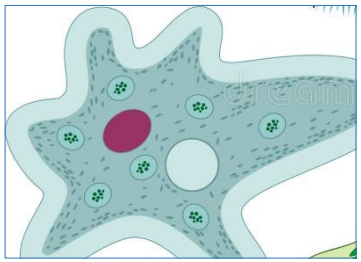


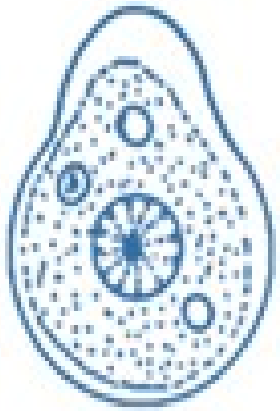
Introduction to Protozoa

Professor Dina Moustafa Abou Rayia

Medical Microbiology and Immunology Department

2024-2025

- **Definition:** Protozoa are unicellular organisms capable of performing all life functions.
- **Morphology:**



❖ **Plasma membrane.**

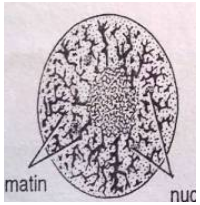
❖ **Ectoplasm:** hyaline, non-granular outer layer and responsible for locomotion, feeding, excretion and protection

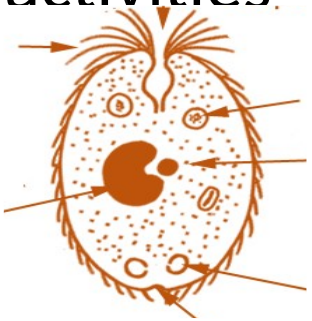
❖ **Endoplasm:** granular, responsible for metabolism. It contains food vacuoles, food reserves and contractile vacuoles

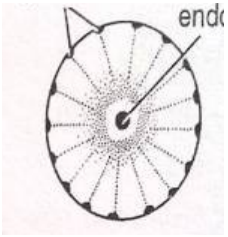
General characters:

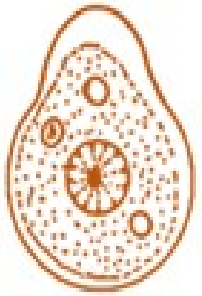
❖ Nucleus:

- One or more,, located in the endoplasm.
- Responsible for reproduction and regulates activities of the cell.
- **Consists of:**
  - Nuclear membrane.
  - Nucleoplasm.
  - Chromatin network.
  - Karyosome (endosome or nucleolus)



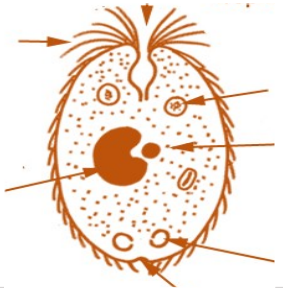






Biology:

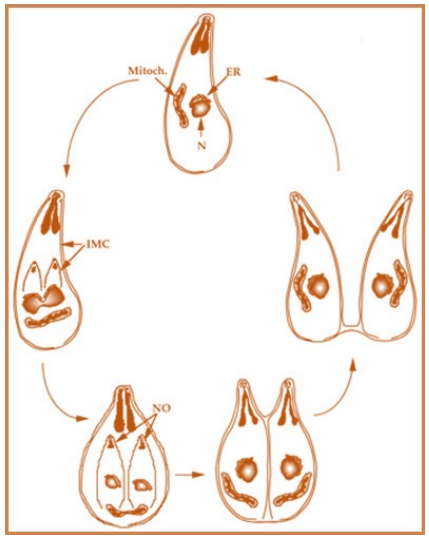
- **Locomotion:** by pseudopodia, flagella or cilia.
- **Nutrition:** by absorption of liquid food from the surface (saprozoic) or ingestion of solid particles (holozoic) through the cytostome or by pseudopodia.
- **Excretion:** by diffusion, contractile vacuoles or cytopyge.
- **Secretion:** enzymes, toxins, and materials for cyst walls.
- **Encystation:** formation of cysts, to resist unfavourable conditions and facilitate transfer

