

Doctor 021

# MICROBIOLOGY

Sheet no. 15



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# PROTOZOAL INFECTIONS

## ❖ Introduction:

In parasitic infections in general: the prevalence and incidence differ between different parasites, also parasites differ by geographic areas.

### Ex:

Malaria (protozoal infection): have a high prevalence.

Intestinal infection by parasites such as Ameba (Amoeba) & Giardia is very common in our country (third world countries) and it is related to sanitation.

## ❖ **Classification of Protozoa:**

1- According to organ of locomotion & sexual reproduction (كيف يتحركو (باي عضو) + كيف يتكاثروا):

1) **Rhizopoda**: organ of locomotion → pseudopods

Reproduction → Asexually (binary fission)

Ex: **Amoeba** (Entamoeba histolytica: the only pathogenic (intestinal infection))

2) **Class Ciliata**: Organ of locomotion → cilia

Reproduction → Asexually (binary fission)

Ex: **Giant Balantidium Coli, Ciliates**

3) **Zoomostigophora**: Organ of locomotion → Flagellates

Reproduction → Asexually (binary fission)

Main family → flagellates

Ex: a) blood → **Trypanosoma, Leishmania** / b) tissue → **Toxoplasma**

4) **Sporozoa**: they don't possess an organ of locomotion

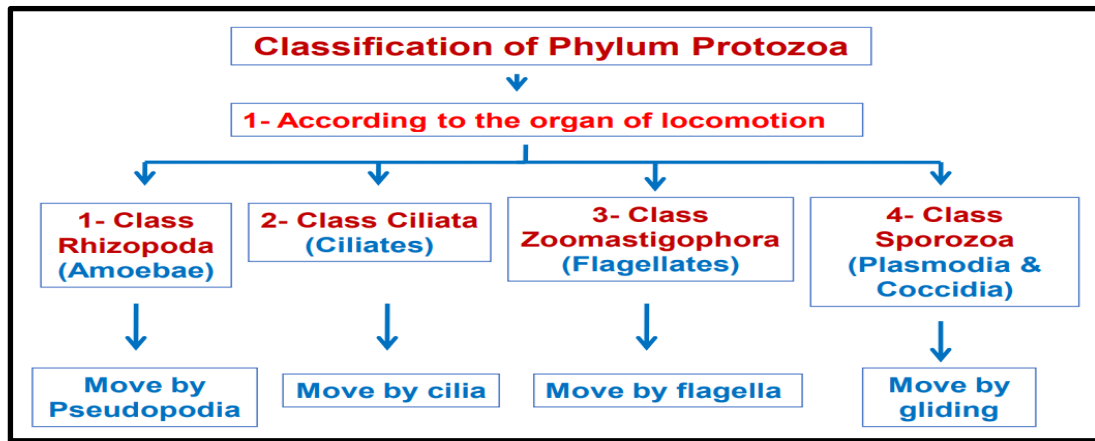
Move by → Gliding

Reproduction (الي بميزهم و بخليهم بعائلة واحدة) → Sexually & Asexually

Ex: a) **Plasmodia** ---> plasmodium (causative agent of **Malaria**)

b) **Coccidia** ---> toxoplasma gondii (tissue coccidia)

- I recommend you to see photos of each type ( even things we will study later).



2- According to the habitat they live in: (what infection it gives)

### 1) Intestinal protozoa:

- a) Amoeba (*Entamoeba histolytic*) → Amoebiasis (Amoebic dysentery)
- b) Ciliates (*Blantidium coli*: Blantidiasis)
- c) Flagellates (*Giardia lamblia*: Giardiasis)
- d) Coccidia (*Cryptosporidium*: Cryptosporidiosis)  
(*Cyclospora*: Cyclosporiasis)  
(*Isospora (cystoisospora)*: Cystoisosporiasis)

### 2) Blood protozoa:

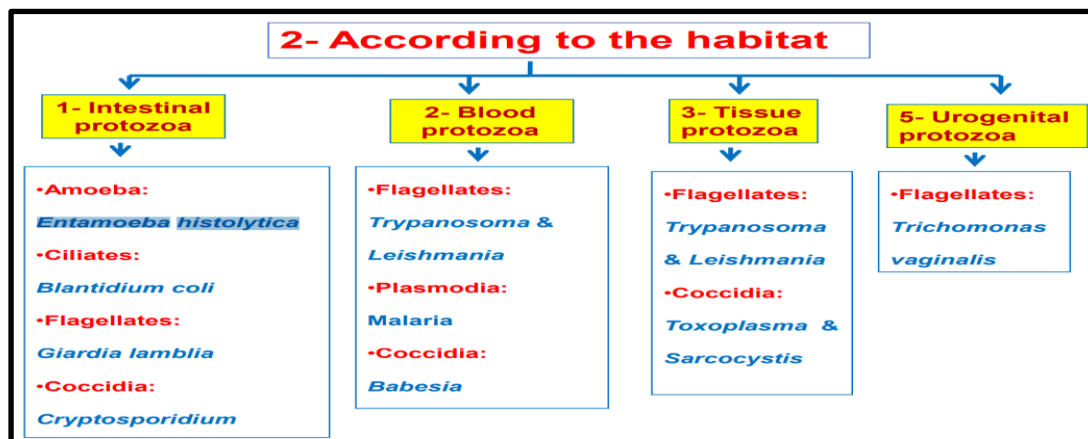
- a) Flagellates (*Trypanosoma*: Trypanosomiasis)  
(*Leishmania*: Leishmaniasis)
- b) Plasmodia (Malaria)
- c) Coccidia (*Babesia microti*: Babesiosis)

