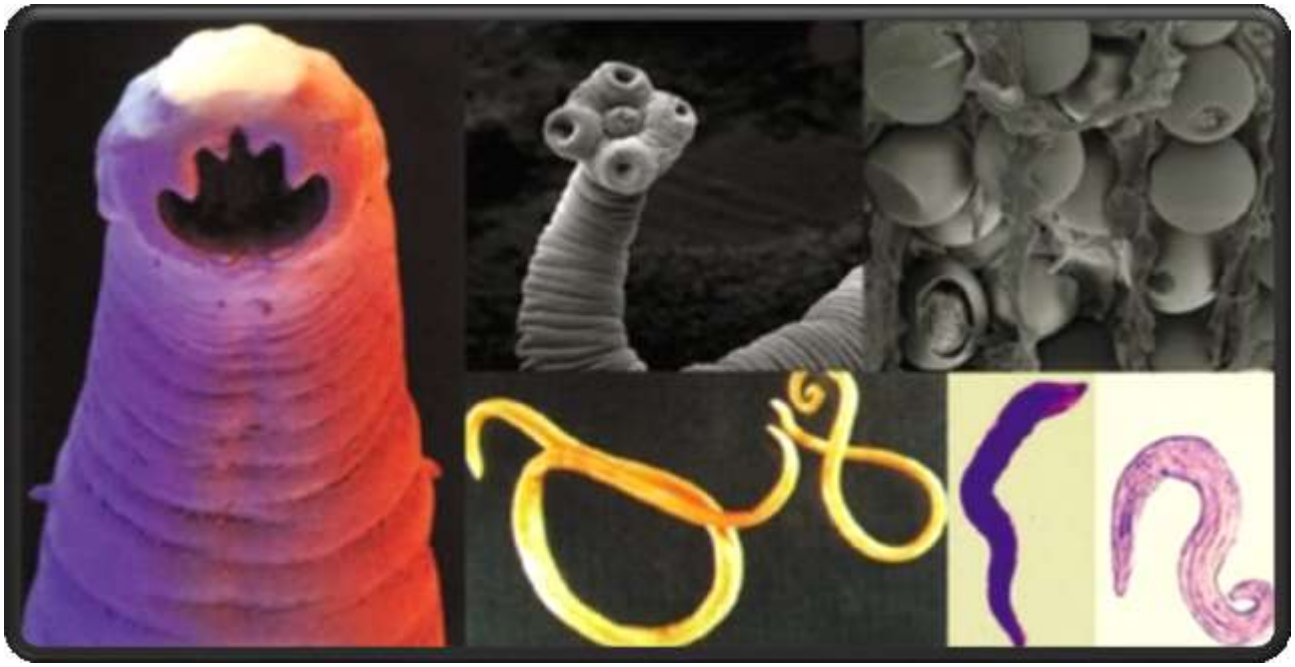
Parasitology



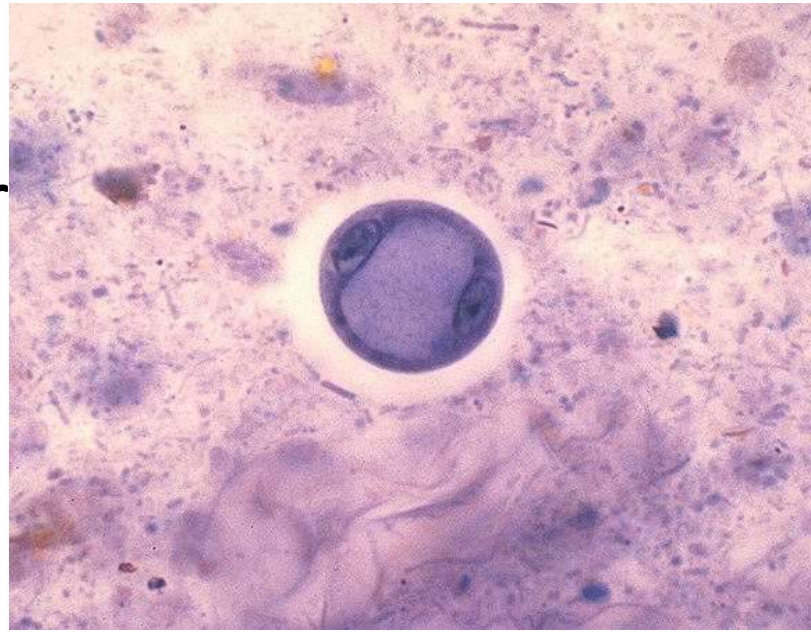
Lec.Dr.Ruwaidah F. Khaleel

Introduction

- **Parasites** are traditionally considered to be protists, worms and arthropods, adapting themselves to live in or on another organisms termed host
- The relationship between two dissimilar organisms that are adapted to living together is called symbiosis and the associates are symbionts
- Textbooks on parasitology frequently distinguish among the fallowing three general kinds of symbiosis: **commensalism, mutualism and parasitism**

Commensalism:

- this occur when one member of association pair, usually the smaller, receives all the benefit and the other member is neither benefited nor harmed, the commensai organism referred as non-pathogenic, exp. **Entamoeba. coli**

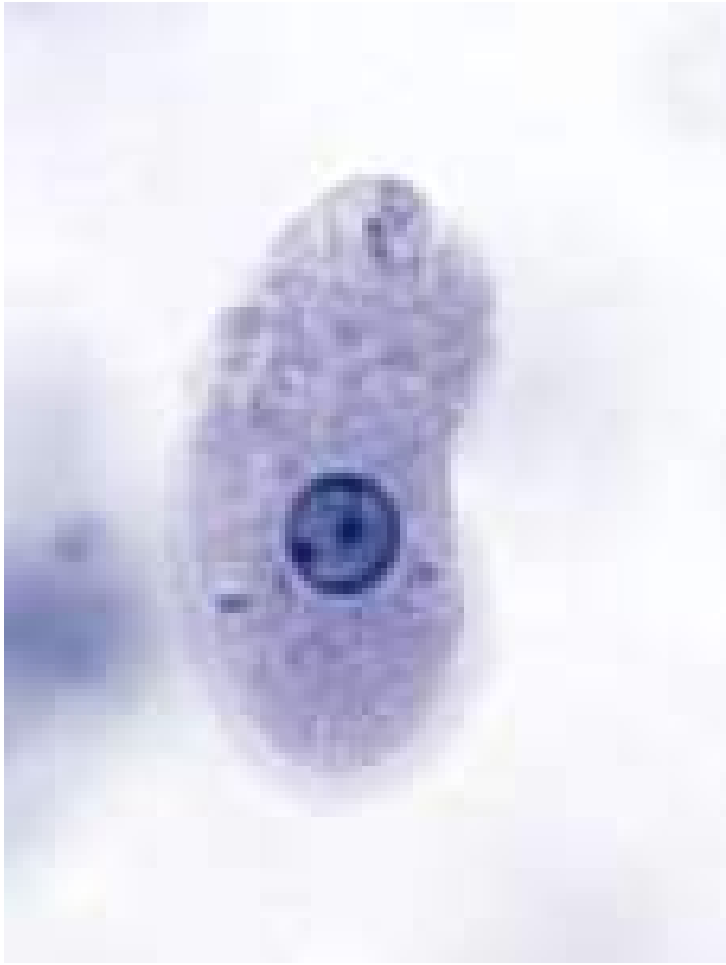


Mutualism:

- This occurs when each member of association benefits the other exp. **Termites and their flagellates.**

Parasitism :

- The original meaning of the word parasite (from the Greek parasitos) was one who eats at another′s table, or one who lives at another's expense. Parasite benefits, gain shelter and nutrition on the expanse of the other (host) the host may suffer from wide range of functional and organic disturbance due to such association. The parasitic organism referred as pathogenic, exp. **Entemeoba histolytica** (pathogenic).

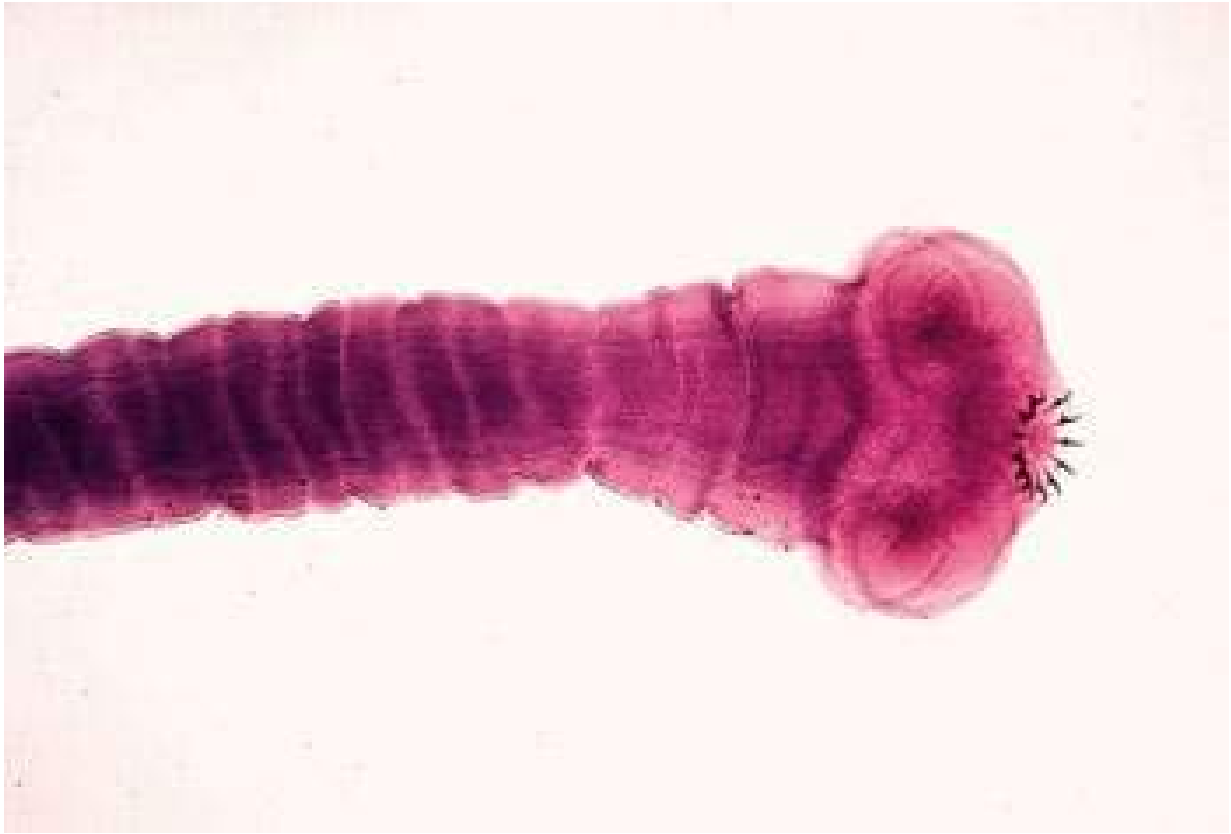


Type of parasites

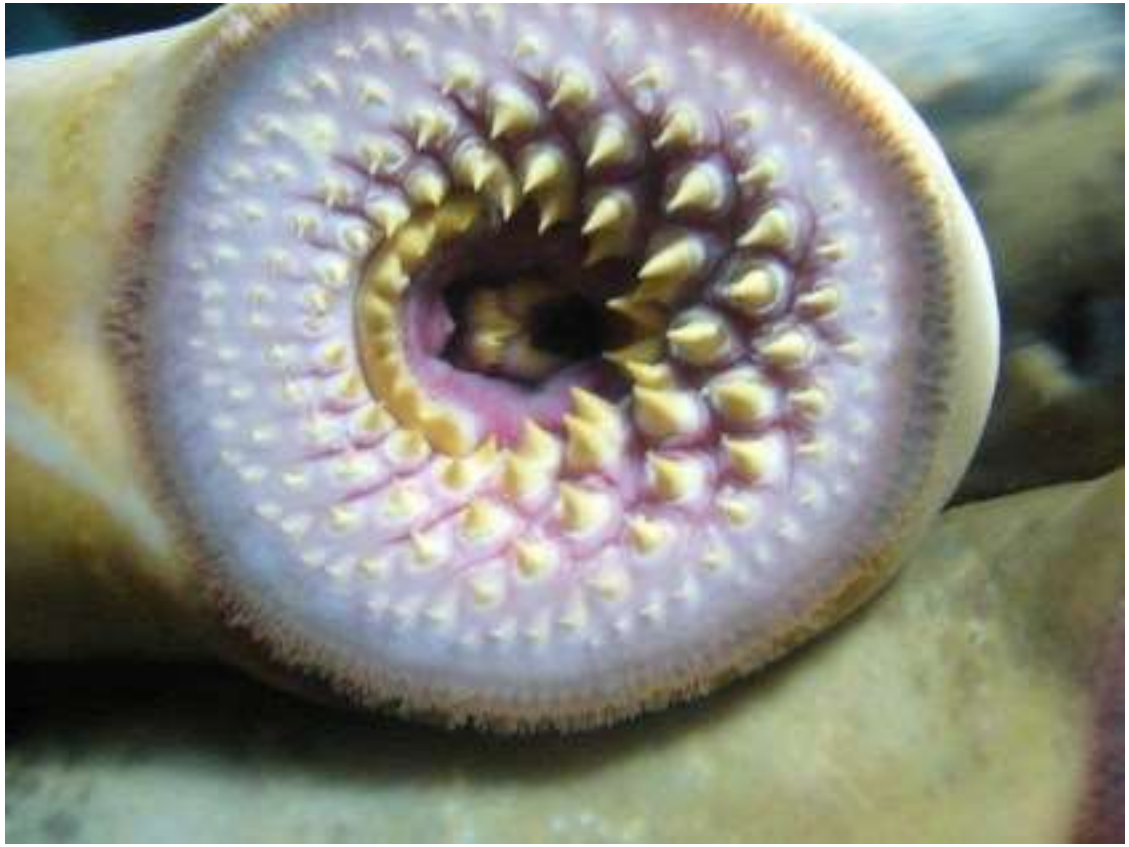
Various descriptive names denote special type or functions of parasites such as:

1. **Ectoparasites:** lives on the outside of the body (on the surface) and the relationship called infestation most parasitic arthropods belong to this category

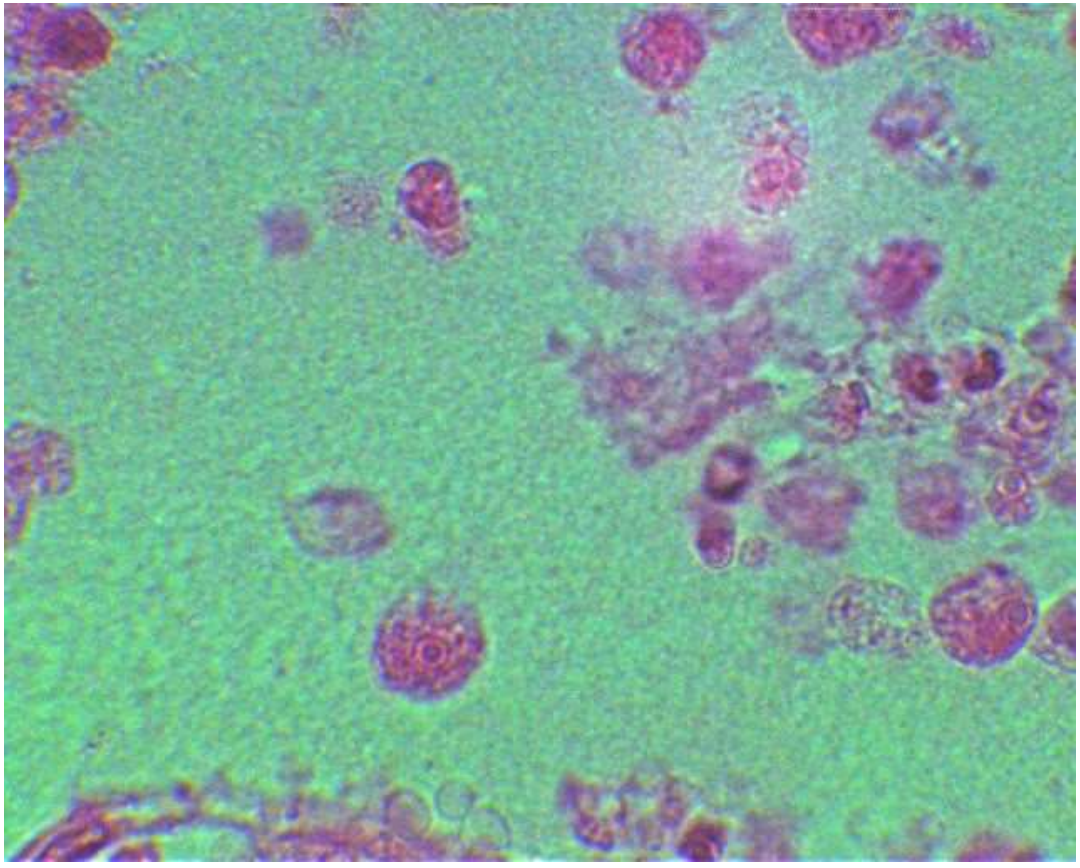
2. Endoparasites: lives within the body of the host(infection).



3. Temporary or intermitted parasite: visit the host from time to time for food.



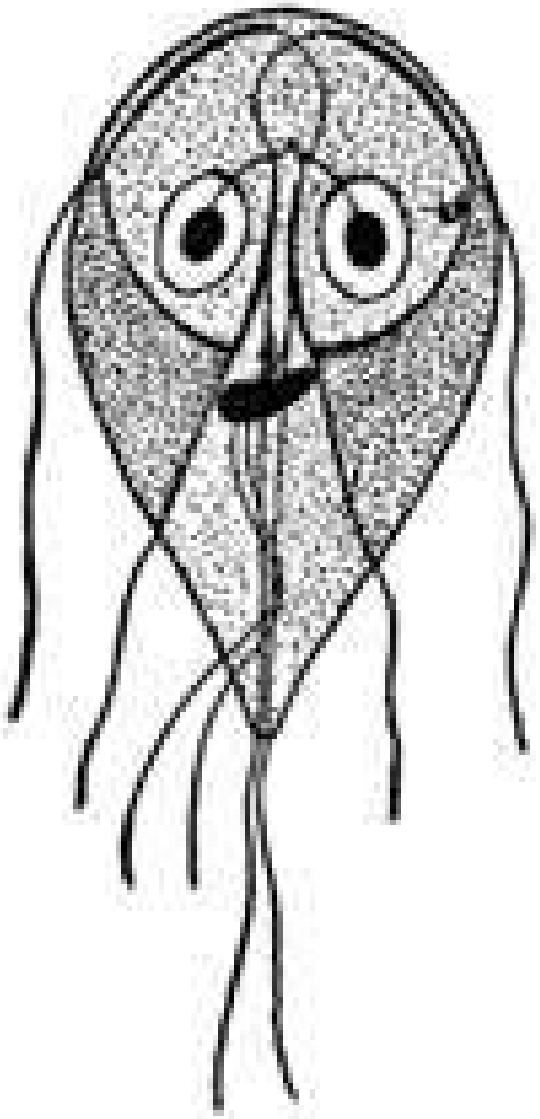
4. Facultative parasites: organism that can exist in a free living state or as a parasite.



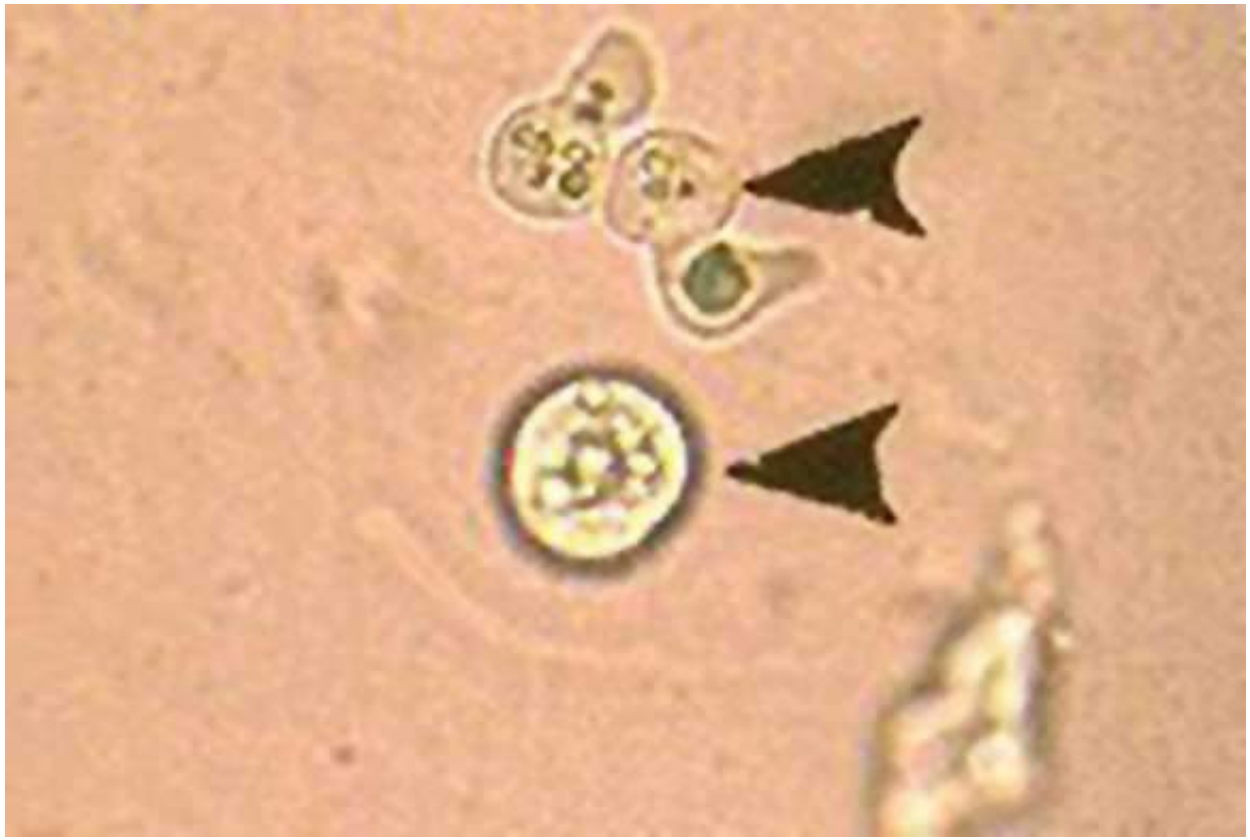
5. Obligatory parasite: cannot survive without the host.