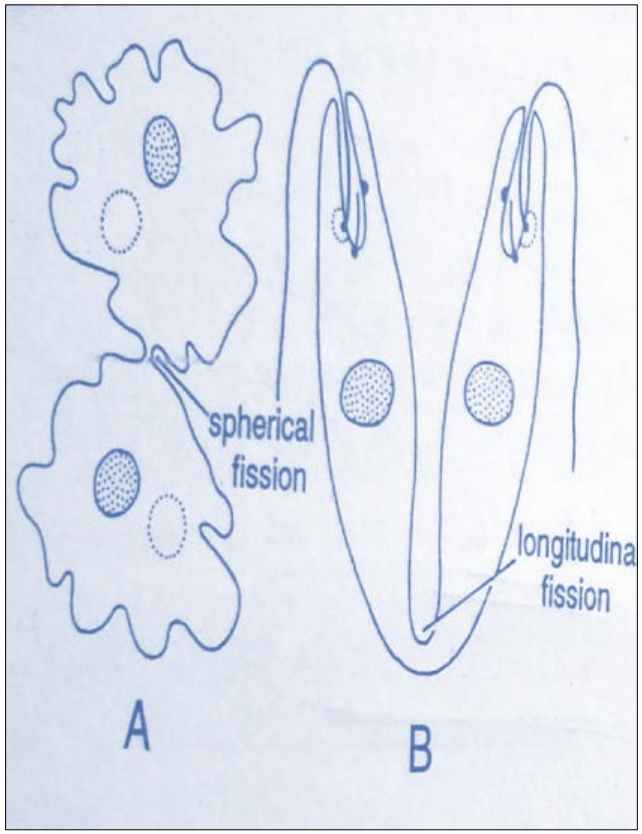


Reproduction:

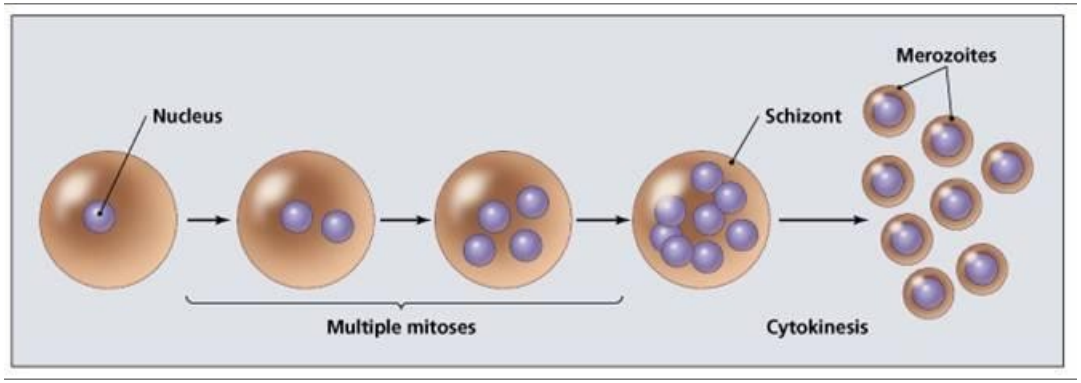
- Asexual:

Endodyogony





Simple binary fission



Multiple fission (schizogony)

Reproduction:

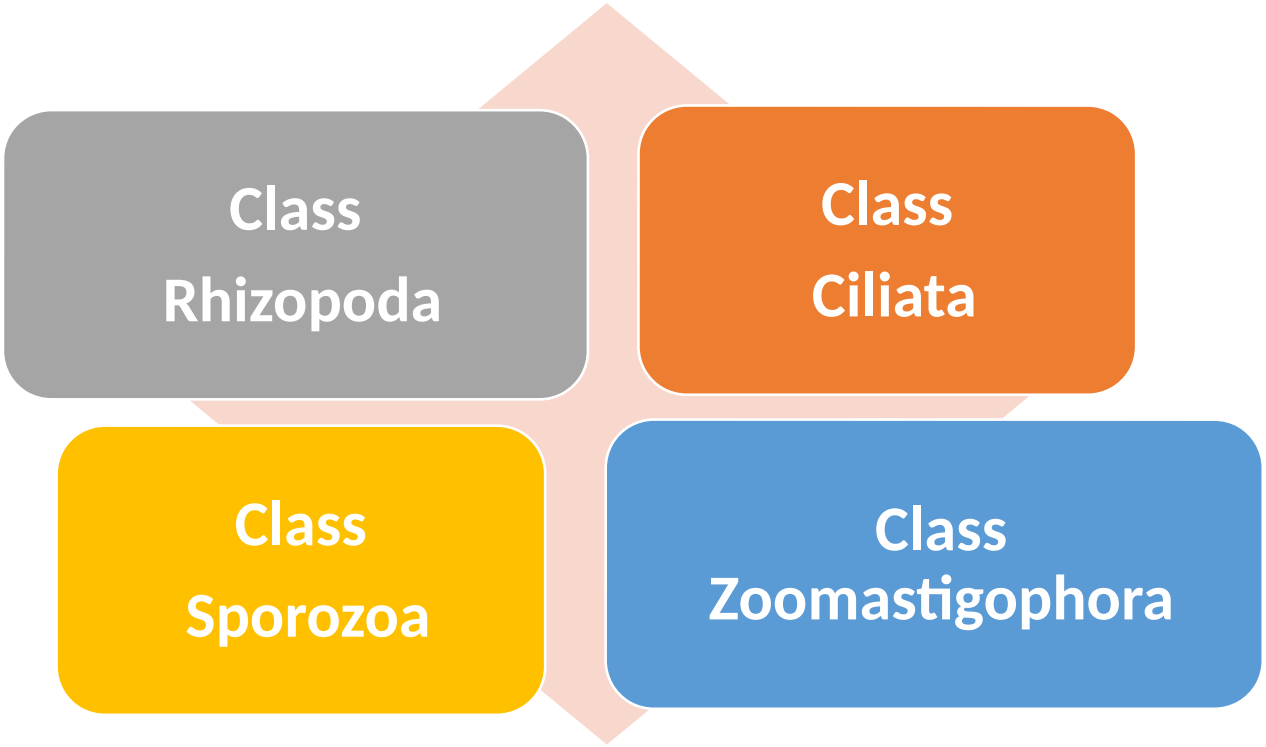
❖Sexual:

- **- Syngamy:** permanent union of gametes for formation of a zygot.

- **- Conjugation:** temporary union of two organisms for exchange of nuclear material as in *Balantidium coli*.

Protozoa classification

According to the mode of locomotion



Class Rhizopoda (Ameobae)

- **General Characters:**

(1) Move and feed by pseudopodia.

(2) Multiply by binary fission.

(3) Have trophozoite and cystic stages

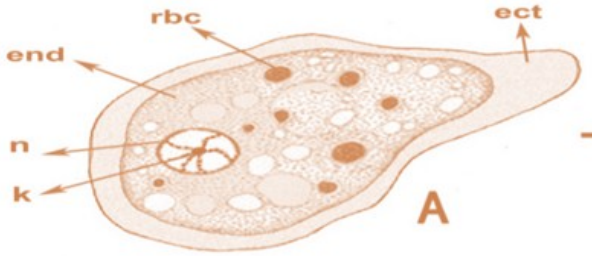
- **They may be:**

A) Pathogenic: *Entamoeba histolytica*

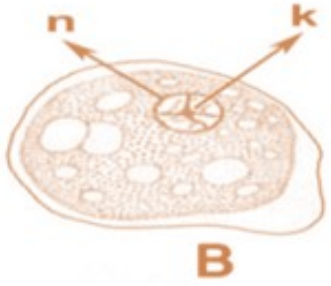
B) Non-pathogenic: e.g. *Entamoeba coli*

Entamoeba histolytica

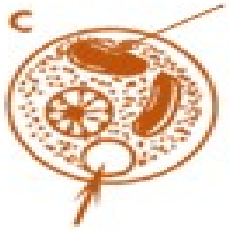
Entamoeba histolytica



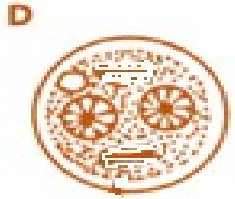
Trophozoite



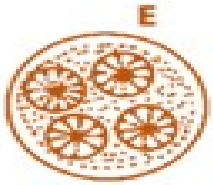
Pre-cyst



Uninucleated cyst



Binucleated cyst



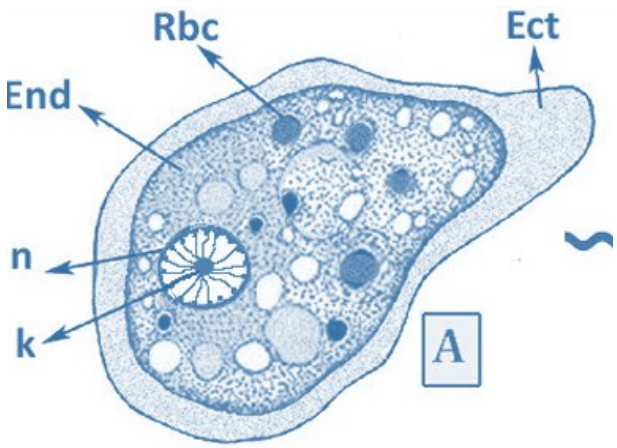
Quadrinucleated mature cyst

Morphology

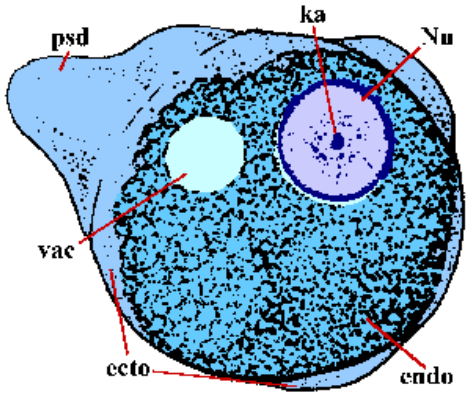
- **Trophozoite:** It has clear ectoplasm and granular endoplasm with food vacuoles containing RBCs in the invasive form. The nucleus has central fine karyosome and fine regularly arranged chromatin dots on the inner surface of the nuclear membrane.