### 3) Tissue protozoa:

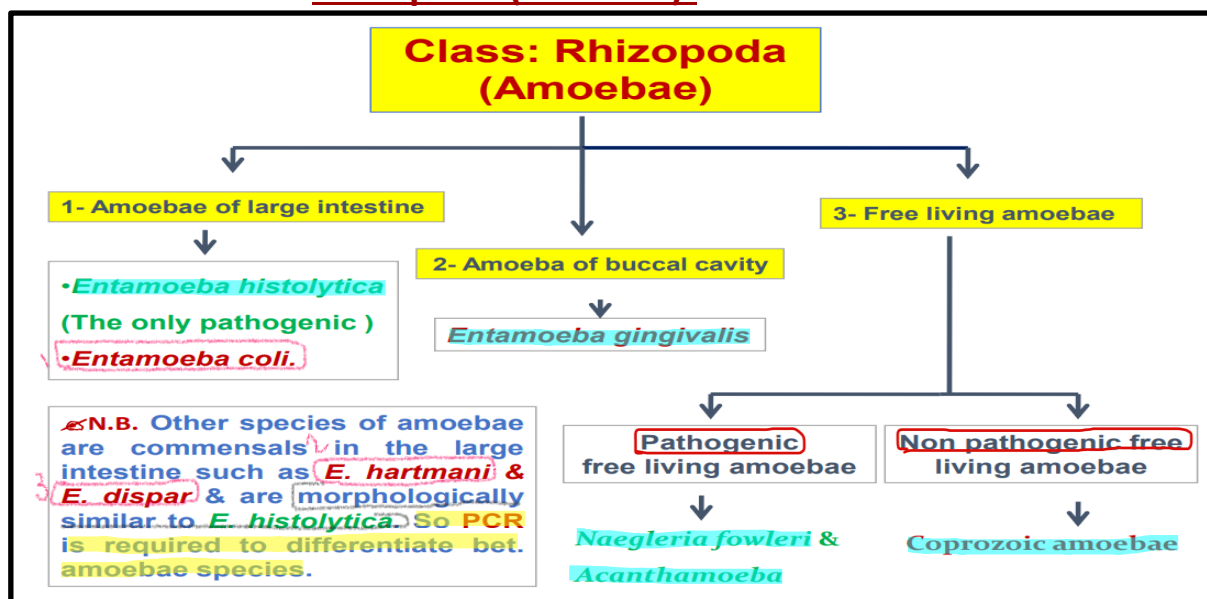
- a) Flagellates (*Trypanosoma*: intr. infection mainly in heart muscle: Chagas disease)  
(*Leishmania donovani*: intr. In the reticular endothelial system: Kala-Azar)
- b) Coccidia (*Toxoplasma brodii*: toxoplasmosis)-(Sarcocystis)

### 4) Urogenital protozoa:

- a) Flagellates (*Trichomonas vaginalis*: causative agent of trichomoniasis (STD))



So let's start with Rhizopoda (Amoeba):



-you have to differentiate between **Amoebes** to know that patient has **Amoebiasis**

- buccal cavity has normal flora (majorly)

- **Naegleria fowleri (brain-eating amoeba)**: from polluted water, one of the worst diseases in prognoses. The death rate is 100% but is very rare.

- **Acanthamoeba**: corneal ceratitis – granulomatous encephalitis

### ❖ **Entamoeba histolytica:**

- The only pathogenic one of rhizopods
- Forms pseudopods and moves through them
- Multiplies by binary fission (asexual reproduction)

- **Disease caused:** Amoebiasis or Amoebic dysentery (الزحار الاميبي)

## ## What is Dysentery (زحار)?

Diarrhea containing blood and mucus. So there is an invasion of mucosa and submucosa. That's why RBC, stool, and mucus **بطالعو**. So they will have abdominal pain.

**-Geographical distribution: Worldwide** (not restricted to any region) **especially in the temperate zone and more common in areas with poor sanitary conditions & poor hygiene.**

**Habitat: Large intestine (caecum, colonic flexures and sigmoidorectal region).** In places where there is fetal stasis (they are corners) in the large intestine. giving a chance for invasion.

**reservoir host (R.H): Dogs, pigs, rats, monkeys,** and also a man.

**definite host (D.H): Man.**

### Morphological characters:

#### 1- Trophozoite stage (Vegetative form or tissue form)

-The active, motile, feeding form, multiplying form inside the body.

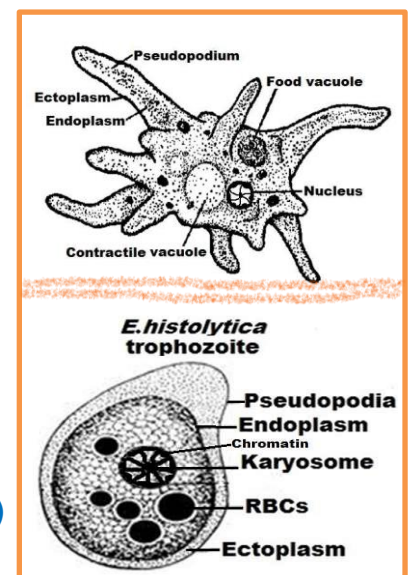
-It has a plasma membrane

-ectoplasm

-endoplasm: 1- reticular (a lot of granules)

2- Karyosome (nucleus) has chromatin

3- RBCs (pathognomonic) differentiating feature (**حصري**)



#### 2- Cyst stage (Luminal form):

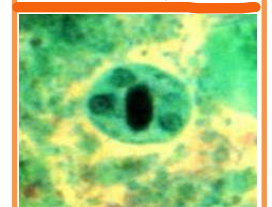
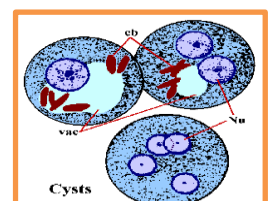
When it is outside the body, Quiescent resistance stage.

It is encysted to protect us from changes in temp. , moisture.

When a cyst enters the body.. becomes a trophozoite.

**(a) Immature cyst (Uninucleate cyst and Binucleate cyst)**

**(b) Mature cyst (Quadrinucleate cyst) (infectious stage) +RBC**



## Modes of infection:

Transmission is fecal-oral (that's why it's related to sanitation)

**1- Contaminated foods (ex. green vegetables) or drinks or hands with human stool containing mature cyst.** In the case of acute diarrheal stool (acute amoebiasis) trophozoite can leave with it before it gets destroyed.

**2- Handling food by infected food handlers as cooks and waiters.**

Make a problem in the reservoir and make the amoebiasis persistent in nature (طبيعة مقاومة) ... Asymptomatic carrier .. find it on food, fruit, and vegetable. That's why you wash it. but what if this water has amoebiasis? Then GG.

**3- Flies and cockroaches that carry the cysts from feces to exposed food..** and this food is eaten by a susceptible person.

**4- Autoinfection (fecal-oral or hand-to-mouth infection).** These people have poor hygiene → scratching perianal area (ex: mother changing diaper to her baby .. or someone scratching himself). Then put hand on mouth or head.

