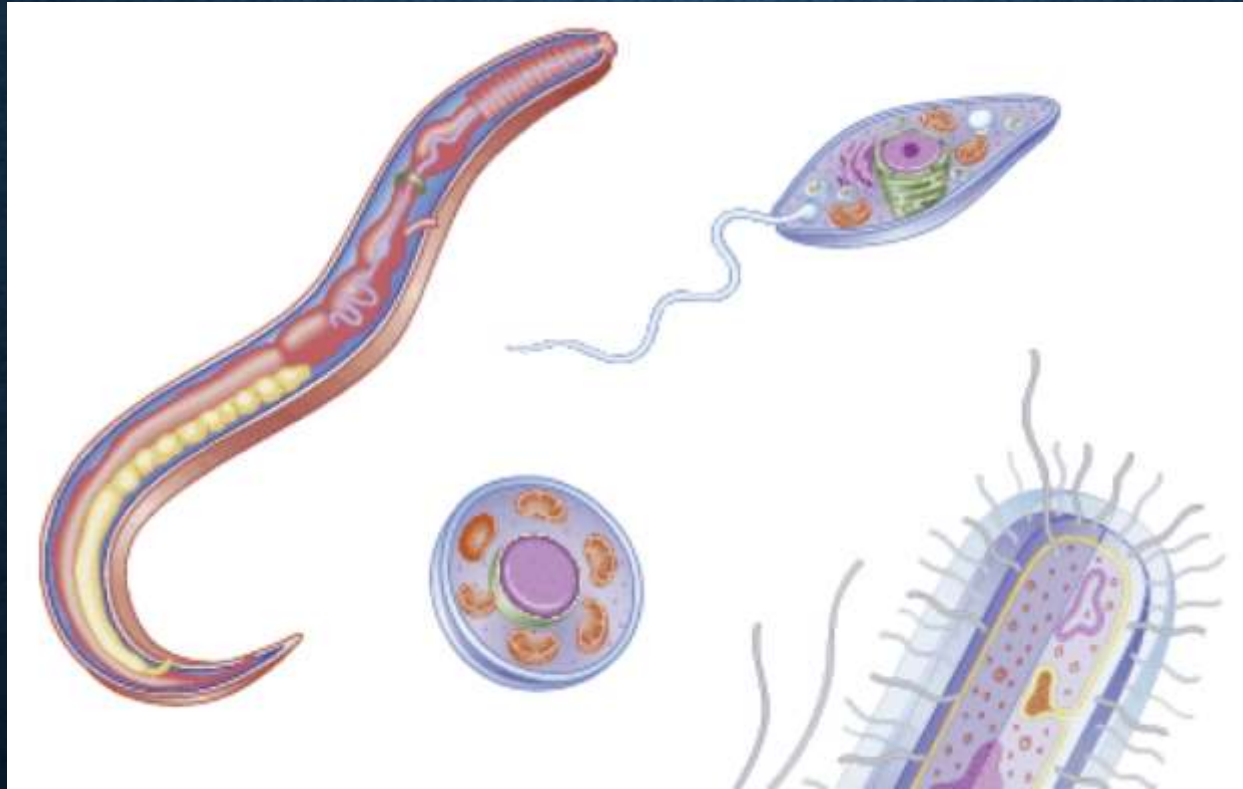


INTESTINAL PROTOZOAN INFECTIONS



TOPICS

- Intestinal amoebiasis
- Amoebae resembling *E.histolytica*
- Giardiasis
- Opportunistic intestinal coccidian parasitic infections
- Balantidiasis

INTESTINAL PROTOZOAN INFECTIONS

The protozoan parasites which cause intestinal infection include:

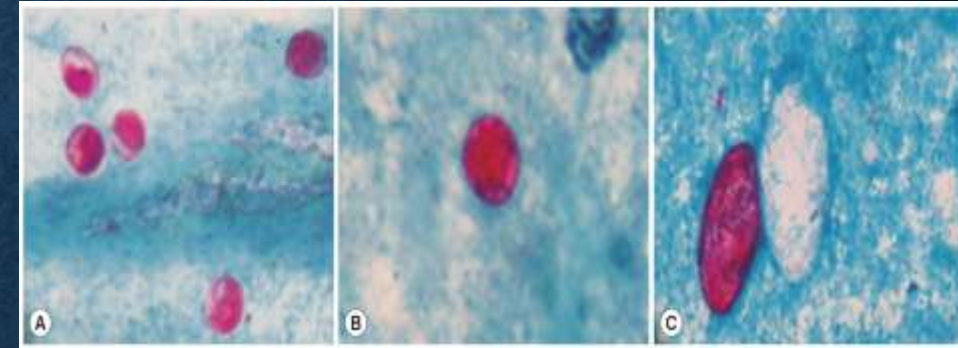
- Intestinal amoebae: *Entamoeba histolytica*
- Intestinal flagellate: *Giardia lamblia*
- Opportunistic intestinal coccidian parasites: *Cryptosporidium*, *Cyclospora*, and *Cystoisospora*
- Others: *Balantidium coli*, *Blastocystis hominis*, *Sarcocystis* and Microsporidia (now considered as fungi).



Entamoeba histolytica



Giardia lamblia



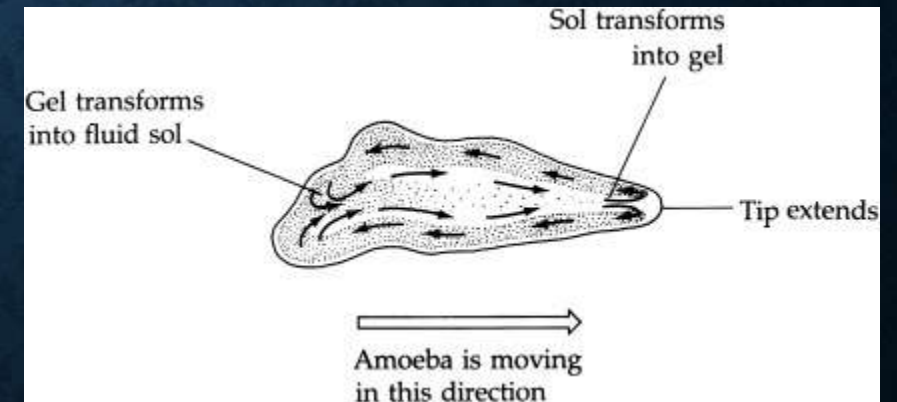
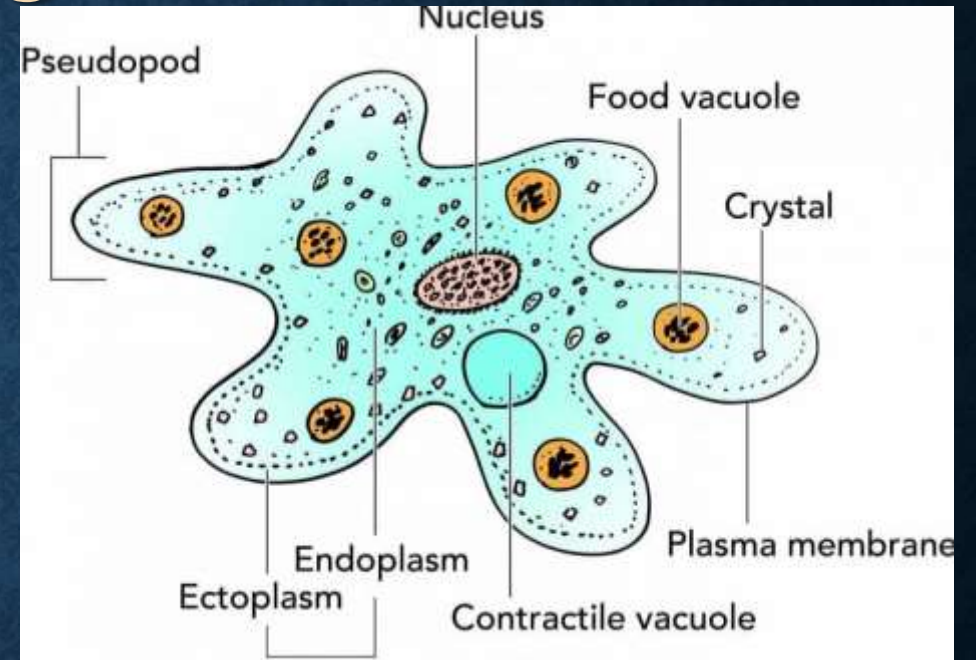
Cryptosporidium, *Cyclospora*, and *Cystoisospora*



Balantidium coli

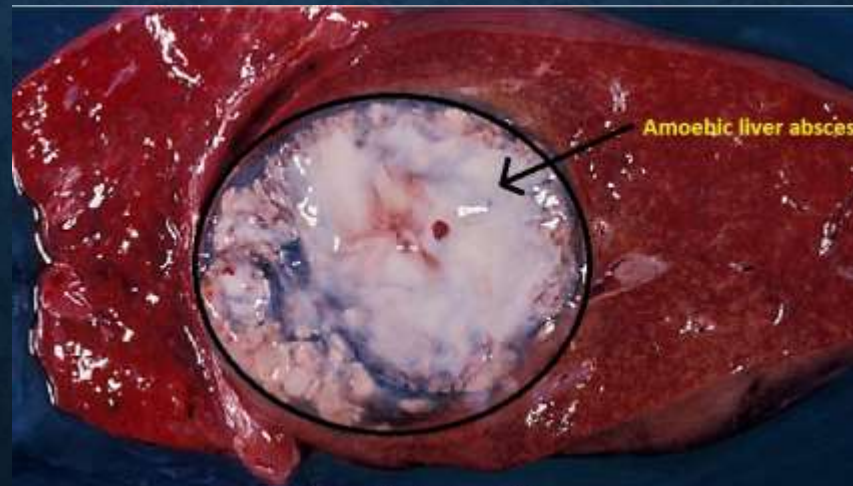
INTESTINAL AMOEBIASIS

- Amoebae (meaning “change”) - single-celled protozoan parasites that constantly change their shape - due to presence of an organ of locomotion called as “pseudopodium”.
- Based on the habitat they are of two types -
 - (i) intestinal amoebae, and
 - (ii) free-living amoebae.