6. parasitoids: fails to reproduce in its host but merely eats it from the inside.

7. Coprozoic parasites: are species that have passed through the alimentary canal without infecting the host

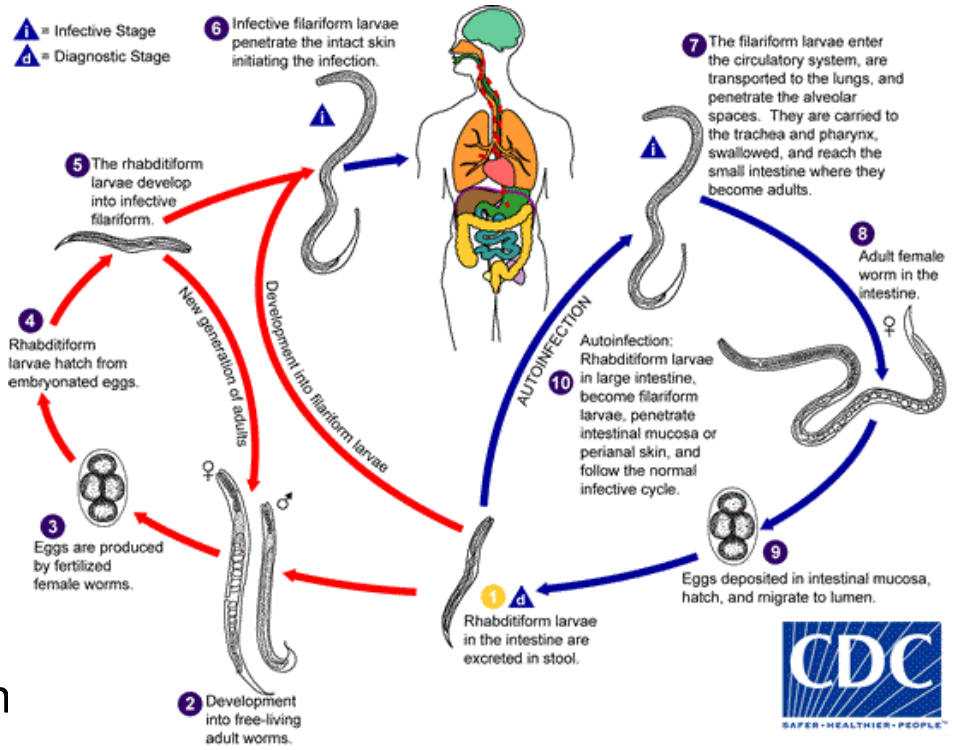


8. Pseudoparasites: are artifacts mistaken for parasites.



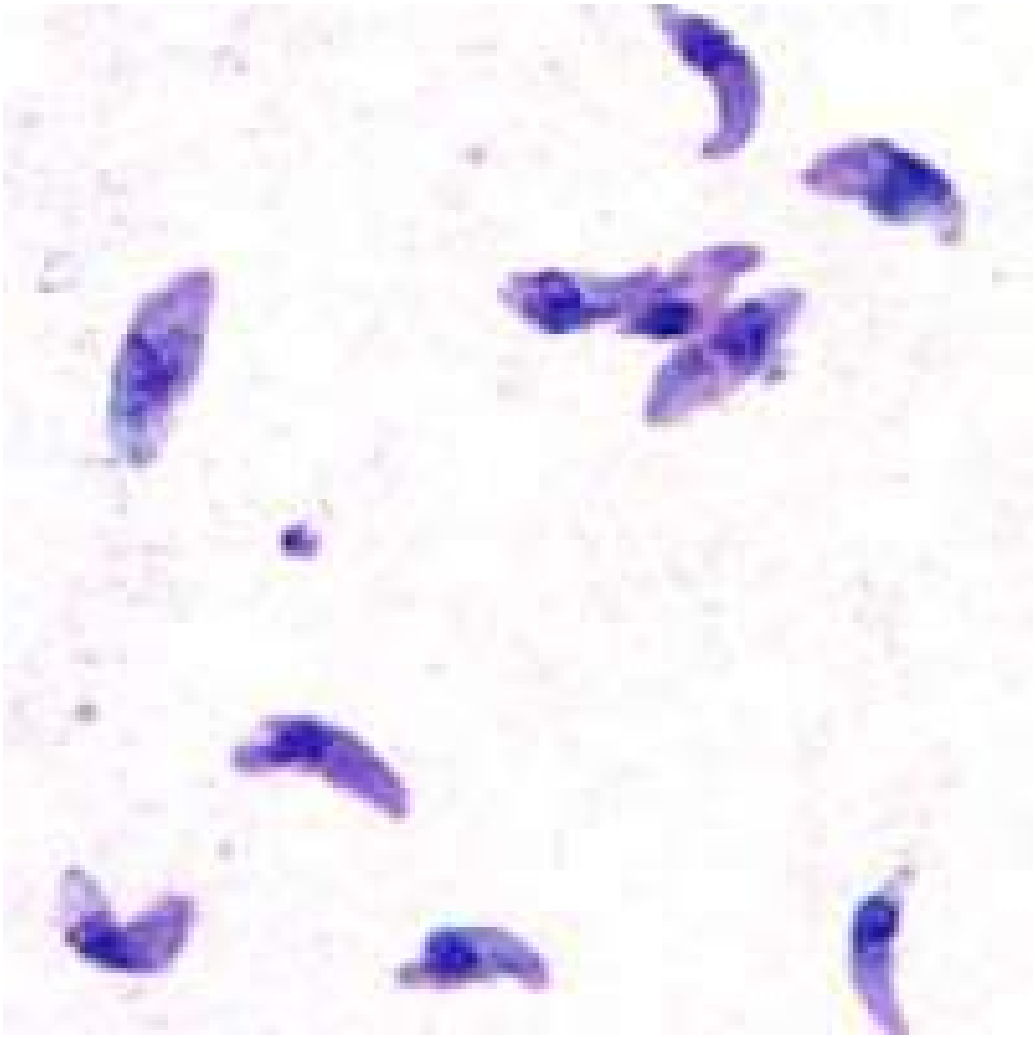
Types of hosts

1. **Definitive or final host:** host with adult stage or sexually reproducing forms of the parasite.
2. **Intermediate host:** host with a larval stage or a sexually reproducing forms of parasites.
3. **Reservoir host:** is an animal that harbors the same species of parasite as man.
4. **Carrier host:** human who carries parasites and can pass them on to others.
5. **Vector host:** is an invertebrate organism that carries the parasite from one host to another. The vector may either be a **mechanical vector**, in which no development or multiplication takes place, or a **biological vector** in which either multiplication or development occurs.

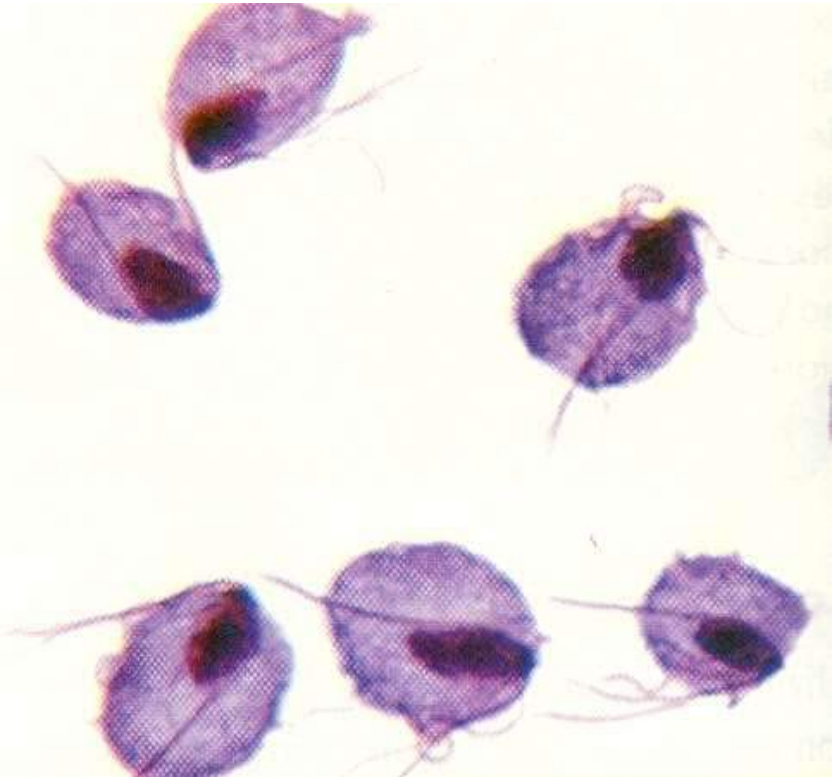


Mode of parasitic infection :

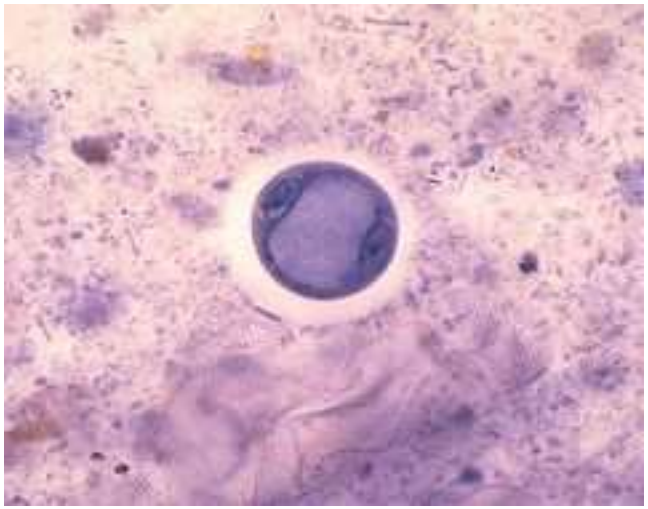
1. **Congenital…..**from mother to fetus (e.g: **Toxoplasma**)



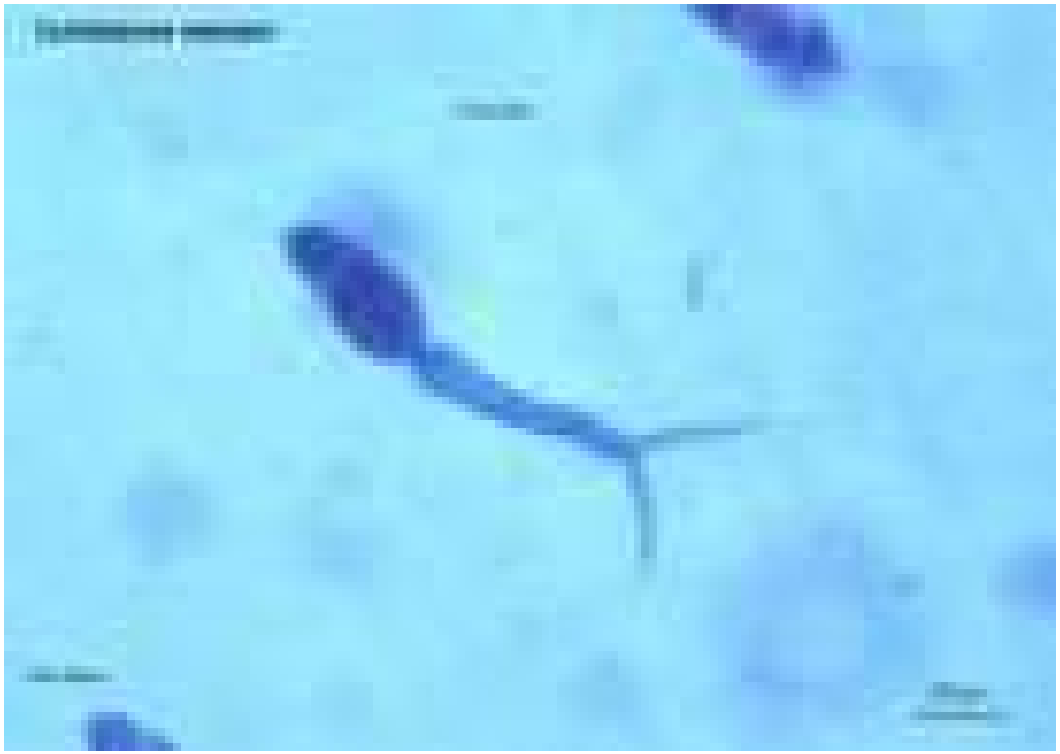
2. Direct contact…..through the skin (e.g: **Mites**) or sexually e.g. **Trichomonas vaginalis**)



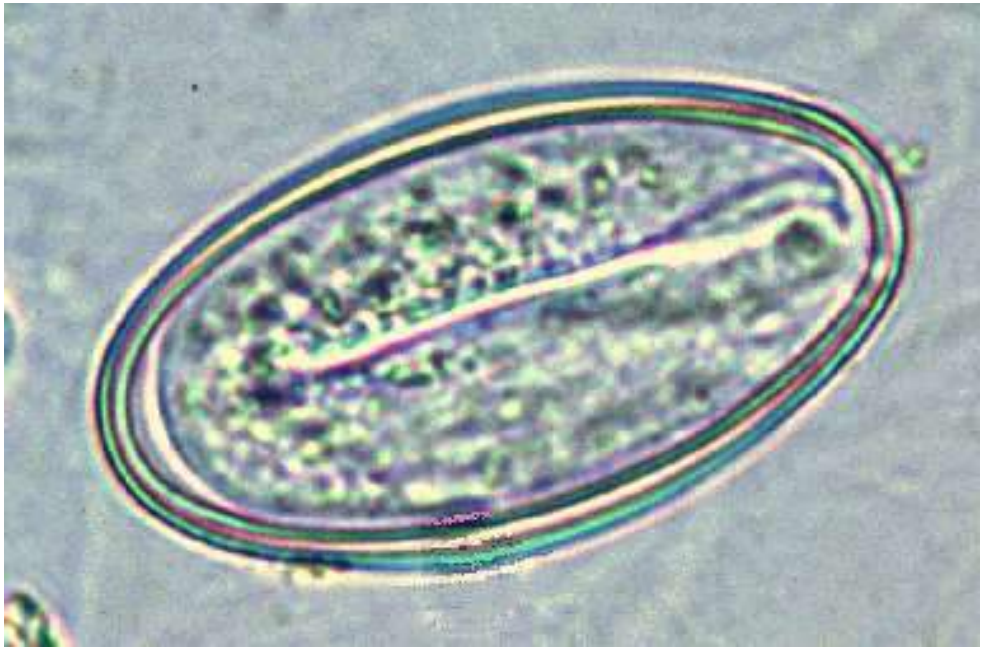
3. Ingestion of contaminated food and water…..(e.g: **Entamoeba**) or uncooked meat in which the infective stage has developed (e.g. **Taenia**)



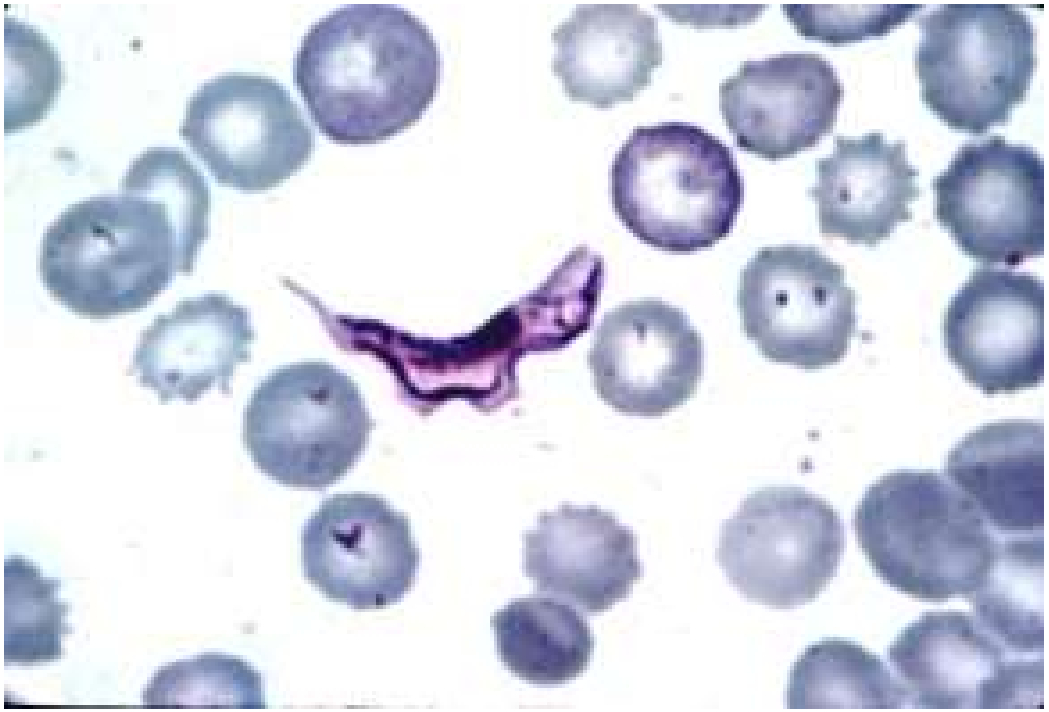
4. Penetration of the skin…. Due to contact with infective soil (e.g: **Schistohsoma cercaria**)



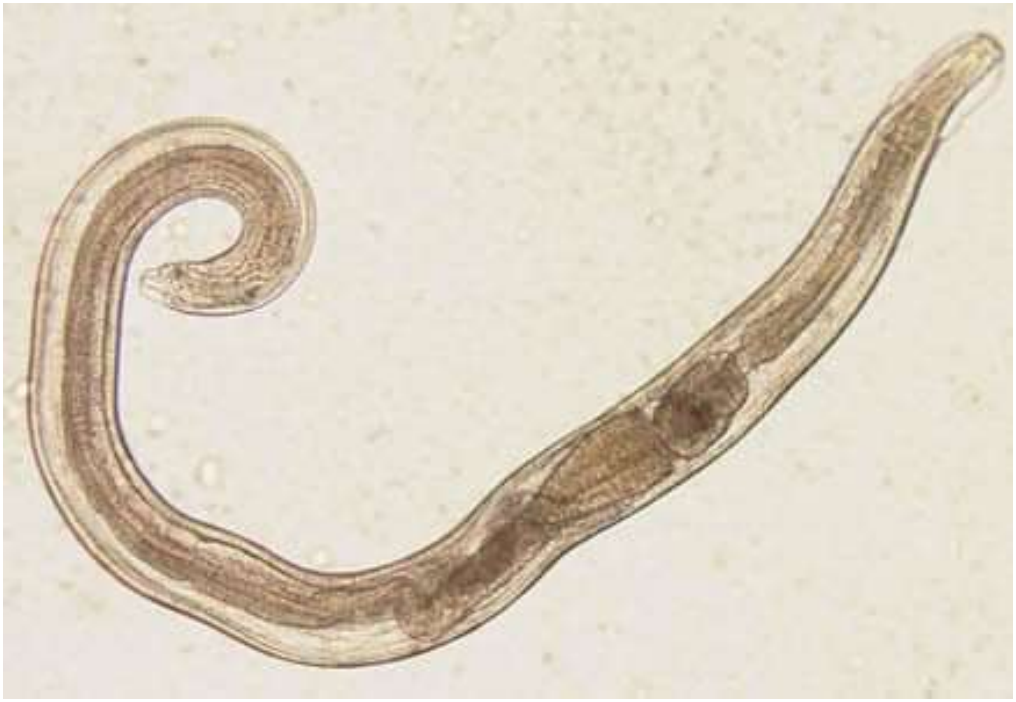
5. Inhalation … of dust carrying the infective stages of parasite (e.g: **Enterobius vermicularis eggs**)