**5- Homosexual transmission.** Anal-oral transmission (direct).

## The life cycle of E. Histolytica:

Ingestion of mature E.histolytica cyst ..... once it reaches the small intestine it goes excystation where it produces 8 trophozoites.....then it can go :

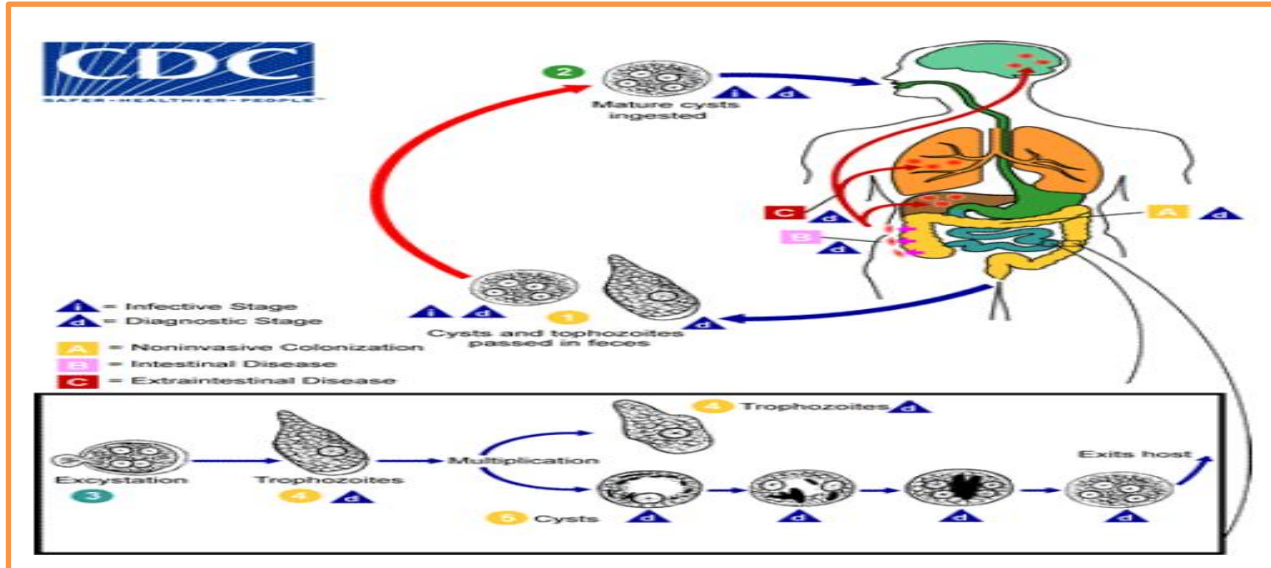
- a) noninvasive colonization. ↪ + chronic
- b) intestinal disease (lumen: acute amoebiasis or mediastinal carrier).
- c) extraintestinal disease (liver, lung, or even brain (amoebic encephalitis)).
- d) convert to cyst and leave the body with feces.

**In the case of acute diarrhea stools** ... it comes with: RBC, mucus, cyst, and trophozoite (since it is fresh) ... stay a couple hours then get destroyed.

That's why both stages are considered diagnostic stage.

i= infective stage, The stage in the life cycle of an endoparasite wherein it can initiate infection to its host.

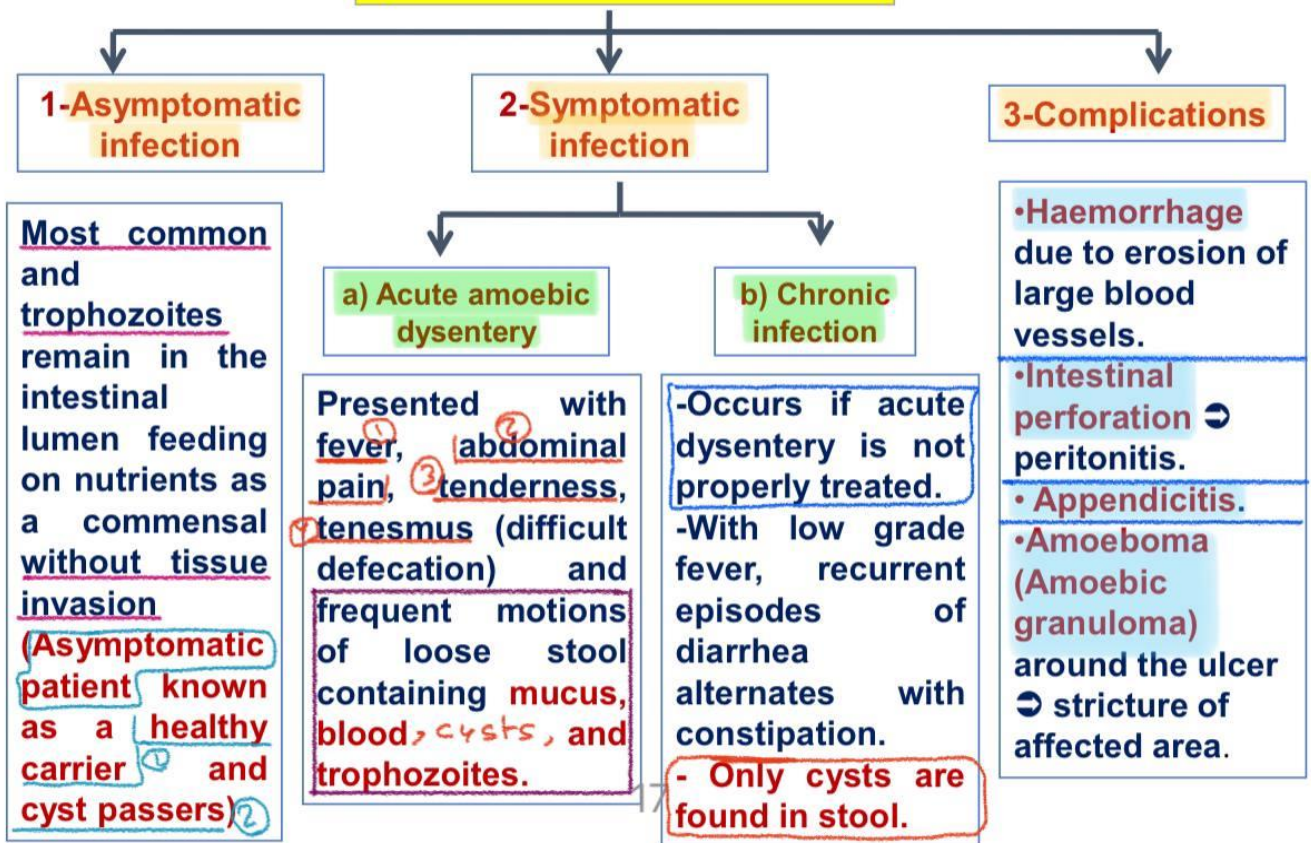
d=diagnostic stage, the stage where symptoms start to show, and the disease can be diagnosed.



Clinical pictures:

## Clinical pictures

### I) Intestinal amoebiasis



1- we are afraid of them because it makes amoebiasis persistent in nature. (Handling food by infected food handlers as cooks and waiters) and they don't feel ill.

2- in case of invasion. (tenesmus means: تننائة )

The pathologic picture (in case of invasion) **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 cytolysed cells, mucus, and trophozoites.

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

## 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:

### a) Liver:

-Amoebic liver abscess or diffuse amoebic hepatitis.

❖ -Affect commonly **right lobe** either due to (hematgenously) spread via portal vein or (direct) extension from perforating ulcer in right colonic flexure.

-CP: include fever, hepatomegaly, and pain in right hypochondrium.



### b) Lung:

- Lung abscess → pneumonitis with chest pain, cough, fever.
- Amoebic lung abscess usually occurs 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.