ENTAMOEBA HISTOLYTICA

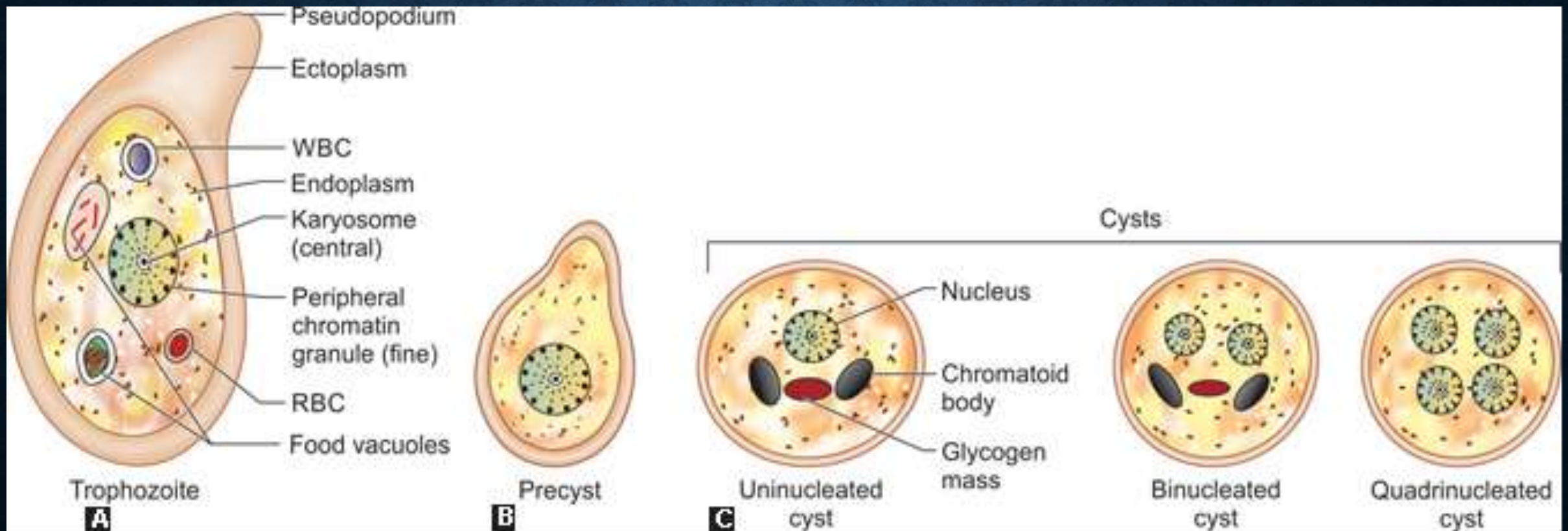
- *E. histolytica* causes amoebic dysentery and a wide range of other invasive diseases, including amoebic liver abscess.
- There are three other species of *Entamoeba* such as *E. dispar*, *E. moshkovskii* and *E. bangladeshi* which are morphologically similar to *E. histolytica*.



EPIDEMIOLOGY

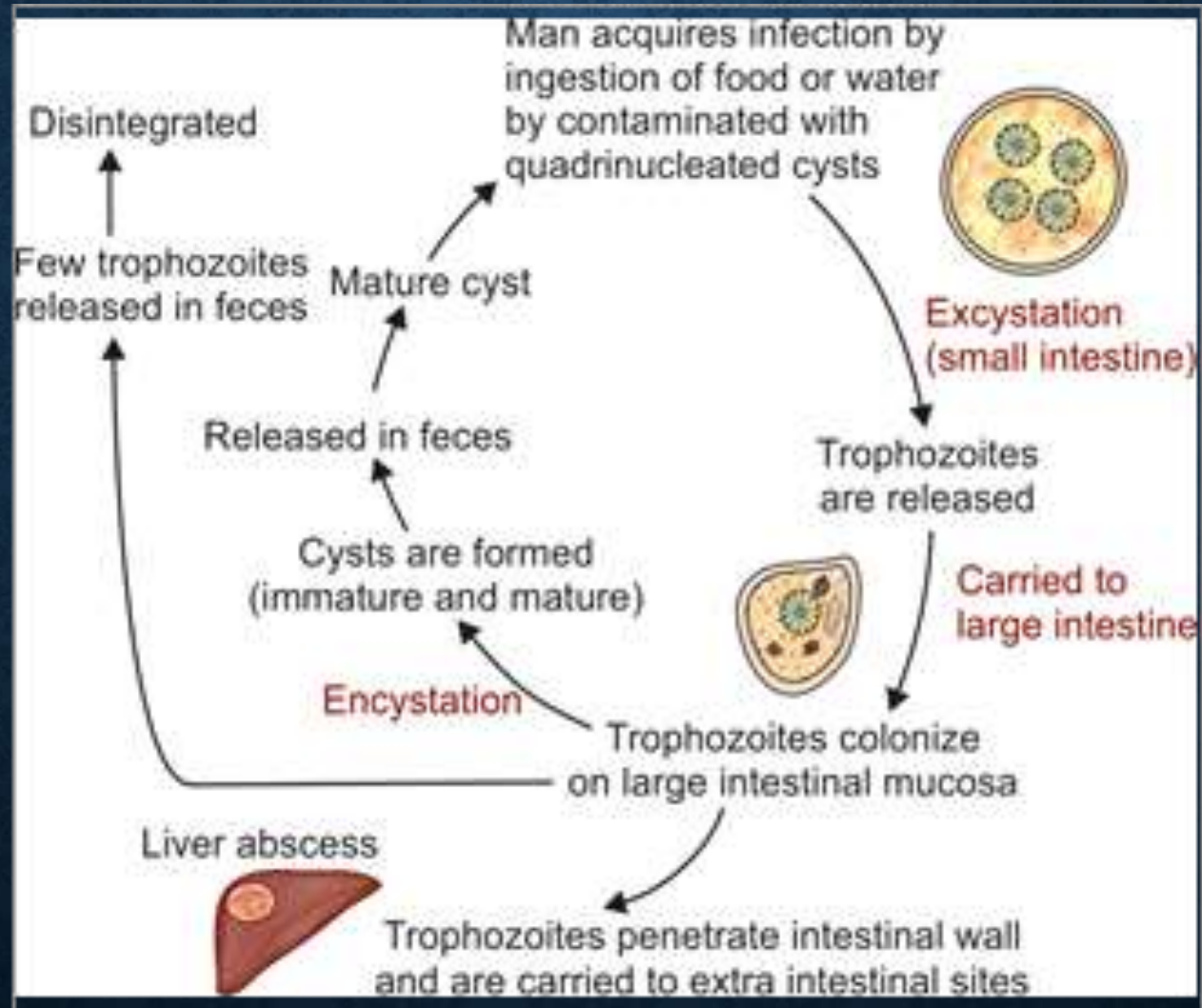
- *E. histolytica* is worldwide in distribution but more common in tropical and subtropical countries.
- Largest burden of the disease - tropics of China, Central and South America, and Indian subcontinents affecting 10% of the world's population (500 million)
- Third most common parasitic cause of death in the world.

MORPHOLOGY



Infective form

LIFE CYCLE

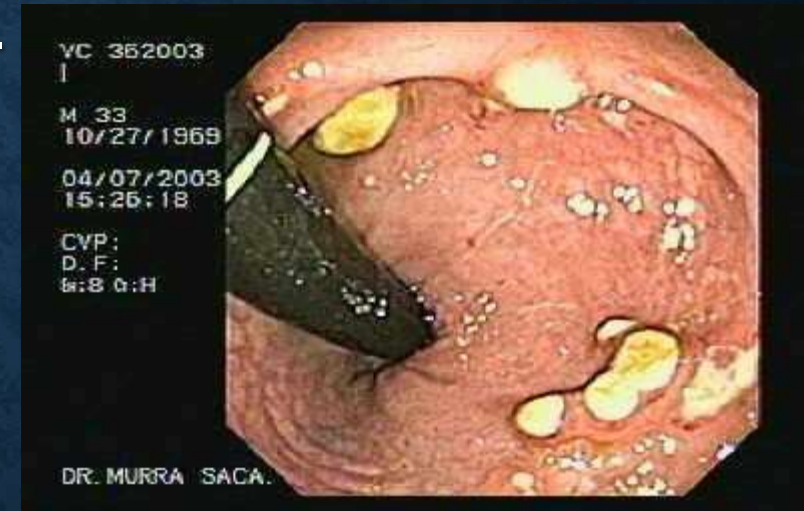


VIRULENCE FACTORS

- Trophozoite of *E. histolytica* - invasive form and possesses many virulence factors that play an important role in the pathogenesis.
 - Amoebic lectin antigen
 - Amoebapore
 - Cysteine proteases and hydrolytic enzymes
 - Neuraminidase and metallocollagenase

PATHOGENESIS

- **Colonization:** Trophozoites first colonize the intestinal mucosa.
- **Adhesion:** Then the trophozoites penetrate the mucus layer and adhere to the large intestinal mucosa by - Gal/GalNAc lectin molecules
- **Flask-shaped ulcers**
- **Invasion:** Amoebae then invade the large intestinal wall - migrate to extraintestinal sites.
- **Intestinal complications**