- **Cysts:** Surrounded by a cyst wall. Contain one nucleus, two nuclei or four nuclei (mature quadrinucleated cysts) which resemble that of the trophozoite. They usually contain a glycogen mass and cigar-shaped chromatoid bodies which represent stored food and present in young cysts

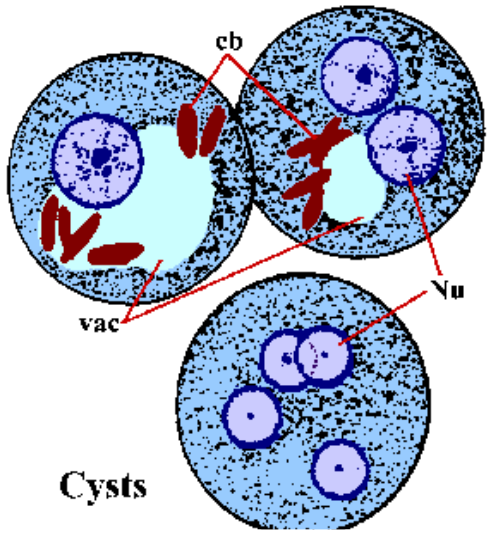
Trophozoite

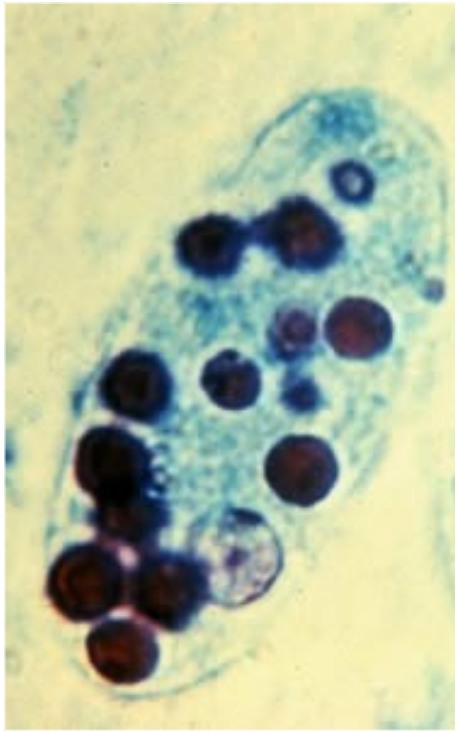


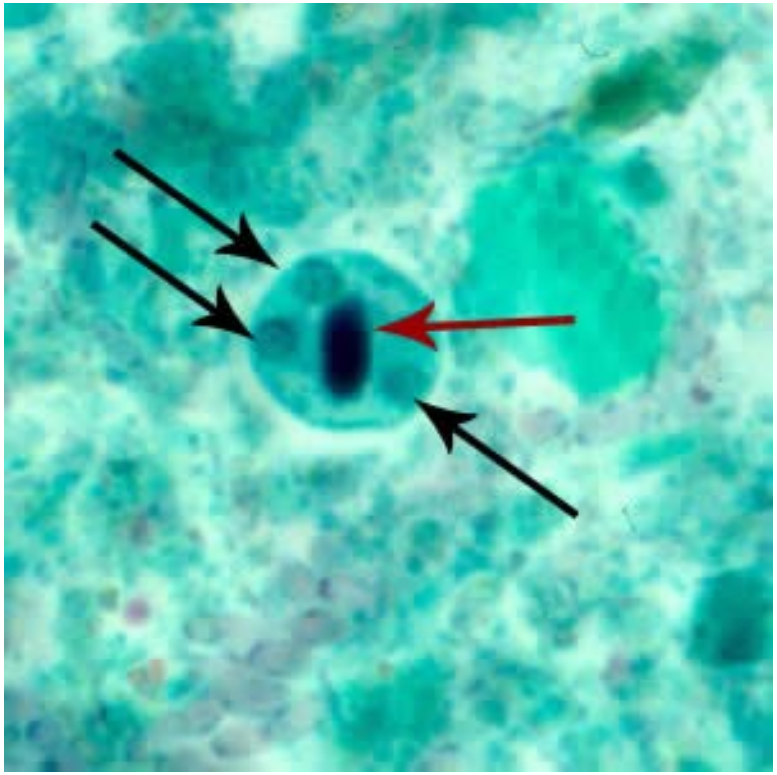
Precyst

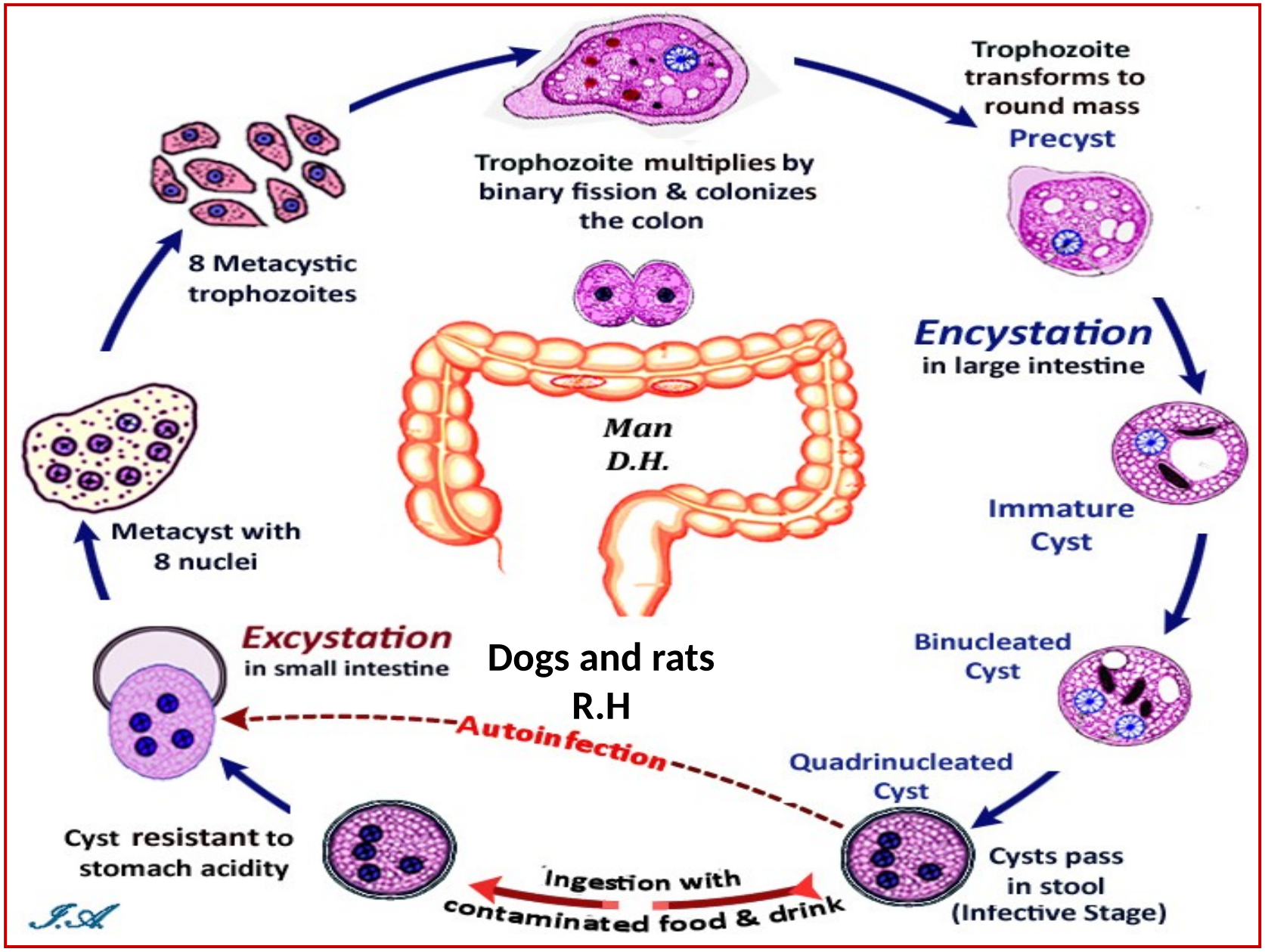


cyst








Trophozoite transforms to round mass
Precyst
Trophozoite multiplies by binary fission & colonizes the colon
8 Metacystic trophozoites
Encystation
in large intestine
Man
D.H.
Immature Cyst
Metacyst with 8 nuclei
Excystation
in small intestine
Dogs and rats
R.H
Binucleated Cyst
Autoinfection
Quadrinucleated Cyst
Cyst resistant to stomach acidity
Ingestion with contaminated food & drink
Cysts pass in stool (Infective Stage)

Life cycle

- The trophozoites inhabit the colon and multiply by simple binary fission, colonize and/or invade the large intestinal epithelium, formation of flask-shaped ulcers.

- When the trophozoites pass in diarrhoeic stool, they disintegrate rapidly in the external environment.

- The trophozoites are dehydrated in the lumen of the large intestine and undergo encystation with the passage of cysts in the stool.

- Human infection is by ingestion of the quadrinucleated cyst, excystation in the ileum, metacyst immediately undergoes mitosis, eight small metacystic trophozoites , pass downward to the large intestine, trophozoites feed, grow and reproduce.