### c) Skin: Cutaneous amoebiasis (Amoebiasis cutis) due to:

- 1- extension of acute amoebic colitis to the perianal region
- 2- through rupture on the abdominal wall from hepatic, colonic, or appendicular lesions.

### d) Brain: Brain abscess → encephalitis (fatal).

The first type is finished (the doctor said we will talk about it more in GI)  
(BRUH)

## **GIARDIA LAMBLIA (INTESTINAL FLAGELLATE)**

**Giardia lamblia** (also referred to as **Giardia duodenalis** or **Giardia intestinalis**) (indicates the site of pathogenesis) **is the causative agent of giardiasis and is the only common pathogenic protozoan found in the duodenum and jejunum of humans.**

- The main differences between *G.Lamblia* and *E.histolytica* are:
  - 1- The site of pathogenesis. In *G.Lamblia* the site of pathogenesis is small intestines.
  - 2- In *G.Lamblia*, there is no invasion, it is just chronic irritation for the epithelial lining of small intestines and this will lead to malabsorption of fats.
- **Giardia exists in two forms: the trophozoite and the cyst forms.**
- Side note: in U.S. and Canada, the disease is called "beaver fever".

# MORPHOLOGY

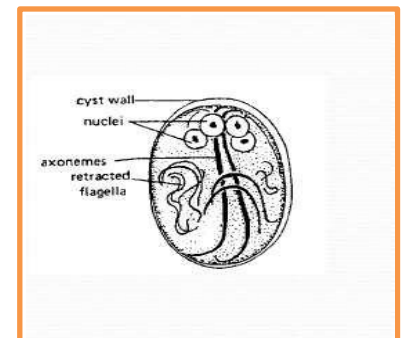
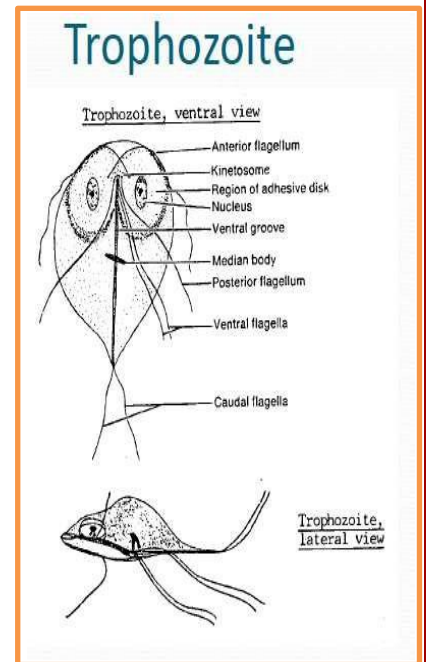
The trophozoite of *G lamblia* is a heart-shaped organism, has four pairs of flagella, 2 nuclei with prominent central karyosome, and 2 axostyles, and is approximately 15  $\mu$  m in length.

A large concave sucking disk on the ventral surface helps the organism to adhere to intestinal villi. As the parasites pass into the colon, they typically encyst, and the cysts are passed in the stool.

The swaying or dancing motion of the trophozoites in fresh preparations is unmistakable.

It uses its ventral bodies for attachment to the duodenum and jejunum.

- Cysts are found in the stool –often in enormous numbers. As the parasites pass into the colon they typically encyst.
- They are ellipsoid, thick-walled, highly resistant, and 8–14  $\mu$  m in length; they contain two nuclei as immature forms and four as mature cysts.



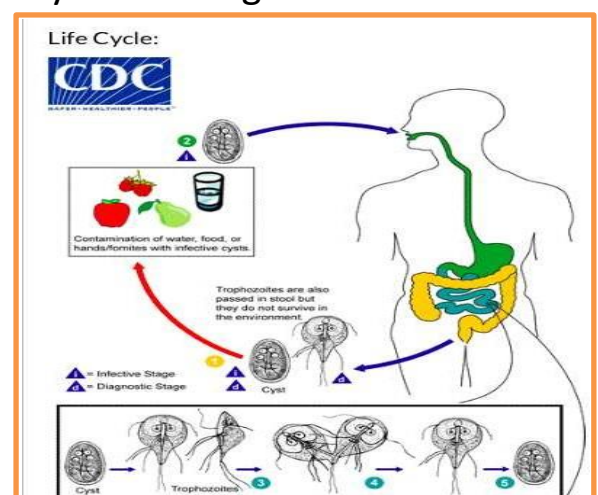
# PATHOLOGY AND PATHOGENESIS

- Giardia lamblia is usually only weakly pathogenic for humans.
  - Cysts may be found in large numbers in the stools of entirely asymptomatic persons.
  - In some persons, however, large numbers of parasites attached to the bowel wall may cause irritation and low-grade inflammation of the duodenal or jejunal mucosa, with consequent acute or chronic diarrhea associated with crypt hypertrophy, villous atrophy or flattening, and epithelial cell damage.
- Remember that the *G.Lambila* infects the small intestines

- Please notice that the G.Lambila doesn't invasive, it just attaches, and attachment will cause villous atrophy, which will cause irritation, and this irritation will cause steatorrhea.
- Steatorrhea: a term used to describe fatty stool.

## TRANSMISSION

- **Humans are infected by ingestion of fecally contaminated water or food** (the most common is water contaminated by the giardial cyst) **containing giardia cysts or by direct fecal contamination, as may occur in daycare centers, refugee camps, and institutions, or during oral–anal sex.**
- **Epidemic outbreaks have been reported at resorts, where overloading of sewage facilities or contamination of the water supply has resulted in sudden outbreaks of giardiasis.**
- **Cysts can survive in water for up to 3 months** viable and infectious.
- It resists chlorination under safe concentration. If you want to sterilize water by chlorination, you have to increase the concentration of chlorine and this makes it unsafe.
- The only way to sterilize water from the cyst is boiling.
- The infective stage is a cyst.
- The diagnostic stage is trophozoite (acute diarrheal stool) and cyst.
- The Excystation occurs in small bowel (duodenum and jejunum).
- Each cyst converts to 2 trophozoite



# GIARDIASIS: CLINICAL ASPECTS

- **The spectrum varies from asymptomatic carriage to severe diarrhea and malabsorption. Subclinical infections common in endemic areas.**
- **In acute outbreaks, Stools may be watery, semisolid, greasy, bulky, and foul-smelling at various times during the course of the infection.**
- **The diagnosis of giardiasis is made by finding the cyst in the formed stool or the trophozoite in diarrheal stools, duodenal secretions, or jejunal biopsy specimens.**
- **Commercially available, enzyme immunoassays(EIAs) detect Giardia antigen in stool**, also use enterotest which is a gelatin capsule surrounding the end of a string is swallowed by the patient; the other end of the string is taped to their cheek.
- In general, it is mild, self-limited, and doesn't cause significant problems.
- Some people suffer from steatorrhea which is a watery, semisolid, greasy, bulky, and foul-smelling stool, and sometimes this leads to dehydration.
- Don't forget that there is no invasion, so there is no bloody stool.