6. Vectors… through the bite or feces of infected vector (e.g: **Trypanosome spp.)** or by swallowing the vector.

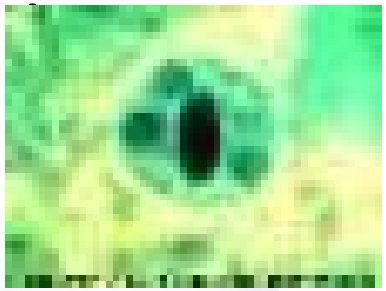


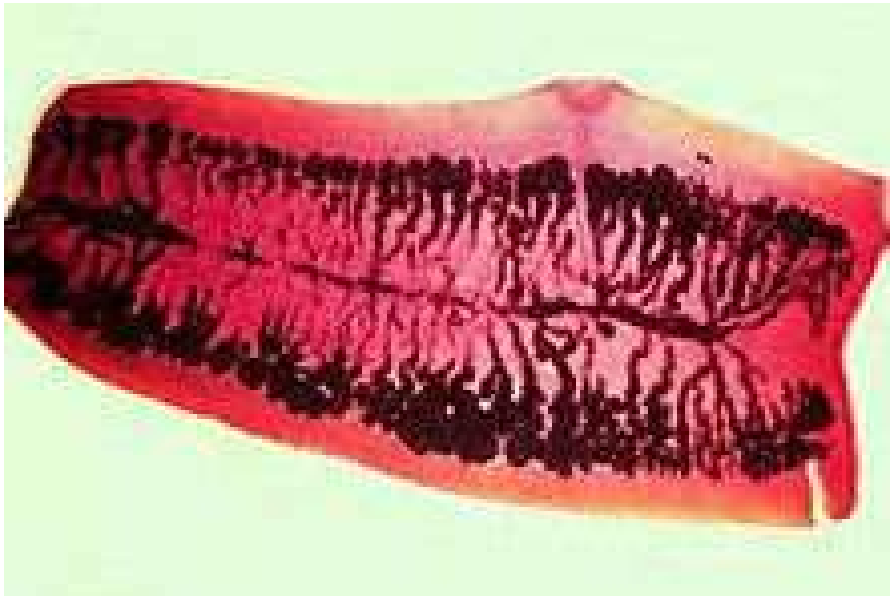
7. Autoinfection…occurs when infective stages are carried from feces to mouth person (e.g: **Enterobius vermicularis**)

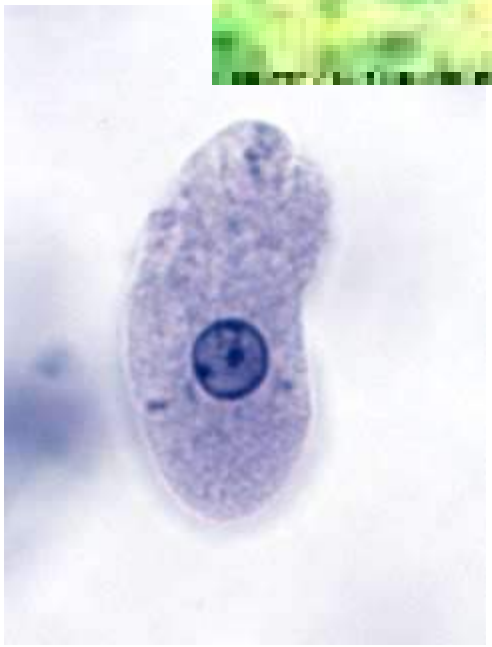


Sources of parasitic infection

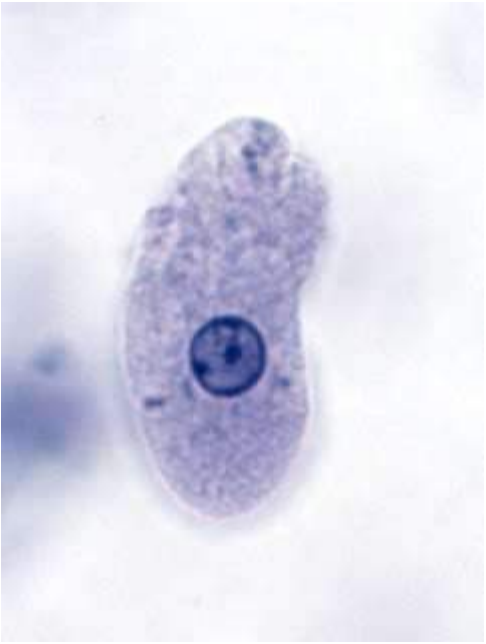
1. Food: meat (**Taenia saginata larvae**)
Vegetables.. (**E. histolytica**, **cysts**)

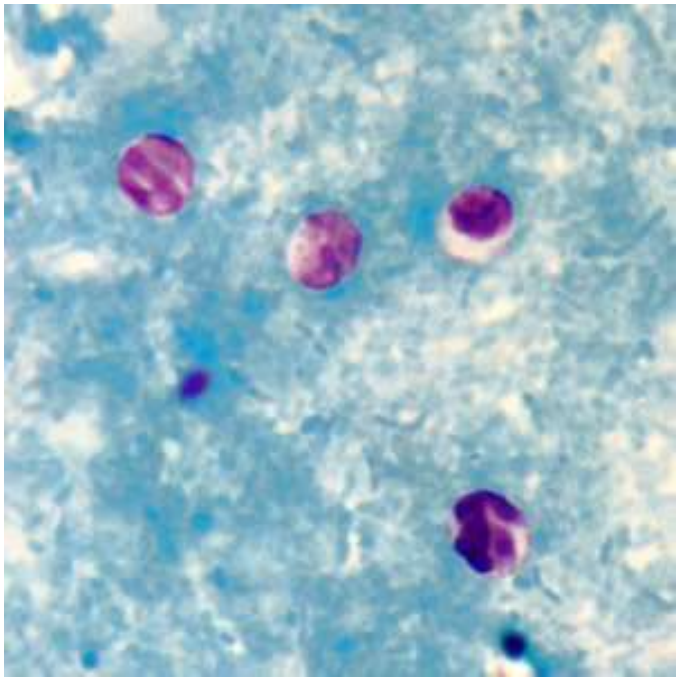




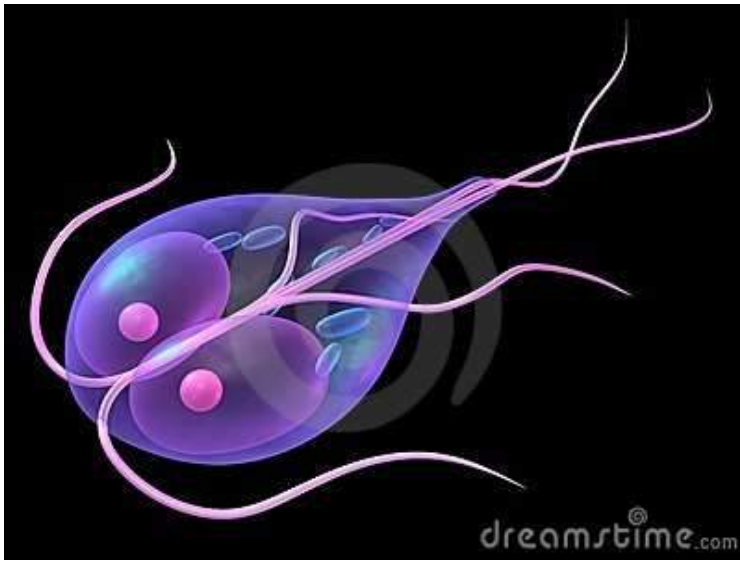


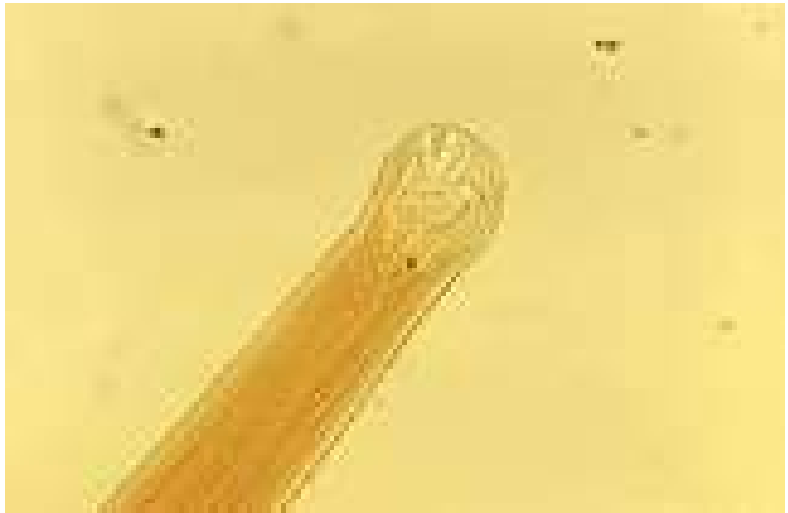
2. Water… protozoal cysts (E. histolytica, Cryptosporidium), Cercaria (Shistosoma), cyclop (Dracunculus medinensis)



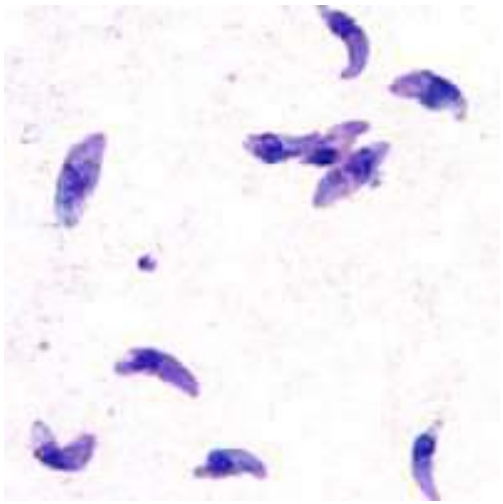


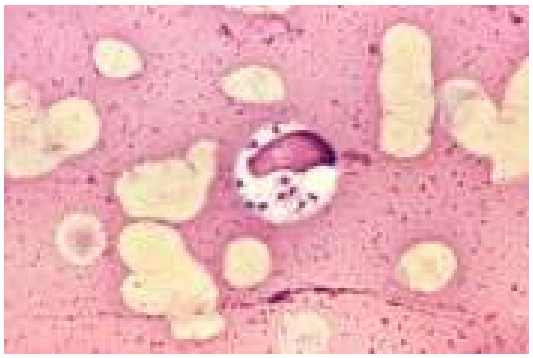
3. Soil… contaminated with feces contained (Ascaris, Ancylostoma, Giardia)



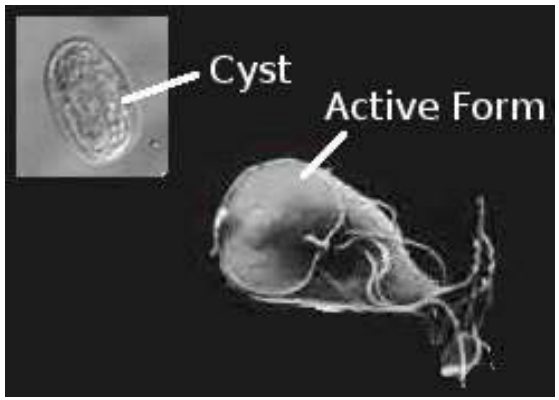


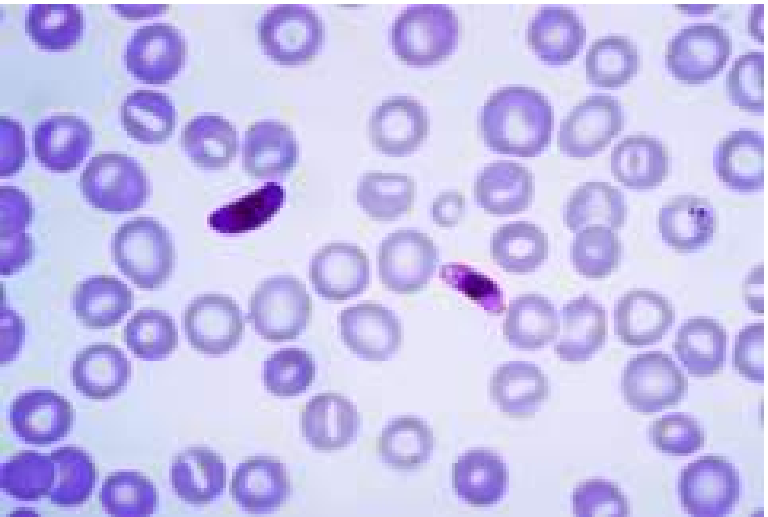
4. Association with domestic animals… Dogs: (**Echinococcus granulosus**, **Toxocara**, and **Leishmania**). Cats: (**Toxoplasma)**.

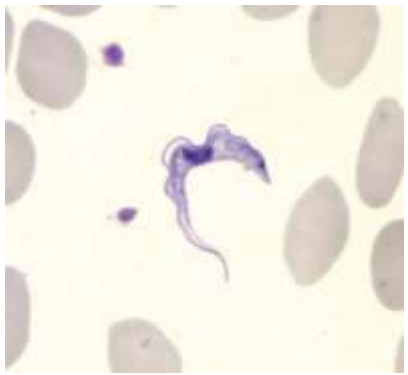


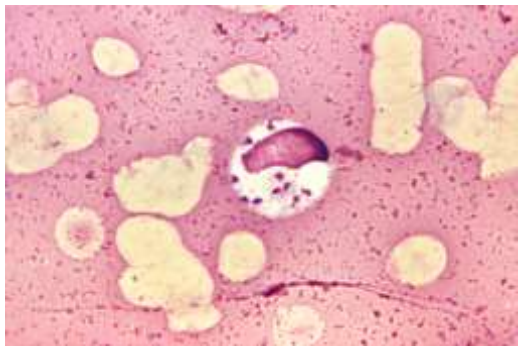


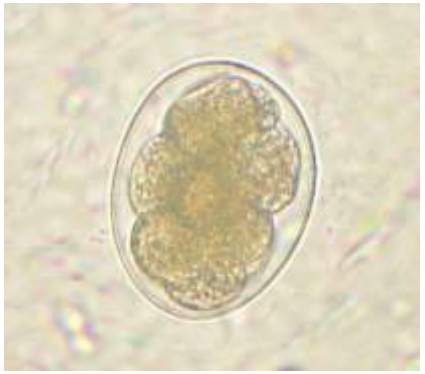
5. Arthropods… blood sucking (Plasmodium, Trypanosome, Leishmania) .mechanical transmission (Worms ova, **Protozoal cysts**).



