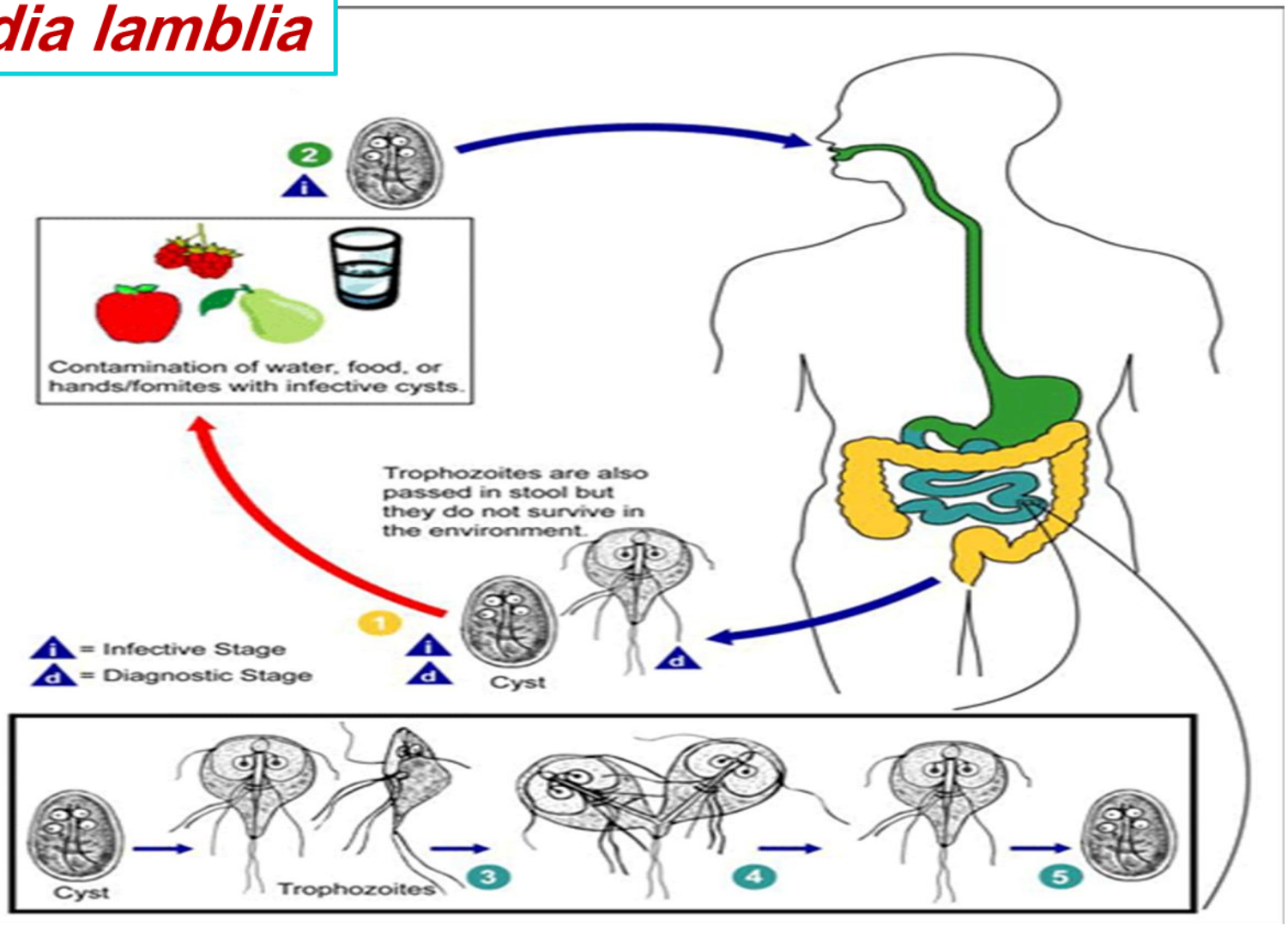


Giardia lamblia



Mode of transmission:

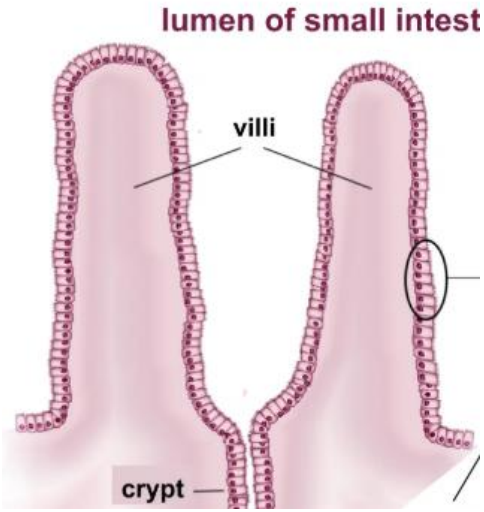
The same as *Balantidium coli*

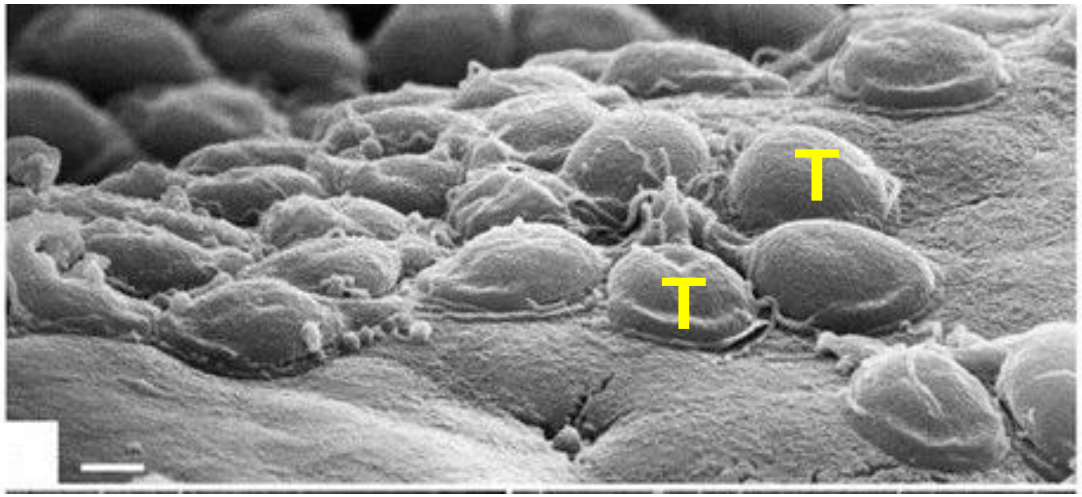
Pathogenesis and symptomatology

Disease: Giardiasis

Predisposing factors for the development of infection with *G. lamblia*

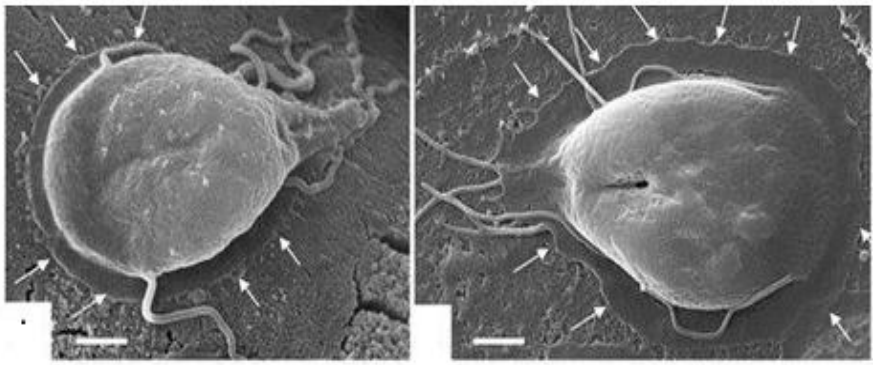
- Hypogammaglobulinaemia.
- Low level of secretory IgA in the gut.
- Achlorhydria (decreased HCL).
- Malnutrition.





Trophozoites (T) attaching to the intestinal surface of an experimentally infected mouse.

“carpet-like layer”



Higher magnification view of a trophozoite attaching to the mouse and to human intestinal surface cells

Treatment

1- Metronidazole (Flagyl).

2-Nitazoxanide

Clinical pictures

1-Asymptomatic infection

Most common. (Asymptomatic patient known as a healthy carrier).