# GIARDIASIS TREATMENT

- **Quinacrine and metronidazole are effective (70%-95%) and are preferred for patients capable of ingesting tablets.**
- **Tinidazole** single injection.
- **Furazolidone is used by pediatricians.**
- **Close contacts should be examined**
- We use Flagyl (metronidazole) for anaerobic bacteria and protozoal infection and it has side effects which are disulfiram-like reactions.

# CRYPTOSPORIDIUM

Cryptosporidium species, typically *C. hominins*, *C. parvum* can infect the intestine in immunocompromised persons (eg, those with AIDS) and cause severe, intractable diarrhea, it isn't fatal, but causes dehydration.

- They have long been known as parasites of rodents, fowl, rhesus monkeys, cattle, and other herbivores and have probably been an unrecognized cause of self-limited, mild gastroenteritis and diarrhea in humans.
- Cryptosporidium inhabits the brush border of mucosal epithelial cells of the gastrointestinal tract, especially the surface of villi of the lower small bowel, NO INVASION.
- Crypto: crypts of the villi in the intestine  
Sapporo: spores within these crypts

## CLINICAL ASPECTS

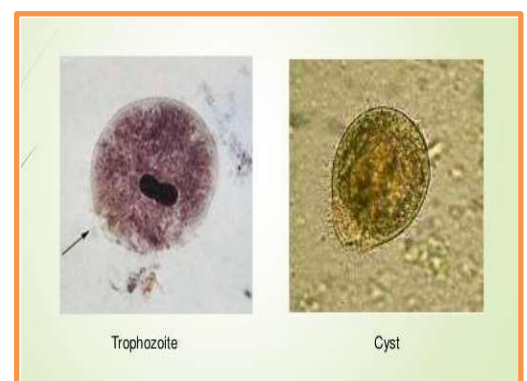
- Clinically, range from self-limited watery diarrhea (immunocompetent) to chronic, severe, non-bloody diarrhea with nausea, vomiting, abdominal pain, and anorexia resulting in weight loss and death (immunocompromised).
- Diagnosis depends on detection of oocysts in fresh stool samples.
- Stool concentration techniques using a modified acid-fast stain are usually necessary. stool antigen detection by direct fluorescent antibody or EIA tests is now commercially available.
- Nitazoxanide, a synthetic drug, has been approved for use in all patients over 1 year of age in the United States and is reported to have a cure rate of 72% to 88% by the CDC.
- Notice that CRYPTOSPORIDIUM is SPOROZOA (there is sexual multiplication)
- Oocyst is infectious so the person-to-person transmission is well documented.
- CRYPTOSPORIDIUM is diagnosed by modified acid-fast stain, the oocyst appears red with acid-fast staining

# CYCLOSPORA

- The life cycle of Cyclospora is similar to that of Cryptosporidium and appears to involve only a single host. Cyclospora, however, differs from Cryptosporidium in that Cyclospora oocysts are not immediately infectious when passed in stools because it needs sporulation.
- Pathogenesis: Altered mucosal architecture with shortening of intestinal villi due to diffuse edema and infiltration of inflammatory cells leads to diarrhea, anorexia, fatigue, and weight loss.
- when examining stools for oocysts (8–10  $\mu$  m), which are acid-fast positive (reddish).
- Cyclospora infections are treatable with trimethoprim-sulfamethoxazole (TMP-SMZ).

# BALANTIDIUM COLI

- It causes balantidiasis or balantidial dysentery, which is the largest intestinal protozoa of humans.
- The trophozoite is a ciliated oval organism 60 X 45  $\mu$  m or larger. It has a steady progression and rotation around the long-axis motion.
- Most infections are apparently harmless. However, rarely, the trophozoites invade the large bowel and terminal ileum causing erosions and ulceration **NO INVASION**
- Treatment: Oxytetracycline, may be followed by Iodoquinol or Metronidazole.
- It is ciliated protozoa
- The patient suffers from watery diarrhea.
- It has two nuclei in both cyst and trophozoite, macronucleus (bean shaped) and micronucleus.
- The cyst has a cytosome which is considered a mouth of it.



The second type is over.

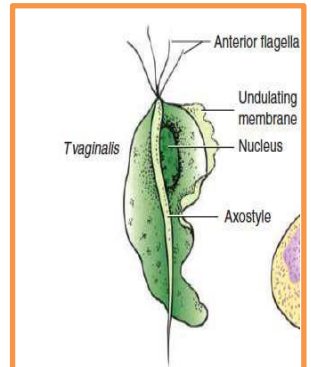
# SEXUALLY TRANSMITTED PROTOZOAN INFECTION

## ❖ TRICHOMONAS (Urogenital flagellated protozoa):

**Trichomonads are flagellated protozoa with 3-5 anterior flagella.** 4 anterior + 1 axostyle continue posteriorly.

(tri but 5 flagella. Hmm, it's a misleading name).

-Only a trophozoite form (no cyst form). has bile form.



It is pear-shaped with an undulating membrane lined with the flaellum and 4 anterior flagella. It is about 5-30 X 2-14 µm. it moves with a wobbling or rotating motion.

Three members of the genus *Trichomonas* parasitize humans:

1) *Trichomonas hominis* (intestinal cavity)    2) *Trichomonas Tenax* (oral cavity)    3) *Trichomonas vaginalis*

-but only T vaginalis is an established pathogen.

*Trichomonas vaginalis* cause trichomoniasis in human (STD). Since it is only a trophozoite form it can't go outside, it needs direct intimate contact (sexual contact). thus, leading to STD.

Direct contact of T vaginalis with the squamous epithelium of the genitourinary tract results in the destruction of the involved epithelial cells and the development of a neutrophilic inflammatory reaction and petechial hemorrhages.

In females, trichomoniasis is more symptomatic than in males. Where more than 50% of females carrying the disease are symptomatic while only 10% of males are symptomatic.

(In females) The most common symptom is: Itchy followed by dysuria (pain during urination) & dyspareunia (pain during sex) ..**Low-grade inflammation limited to vulva (vulvitis) vagina (vaginitis) and cervix (cervicitis) causing frothy yellow or creamy discharge (foul (fish) smell).**

In males, it may infect the prostate (prostatitis), seminal vesicles, and urethra (urethritis).

**Wet mount examination** (in the vagina) **for motile trophozoites is sufficient in most symptomatic cases.**

If a patient came to you and wants treatment, it's not enough to be treated alone. You have to treat both patient and their sexual partner.