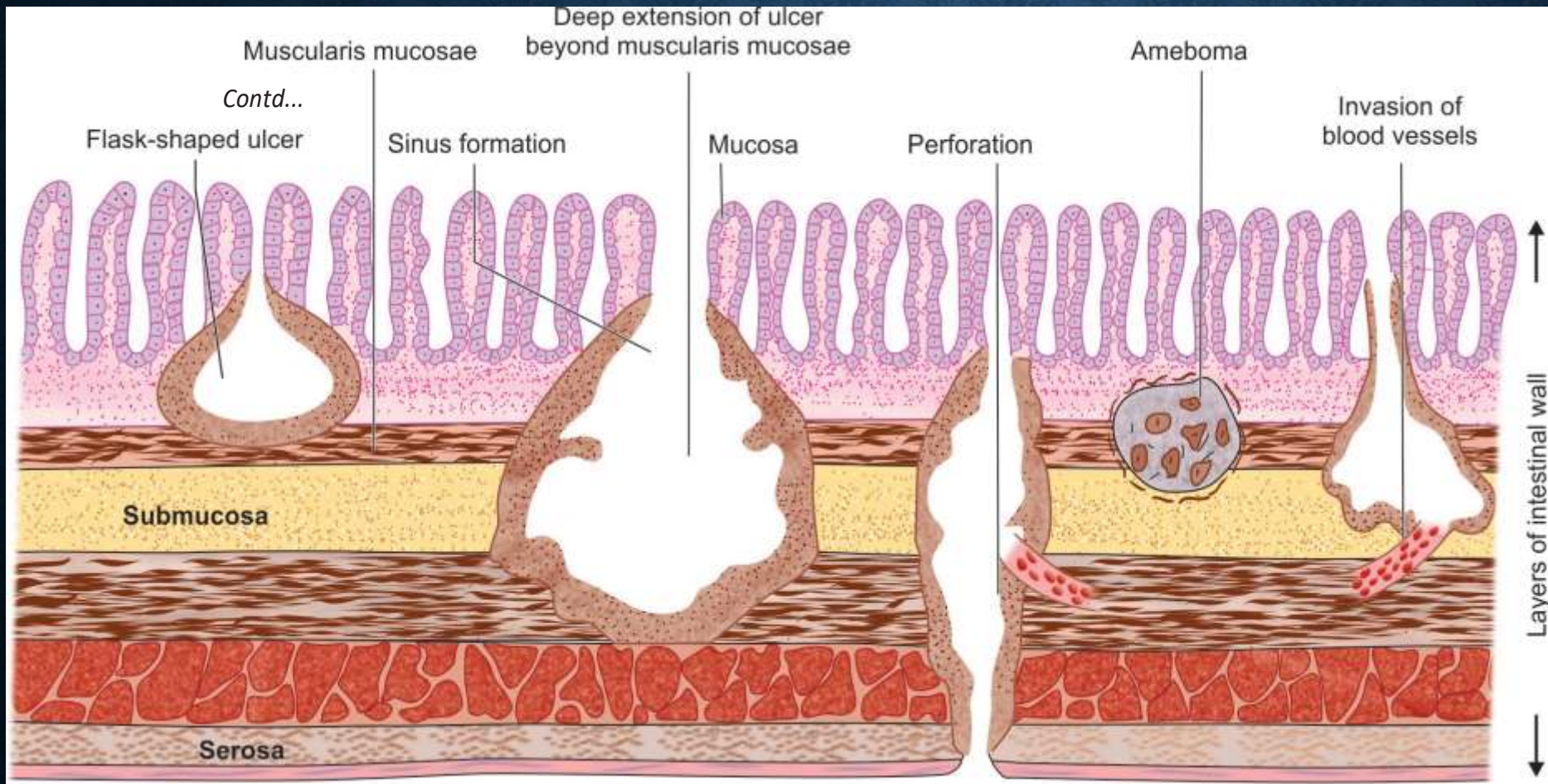


Flask-shaped ulcers

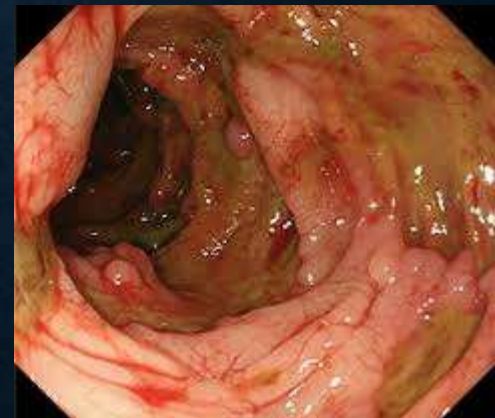
CLINICAL MANIFESTATIONS

- Incubation period - one to four weeks.
- Males and females are affected equally.
- Majority of infections (90%) result in asymptomatic cyst passers.
- The remaining (10%), develop intestinal amoebiasis:
 - ❑ **Amoebic dysentery**
 - ❑ **Amoebic appendicitis**
 - ❑ **Amoeboma**
 - ❑ **Fulminant colitis**

COMPLICATIONS OF INTESTINAL AMOEBIASIS



Amoeboma



Fulminant colitis

DIFFERENCES IN STOOL CHARACTERISTICS BETWEEN AMOEBIC DYSENTERY AND BACILLARY DYSENTERY

Character	Amoebic dysentery	Bacillary dysentery
<i>Pathology</i>		
Ulcer	Deep	Shallow
Margin	Ragged and undermined	Uniform
Intervening mucosa	Normal	Inflamed
Cellular response	Mononuclear	Polymorphonuclear

DIFFERENCES IN STOOL CHARACTERISTICS BETWEEN AMOEBIC DYSENTERY AND BACILLARY DYSENTERY

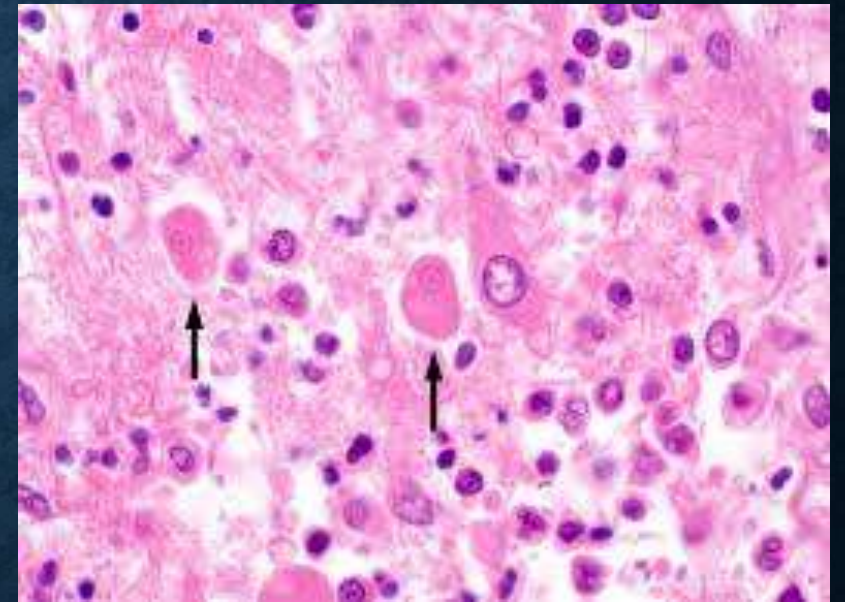
Character	Amoebic dysentery	Bacillary dysentery
<i>Stool macroscopic feature</i>		
Number of motion	6-10/day	> 10/day
Amount	Copious amount	Small quantity
Color	Dark red	Bright red
Odor	Offensive	Odorless
Reaction	Acidic	Alkaline
Consistency	Not adherent to the container	Adherent to the container

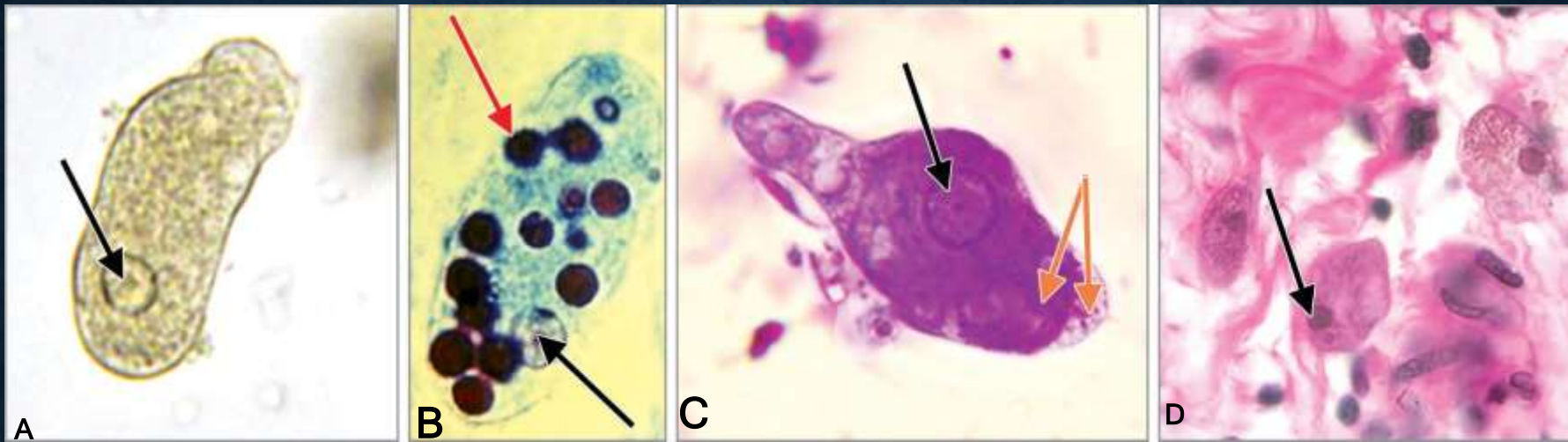
DIFFERENCES IN STOOL CHARACTERISTICS BETWEEN AMOEBIC DYSENTERY AND BACILLARY DYSENTERY

Character	Amoebic dysentery	Bacillary dysentery
<i>Stool microscopic feature</i>		
Red blood cells (RBCs)	In clumps	Discrete or in rouleaux
Pus cells	Few	Numerous
Macrophages	Few	Numerous*
Eosinophils	Present	Absent or rare
Charcot-Leyden crystal	Present	Absent
Pyknotic body**	Present	Absent
Organism detected	<i>E. histolytica</i> cyst or trophozoite	Bacteria (e.g. <i>Shigella</i>)

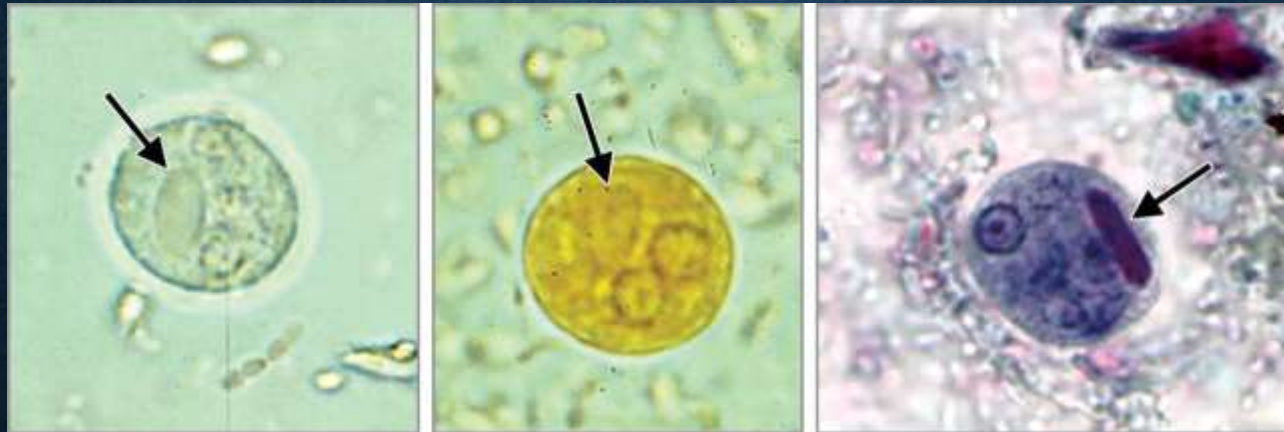
LABORATORY DIAGNOSIS OF INTESTINAL AMOEBIASIS

- **Stool microscopy** by wet mount, permanent stains, etc.— detects cysts (round, 1–4 nuclei) and trophozoites (with finger like pseudopodia)
- **Histology**—Intestinal biopsies stained with PAS or H&E stains reveal trophozoites
- **Stool culture**—Polyxenic and axenic culture





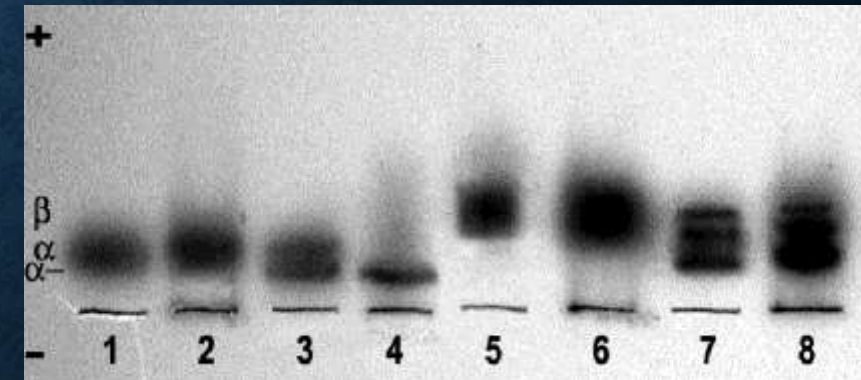
Entamoeba histolytica trophozoites: A. Saline mount; B and C. Trichrome stain, note the single nucleus (black arrow) RBCs (red arrow) and ectoplasm and endoplasm (orange arrows); D. Trophozoites in colon tissue stained with H&E.