2-Symptomatic infection

Acute giardiasis

Risk: children, travelers
Diarrhea: watery, **mucus**, **NO blood**
Symptoms: bloating, flatulence, abdominal pain
Late: **steatorrhea → weight loss**
Dx: trophozoites in stool
Rare: biliary → jaundice
Course:
Normal → self-limited
IgA ↓ → chronic, severe malabsorption
Watery non-bloody + fatty stool = Giardia

Chronic giardiasis

complications

2-Symptomatic infection

- **Acute giardiasis**
- **Chronic giardiasis**
  - **- Common in adults.**
  - **-The patient suffers from anorexia, epigastric pain, dyspepsia, nausea, vomiting & diarrhea alternating with constipation. Only cysts are found in stool.**
- **Complications**
  - **1- Retardation of growth & development in infant and young children.**
  - **2- Malnutrition and malabsorption syndrome.**
  - **3- Biliary tract disease.**

Giardia and Steatorrhea

Giardia + Bile Salts

Normal

- Liver conjugates bile acids (glycine/taurine)
  -makes them **water-soluble + strong detergents**
  -forms **micelles → fat absorption**

Giardia effect

- Duodenal attachment → **mucosal injury + stasis**
- **Bacteria effect (SIBO-like)**
- Overgrowth → **bile salt hydrolase (BSH)= deconjugates bile salts**

Final result

↓ detergent activity

↓ micelles

↓ fat absorption

= **Steatorrhea**

Laboratory diagnosis

- Stool O&P: cysts (formed stool), trophozoites (watery stool)
- Stool antigen test (ELISA/DFA): most sensitive
- Duodenal aspirate / string test: if stool negative
- Intermittent shedding → repeat stool samples (×3)

USMLE Case: *Giardia lamblia*

A 28-year-old man returns from a camping trip and presents with **2 weeks of foul-smelling, bulky, greasy diarrhea**. He also reports **abdominal bloating, flatulence, and weight loss**. No blood or fever. Several others who drank from the same stream have similar symptoms.

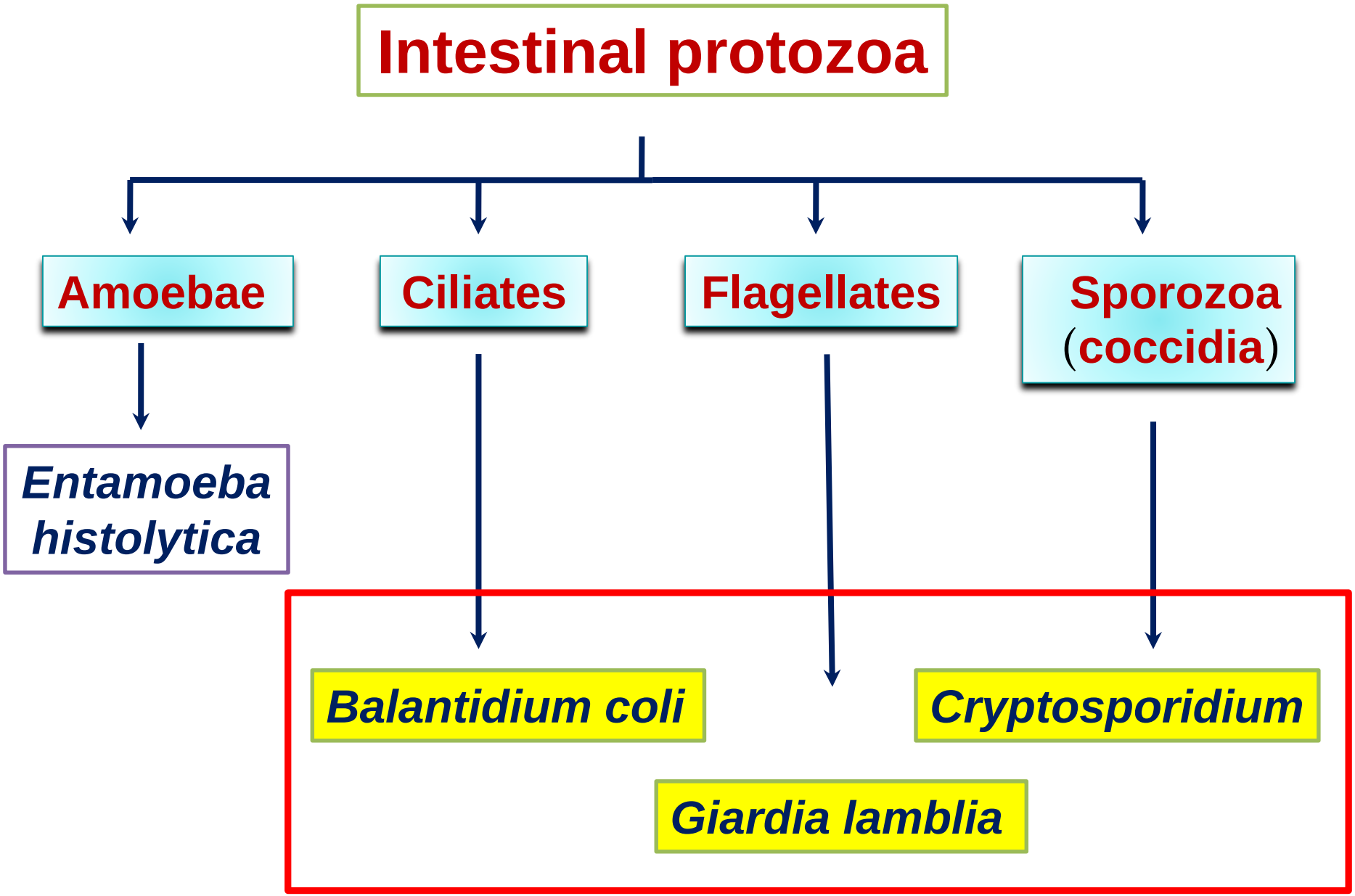
On exam: mild dehydration. Stool exam shows **pear-shaped trophozoites with flagella**.