Habitat:	Large intestine (mainly caecum)
Hosts:	**D.H.:** Man
	R.H.: Dog and rat
Diagnostic stage:	In formed stool: Cyst forms
	In diarrhoeic stool: Trophozoite
Infective stage:	Mature quadrinucleated cyst
Mode of infection:	Ingestion of cysts: 1) In contaminated food and drinks 2) Faeco-oral transmission (hand to mouth): • From person to person • By autoinfection

Pathogenesis

With heavy infection and lowering of host immunity

The trophozoites of *E. histolytica* invade the mucosa and submucosa of the large intestine by secreting lytic enzymes ⌙ amoebic ulcers

The ulcer is flask- shaped with deeply undermined edges containing cytolyzed cells, mucus and trophozoites.

The most common sites of amoebic ulcers are caecum, colonic flexures and sigmoidorectal regions due to decrease peristalsis & slow colonic flow at these sites that help invasion.

Clinical pictures

I) Intestinal amoebiasis

1-Asymptomatic infection

Most common and trophozoites remain in the intestinal lumen feeding on nutrients without tissue invasion (Asymptomatic patient known as a healthy carrier and cyst passers)

2-Symptomatic infection

a) Acute amoebic dysentery

Presented with fever, abdominal pain, tenderness, tenesmus (difficult defecation) and frequent motions of loose stool containing mucus, blood and trophozoites.

b) Chronic infection

-Occurs if acute dysentery is not properly treated.
-With low grade fever, recurrent episodes of diarrhea alternates with constipation.
- Only cysts are found in stool.

3-Complications

- Haemorrhage due to erosion of large blood vessels.
- Intestinal perforation ⌙ peritonitis.
- Appendicitis.
- Amoeboma (Amoebic granuloma) around the ulcer ⌙ stricture of affected area.

II) Extra-intestinal amoebiasis

Due to invasion of the blood vessels by the trophozoites in the intestinal ulcer ⌙ reach the blood ⌙ to spread to different organs as:

→ Liver →

-Amoebic liver abscess or diffuse amoebic hepatitis.
-Affect commonly right lobe either due to spread via portal vein or extension from perforating ulcer in right colonic flexure.
-CP: include fever, hepatomegaly and pain in right hypochondrium.

→ Lung →

•Lung abscess ⌙ pneumonitis with chest pain, cough, fever.
•Amoebic lung abscess usually occur in the lower part of the right lung due to direct spread from the liver lesions, through the diaphragm or very rarely trophozoites may reach the lung via blood.

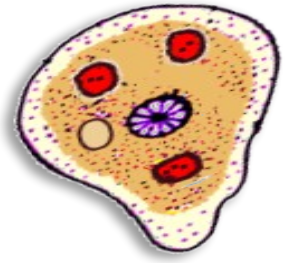
→ **Brain** → Brain abscess ⌐ encephalitis (fatal).

→ **Skin** →

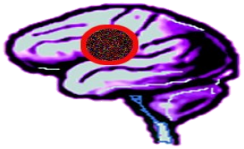
Cutaneous amoebiasis (Amoebiasis cutis):

- when the invasive amoebae escape from the large gut and stick to adjacent skin, usually the perianal and perigenital area.

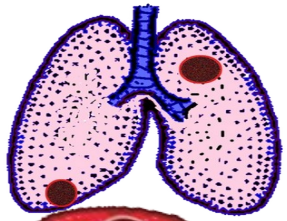
Pathogenesis of amoebiasis



Cerebral amebiasis



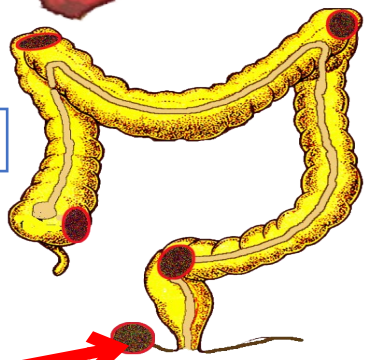
Pleuropulmonary amebiasis



Hepatic amoebiasis

Amoebic hepatitis

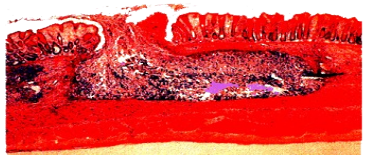
Amoebic liver abscess



Intestinal amoebiasis

Acute amoebic dysentery

Chronic intestinal amoebiasis



Flask-shaped ulcers

Coetaneous amoebiasis

Laboratory diagnosis

I)Intestinal amoebiasis

a) Direct

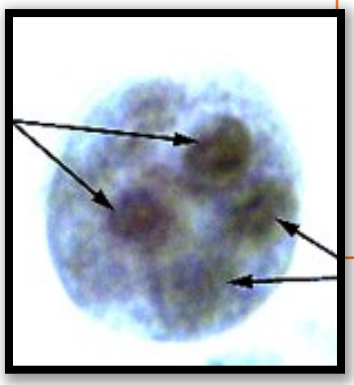
•**Macroscopic:** Offensive loose stool mixed with mucus and blood.