Cyst of *E. histolytica*: A. Saline mount; B. Iodine mount; C. Trichrome stained smear. Note the chromatoid body with blunt ends (arrow).

LABORATORY DIAGNOSIS OF INTESTINAL AMOEBIASIS

- **Stool antigen detection** (copro-antigen)—ELISA (detecting 170-kDa of lectin Ag) and ICT (detecting 29-kDa surface Ag).
- **Serology**
 - Amoebic antigen—ELISA (170-kDa of lectin Ag)
 - Amoebic antibody—ELISA
- **Isoenzyme** (zymodeme) analysis
- **Molecular diagnosis**—Nested multiplex PCR and real time PCR (18S rRNA) and Biofire FilmArray



Isoenzyme (zymodeme) analysis

TREATMENT OF INTESTINAL AMOEBIASIS

- **Asymptomatic carriage:** Treatment helps in reducing passage of cysts in stool and thereby preventing the disease transmission.
- Luminal agents are used for treatment.
 - Iodoquinol (for 20 days) or
 - Paromomycin (for 10 days)

TREATMENT OF INTESTINAL AMOEBIASIS

- **Amoebic dysentery:** Treatment consists of:
 - *Tissue agents:* Metronidazole (for 5–10 days) or tinidazole (for 3 days) with
 - Luminal agents
 - **Other measures** include fluid and electrolyte replacement and symptomatic treatment.

PREVENTION

- Preventive measures:
- Avoiding ingestion of food and water contaminated with human feces
- Treatment of asymptomatic carriers.

AMOEBAE MORPHOLOGICALLY RESEMBLING *E. HISTOLYTICA*

- *E. dispar*, *E. moshkovskii* and *E. bangladeshi* - amoebae which are morphologically (both cyst and trophozoite) similar to that of *E. histolytica*.
 - ❑ *E. dispar* - non-pathogenic commensal found in human intestine
 - ❑ *E. moshkovskii* - primarily a free-living amoeba, found in polluted water, may cause occasionally non-invasive diarrhea in infants
 - ❑ *E. bangladeshi* have been recovered from asymptomatic individuals, as well as those with diarrhea

- **Diagnosis:** They can be differentiated from *E. histolytica* by various methods
 - PCR amplifying small subunit rRNA gene—most reliable method
 - Detection of lectin antigen in stool
 - RBC inside trophozoites—present only in *E. histolytica* (marker of invasiveness)
 - Zymodeme study.

AMOEBAE MORPHOLOGICALLY RESEMBLING E. HISTOLYTICA

NONPATHOGENIC AMOEBAE

- *Entamoeba coli*,
- *E. hartmanni*,
- *E. gingivalis*,
- *E. polecki*,
- *Endolimax nana*
- *Iodamoeba butschlii*.

DIFFERENCES BETWEEN ENTAMOEBA HISTOLYTICA AND ENTAMOEBA COLI

<i>Entamoeba histolytica</i>		<i>Entamoeba coli</i>
<i>Trophozoite</i>		
Size	15-20 mm	20-25 mm
Motility	Very active and unidirectional purposeful motility Pseudopodia with finger-like projection	Sluggish, nonpurposeful and aimless motility in any direction Blunt pseudopodia
Cytoplasm	Clearly differentiated to ectoplasm and endoplasm	Not differentiated
Cytoplasmic inclusions	RBC, leukocytes, tissue debris and bacteria	Same except it does not contain RBC
Nucleus	Karyosome is small and central Nuclear membrane is thin and lined by fine chromatin granules	Karyosome is large and eccentric Nuclear membrane is thick and lined by coarse chromatin granules
<i>Cyst</i>		
Size	12-15 mm	15-25 mm
Nucleus	Same as trophozoite	Same as trophozoite
Nuclei	1-4 in number	1-8 in number
Chromatoid body	Thick bars with rounded ends	Filamentous and thread like ends

ENTAMOEBA COLI

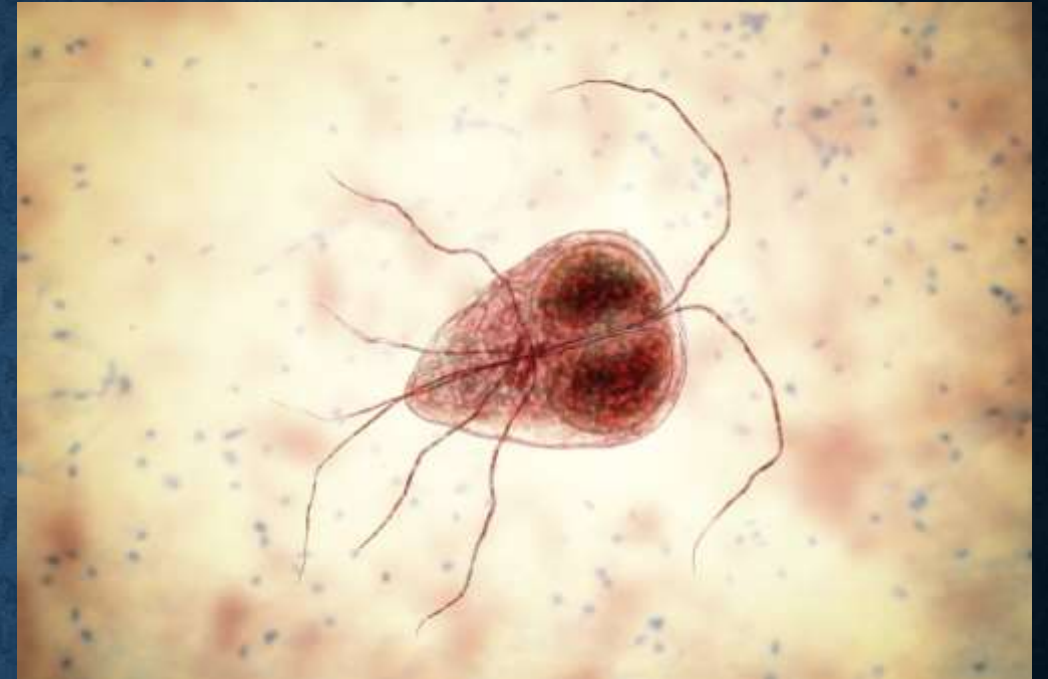


- A. Trophozoite (iron hematoxylin stain) shows nucleus with coarse peripheral chromatin and abundant food vacuoles in the cytoplasm containing fecal debris;
- B. Cyst (iodine mount) containing several nuclei.

INTESTINAL FLAGELLATE INFECTIONS

GIARDIASIS

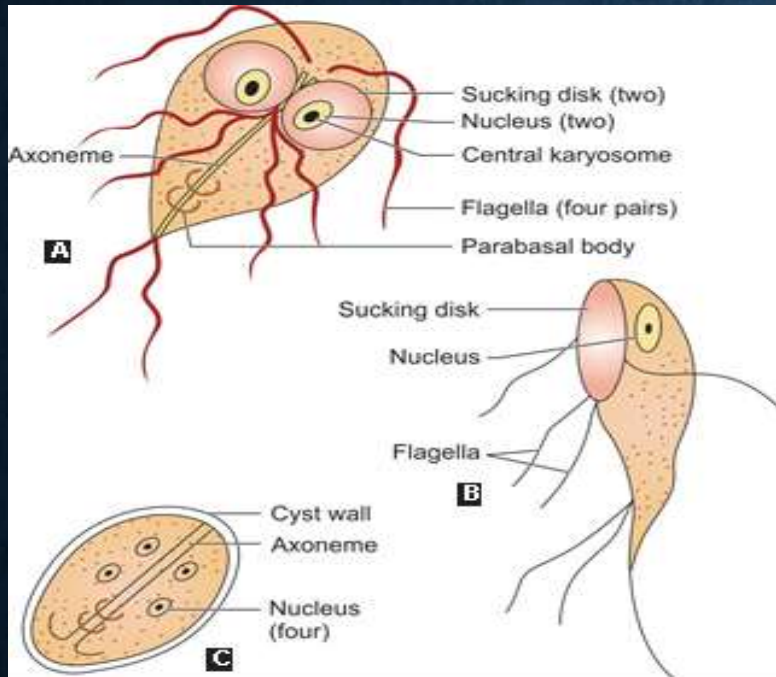
- Giardiasis - caused by *Giardia lamblia*, also known as *G. intestinalis* or *G. duodenalis*.
- It was first observed by AV Leeuwenhoek in 1681 while examining his own stool.
- Later it was described and named after Dr F Lambl and Professor A Giard (1859).



EPIDEMIOLOGY

- *G. lamblia* - worldwide in distribution.
- Considered as one of the most common parasite—causes both endemic and epidemic intestinal disease and diarrhea.
- **Geographical area:** Warm climate of tropics and subtropics.
- **Genotypes:** A and B - further classified into eight assemblages (A to H).

MORPHOLOGY



A. Trophozoite front view; B. trophozoite lateral view; C. Cyst.

- **CYST**

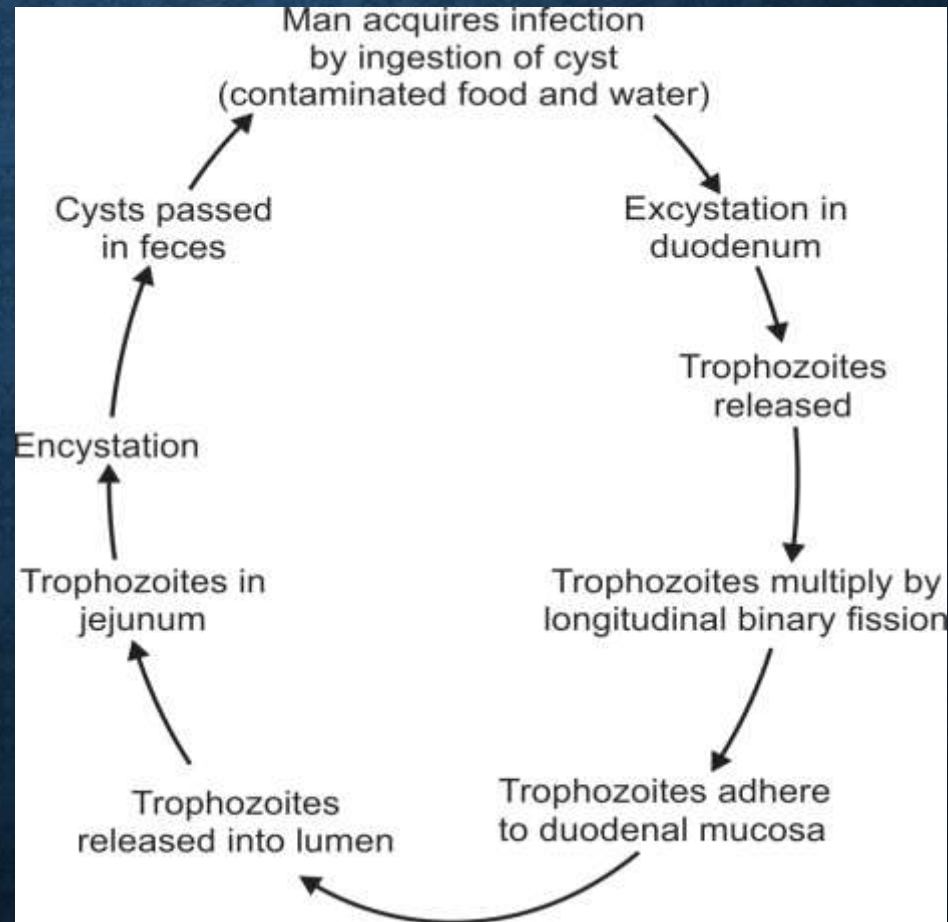
- ❖ Oval and measures $11-14 \times 7-10 \mu\text{m}$
- ❖ Immature cysts may have two nuclei, but a fully mature cyst is quadrinucleate—the four nuclei are seen at one end in pairs

- **TROPHOZOITE**

- ❖ Pear-shaped
- ❖ Bilaterally symmetrical structure that measures about $10 \times 15 \mu\text{m}$ exhibits a '**falling leaf**' motility
- ❖ It resembles a tennis racket and has a sucking disc on its concave ventral surface
- ❖ The parasite has two nuclei and two parabasal bodies present on axostyles

LIFE CYCLE

- **Host:** *Giardia* completes its life cycle in one host.
- **Infective form:** Cysts are the infective form.