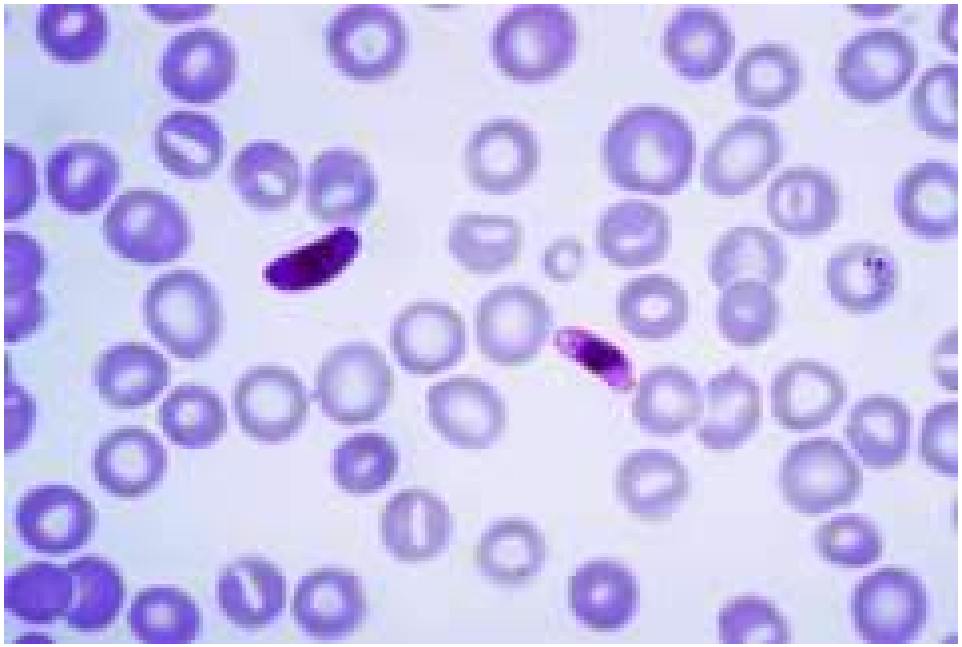




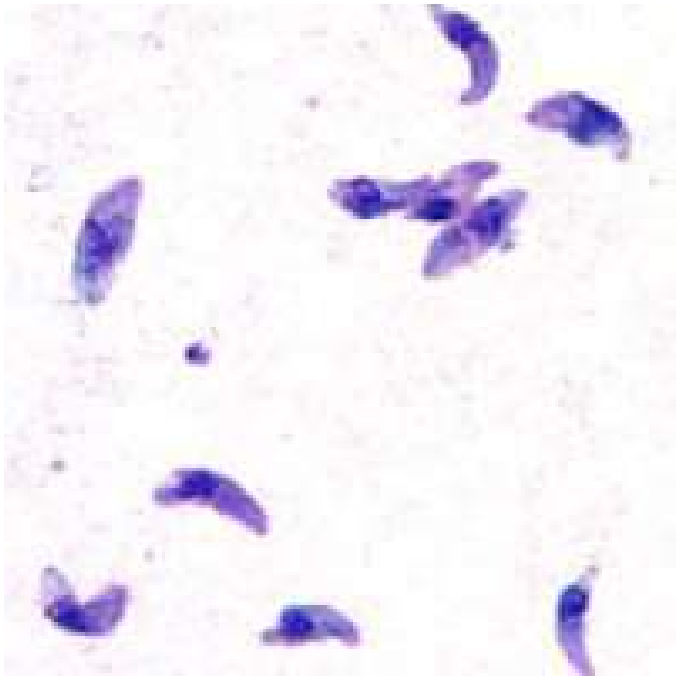


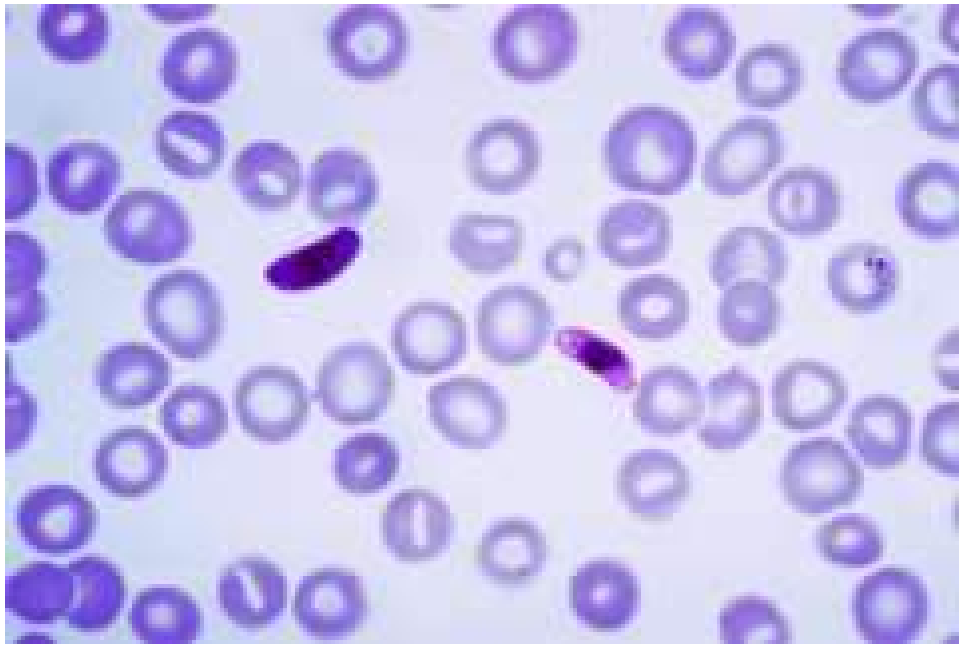


6. Blood transfusion ….(Erythrocytic stages of plasmodium).

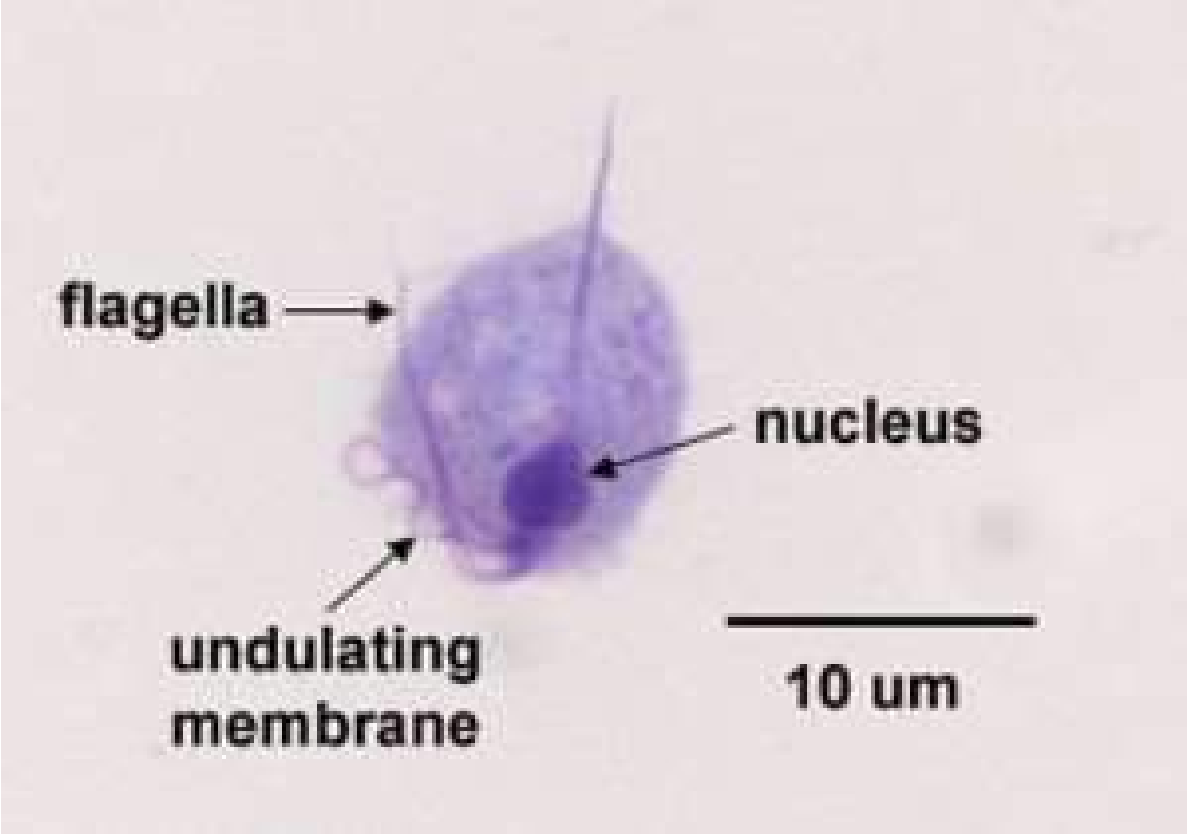


7. Congenital transplantation …..(Toxoplasma. Plasmodium).

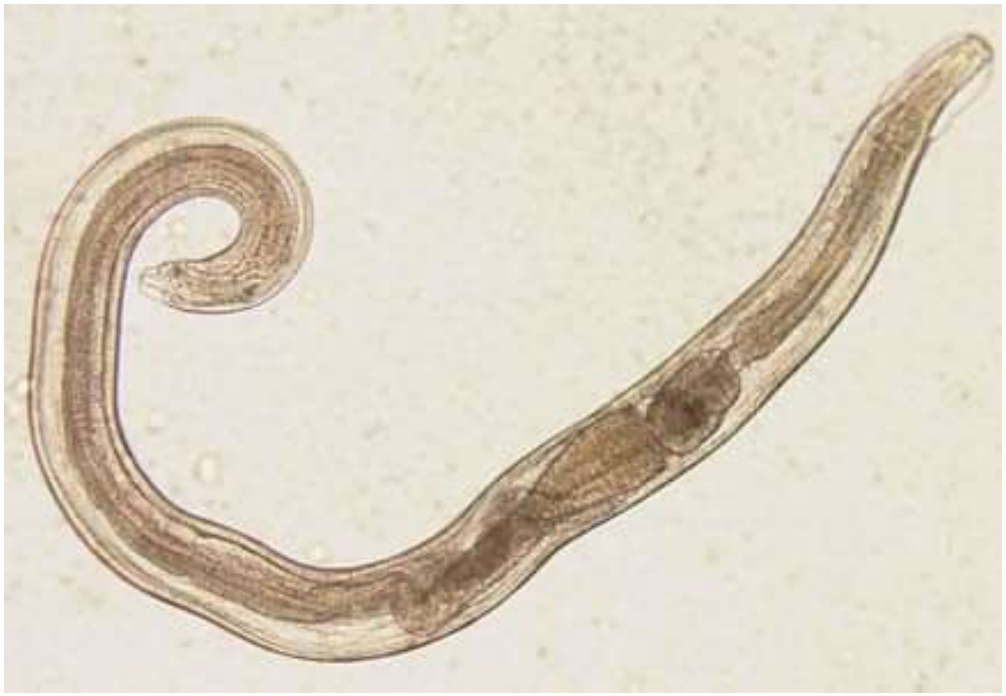




8. Sexual intercourse ….(Trichomonas vaginalis).

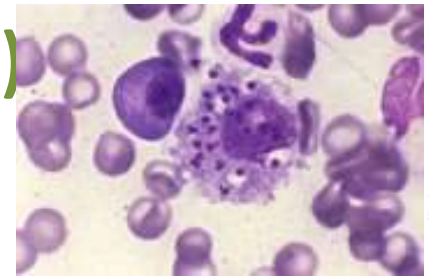


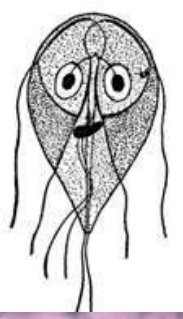
9. Inhalation of dust ….(Enterobius vermicularis).

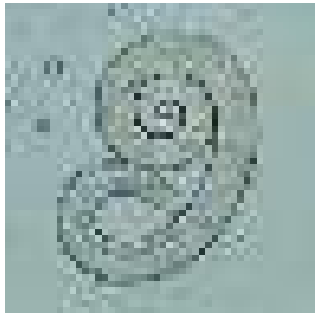


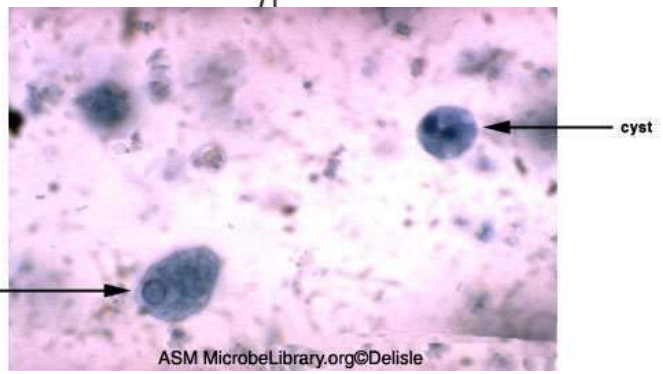
The habitat is the site where the parasite lives and multiplies in host, such as:

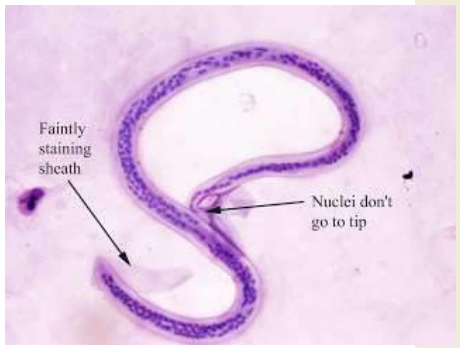
1. Small intestine .. (Giardia)
2. Large intestine… (Entamoeba histolytica)
3. Blood vessels…(Schistosoma spp.)
4. Muscles…(Trichinella spiralis)
5. Lymphatic.. (Wucheraria bancrofti)
6. Reticulo-endothelial system…(Leishmania)

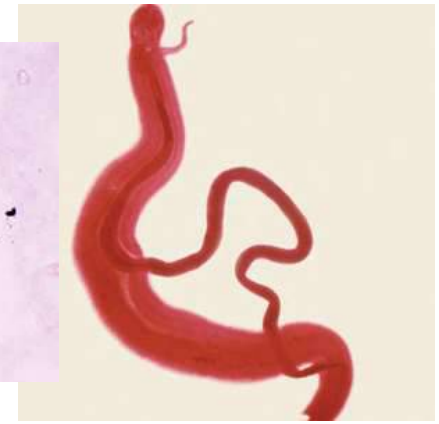






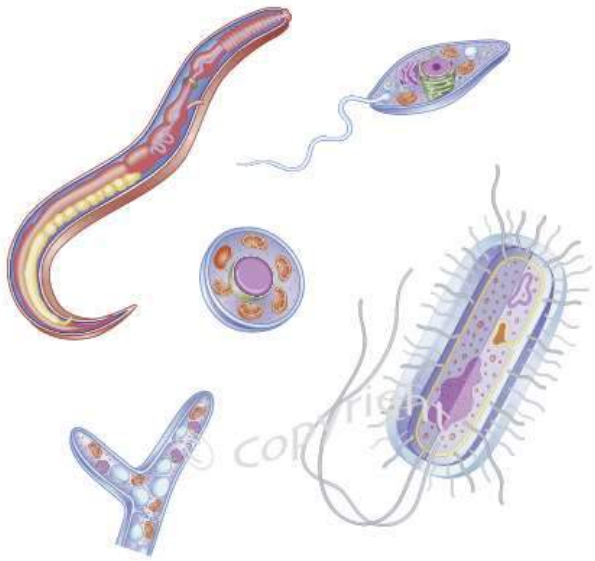




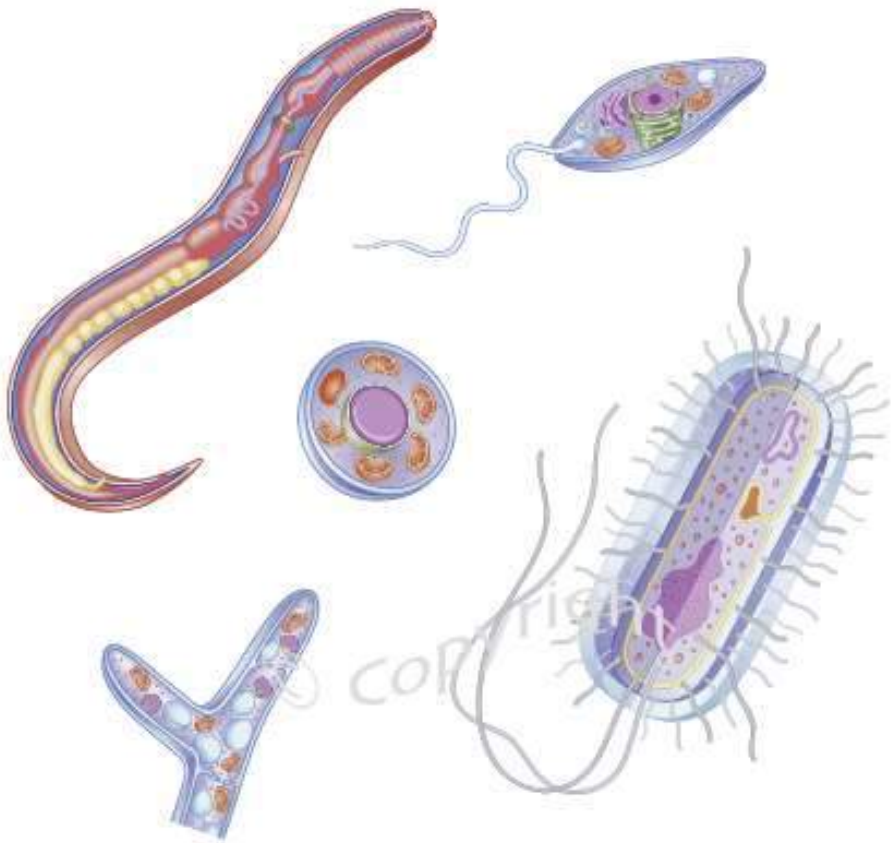


Definitions

1. **Host…**organism harboring a parasite
2. **Parasitic infection…**invasion by endoparasites (protozoa and helminthes)
3. **Parasitic disease..** invasion and pathology produced by endoparasites.
4. **Parasitic infestation …** external parasitism by ectoparasites (arthropods)
5. **Prevalence..**percentage (%) of infection
6. **Intensity…**number of parasite in the host



7. **Incidence…** percentage of new infection.
8. **Endemic..** A disease or disease agent that occurs in a human community at all time
9. **Enzootic…** a disease agent that occurs in an animal population at all time.
10. **Epidemic..** a disease or disease agent that spreads rapidly through a human population
11. **Epizootic..** A disease or disease agent that spreads rapidly through an animal population

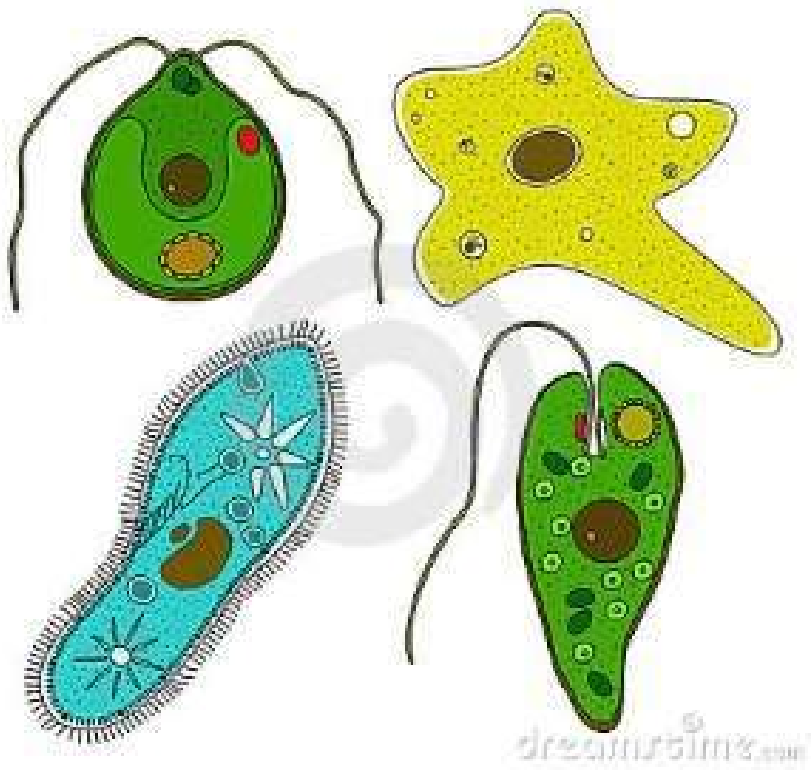


Taxonomy

Classification	name	Example genus
Kingdom	animalia	
Subkingdom	protozoa	
Phylum	sarcomastigophora	
Subphylum	sarcodina	Entamoeba histolytica
Subphylum	mastigophora	Giardia
Phylum	apicomplexa	Plasmodium (malaria)
Phylum	ciliophora	Balantidium
Phylum	microspora	Enterocytozoan

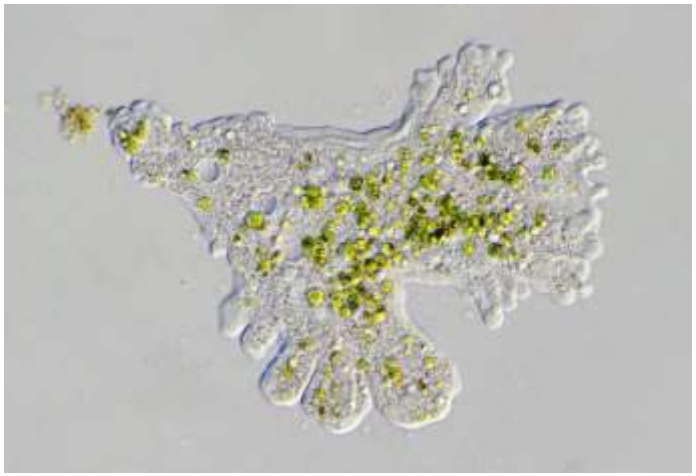
Traditional classification

- Diverse structure of protozoa have developed to aid in movement and feeding in a great many environment. Traditional classification of protozoa are based mainly of their **structure morphology** and **ways of moving**



The traditional four phyla classification, based largely on movement, includes the following groups:

- **Amoebae-** uses psudopodial structures, flowing cytoplasm
- Amoebae are protozoa that move by employing pseudopodia. Many amoebaes also employ their pseudopodia to engulf food

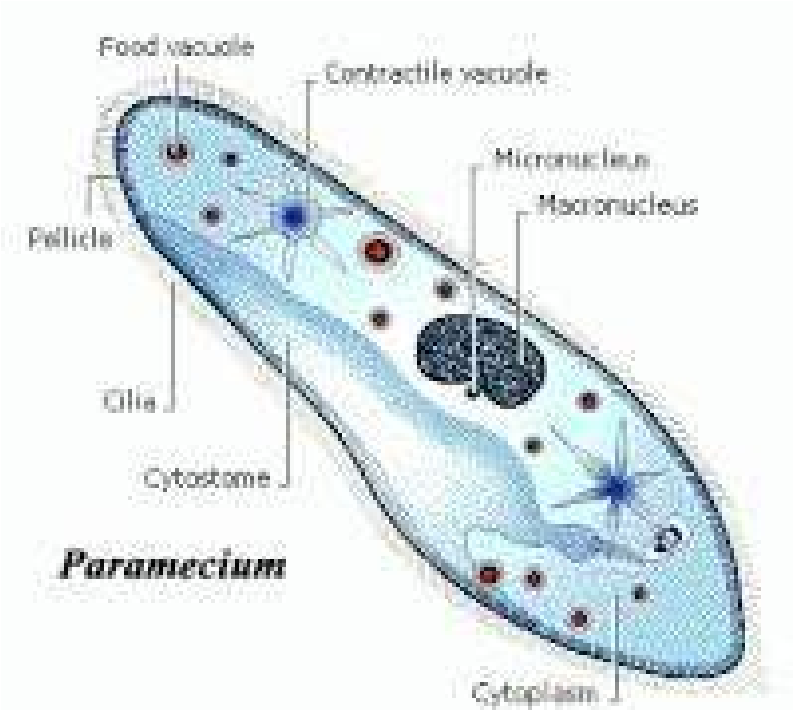


Flagellates- use flagella

- **Flagellates** are protozoa that move by means of flagellar action. Some flagellates have their flagella in a structure called an undulating membrane

Sporozoans-bending, creeping or glinding

- The sporozoans are parasitic spore formers that do not move under their own power.

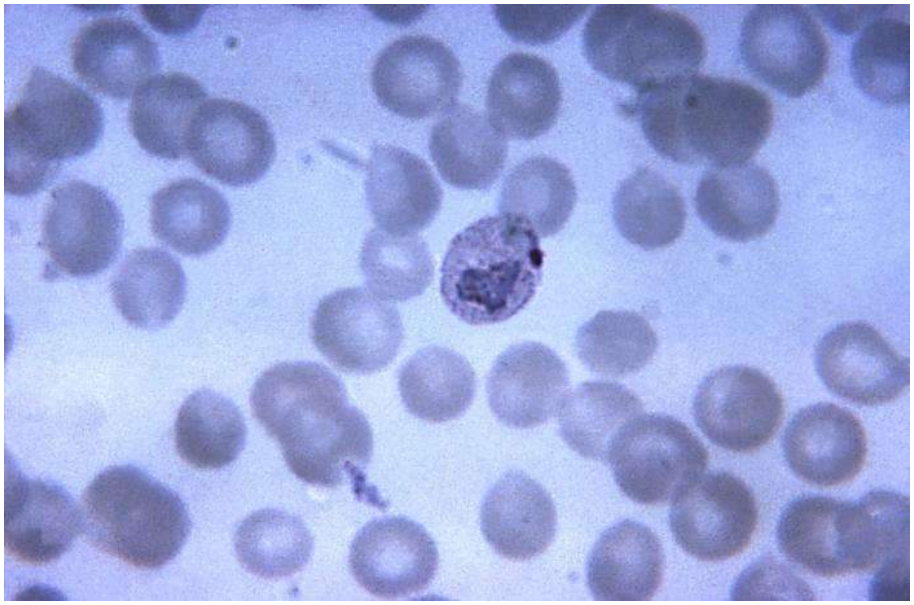


Ciliates-use cilia

- The ciliates are protozoa that move by means of cilia action. Recall that the difference between eukaryotic flagella and cilia is one of size and number. **Cilia are small and numerous flagella are large and few.**

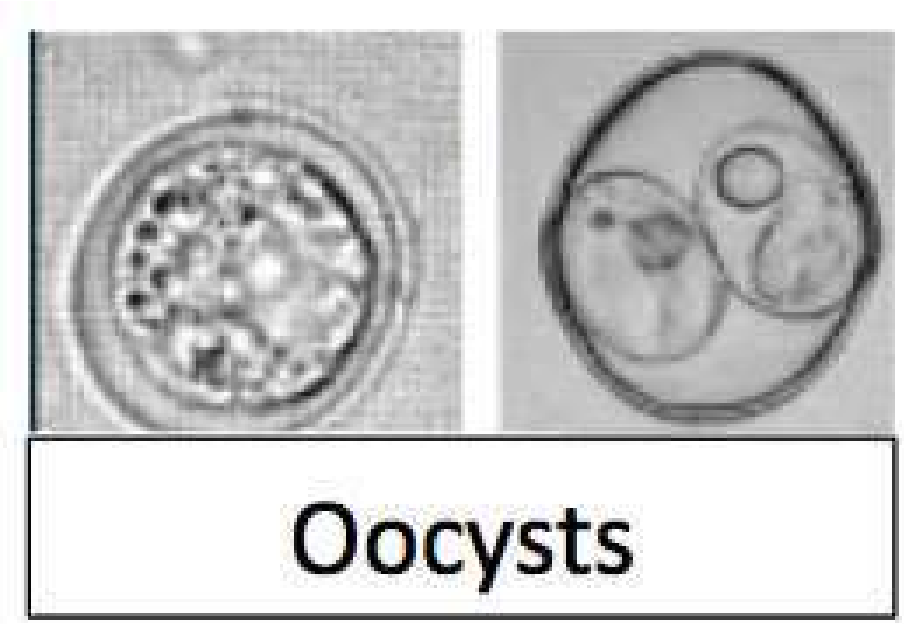
Life cycle stages

- During its life cycle, a protozoan generally passes through several stages that differ in structure and activity.
- **Trophozoite..** is a general term for the active, feeding, multiplying stage of most protozoa

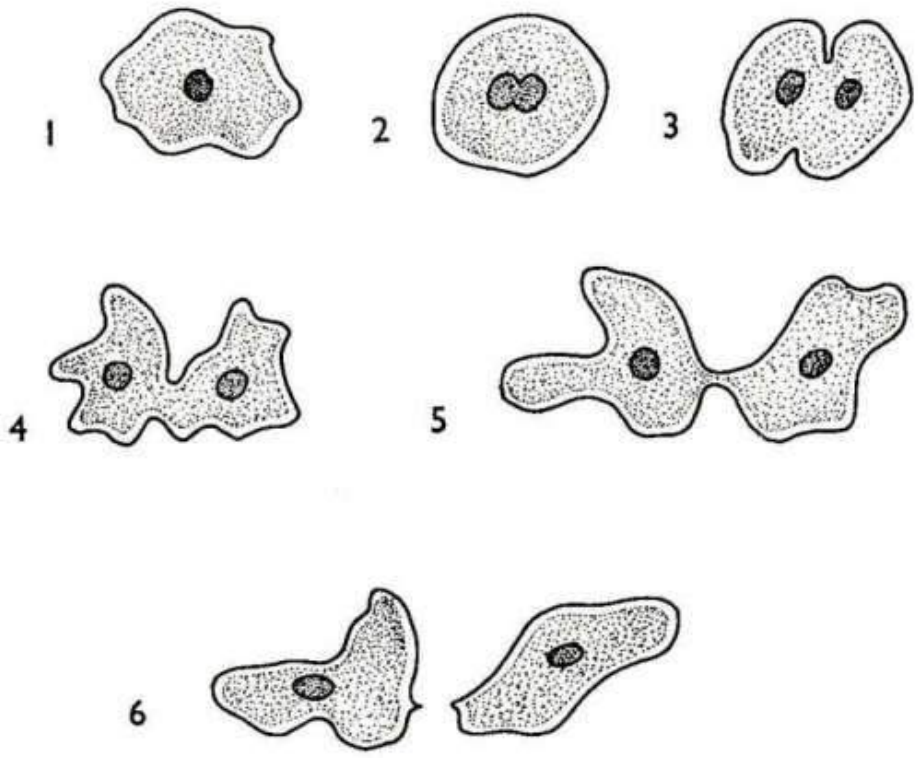


- **Cyst..** a non motile stage which is protected by a cell wall, protozoan cysts that must survive outside the host usually have more resistant walls than cysts that form in tissue, cysts contain one or more infective form. Multiplication occurs in the cysts of some species so that excystation releases more than one organism.