(لو لحاله حيضل المرض يرجع)

**-Treatment: Topical and systemic Metronidazole (Flagyl).**

**Tinidazole and Ornidazole are equally effective with fewer side effects.**

## BLOOD AND TISSUE PROTOZOAN INFECTIONS

**Haemflagellate:**

**1- Trypanosoma (Trypanosomiasis)**

**2- Leishmania (leishmaniasis or leishmania)**

They move by the means of flagella and exist in the blood.

4 morphological forms: trypomastigote, epimastigote, promastigote, and amastigote.

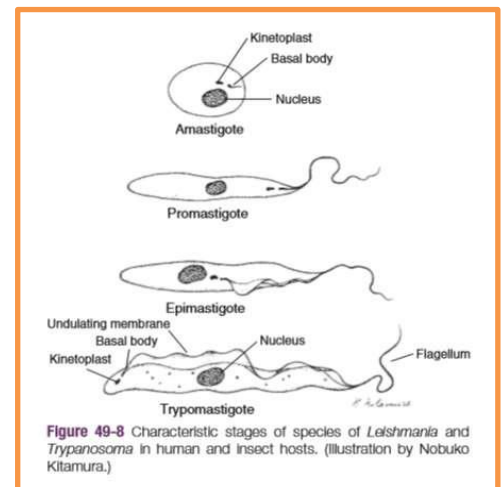


Figure 49-8 Characteristic stages of species of *Leishmania* and *Trypanosoma* in human and insect hosts. (Illustration by Nobuko Kitamura.)

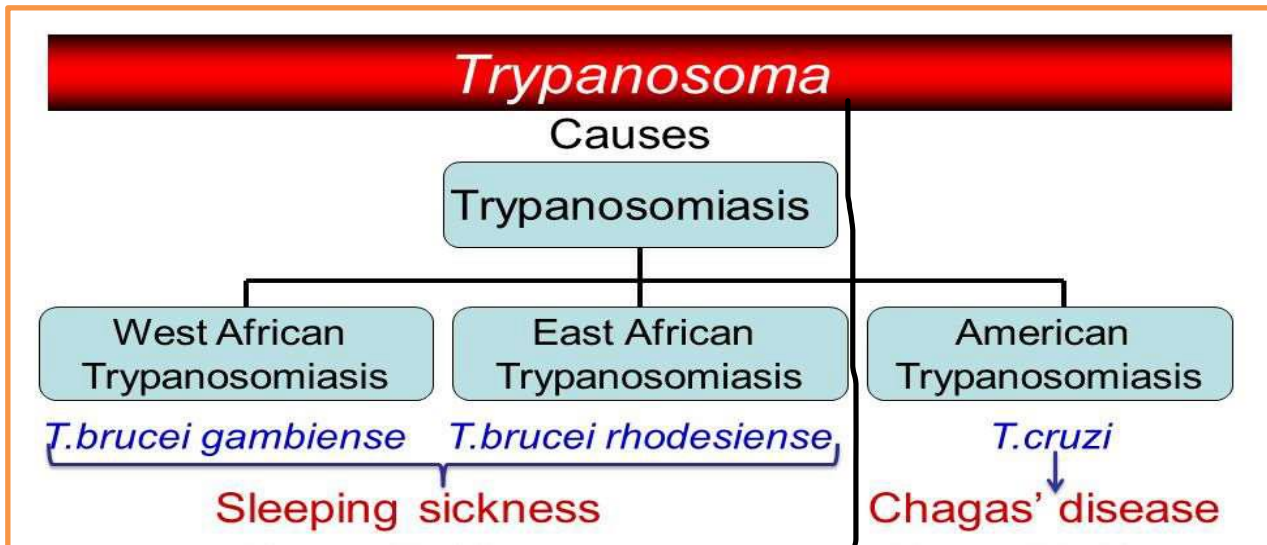
***Promastigote* and *epimastigote* are found in the vector (since it is vector-borne diseases: infections transmitted by the bite of infected arthropod species, such as mosquitoes, ticks, triatomine bugs, sandflies, and blackflies (google).)**

***Amastigote* is a round intracellular form. The diagnostic stage in case of intracellular diseases (some Trypanosomiasis and leishmaniasis)**

***Trypomastigote* is found in the human host. The diagnostic stage and the infective stage in case of Extracellular diseases. And infective stage in intracellular diseases (can be diagnostic also يعني بس في اماكن معينة بالجسم)**



❖ **Trypanosoma:**



In microbiology it is trypanosomiasis. But in clinical: African and American.

**African trypanosomiasis:** extracellular stages, **African sleeping sickness** as the patient in the last levels of disease will sleep anywhere because it reaches the brain (fetal disease). **caused by T. Brucei**

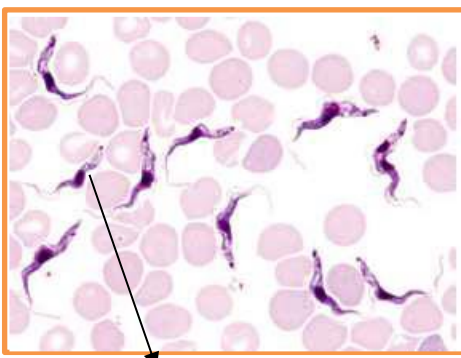
**American trypanosomiasis (Chagas' disease):** intracellular stages. **caused by T. Cruzi.**

**Morphology:**

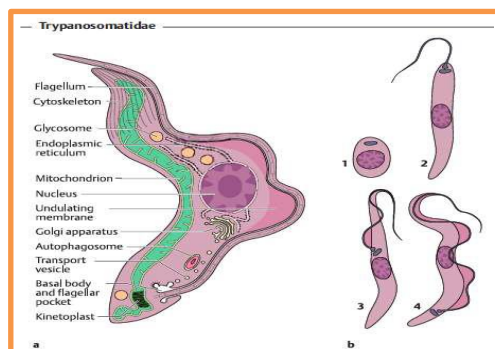
-spindly, uniflagellate stages (trypomastigote, epimastigote, promastigote)

-rounded, amastigote form

-African Trypanosomiasis is caused by an extracellular parasite



Trypomastigote (African Trypanosomiasis)



## African Trypanosomiasis:

- Is caused by 2 sub spp. :

### a) *T. brucei gambiense* :West African trypanosomiasis ,

The R.H: another infected human.

### b) *T. brucei rhodesiense*: East African trypanosomiasis

The R.H: infected humans and animals. The progression of the disease is worse and more complications occur. Fertility is higher.



- **Vector: tsetse fly (*Glossina* spp.). found only in rural Africa.**

### a) *Glossina palpalis* transmits *T. b. gambiense*

### b) *Glossina morsitans* transmits *T. b. rhodesiense*

- **Works by:** injection (هي بتغزك و بتطلع المادة و بتنتشر بجسمك , عكس الاميريكان)
- **infective stage + diagnostic stage:** trypomastigote.