Most likely organism?

Giardia lamblia

Why this is Giardia (exam reasoning)

- **Camping / untreated stream water**
- **Greasy (steatorrhea)** → fat malabsorption
- **No blood, no fever** → non-invasive
- **Bloating + flatulence** → carbohydrate malabsorption
- **Outbreak in group** → waterborne spread


Intestinal protozoa
Amoebae
Ciliates
Flagellates
Sporozoa (coccidia)
Entamoeba histolytica
Balantidium coli
Giardia lamblia
Cryptosporidium

Cryptosporidium

Geographical distribution: Cosmopolitan especially among immunosuppressed patients.

D.H & I.H: Man

R.H: Domestic animals such as cattle, sheep, goats & dogs.

Habitat:

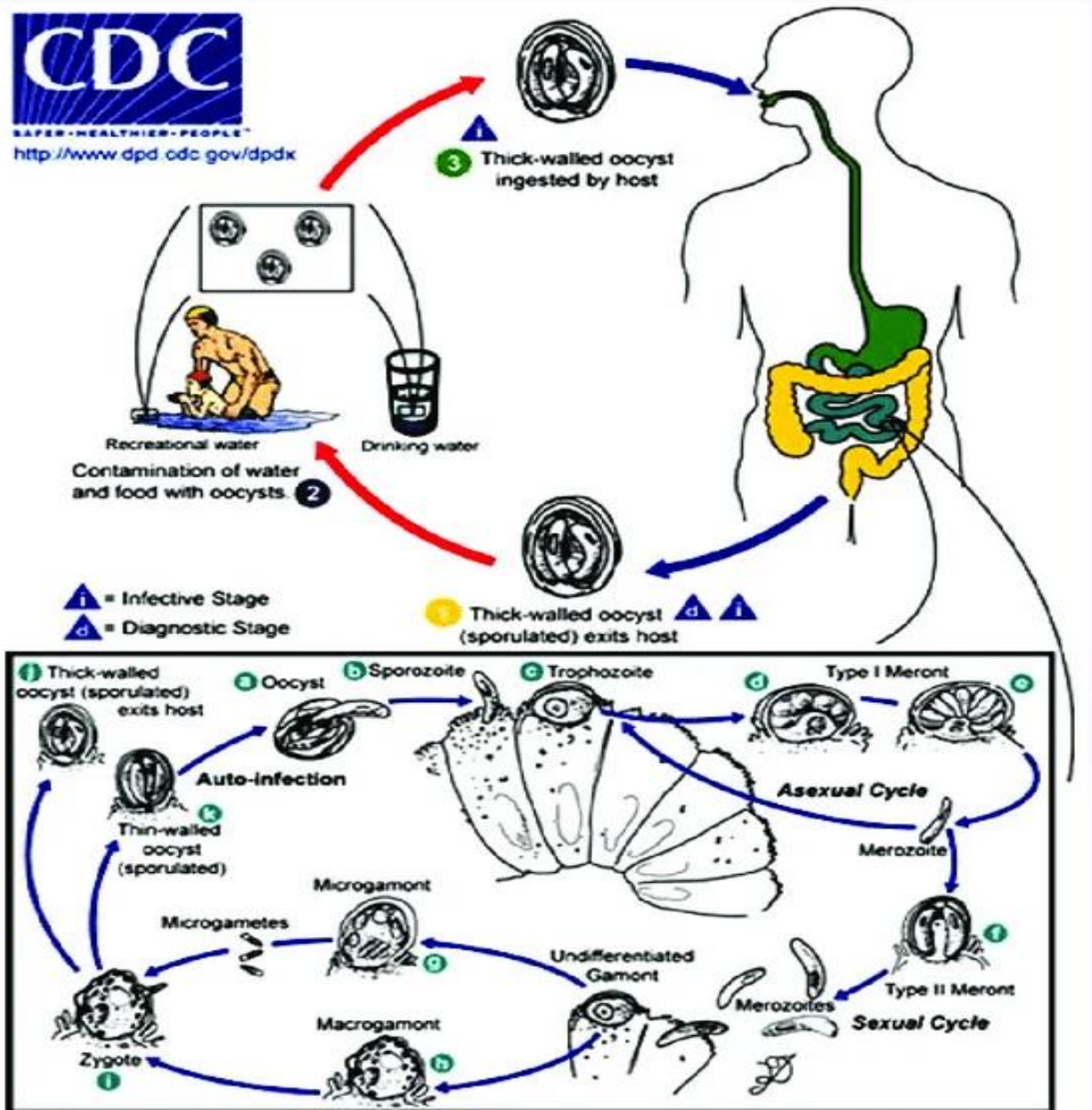
-Mainly in the brush border of the small & large intestine.

-In the epithelium of the respiratory and biliary tracts (in immunocompromised individuals).

***Cryptosporidium* species are:**

1- *C. parvum*. 2- *C. muris*. 3- *C. bovis*.

Cryptosporidium



Cryptosporidium

What is it?

- A protozoan parasite that infects the small intestine
- Causes watery diarrhea
- Important species: *Cryptosporidium parvum* and *Cryptosporidium hominis*

Cryptosporidium

Transmission

- Fecal–oral route