- **Oocysts..** are stages resulting from sexual reproduction in the apicomplexa

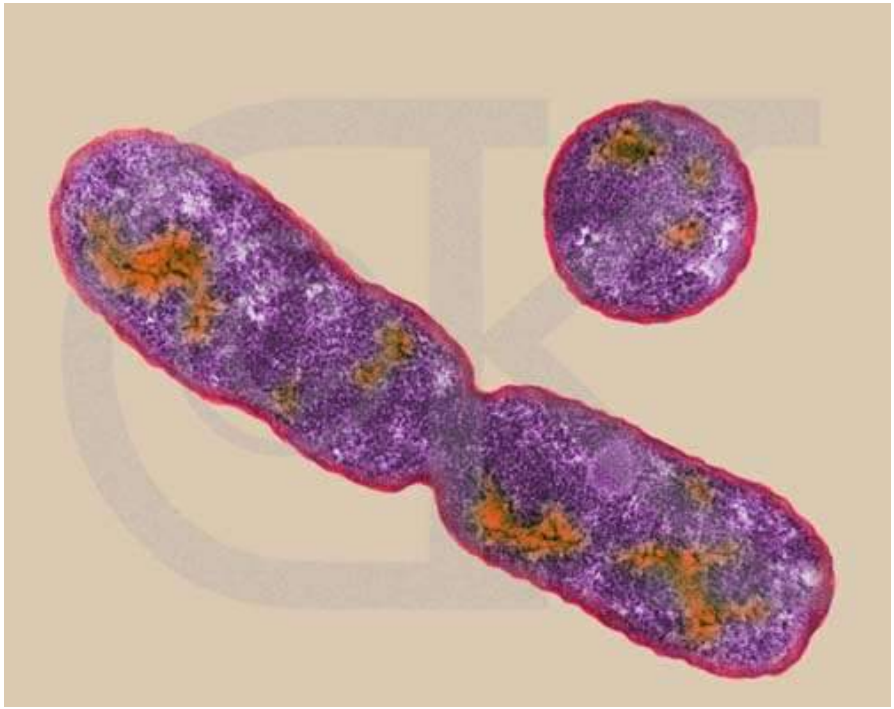


- **Reproduction…**
- Reproduction in the protozoa may be a sexual, as in the amebas and flagellates that infect humans, or both asexual and sexual, as in the Apicomplexa of medical importance.

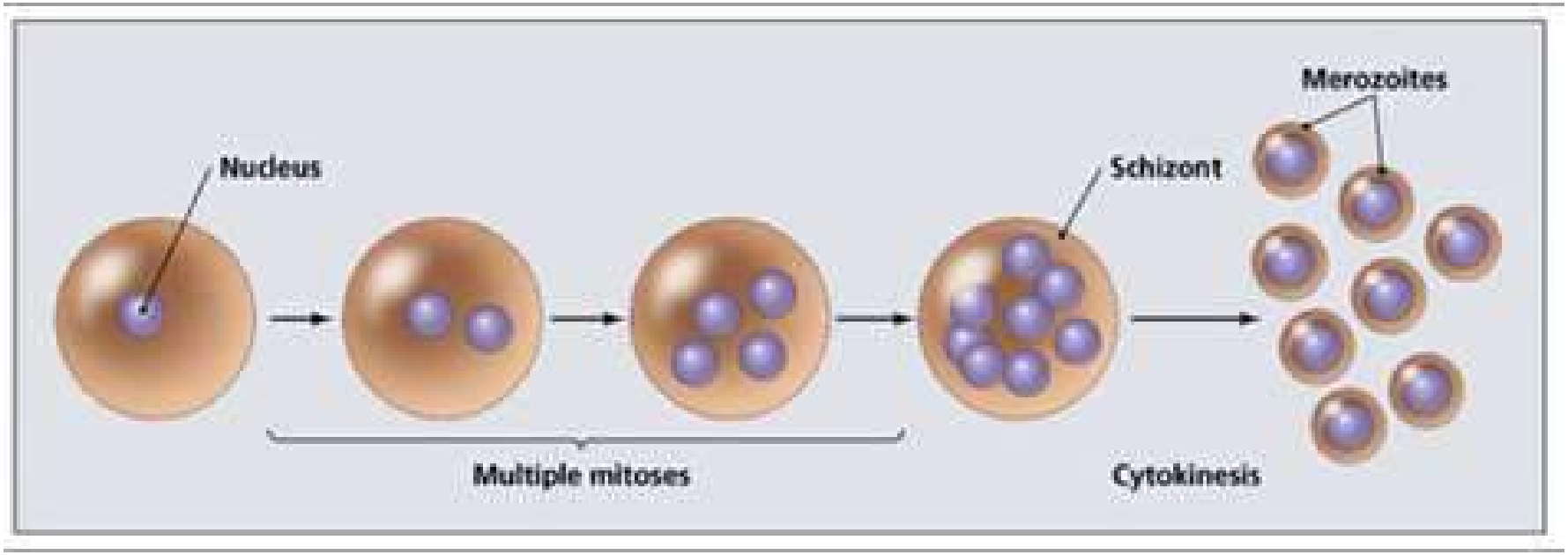


The most commen type of asexual multiplication is :

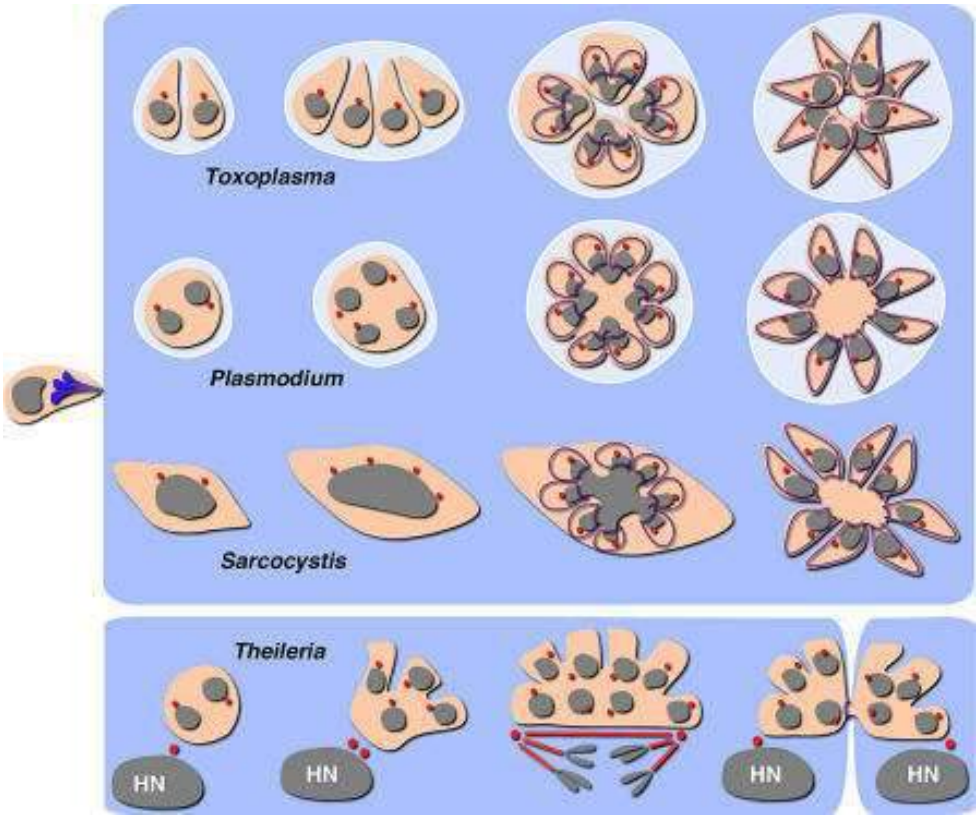
1. **Binary fission…** in which the organelles are duplicated and the protozoan then divides into two complete organisms, division is either longitudinal in the flagellates or transverse in the ciliates, amebas have no apparent anterior-posterior axis.



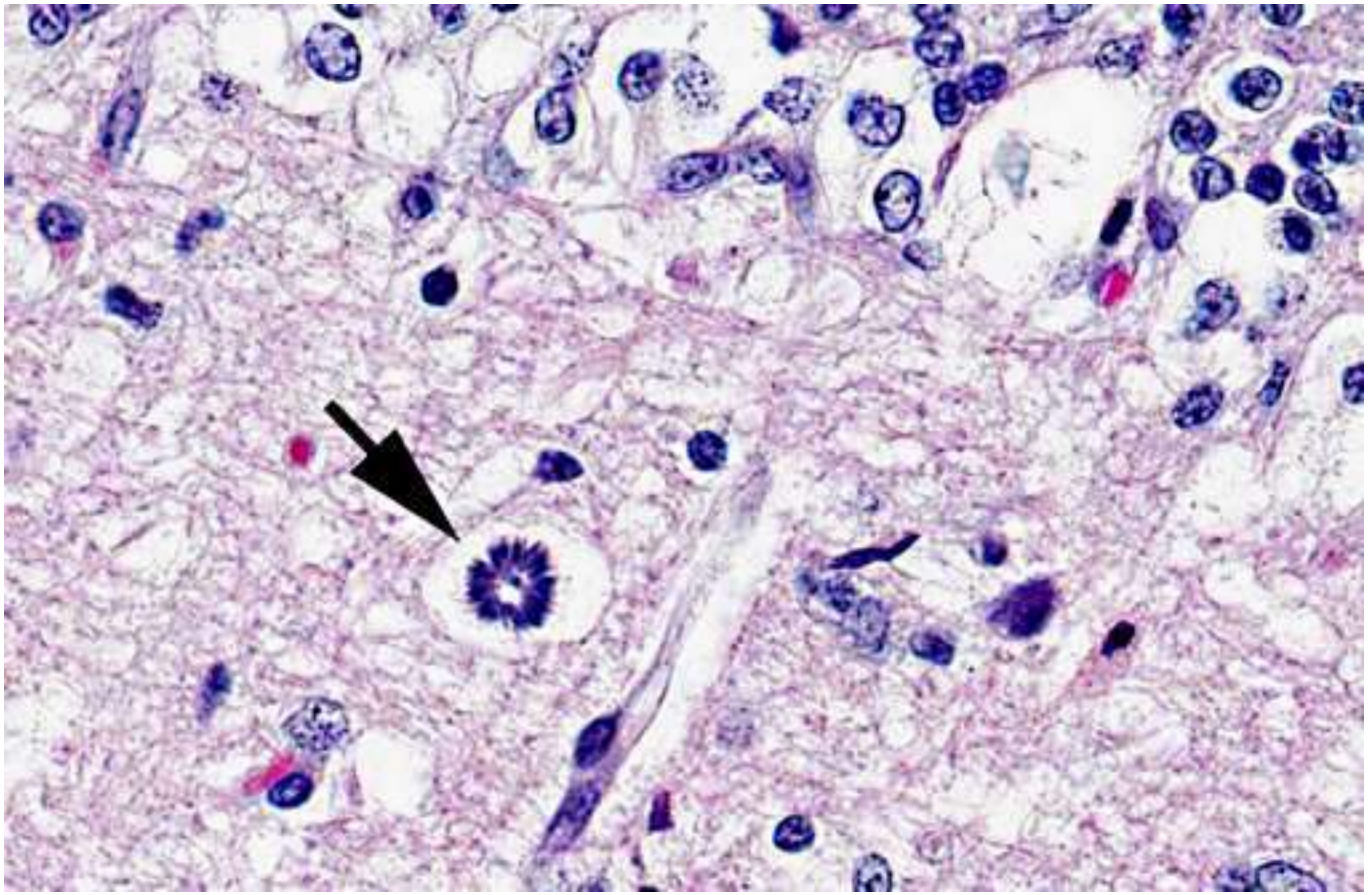
2. Multiple fission (shizogony)… a common form of asexual division in the Apicomplexa, the nucleus divides a number times, and then the cytoplasm divides into smaller uninucleate, merozoites.



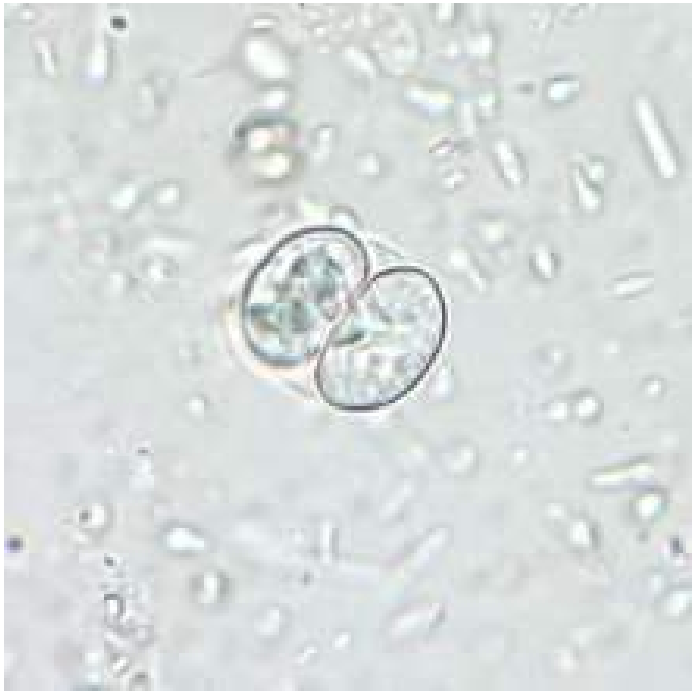
3. Endodyogeny (budding)… is a form of asexual division seen in toxoplasma and some related organisms. Two daughter cells within the parent cell, which then ruptures, releasing the smaller progeny which grow to full size before repeating the process



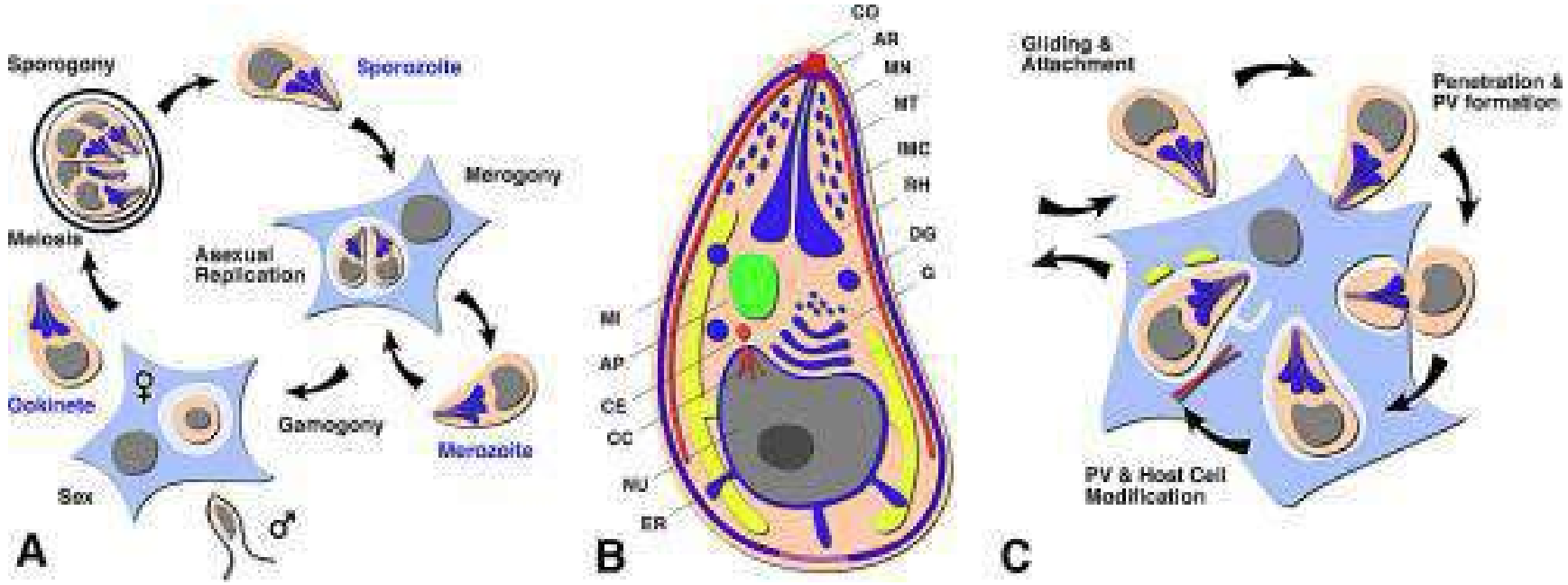
4. Endopolygeny… more than two cells resulting from internal budding of parent.



5. Gametogony (gamogony)= formation of gametes

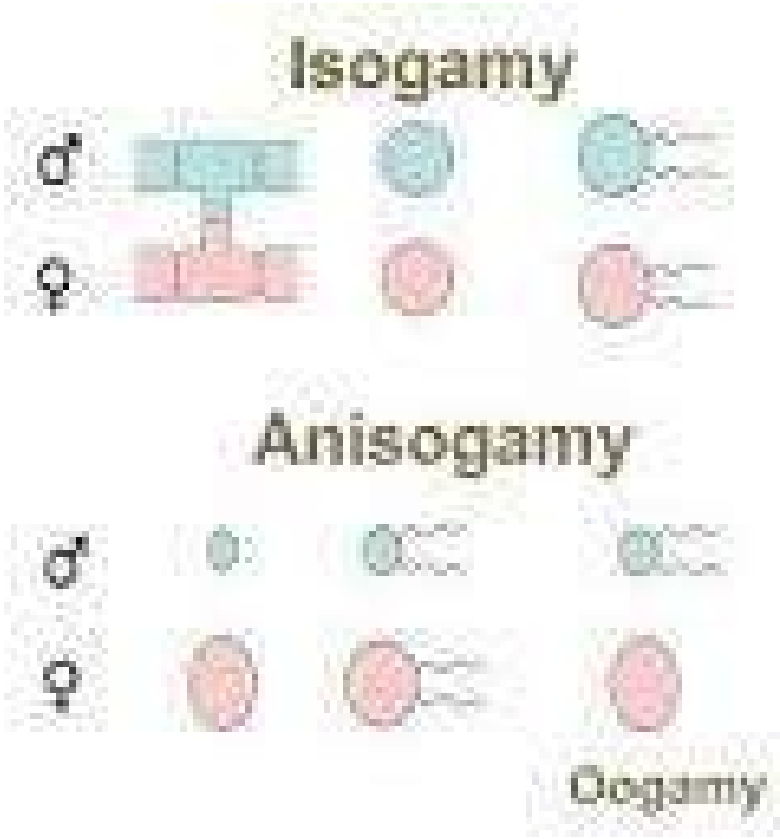


- **Sexual reproduction or multiplication,** which is usually takes place in the invertebrate host, is known as sporogony



- **Sexual union (syngamy)…**fusion of two gametes may be

A. **Isogamy…**union between cells of equal size and structure (similar gametes
B. **Anisiogamy….**Union between dissimilar cells (dissimilar gametes), male microgamets and female macrogamets which produces a zygote



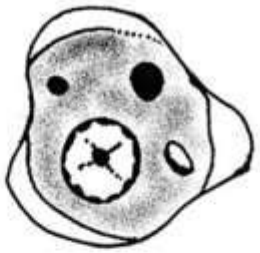
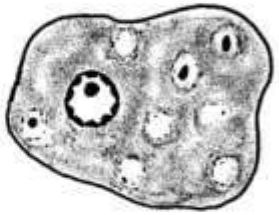
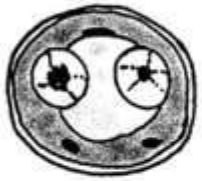
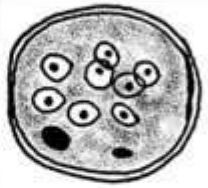
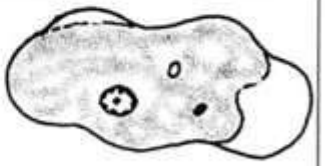
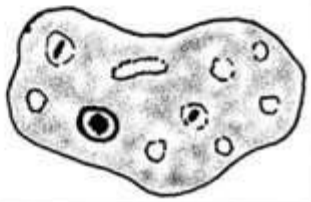
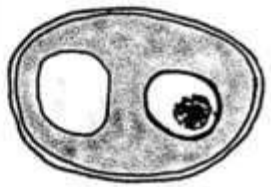
- **Another type of sexual multiplication is**
- **Conjugation…** occurs in ciliate, it is a temporary union of two ciliated organisms for the exchange of nuclear material.