## American trypanosomiasis (Chagas' disease):

- **Caused by:** *Trypanosoma cruzi*

- **Zoonosis**

- **Transmitted by vector: reduviid bugs.** (kissing bug)

- **Works by: defecates while taking a blood meal** (eggs full of trypomastigote (infective stage)) (هون هي بتغز فقط بتحط البيض.. بعدها بالحكة تنتشر)

- **Definitive host: Human, dog, cat, rats.**

- **Habitat in the Definitive host** (diagnostic stage):

**Trypomastigote in blood, Amstigote in tissue**

(Trypomastigote gets intracellular to heart muscle specifically)



It is called (kissing bug) because it prefers face area..when defecation and scratch it .. it gets access to the conjunctiva of the eye not site of the bite.

In acute infection → unilateral eye swelling (romaña sign).

## ❖ Leishmania:

Life cycle requires two hosts:

a) **vertebrate: mammalian host**

b) **Invertebrate vector: female sand fly.**



### • Obligate intracellular organism

- **Infects primarily: phagocytic cells and macrophages.**
- **The incubation period ranges from: 10 days to 2 years.**
- We only have one form of it in Jordan is cutaneous leishmaniasis.
- **diagnostic stage:** amastigote.
- **infective stage:** promastigote.

### • **Transmission (blood-borne infection):**

1. **Bite of sand fly.**
2. **Transfusion blood and transplantation** (from an infected person)
3. **Mother to baby** (transplacentally).
4. **Direct contact; from man to man through nasal secretion.**



(ex : cutaneous leishmaniasis)

### • **Leishmaniasis is divided into clinical syndromes according to what part of the body is affected most:**

#### 1. **Cutaneous Leishmaniasis** (*L.tropica*, *Leishmania major*)

It can be anywhere but mostly in the face. They might heal but take a long time so they leave a scar. The patient come for cosmetic reasons.

**Other names:** allebo boil, Baghdad boil, oriental sore.



#### 2. **Mucocutaneous leishmaniasis** (*L.braziliensis*)

On mucus membrane.

**Another name:** rhincerebral leishmaniasis.

Lesions on the nose and destruction of the nasal septum.



### 3. Visceral Leishmaniasis (*L. donovani*).

Deeper intracellular infection by Leishmania. It's not only affecting macrophages and APCs of skin and mucus membrane, but it also reaches bone marrow, and liver leading to abdominal distention and accumulation of body fluid.

**Diagnostic form:** amastigote in bone marrow

**Other names:** Kala-avar , domdom disease



## PLASMODIUM (BLOOD SPOROZOA)

**Plasmodium:** is a genus of parasitic alveolates, many of which cause malaria in their hosts.

The parasite always has two hosts in its life cycle: A dipteran insect host and a vertebrate host.

#### • Species:

1. *P. falciparum* (the major species associated with deadly infections throughout the world). The most important . causes malignant tertian malaria → high temperature (above 42) + complications: cerebral malaria (the most dangerous type of malaria).

2. *P. malariae*: the most ancient (now is very rare) → Quantal malaria (with a cycle of 72 hours).

3. *P. vivax*

4. *P. ovale*

⊖ The most common. causes benign tertian malaria

Benign: not high complications nor temp. , tertian: has a cycle (48 hours).

5. *Plasmodium knowlesi*: malaria only in monkeys. But some countries live in places near monkeys so it may transfer to humans causing the 5<sup>th</sup> malaria Or semen malaria.

## Mechanism of Infection:

Remember it is Sporozoa (Asexual and sexual multiplication).

In the vector sexually, in humans Asexually.

- **The vector for malaria is the female anopheline mosquito.**

**When the vector takes a blood meal, sporozoites contained in the salivary glands of the mosquito are discharged into the puncture wound.**

So the infective stage is sporozoites.

**Within an hour, these infective sporozoites are carried via the blood to the liver (where they do Asexual multiplication), **where they penetrate hepatocytes and begin to grow, initiating the pre-erythrocytic or primary exoerythrocytic cycle (in the liver)****

**The sporozoites become round or oval and begin dividing (Asexually) repeatedly.**

**Schizogony (merogony), when hepatocytes get full and explode **results in** releasing **large numbers of exoerythrocytic merozoites**.**

**Once these merozoites leave the liver, they invade the red blood cells (RBCs) of the peripheral blood, initiating the intraerythrocytic cycle.**

**Once the RBCs and reticulocytes have been invaded, the parasites grow and feed on hemoglobin. and hemolysis when explode which leads to hemolytic anemia.**

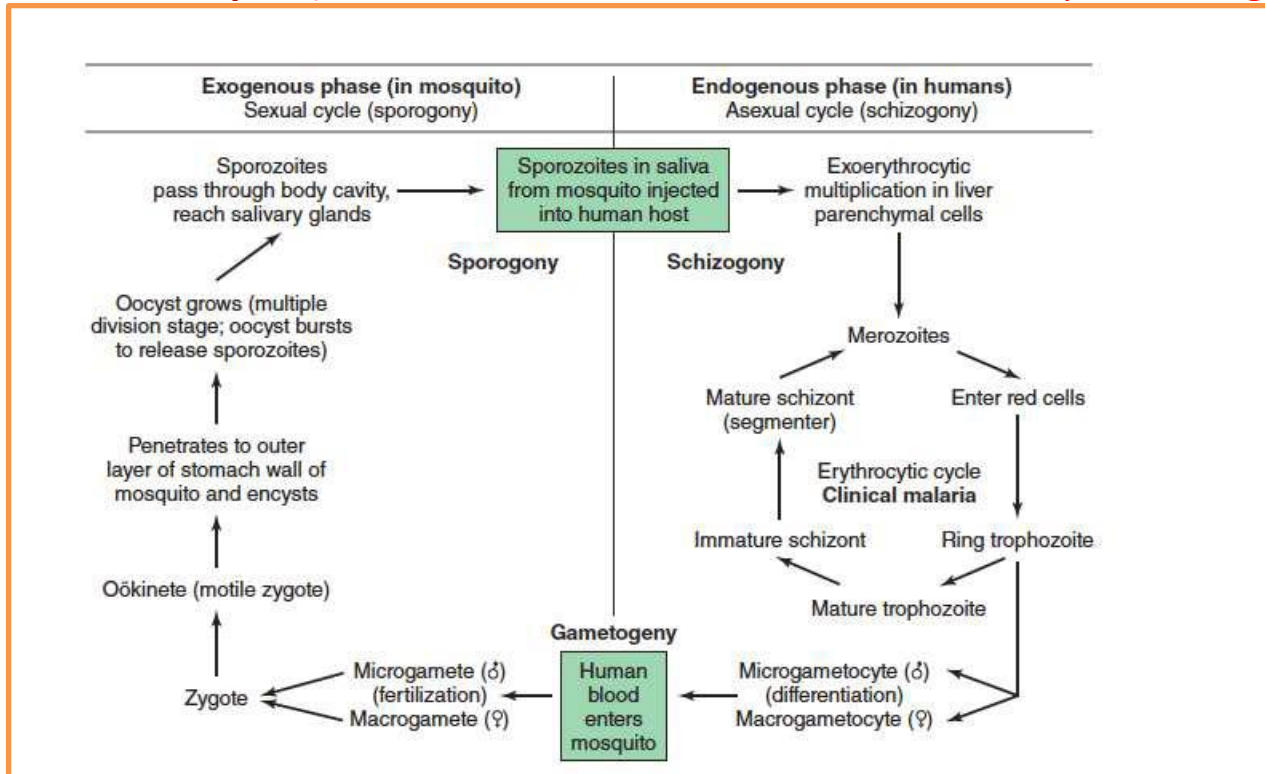
There is a problem when RBCs is infected by *P. falciparum* as they tend to become abnormal → clump (cytoadherence) which leads to ischemia in the blood going to the brain → cerebral malaria