PATHOGENICITY

- **Infective dose:** As few as 10–25 cysts can initiate the infection
- **Risk factors:** Children are more commonly affected. Other high-risk groups - elderly debilitated persons, poor hygiene, and immunodeficient individuals.
- It does not appear to be associated with HIV/AIDS
- **Pathogenic form:** Trophozoites - adhere to the duodenal mucosa, cause disruption of the intestinal epithelium that leads to increased permeability and malabsorption.
- Trophozoites do not invade the mucosa, but feed on mucus secretions

- **Malabsorption** may be of various types such as:
 - Malabsorption of fat (steatorrhea)—leads to foul smelling profuse frothy diarrhea
 - Disaccharidase deficiencies (lactose, xylose)— leading to lactose intolerance
 - Malabsorption of vitamin A, B12 and iron
 - Protein losing enteropathy.
- **Antigenic variation:** *Giardia* undergoes frequent antigenic variations due to a cysteine rich protein on its surface called **variant surface protein (VSP)**.

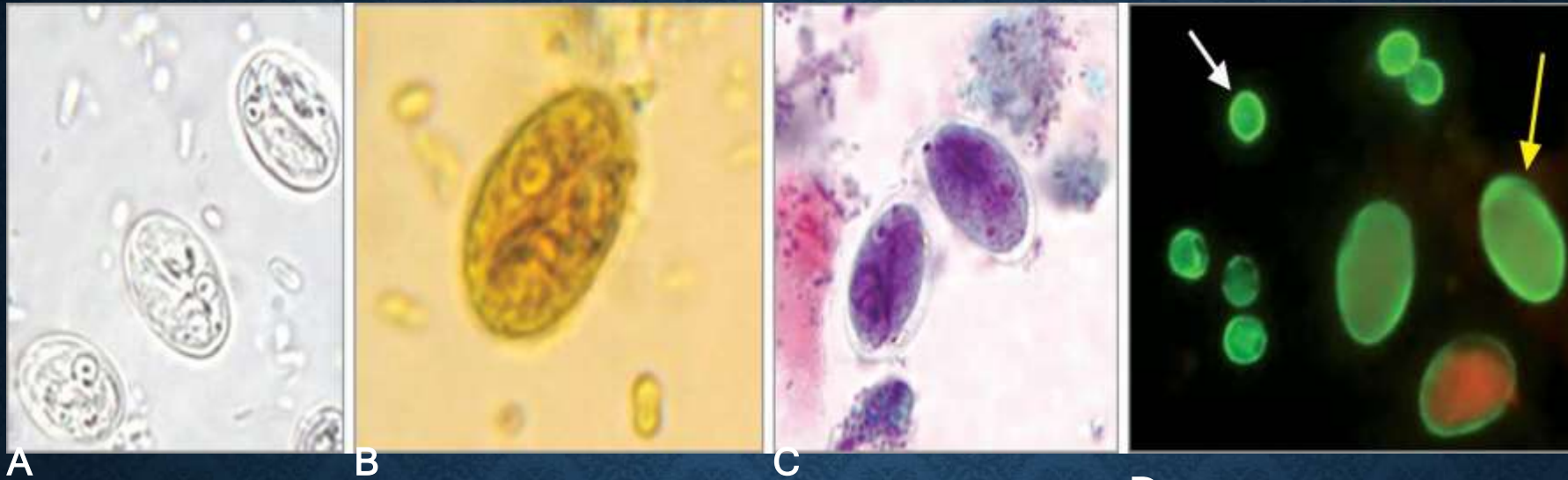
CLINICAL FEATURES

- **1. Asymptomatic carriers:** Most infected persons are asymptomatic, harbor the cysts in the gut and spread the infection
- **2. Acute giardiasis:** Incubation period varies from 1 week to 3 weeks
 - ❖ **Acute presentation:** Mild diarrhea to severe malabsorption syndrome; Loose motions are foul-smelling and contain mucus without blood
- **3. Chronic giardiasis:** It may present with or without a previous acute symptomatic episode
 - **Chronic presentation:** Intermittent or recurring
 - Foul-smelling diarrhea with weight loss due to malabsorption syndrome
 - -Extraintestinal manifestations such as urticaria, anterior uveitis or arthritis

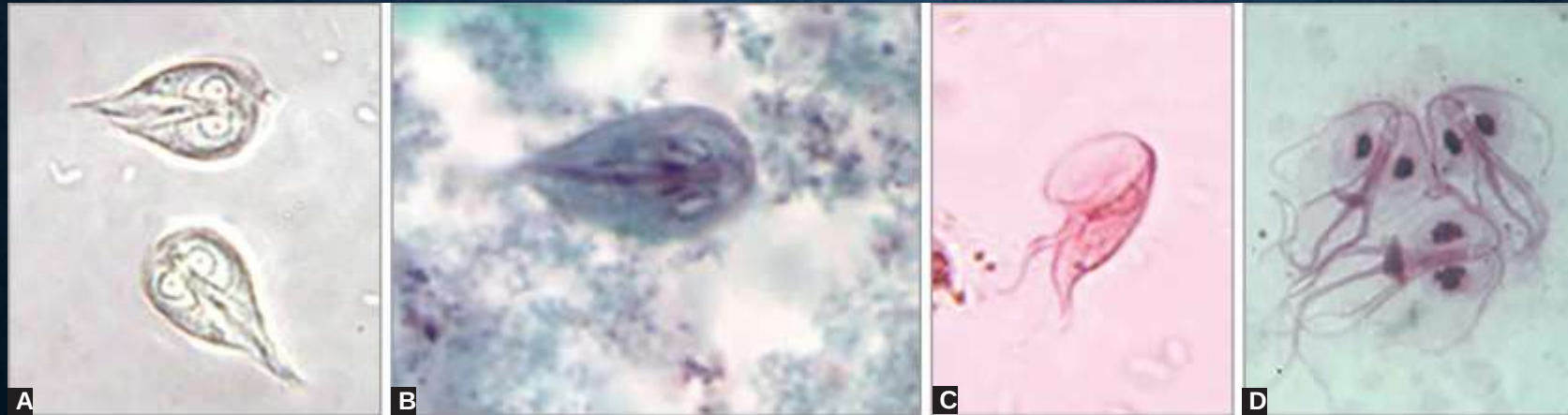
LABORATORY DIAGNOSIS

Stool Examination:

- Gold standard for diagnosis of giardiasis.
- Detects cysts and trophozoites
- Pus cells or blood (RBCs) - never be seen in stool microscopy in case of giardiasis. If found; suggests alternative diagnosis.



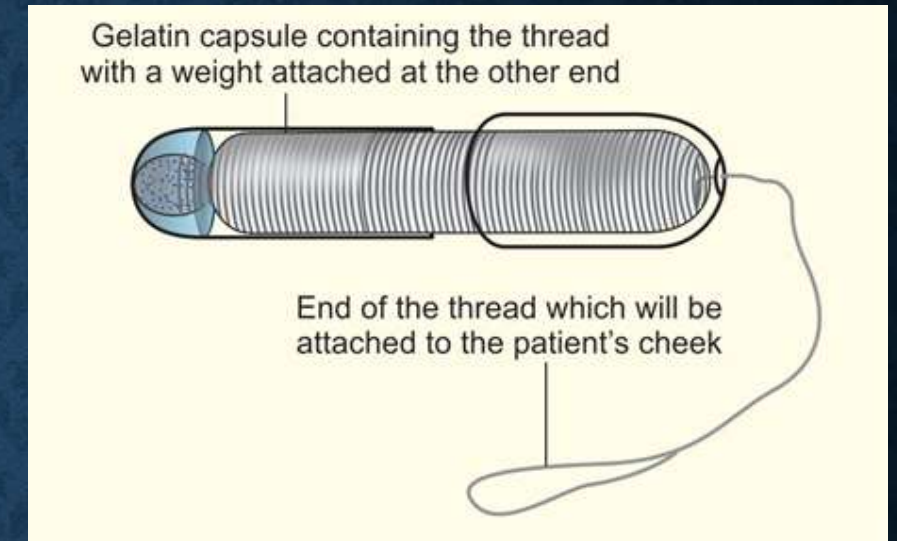
Cysts of *Giardia lamblia*: A. Saline mount; B. Iodine mount; C. Trichrome stain; D. Direct immunofluorescence assay.



Trophozoites of *Giardia lamblia*: A. Saline mount front view; B. Trichrome stain front view; C. Merthiolate iodine formalin (MIF) stain lateral view (spoon shaped); D. Giemsa stained mucosal imprint (front view).

LABORATORY DIAGNOSIS

- ***Entero-test (or String Test)*** – duodenal sampling – with the help of gelatin capsule attached to thread.
- ***Histopathology*** - Endoscopy-guided duodenal biopsy tissue can be processed by touch preparation and stained by Giemsa stain to demonstrate the trophozoites



Entero-test equipment showing duodenal capsule



LABORATORY DIAGNOSIS

- ***Antigen Detection in Stool (Coproantigen)*** - ELISA and direct fluorescent antibody tests (DFA), Rapid ICT (e.g. triage parasite panel).
- ***Antibody Detection*** - Indirect fluorescent antibody (IFA) and ELISA.
- ***Culture*** - Cultivated in axenic media - Diamond's media used for *E. histolytica*. Culture is done for research purpose and to prepare the antigens
- ***Molecular Methods*** – PCR, Biofire Filmarray.
- ***Radiological Finding*** - Fluoroscopy , X-ray after barium meal

TREATMENT OF GIARDIASIS

- Tinidazole (2g once orally) - drug of choice
- Metronidazole (for 5 days) or nitazoxanide (for 3 days) is given alternatively
- Furazolidone is given to children and auranofin, paromomycin can be given in pregnancy

TREATMENT OF GIARDIASIS

- In patients with AIDS and hypogammaglobulinemia, giardiasis is often refractory to treatment.
- Prolonged therapy with metronidazole (for 21 days) has been successful
- Metronidazole resistance has been reported in *Giardia*.
- Auranofin is shown to be effective

PREVENTION

- Improved food and personal hygiene.
- Boiling or filtering of potentially contaminated water
- Treatment of asymptomatic carriers
- No vaccine is currently available.