Ciliates

c. Conjugation

Thank you

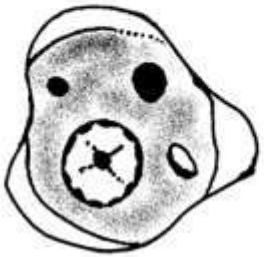
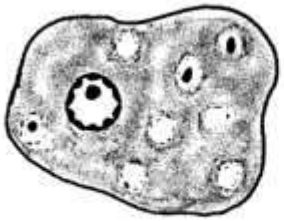
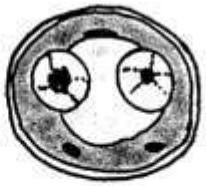
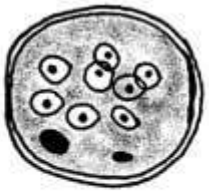
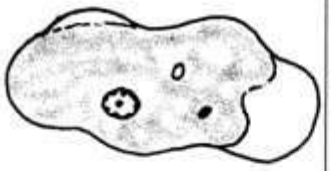
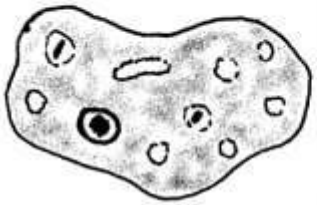
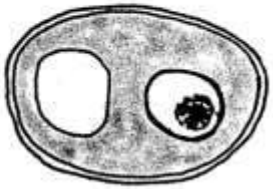
Parasitology

Organism	Trophozoite	Precyst	Cyst
E. histolytica *E. dispar* *E. moshkovskii*			
E. coli			
E. hartmanni			
I. bütschlii			

Lec.Dr.Ruwaidah F. Khaleel

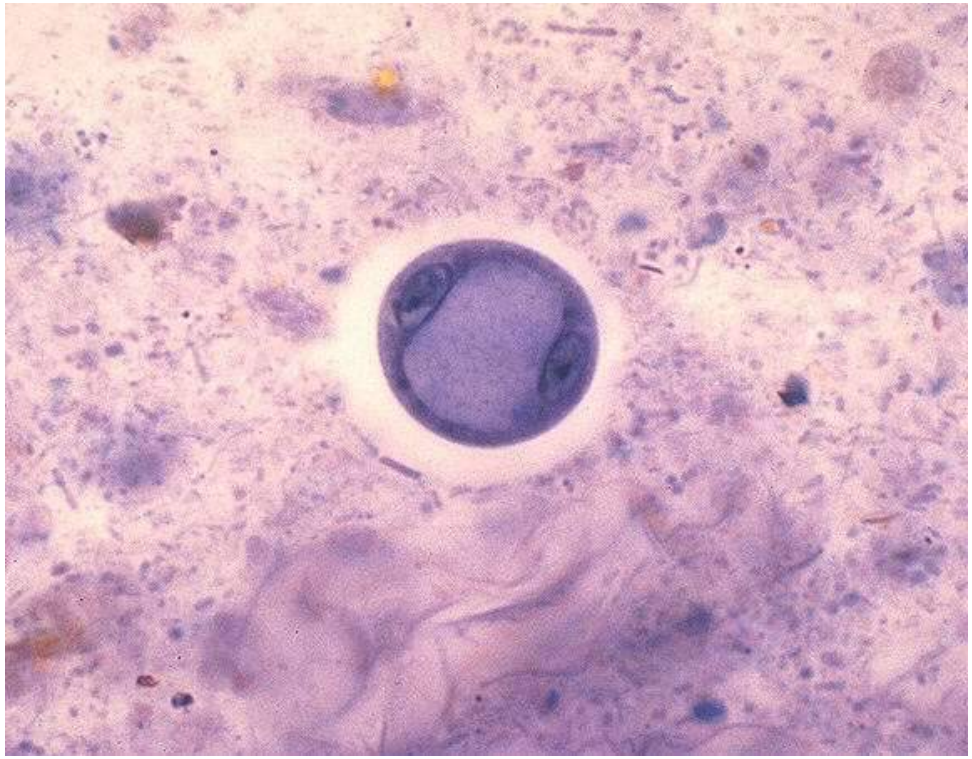
Sub phylum: sarcodin

- **The parasitic amoeba of man**
- At least four genera of amoeba have been definitely established as living in man. Only one species, **Entamoeba histolytica pathogenic** for man. The differential characteristics of parasitic amoeba in man are graphically represented in bellow

Organism	Trophozoite	Precyst	Cyst
E. histolytica *E. dispar* *E. moshkovskii*			
E. coli			
E. hartmanni			
I. bütschlii			

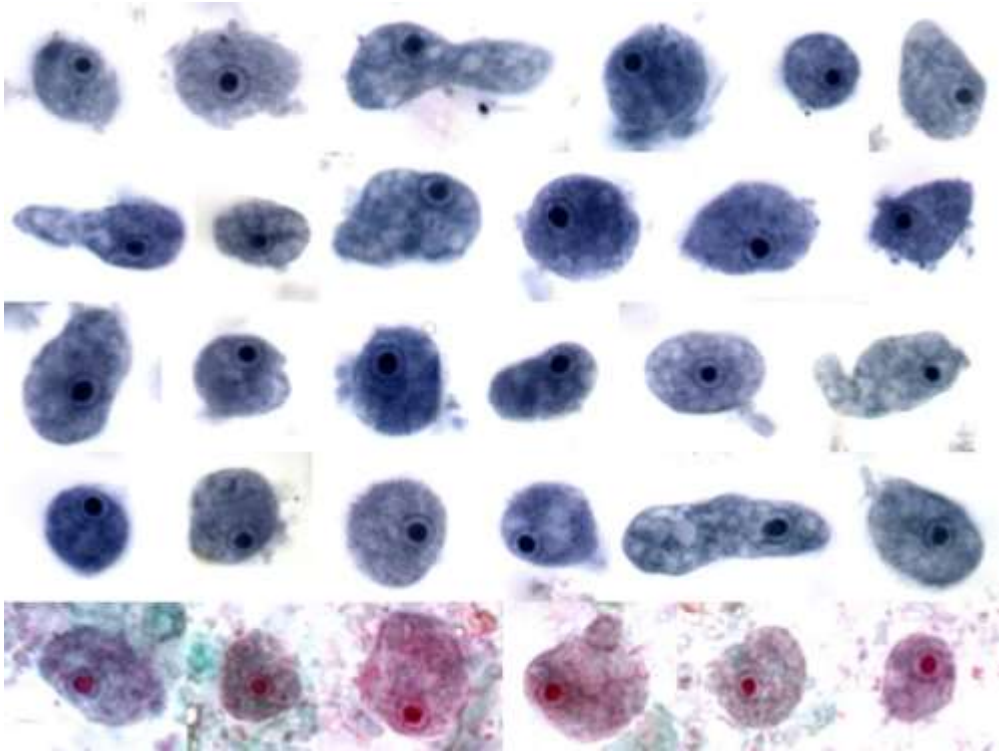
Genus Entemoeba:

- has more or less spherical vesicular nucleus with small, **usually central karyosome**, a **peripheral layer of chromatin granules on the nuclear membrane**
- **Karyosome_ endosome_ nucleolus**



Genus Endolimax:

- are small, and the vesicular nucleus has **a large, irregular, central or eccentric karyosome** that **consists of several portions connected by strands and has linin fibers radiating to the delicate peripheral membrane**.

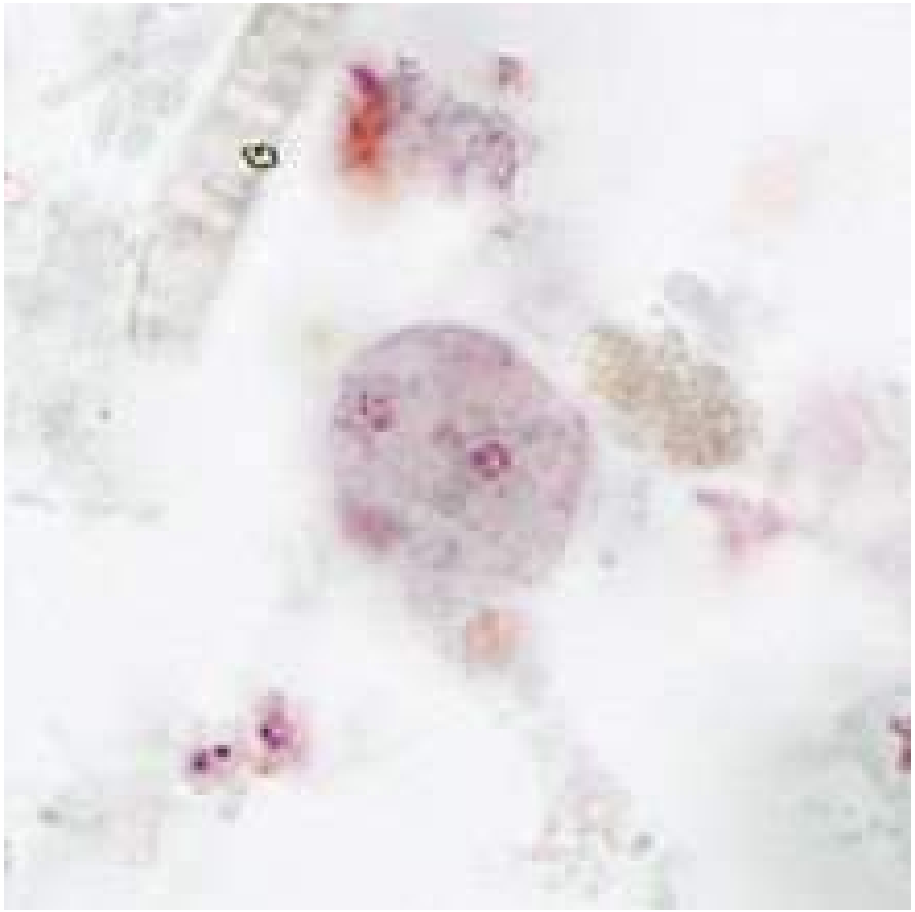


Genus Iodamoeba:

- **a large karyosome, rich in chromatin**, situated more or less centrally in the nucleus, is **surrounded by a layer of achromatic granules** and is **anchored to the nuclear membrane by delicate radiating fibrils**

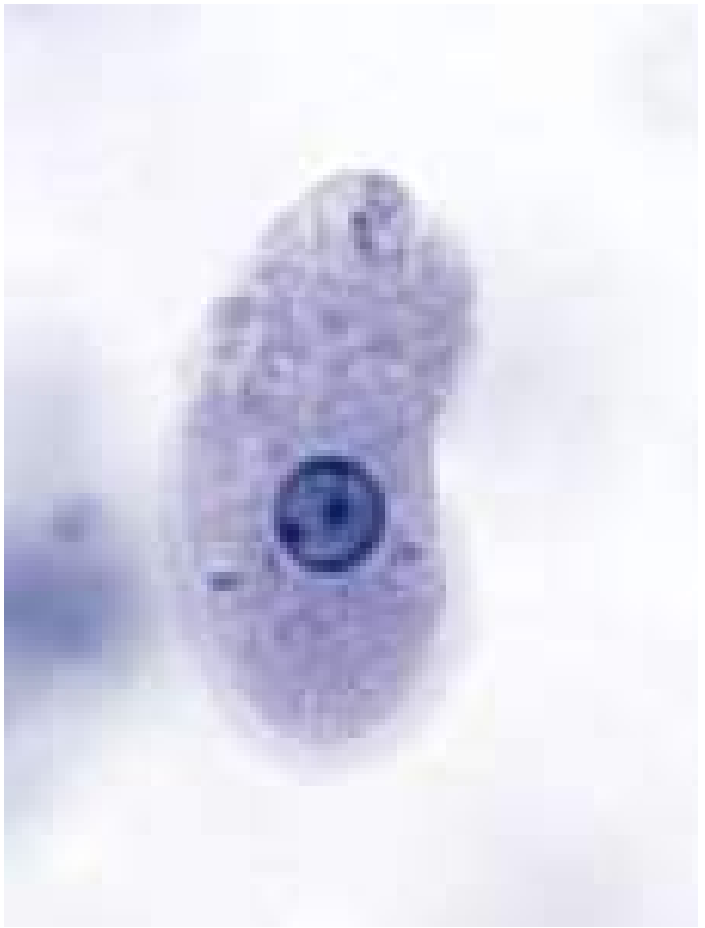
Genus Dientamoeba

- small size, the high percentage of binucleate forms, and the lack of cystic stage. The delicate nuclear membrane **has no peripheral chromatin. In the center of the nucleus is a large karyosome mass** of form four to eight chromatin granules



Entamoeba.histolytica

- **Synonyms:**
- Entamoeba dysenteriae, Entamoeba histolytica
- **Disease:** amebiasis, amebic dysentery, amebic abscess of liver.
- **Geographic distribution:** cosmopolitan.
- Its incidence is higher and its more severe in tropical and subtropical countries and in localities where sanitation is poor.

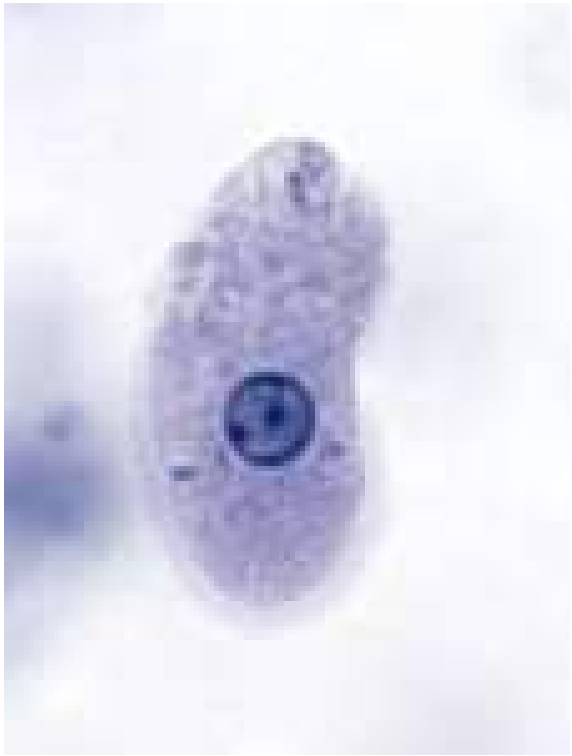


Morphology

E. histolytica may be observed in the feces in three morphologic stages.

A. **Trophozoite:** the vegetative forms range from **10-60m** in diameter.

- The nucleus is spherical; its diameter is one fifth to one third of ameba
- Usually found in liquid or semi fluid feces
- The pseudopodia are broadly fingerlike **(lobo podia)**
- Unusual among eukaryotic organisms in that it lacks mitochondria, and it is anaerobic in its metabolism
- Colonization occurs as a result of repeated binary fission.



b. precyst: round or oval cell

- are smaller than the trophozoite but are larger than the cyst
- lack a cystic wall

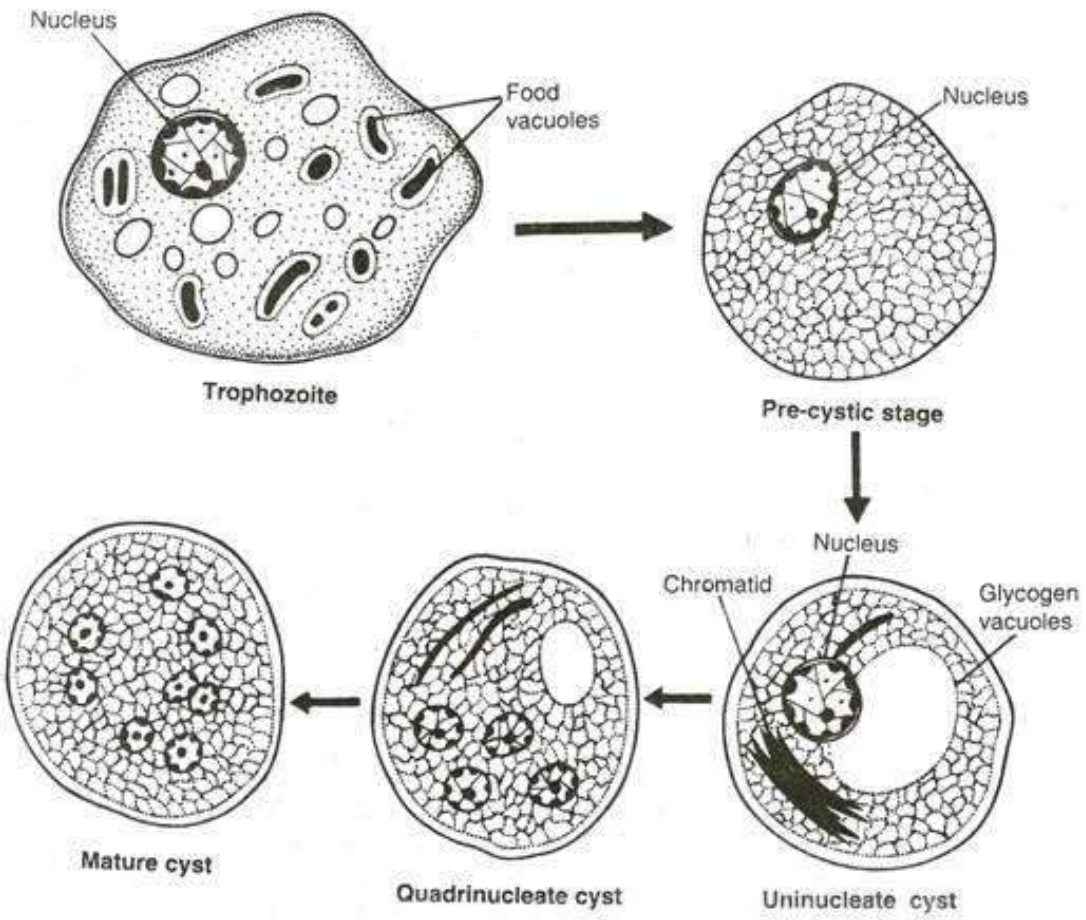
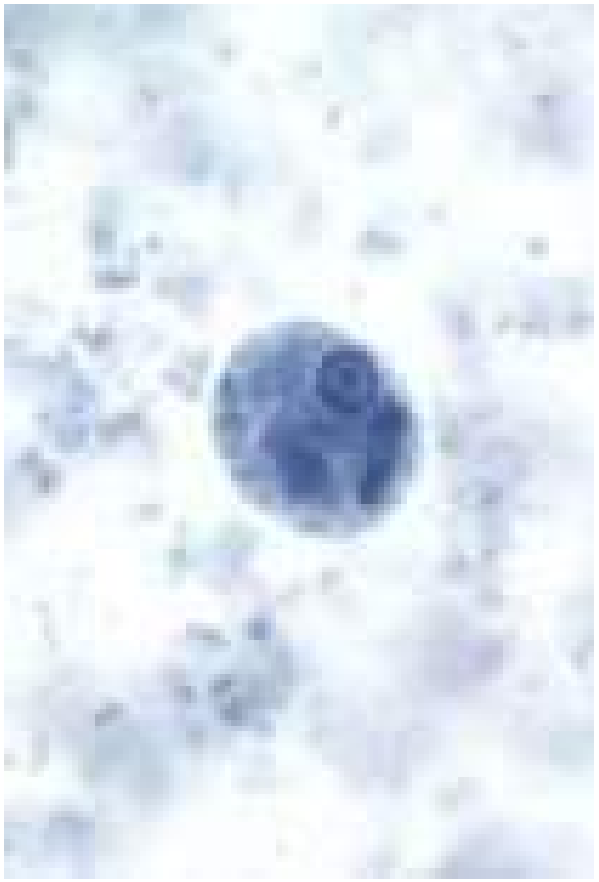


Fig. 177. Stages of life of *Entamoeba coli*.