**Within the RBC, the merozoite (or young trophozoite) is vacuolated, ring shaped, more or less ameboid, and uninucleate.**

**Once the nucleus begins to divide, the trophozoite is called a developing schizont**

**The mature schizont contains merozoites (whose number depends on the species), which are released into the bloodstream.**

## Malaria life cycle (the written above is here + few the doctor pass through)



### A special case!!:

**A dormant schizogony may occur in P. vivax and P. ovale organisms, which remain quiescent in the liver.**

**These resting stages have been termed hypnozoites and lead to a true relapse, often within 1 year or up to more than 5 years later.**

(its treatment is different from other malaria forms)

## TISSUE PROTOZOA

### ❖ Toxoplasma gondii (Tissue sporozoa):

**It is a coccidian protozoa (sexual and Asexual multiplication) with worldwide distribution that infects wide range of animals and birds but does not appear to cause disease in them.**

**The normal final hosts are strictly the cats and its relatives, the only host of which the oocyst-producing sexual stage of toxoplasma can develop.**

Human infection is (اخر احتمال ممكن) . only in eating meat that has the Oocyst like lamb or pork that may eat soil or herb that has cat feces.

There is no problem in immunocompetent individuals.

In immune-compromised people → disseminated toxoplasmosis.  
In pregnant mothers → congenital toxoplasmosis (In the first trimester).  
Which leads to the baby stillbirth or abortion.  
This does happen only in the first (the 2<sup>nd</sup> the baby is immune)  
That doesn't happen in healthy women who are not pregnant.

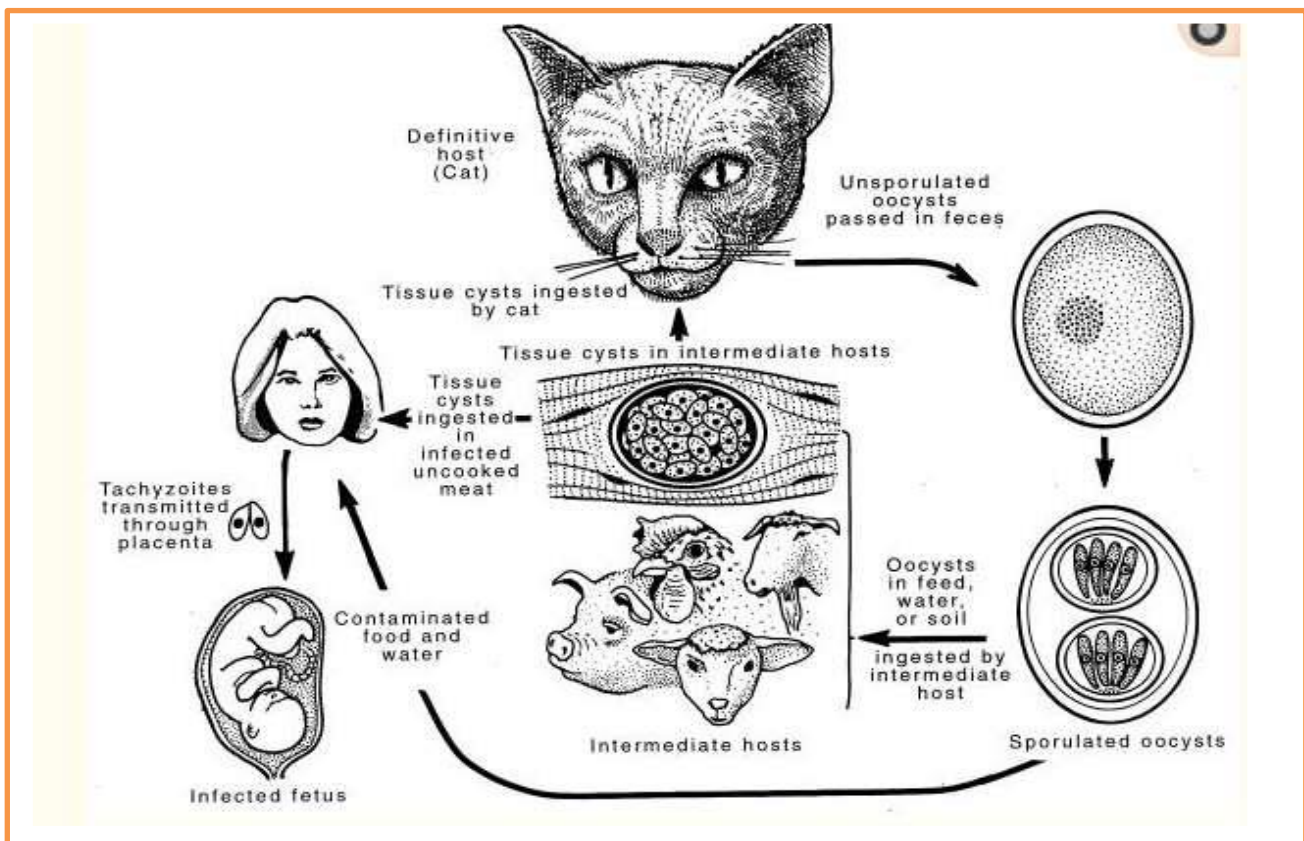
postnatal toxoplasmosis → blindness & mental retardation.

**It produce either congenital or postnatal toxoplasmosis.**

**Congenital infections occur in non-immune mothers during pregnancy.**

**When oocysts are ingested can either repeat their sexual life cycle in a cat or if ingested by a human- can establish an infection in which it can reproduce asexually. Where it opens and releases sporozoites to the duodenum and then invades various cells, especially macrophages where they form tachyzoites which spread the infection to lymph nodes and other organs.**

**Latent infections occur with Toxoplasma (parasites in tissue cysts are called bradyzoites).**



**This is the 2021 sheet, we highly recommend you to at least read it (study it if you only studied the old sheet without watching the lecture)**

**Good luck...**

**THE END**

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**V1**