•**Microscopic:**

1-Stool examination: Reveals either trophozoites (in loose stool) or cysts (in formed stool) by direct smear, iodine stained & culture.

2-Sigmoidoscopy: To see the ulcer or the trophozoites in aspirate or biopsy of the ulcer.

3-X-ray after barium enema: to see the ulcer, deformities or stricture.



b) Indirect

-Copro-antigen detection in stool

-Serological tests: ELISA

¬ N.B. These serological tests are positive only in invasive intestinal amoebiasis but negative in asymptomatic carriers.

II)Extra- intestinal amoebiasis

↓

According to the organ affected

a) Direct

1- X- ray:

In lung ⌙ pleuritis with elevation of the diaphragm and lung abcess

2- Ultrasonography, CT scan& MRI:

For liver abscess.

3- Aspiration of abscess content:

For liver abscess (thick chocolate-colored pus containing trophozoites).

b) Indirect

1- Serological tests: As intestinal amoebiasis. They are positive and can persist for years.

2- Molecular by PCR.

3- Blood examination: Leucocytosis.

4- Liver function tests: Increased in amoebic liver abscess.

Treatment

1) Asymptomatic intestinal carrier

↓

Luminal amoebicides

↓

Paromomycin or

Diloxanide furoate

2) Intestinal amoebiasis

↓

Tissue & luminal amoebicides

↓

Metronidazol

(Flagyl) **is the drug of choice** +

Paromomycin or

Diloxanide furoate

3)Extra-intestinal amoebiasis

↓

Tissue & luminal amoebicides

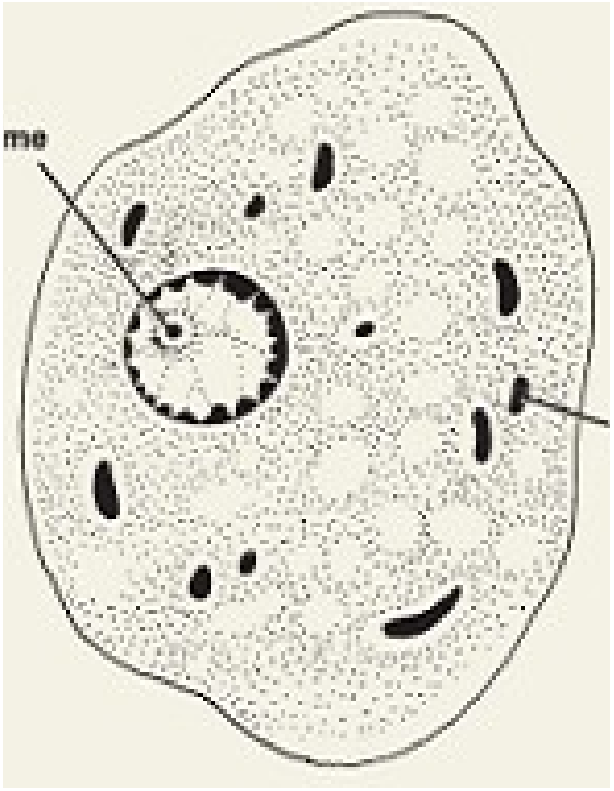
↓

Metronidazol

(Flagyl) +

Paromomycin or

Diloxanide furoate



E. Coli trophozoite

E. Coli cyst

General Mycology

General Mycology

- Mycology is the study of fungi.
- Mycosis is the diseases caused by fungi.

❖ **General characters of fungi:**

1- **Most of them** are saprophyets in soil (consume dead & decaying matter) and **few of them** are parasitic causing diseases in man & animals.

2- They are **eukaryotic cells** (has true nucleus, endoplasmic reticulum & mitochondria).

3- Have **cell wall** consisted primarily of **chitin**. So they resist penicillin and cephalosporins that inhibit the synthesis of cell wall peptidoglycan.

4- Their **cell membrane** contains **ergosterol** in contrast to human cell membrane which contains **cholesterol.**

5- Most of them are **obligate aerobes** & need **carbon for growth** so live on decaying organic matter.

Classification of Fungi

A. Morphological

1- Yeast:

- Oval or rounded.
- Multiply by asexual budding and may form pseudohyphae.

Ex: *Candida albicans.*

2- Mold / Filamentous fungi:

- Have branching filaments (hyphae).
- They may be septate or non septate.
- Reproduce by formation of asexual spores (conidia)
- Ex: *Dermatophytes & Aspergillus.*

3- Dimorphic fungi: occurs in 2 forms:

- Yeast form in tissues or when grown at 37 C.
- Filamentous form at room temperature or in culture when grown at 22 C.
- Ex: *Histoplasma capsulatum.*

Classification of Fungi

Clinically

1- Superficial mycosis:

- Involves stratum corneum without tissue invasion.
- Ex: *Taenia versicolor*.