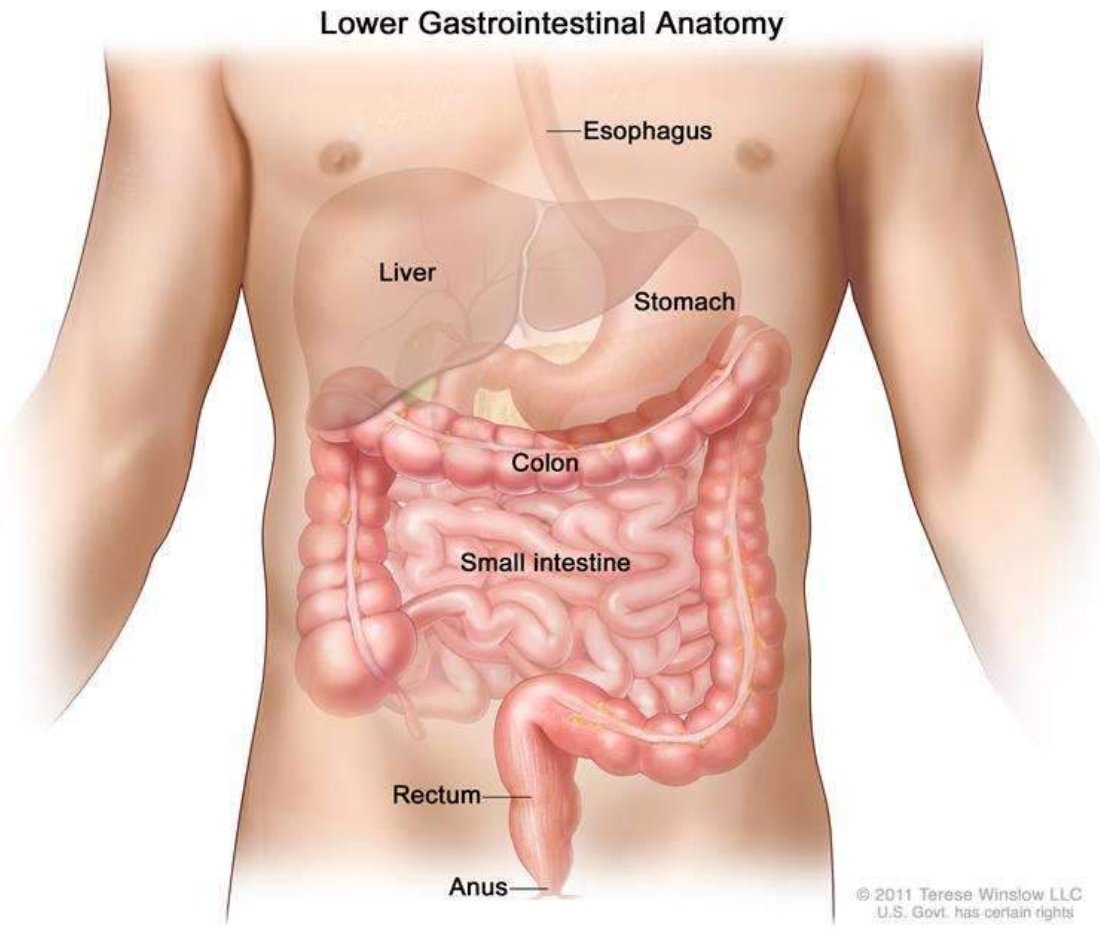
c. cyst: encystation occur in the intestinal lumen, usually during this process:

- Chromatic material is concentrated into bars, rods **(chromatoidal bodies)** with rounded ends in the cytoplasm of the cyst.
- Either before the stool is passed or soon thereafter, the nucleus of the cyst divides into two, then each of the two daughter nuclei divides once again, so the mature cyst typically has four nuclei
- Anaerobic conditions and bacteria are apparently necessary for natural excystation in the intestine
- Immature cysts will not undergo excytation



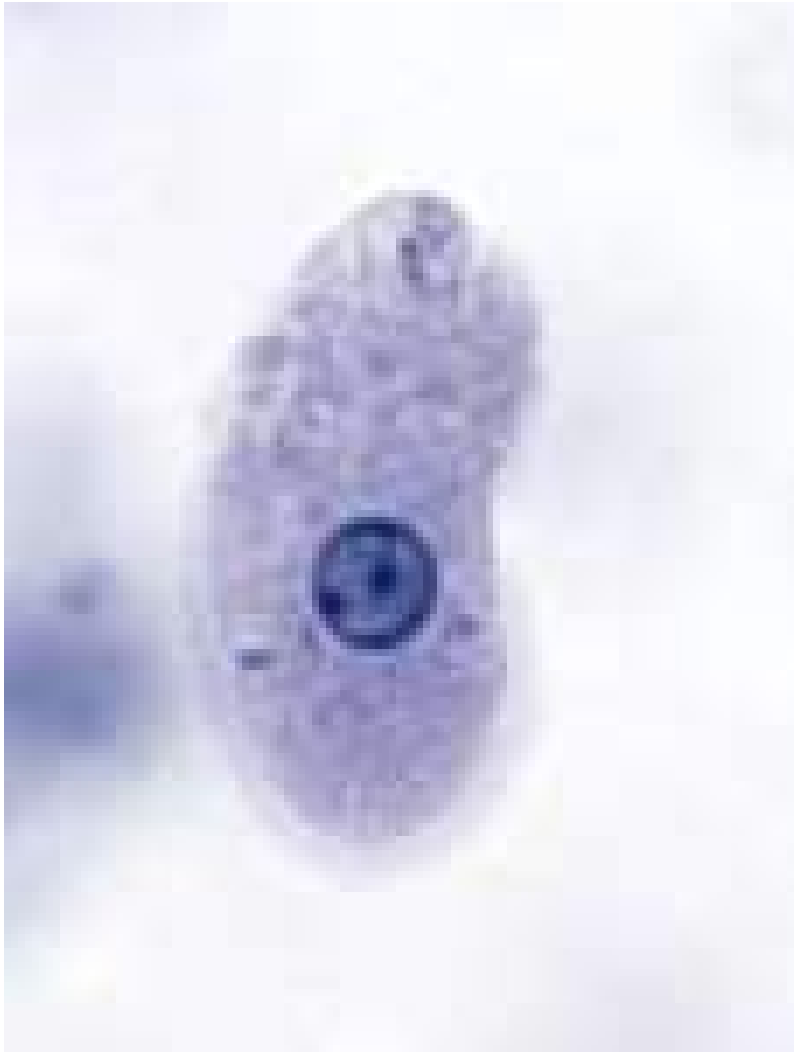
Physiology:

- the intestinal habitat of E.histolytica is the wall of the colon especially in the cecal and sigmoid rectal regions.



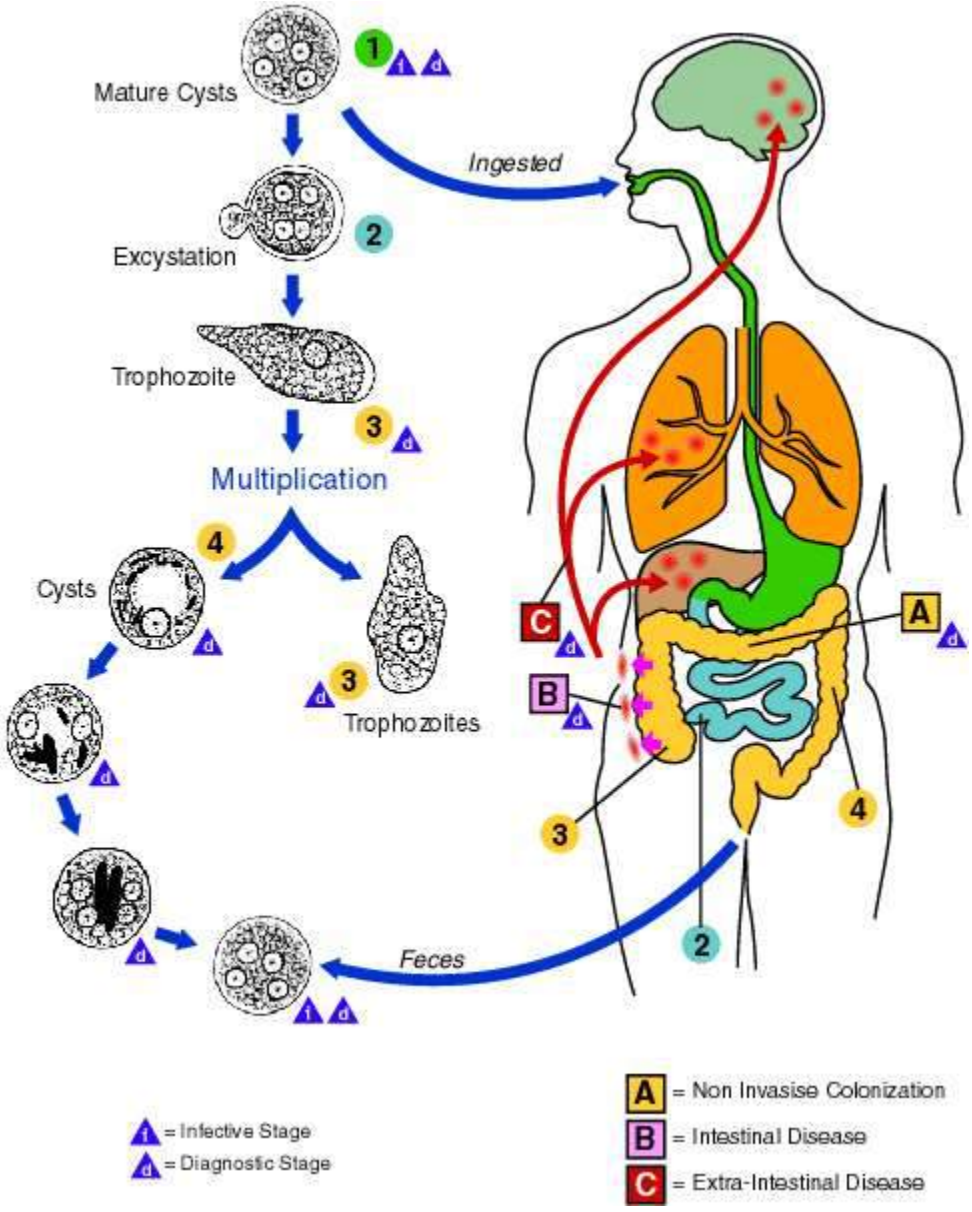
Nutrition:

- in susceptible host, **E.histolytica** absorbs nourishment from the tissues dissolved by cytolytic enzymes and from substances partially synthesized by the or by intestinal bacteria. It also ingests RBCs, hemoglobin, bilirubin.

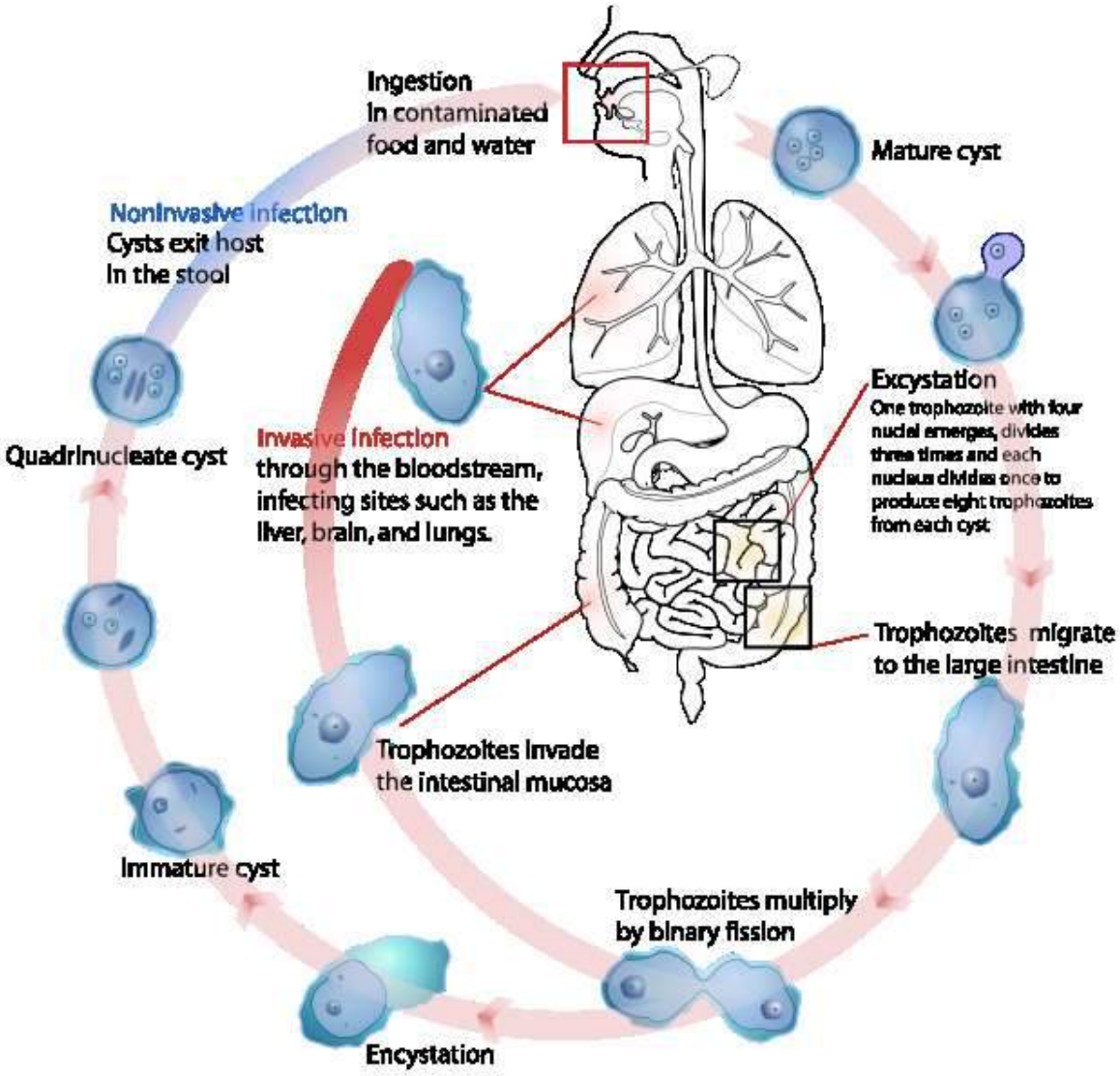


Life cycle

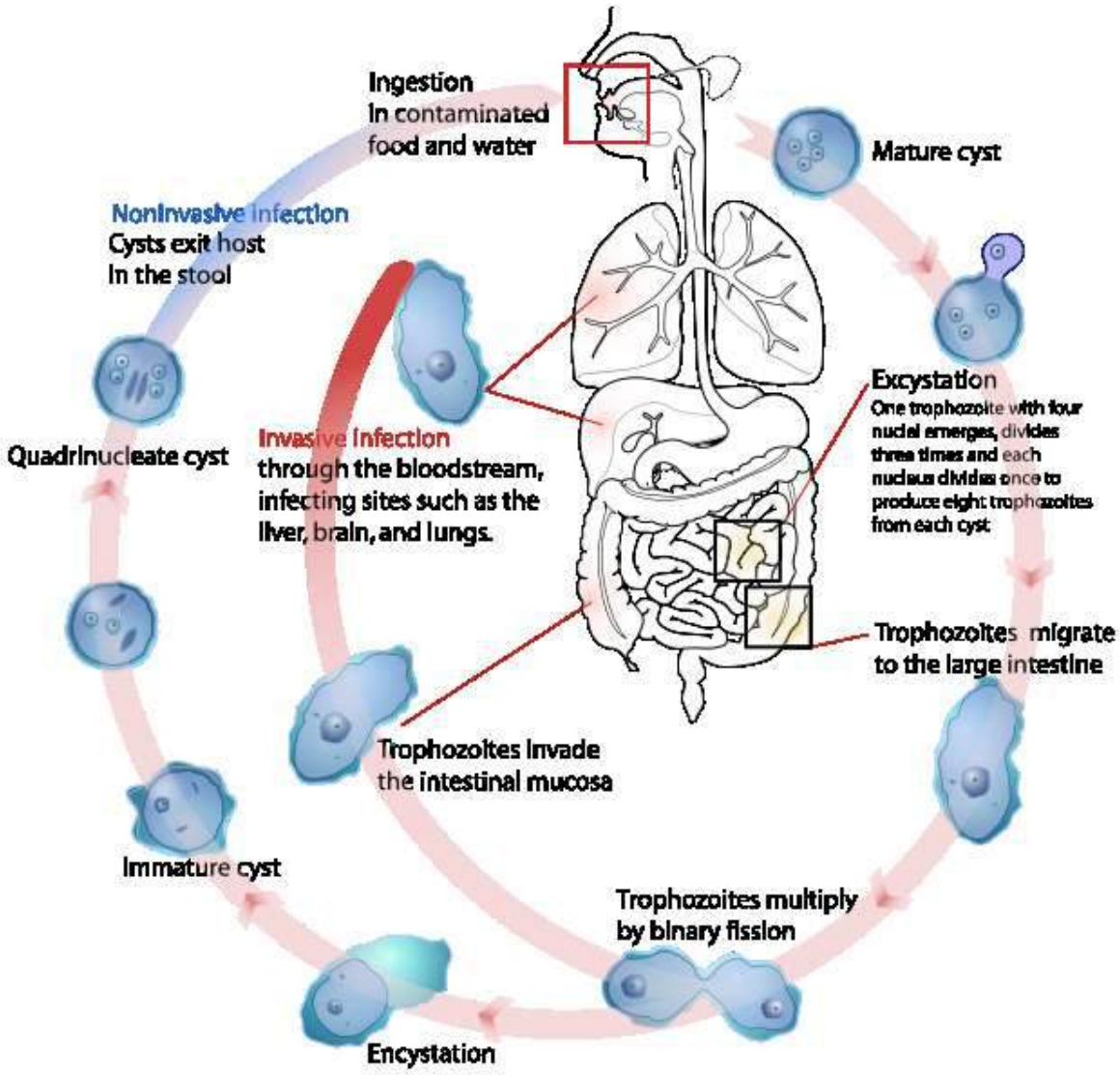
- Man is the principal host and source of infection.
- A certain proportion of amoebae pass from lesions in the intestinal wall into the lumen of large intestine, where they become sub spherical, and secrete a thin tough cystic wall.
- Cysts passed in feces

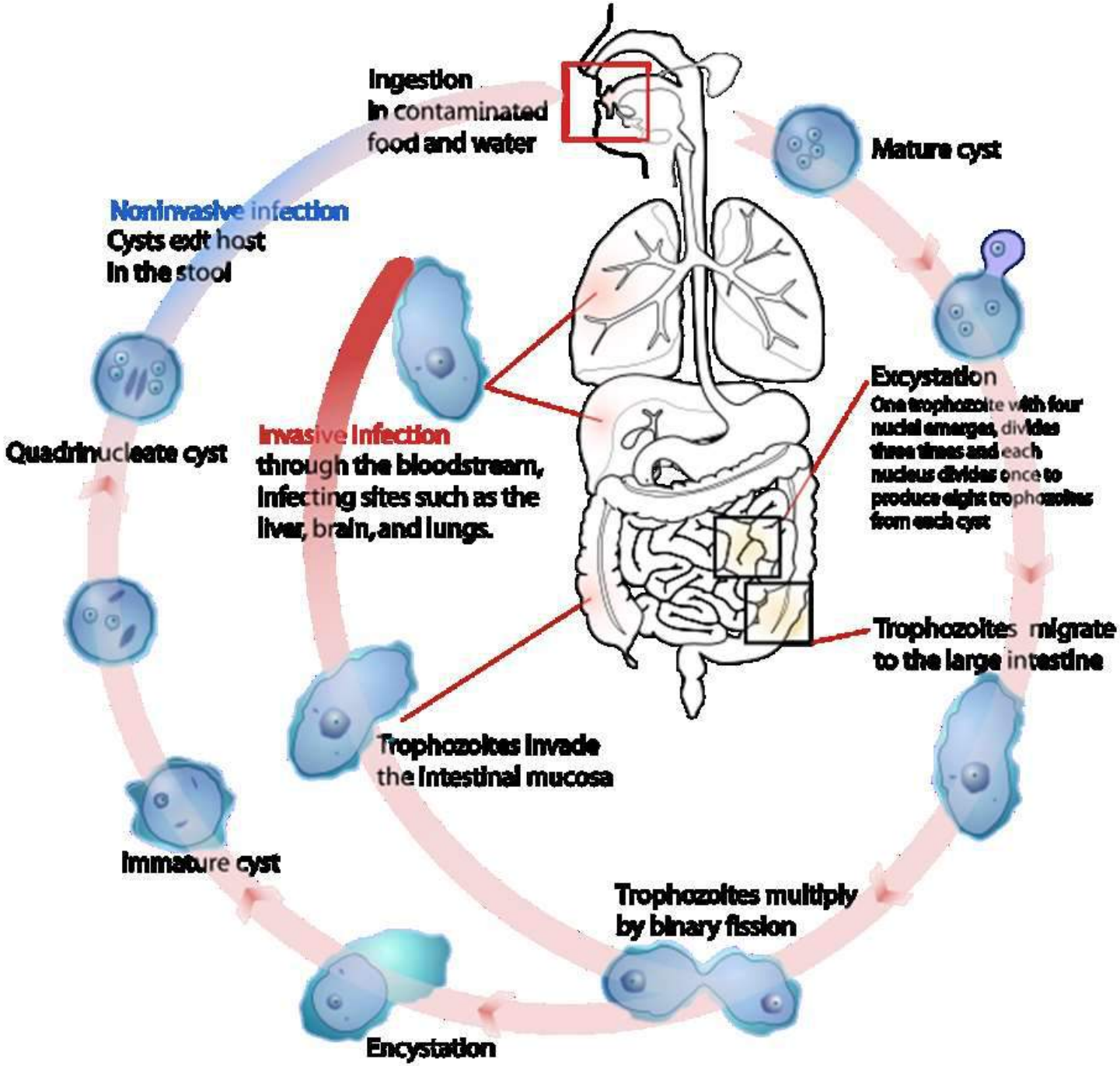


- Mature cyst is the infective stage when ingested with contaminated food or water
- Upon ingestion by a new host, the cysts undergo excitation under the influence o digestive juices.
- The liberated 4-nucleated metacytic ameba divides into eight small trophozoites, the sma immature amoeba move downward to th large intestine.