OPPORTUNISTIC INTESTINAL COCCIDIAN PARASITIC INFECTIONS

OPPORTUNISTIC INTESTINAL COCCIDIAN PARASITIC INFECTIONS

- *Cryptosporidium*, *Cyclospora*, and *Cystoisospora* are the intestinal coccidian parasites - cause opportunistic infections in HIV infected patients producing profuse watery diarrhea.
- Diagnosed by detection of acid fast oocysts in stool microscopy

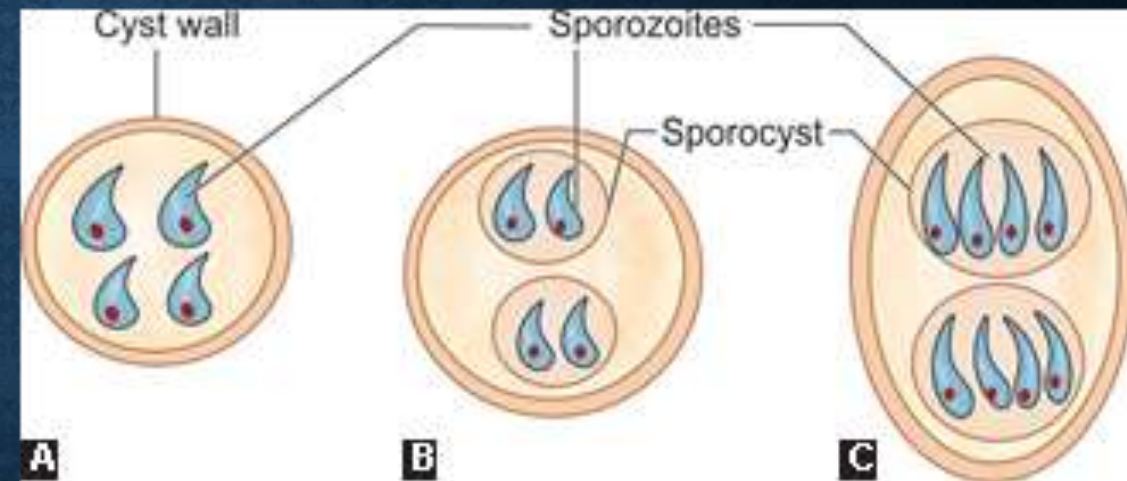
OPPORTUNISTIC INTESTINAL COCCIDIAN PARASITIC INFECTIONS

- *Cryptosporidium* species infecting man – classified as two separate species, *C. parvum* (mammals, including humans) and *C. hominis* (primarily humans).
- Other species infect wide range of mammals, but not man
- *Cyclospora cayetanensis* - recently described coccidian parasite, as human intestinal pathogen.
- *Isospora belli* - recently renamed as *Cystoisospora belli* - only species that infects man.
- No other animal reservoir is known.

MORPHOLOGY

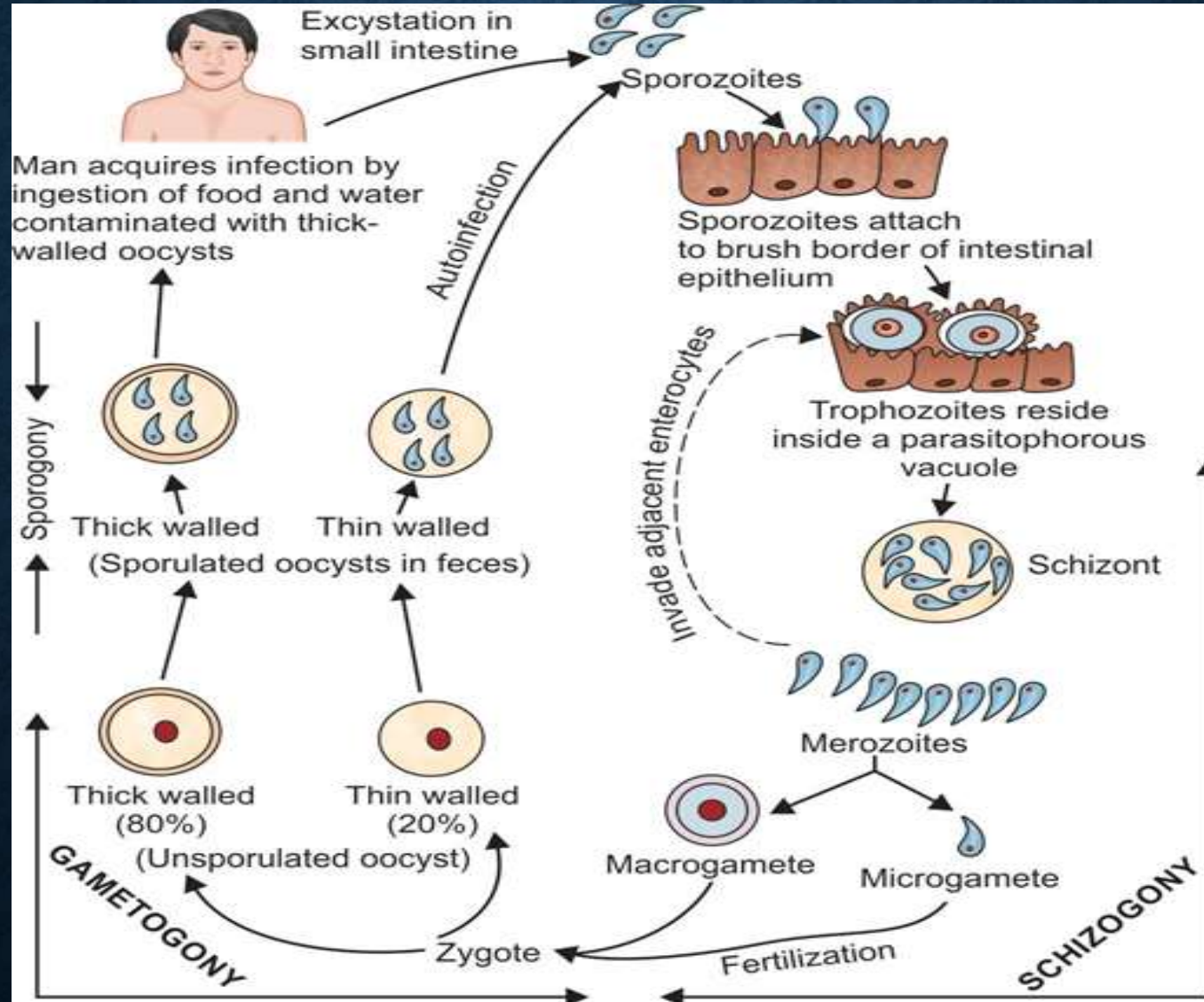
- The life cycle of coccidian parasites passes through various stages - oocyst, trophozoite, schizont (meront), merozoite, and zygote.
- Oocyst is the infective form to man as well as the diagnostic form excreted in the feces.

SPORULATED OOCYSTS



A. *Cryptosporidium*; B. *Cyclospora*; C. *Cystoisospora*

LIFE CYCLE



EPIDEMIOLOGY

- Seen worldwide - more prevalent in tropical and subtropical climates of developing countries from South America, Africa, and Southeast Asia including India.
- Highest prevalence - Haiti.

EPIDEMIOLOGY

- **Risk factors** - More common in area with poor sanitation and HIV infected individuals
- **Peak age:** Infants and children are commonly affected
- **Source of infection** - rainwater lodges and swimming pool recreational water, or contaminated food (e.g. imported foods including raspberries, basil and mesclun lettuce)

PATHOGENESIS

- **Excystation:** Oocyst secretes proteases and aminopeptidases - facilitate excystation in small intestine, releasing sporozoites
- **Attachment:** Sporozoites attach to the brush border epithelium of the small intestine (ileum) -**CP47** (47 kDa *Cryptosporidium* protein)
- **Penetration:** Discharges from the apicomplex present in the anterior end of the sporozoites help in invasion

PATHOGENESIS

- **Vacuole:** Parasitophorous vacuole near the microvilli surface of the host cells
- **Cytokines:** Release cytokines like tumor necrosis factor (TNF)- α , IL-8, prostaglandins, etc. which induce Inflammation
- These factors increase intestinal secretion of chloride and water and decrease the sodium absorption leading to fluid loss and diarrhea.

CLINICAL FEATURES

- **Immunocompetent Hosts:**

- Majority of infections - asymptomatic.
- Some individuals develop symptoms - average incubation period of about 1 week.
- Watery non-bloody diarrhea - common presentation.
- Abdominal pain, nausea, anorexia, fever, and/or weight loss may be present
- Self-limiting - subsides within 1–2 weeks

CLINICAL FEATURES

- **Immunocompromised Hosts (e.g. AIDS):**
 - **Chronic, persistent profuse diarrhea**
 - Severe weight loss, wasting and abdominal pain .
 - **Extraintestinal manifestations** - biliary tract infection (sclerosing cholangitis), respiratory tract infection, and pancreatitis
 - Disease is more severe in patients with AIDS when CD4+ T cell counts become **<100/ μ L**

CLINICAL FEATURES

- **Immunocompromised Hosts (e.g. AIDS)**
- Other immunocompromised conditions – severe combined immunodeficiency syndrome, hematological malignancies and solid organ transplant recipient
- Disease - more severe in *Cryptosporidium* than *Cyclospora* and *Cystoisospora* - attributed to autoinfection seen in the former—a key factor for maintaining the infection, resulting in chronic persistent diarrhea.