2- Cutaneous mycosis:

- Involves skin, nail & hair with tissue destruction.
- Ex: *Dermatophytes & Candida*.

3- Subcutaneous mycosis:

- Involves the subcut. tissues, ms & fascia.
- Ex. madura foot.

- **4- Systemic**: primarily infects the lungs & can be disseminated to different organs mainly in immunosuppressed

❖ **5 There are two other kinds of fungal diseases:**

a- Allergies to fungal spores, particularly those of *Aspergillus*. They cause mainly type I hypersensitivity reactions or atopy manifesting as bronchial asthma, fever , urticaria & eosinophilia.

b- Mycotoxicosis

These are diseases due to the consumption of food containing fungal toxins as:

1- Mycetismus a disease caused by Amanita mushrooms that produce fungal toxins. When ingested, they cause sever fatal damage to the liver and kidney.

2- Ergotism, is caused by the mould *Claviceps purpura*, which infects grains and produces ergotamine alkaloides that cause neurologic effects & gangrenous symptoms (due to vasoconstriction of blood vessels especially fingers & toes).

3-Aflatoxins produced by *Aspergillus flavus* when ingested with spoiled grains and peanuts. They are metabolized in the liver to epoxide, a potent carcinogen. they are hepatotoxic, and are suspected of causing hepatic carcinoma in man.

Diagnosis

1- Direct Microscopic Examination:

- after adding a drop of 10% KOH mount and staining

- Gram stain.
- Giemsa stain.
- Calcofluor white stain.
- India ink.

2- Culture:

- Sabouraud dextrose agar (SDA): either incubated at:

➢ 37 C Body temperature for growth of yeast.

➢ 22 C → Room temperature for growth of mold.

→

3- Serology:

For Abs detection or fungal antigen.

4- Biopsy & histopathology

5- PCR:

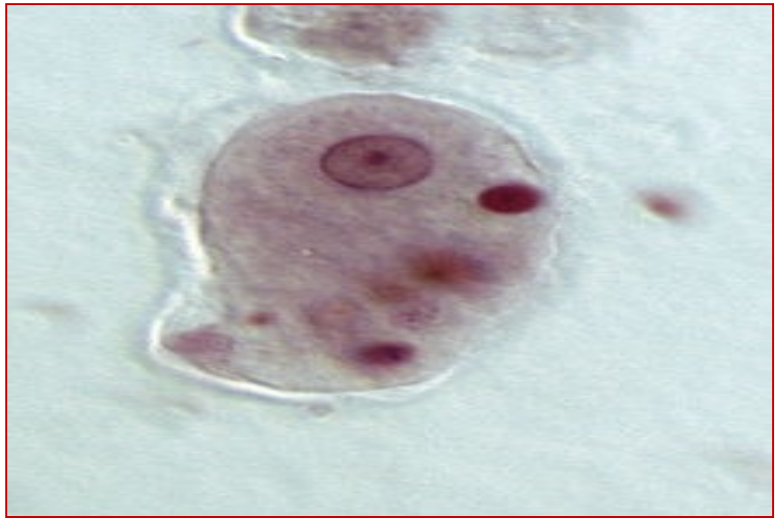
1- Rapid.

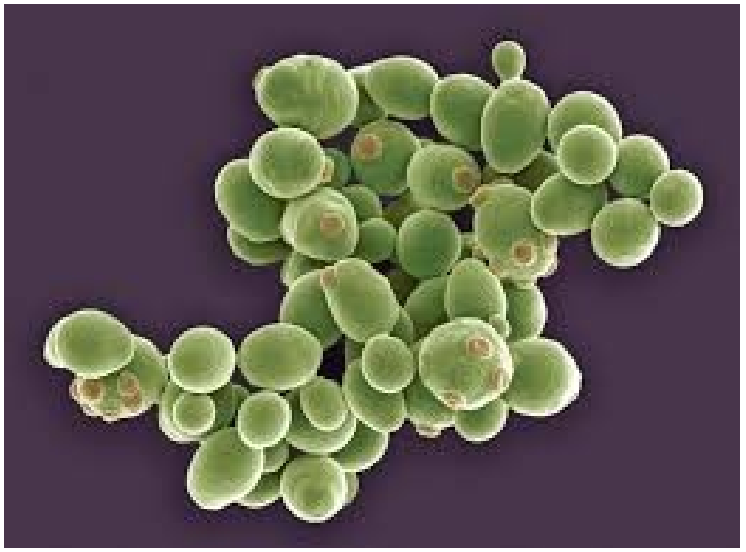
2- Species specific.

3- Expensive.

Parasitology Lab 3

Identify ???






Thank
You