- Contaminated:
  - Water (most common)
  - Food
- Resistant to chlorine → can survive in swimming pools

= “Waterborne parasite”

Cryptosporidium

Pathogenesis:

- Lives on the brush border of intestinal epithelial cells
- Damages absorption → secretory diarrhea
- Does NOT deeply invade like some parasites

Cryptosporidium

Clinical Features

Immunocompetent:

- Self-limited **watery diarrhea**
- Mild:
  - Abdominal cramps
  - Nausea
  - Low-grade fever

Immunocompromised

e.g. **HIV/AIDS patients**

- Severe:
  - **Chronic, life-threatening diarrhea**
  - Dehydration
  - Weight loss

"Mild in healthy, deadly in AIDS"

Cryptosporidium

Diagnosis

- Stool exam shows:
  - **Oocysts**
- Special stain:
  - **Acid-fast stain (modified Ziehl–Neelsen)**

= **"Acid-fast oocysts"**

Treatment

Normal patients:

- Usually **self-limited**
- ± **Nitazoxanide**

Immunocompromised:

- Restore immunity (e.g., **antiretroviral therapy** in HIV)
- Supportive care (fluids

Cryptosporidium vs. Giardia

Feature	Cryptosporidium	Giardia
Stool	Watery	Greasy (fatty)
Blood	No	No
Exposure	Pools/daycare	Streams/camping
Mechanism	Secretory	Malabsorption
Stain	Acid-fast	None

Thank You