LABORATORY DIAGNOSIS

DIRECT MICROSCOPY (STOOL EXAMINATION)

- **Sample collection:** Three stool samples should be collected. Rarely (in HIV positive patients), sputum, bronchial wash, duodenal or jejunal aspirate can be collected
- **Direct wet mount:** Highly refractile, double-walled oocyst

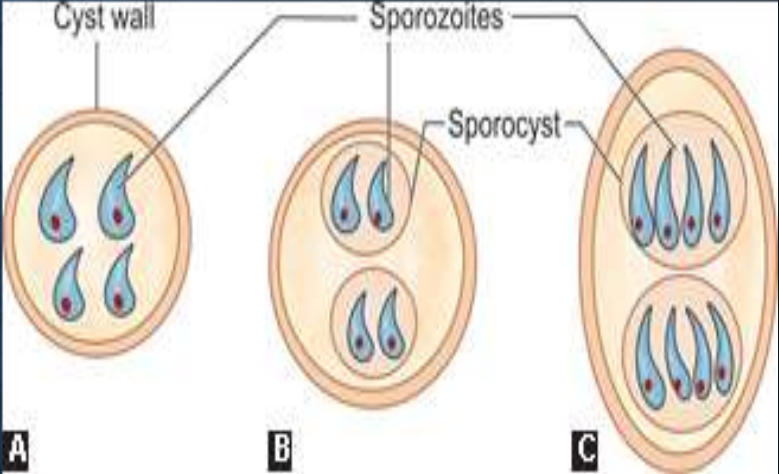
DIRECT MICROSCOPY (STOOL EXAMINATION)

- **Concentration technique:** If the oocyst load is less, then various techniques are used to concentrate the stool sample
- **Acid-fast staining:** Oocysts are acid-fast to 0.5–1% sulfuric acid or acid alcohol and appear as red color oocysts against blue back ground
- **Direct fluorescent antibody staining:** Available for cryptosporidiosis - oocysts can be detected by using fluorescent labeled monoclonal antibody directed against cyst wall antigens.
- **Autofluorescence:** Oocysts of *Cyclospora* fluoresce when viewed under ultraviolet epifluorescence microscopy.
- **Phase contrast microscopy**

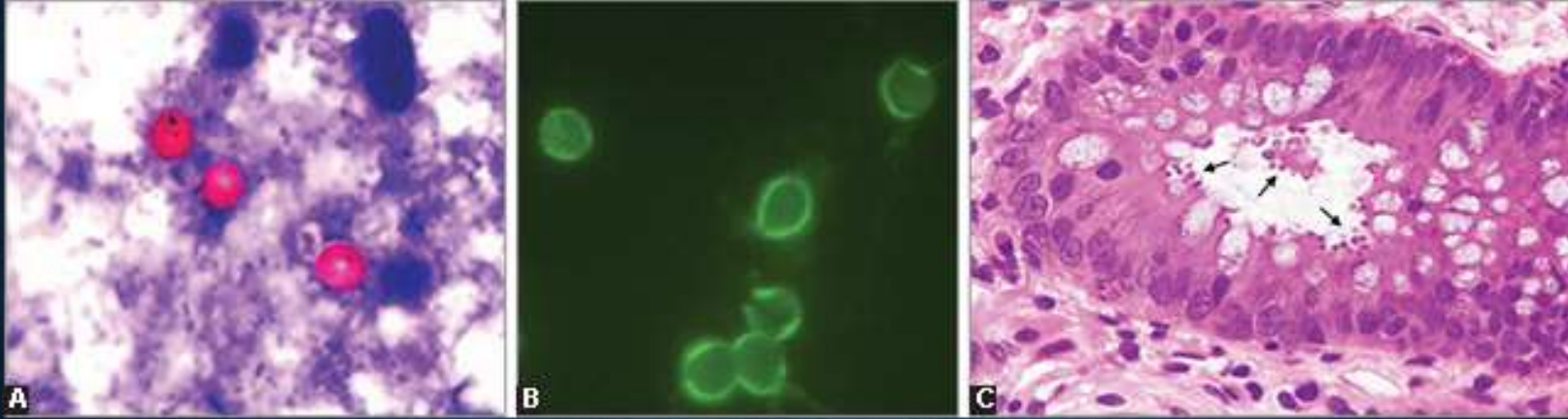
DIFFERENCES BETWEEN *CRYPTOSPORIDIUM*, *CYCLOSPORA* AND *CYSTOISOSPORA*

Property	<i>Cryptosporidium</i>	<i>Cyclospora</i>	<i>Cystoisospora</i>
Infective form	Sporulated oocyst	Sporulated oocyst	Sporulated oocyst
Diagnostic form	Sporulated oocyst	Unsporulated oocyst	Unsporulated oocyst
Outbreaks	Frequent, large	Common, large	Occasional, small
Zoonotic potential	Yes	Uncertain	No
Oocyst size	4-6 mm	8-10 mm	20-33 mm
Oocyst shape	Round	Round	Oval
Sporulated oocyst contain	Four sporozoites	Two sporocysts, each having two sporozoites	Two sporocysts, each having four sporozoites

Property	<i>Cryptosporidium</i>	<i>Cyclospora</i>	<i>Cystoisospora</i>
Acid-fastness	Uniformly acid-fast	Variable acid-fast	Uniformly acid-fast
Autofluorescence	No, but can be stained with fluorescent dye	Autofluorescence ++	Autofluorescence +/-
Sporulation of the oocyst	Occurs inside the host cells (enterocytes)	Occurs in soil (environment)	Occurs in soil (environment)
Treatment	Nitazoxanide	Cotrimoxazole	Cotrimoxazole

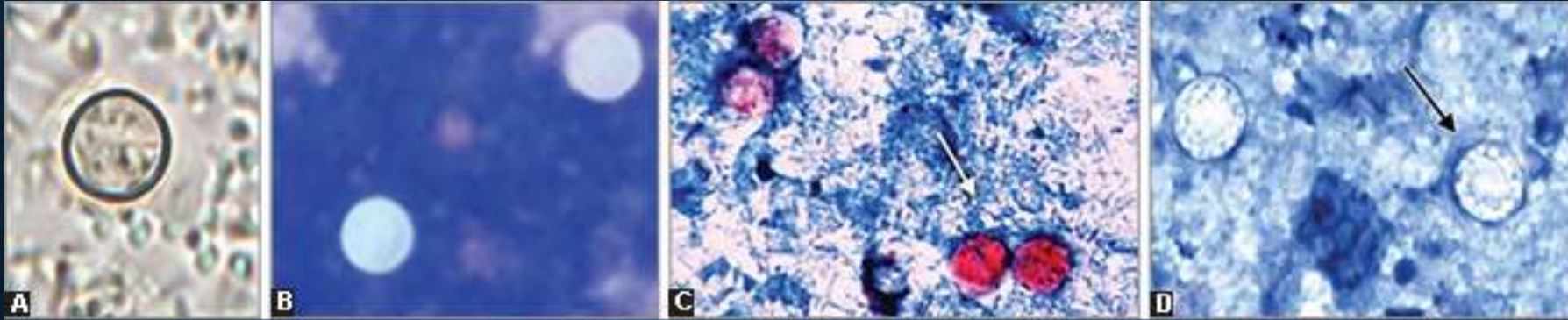


CRYPTOSPORIDIUM SPECIES



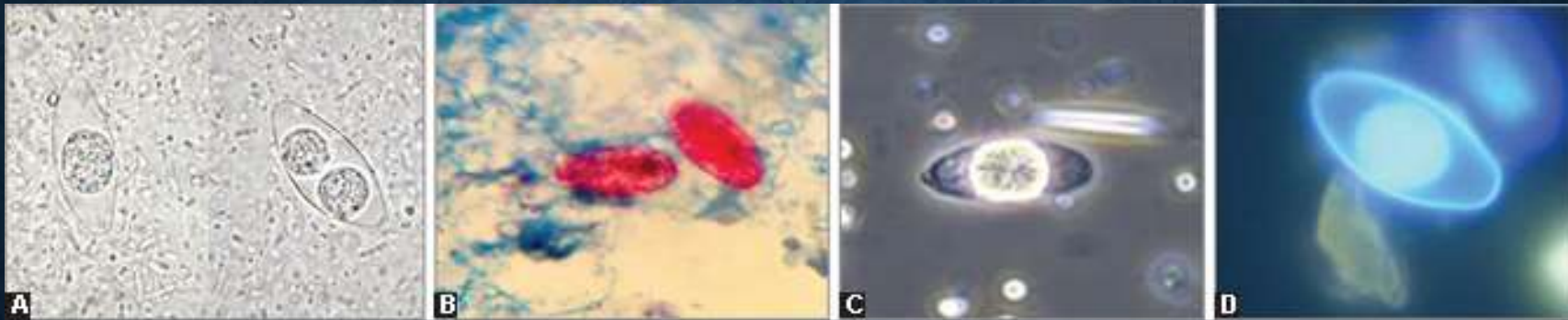
A. Acid-fast stain shows red color oocysts against blue back ground; B. Direct fluorescent antibody staining shows brilliant green fluorescent oocysts; C. Hematoxylin and eosin stain of intestinal biopsy shows numerous oocysts at the luminal surface of the intestinal crypts (marked by arrows).

CYCLOSPORA SPECIES



A. Saline mount preparation showing unsporulated oocyst; B. Epifluorescence microscopy showing autofluorescent oocysts; C. Acid-fast oocysts; D. Non-acid fast oocysts (variable acid-fastness).

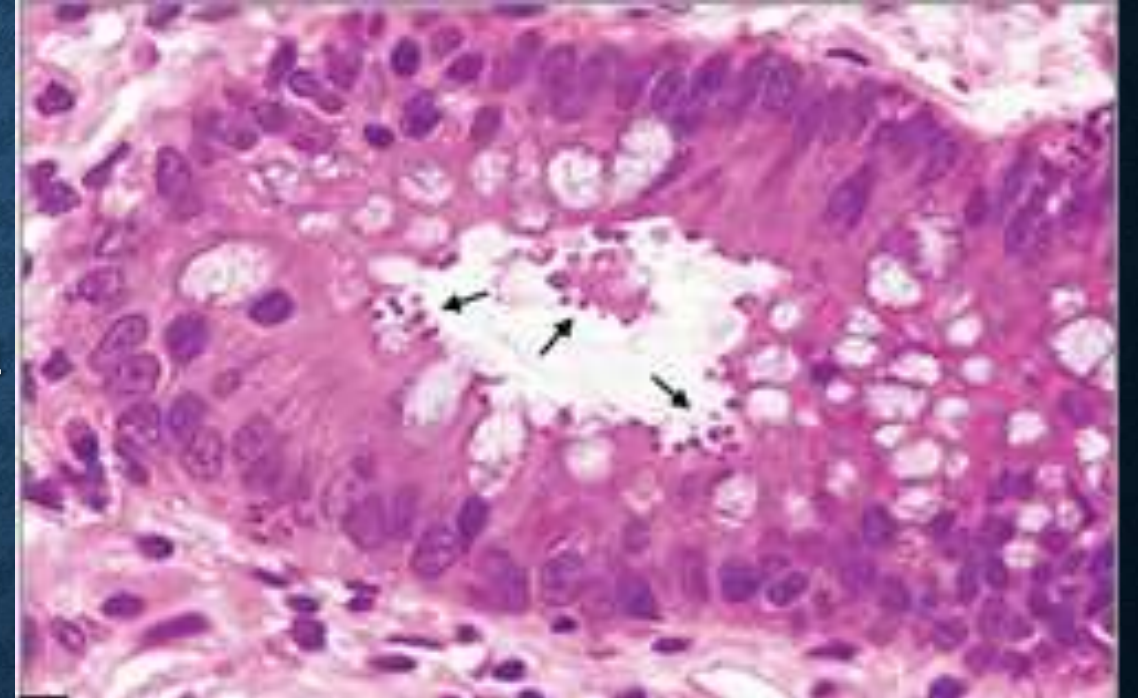
CYSTOISOSPORA BELLI



A. Saline mount preparation shows left—unsporulated oocyst and right—sporulated oocyst; B. Modified acid-fast stain showing unsporulated oocysts; C. Phase contrast microscopy showing unsporulated oocyst; D. Fluorescent stained smears showing unsporulated oocyst.

HISTOPATHOLOGY

- When stained by H&E stain, *Cryptosporidium* appears as 1–3 μm basophilic round bodies within the cell membrane of enterocytes called as “**blue beads**”



ANTIGEN DETECTION FROM STOOL

- ELISA
- ICT

ANTIBODY DETECTION

- ELISA are available detecting antibodies in patient's serum against oocyst antigens of *Cryptosporidium* or *Cyclospora*.
- Useful for sero-epidemiological purpose, not for routine diagnosis.

MOLECULAR DIAGNOSIS

- PCR or real-time PCR can be performed to detect specific genes from both clinical and environmental samples targeting genes such as 18S rRNA or small subunit rRNA.
- BioFire FilmArray

SUPPORTIVE TESTS

- Low CD4-T lymphocyte count (especially in HIV positive patient)
- Fecal leukocyte marker—lactoferrin is increased in 75% of cases indicating increase pus cells in feces.

TREATMENT OF INTESTINAL COCCIDIAN PARASITIC INFECTIONS

Mild cases

- Self-limiting, requires fluid replacement like ORS.
- Lactose-free glutamine supplemented diet is preferred for cryptosporidiosis.

Severe cases

- **Cryptosporidiosis:** Nitazoxanide is given to adults (for 3 days - not effective in HIV-infected patients. Paromomycin - alternate
- **Cyclosporiasis and cystoisosporiasis:** Cotrimoxazole (for 7-10 days). Patients who cannot tolerate cotrimoxazole may be treated with ciprofloxacin or nitazoxanide.

PREVENTION

- Requires minimizing exposure to infectious oocysts in human or animal feces
- Proper hand washing, use of submicron water filters, use of appropriate disinfectants/sterilants to kill the oocysts (such as hydrogen peroxide, steam sterilization), improved personal hygiene are some of the efforts to prevent transmission.

OTHER INTESTINAL PROTOZOAN INFECTIONS

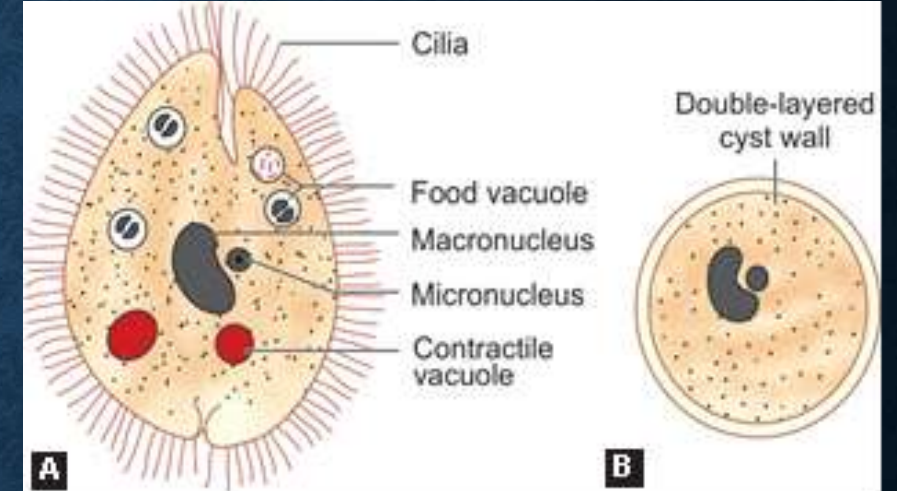
- *Balantidium coli*
- *Sarcocystis* spp.
- *Blastocystis hominis*.
- Involvement of intestinal muscles occurs in chronic Chagas's disease caused by *Trypanosoma cruzi*.

BALANTIDIASIS

- Balantidiasis - intestinal infection caused by a protozoan parasite *Balantidium coli*.
- Largest protozoan and the only ciliated parasite of humans.
- Produces intestinal disease similar to amoebic dysentery.
- **Habitat:** Resides in the large intestine.

BALANTIDIASIS

- **Host:** Pig (main reservoir) and other animals - primary hosts.
- Man - Accidental host
- **Morphology:** Exists in two forms -
 - (1) trophozoite (found in the dysenteric stool),
 - (2) cyst (found in carriers and chronic cases).
- Both forms are binucleated having a large macronucleus and a small micronucleus



Morphology of *Balantidium coli* (schematic diagram)
A. Trophozoite; B. cyst.

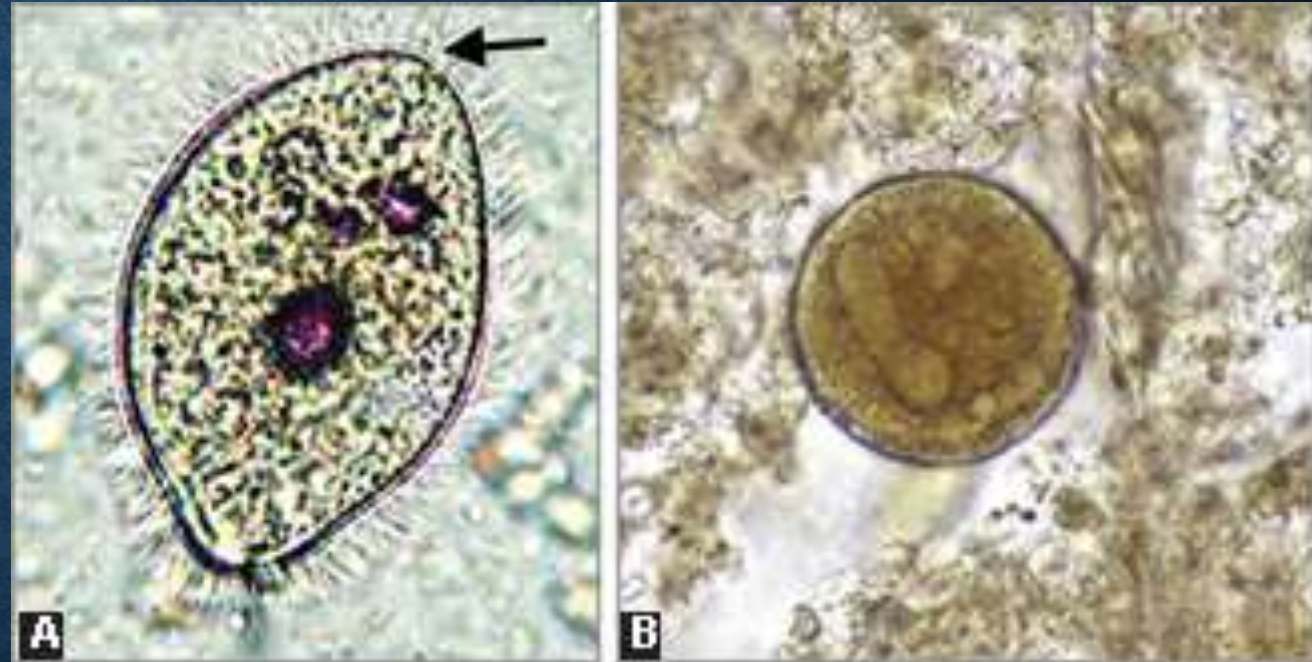
Life cycle:

- Man gets infection - ingestion of food and water contaminated with cysts (infective form)
- Cysts transform into trophozoites - multiply in the large intestine by both asexual mode and sexual mode (conjugation)
- Both trophozoites and cysts - excreted in the feces.
- Trophozoites disintegrate - cysts are resistant and are infective to man and pig.

Clinical features:

- Majority of infected individuals - asymptomatic carriers.
- Small number of people develop acute dysenteric disease -
Characterized by profuse diarrhea and dysentery with mucous and blood, weeks to months later.
- Other features - fever, nausea, vomiting and abdominal pain

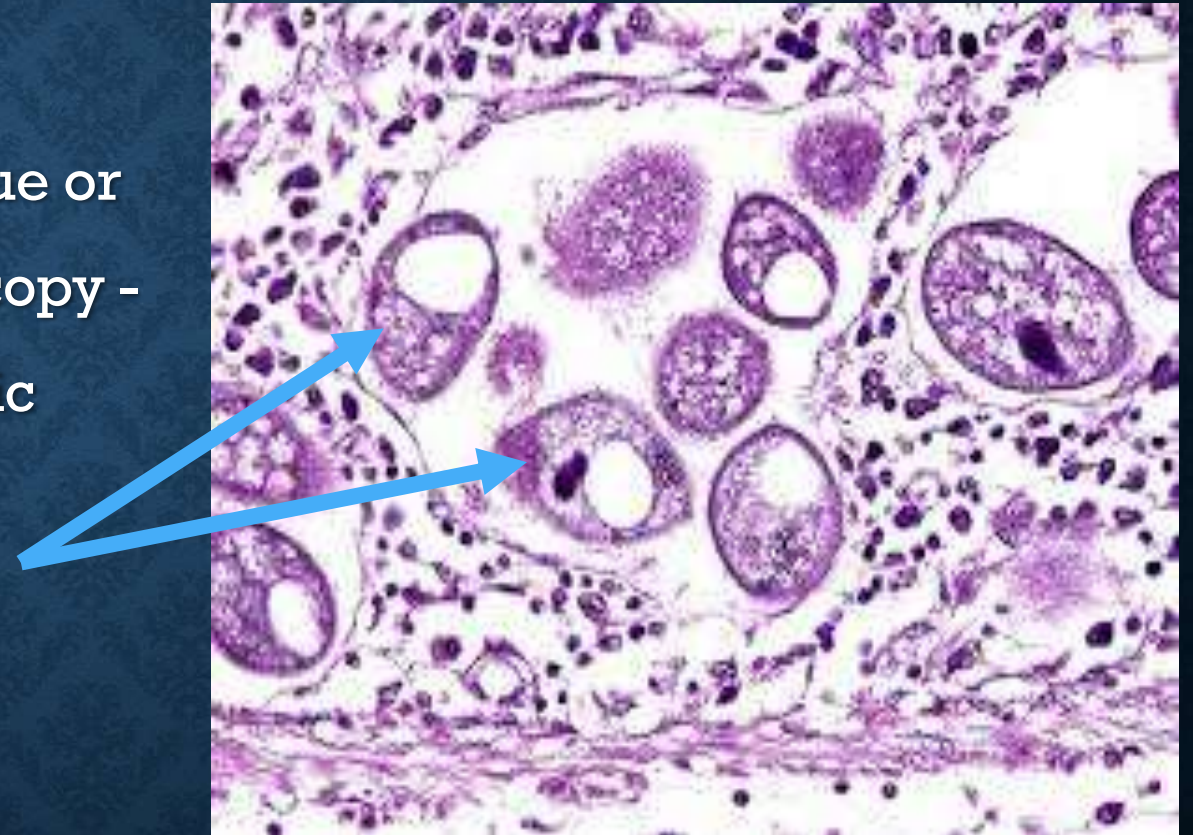
- **Laboratory diagnosis:** Repeated stool examination - as the parasite is excreted intermittently.
- Trophozoites - appreciated in the saline mount.
- Internal structures of cysts - seen more clearly in iodine mount.



Balantidium coli: A. Saline wet mount preparation shows trophozoite with cilia; B. Iron hematoxylin stain shows cyst with prominent macronucleus.

Histopathology:

Histopathological staining of the biopsy tissue or scrapping of the ulcers taken by sigmoidoscopy - cluster of trophozoites, cysts and lymphocytic infiltration in the submucosa.



Treatment:

- Tetracycline - drug of choice, given for 10 days.
- Alternatively, metronidazole can be given

**Thank
you!**