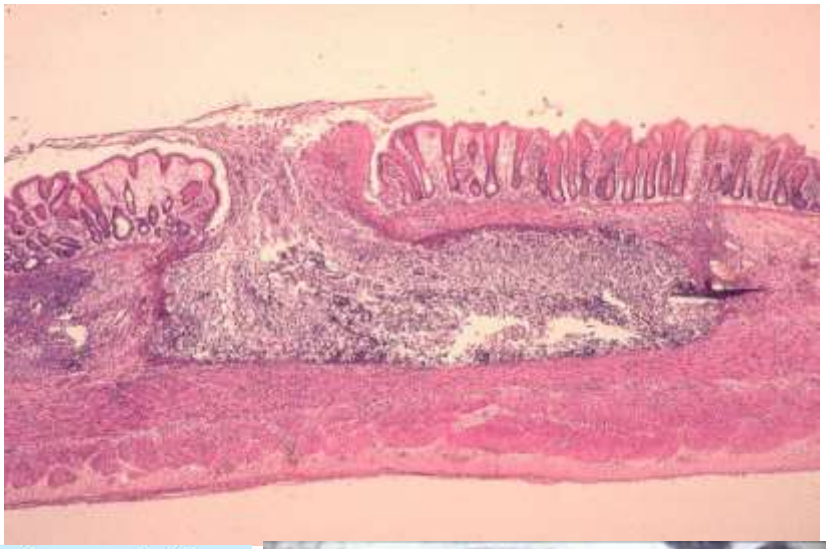
- Intestinal stasis often enables the amoebae to establish a site of infection in the cecal region of the colon
- At the site of localization, the amoebae invade the intestinal mucosa by the action of their cytolytic enzymes and amoeboid movements
- In some cases the infection remain superficial, and in others, the organisms from the intestinal wal the amebae may spread to secondary sites throughout the body

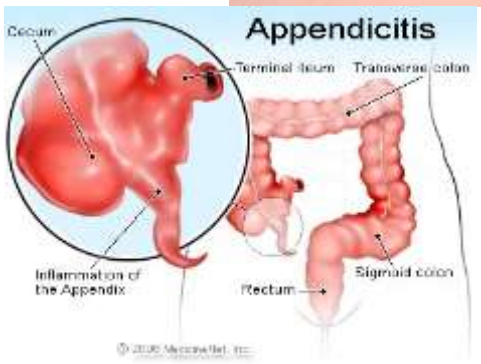


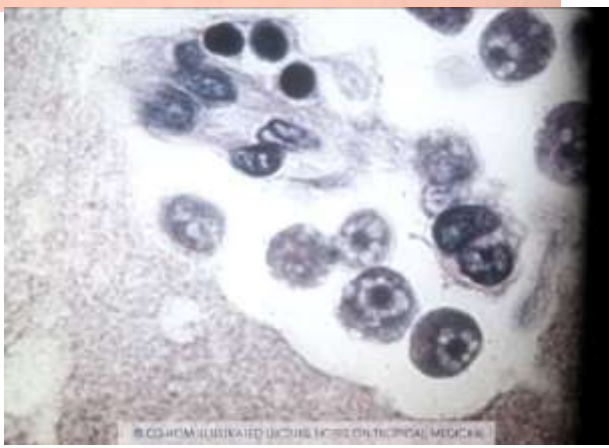

Ingestion
In contaminated
food and water
Mature cyst
Noninvasive infection
Cysts exit host
In the stool
Excystation
One trophozoite with four
nuclei emerges, divides
three times and each
nucleus divides once to
produce eight trophozoites
from each cyst
Quadrinucleate cyst
Invasive Infection
through the bloodstream,
infecting sites such as the
liver, brain, and lungs.
Trophozoites migrate
to the large intestine
Trophozoites invade
the intestinal mucosa
Immature cyst
Trophozoites multiply
by binary fission
Encystation

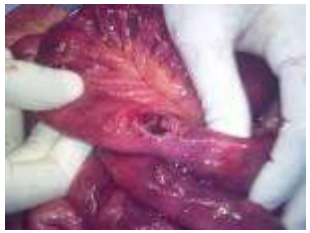
Pathology and symptomatology

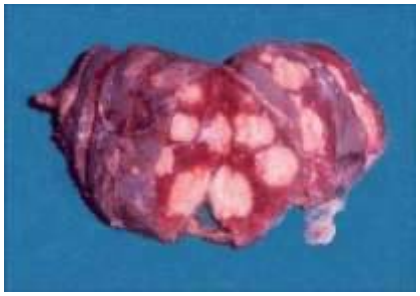
- Primary lesion is ulcer- invasion of the wall of large intestine- ulcer is flask shaped.
- **Complications-**
- Amoeba granuloma (amoeboma); appendicitis, intestinal perforation





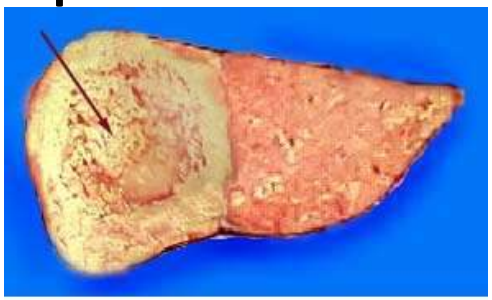




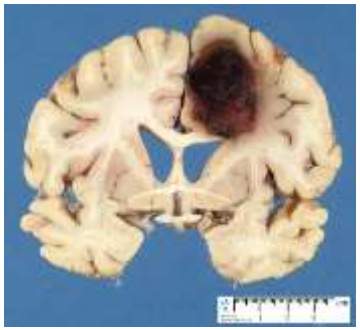


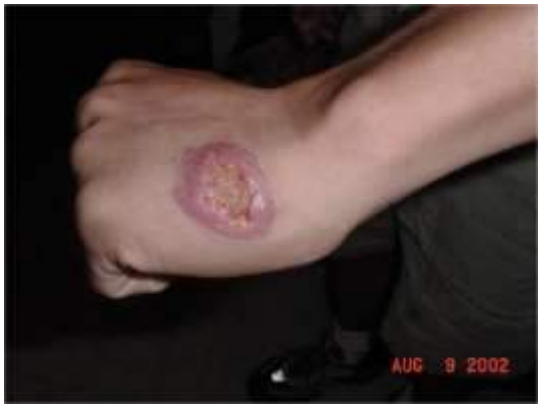
Secondary lesions-

- occur as a result of metastasis of trophozoites to extra intestinal organs-
- **liver is most frequency affected-**
- Hepatic amoebiasis;
- Pulmonary amebiasis
- Cerebral ameobiasis;
- Cutaneous amebiasis;
- Splenic abscess.



Abscess of hepatic amebiasis



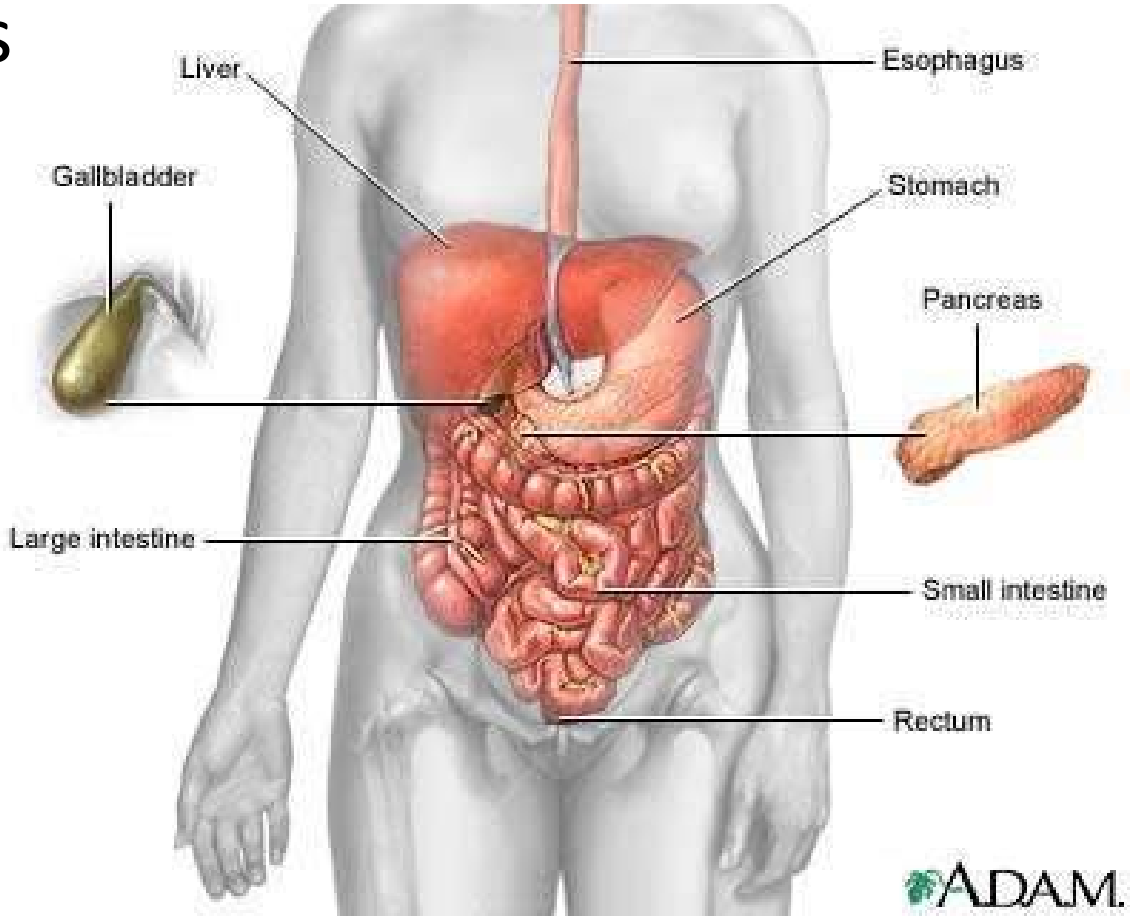


Disease state:

- Asymptomatic carrier
- Symptomatic infection
- **Symptoms:**
- **Diarrhea**
- **Dysentery- stool containing blood, mucous and shreds of necrotic mucosa,**
- **Acute abdominal pain,**
- **Tenderness**
- **Fever.**

Chronic ameobiasis-

- Recurrent attacks of dysentery.
- Abdominal tenderness,
- Hepatomegaly,
- Weight loss and emaciation.

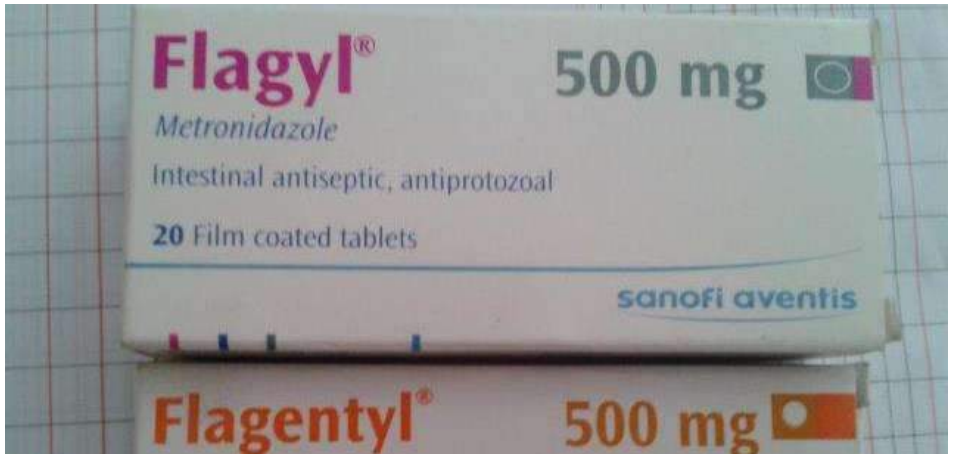


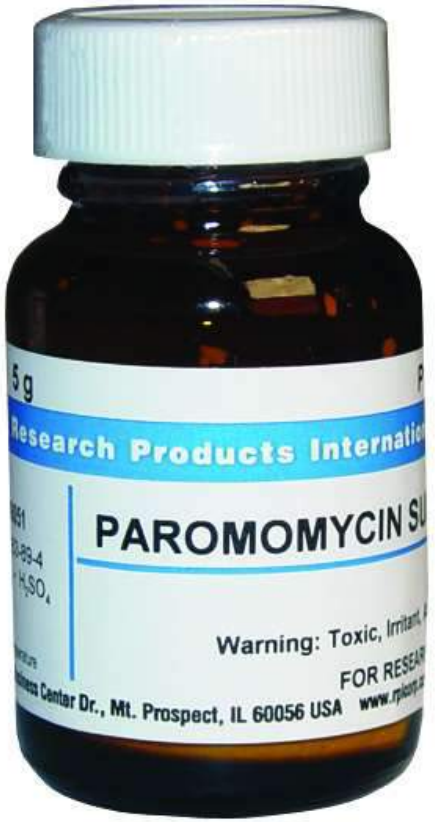
Diagnosis:

- Microscopic stool examination- for trophozoite and cysts
- Amoebic serology
- Abscess aspiration
- Entamoeba dispar a non- pathogenic is indistinguishable by microscopy, PCR are distinguish E. dispar from E.histolytica in heavier infection.

Treatment

- Invasive state (dysentery, liver abscess)…. Metronidazole
- Carrier state..paromycin



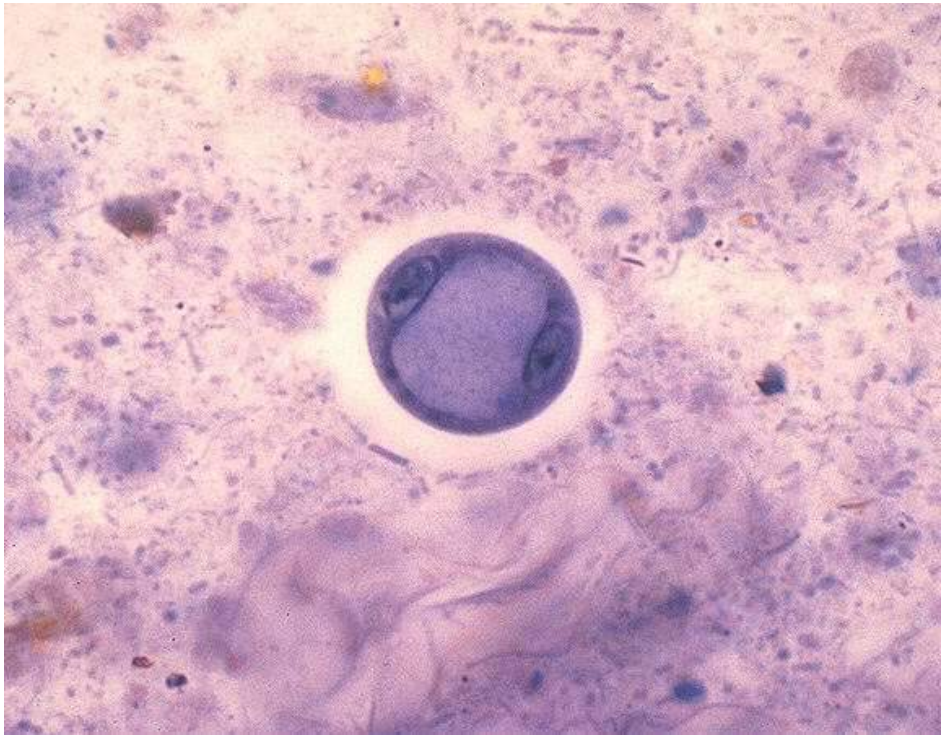


Prevention:

- Personal hygiene
- Food and drinks must be protected from flie (mechanical transmission)
- Avoiding autoinfection by wash hands after defecation
- Human feces should not be used as a fertilizer.

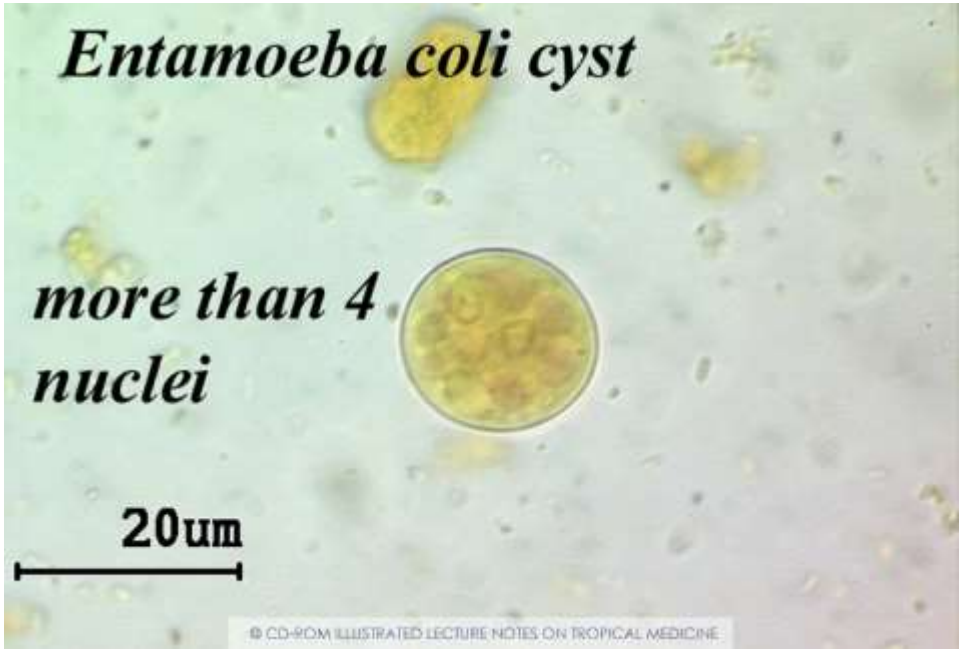
Nonpathogenic amoebae

- **Entamoeba.coli**
- **Synonyms…** Amoebae coli, Entamoeba coli
- **Geographic distribution…**cosmopolitan

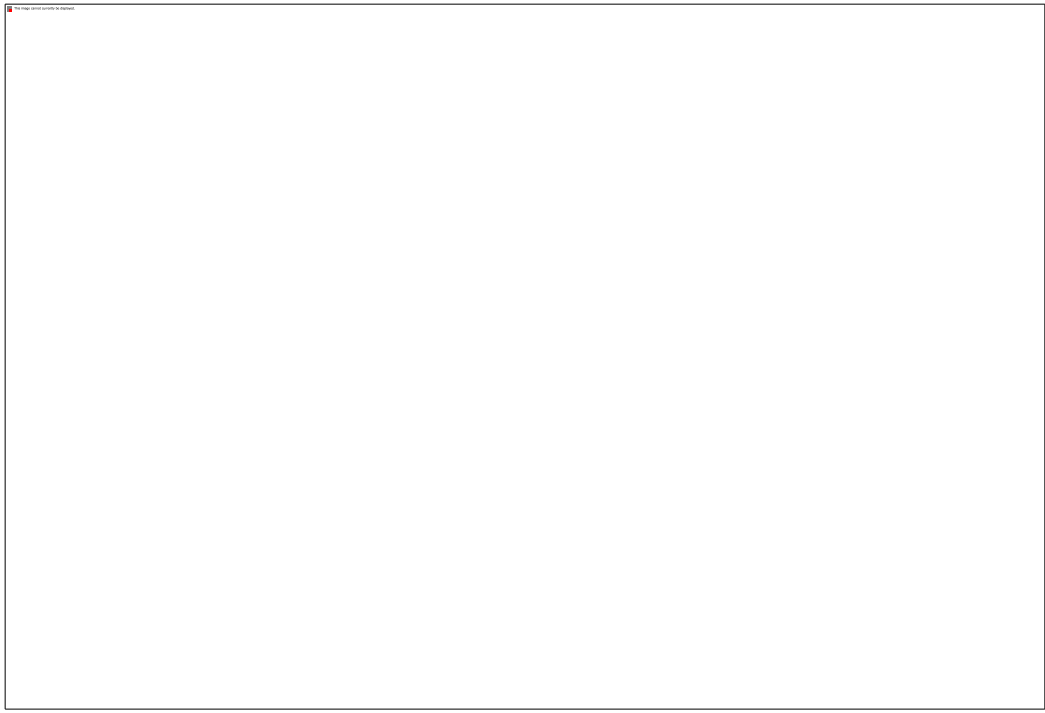


Morphology…

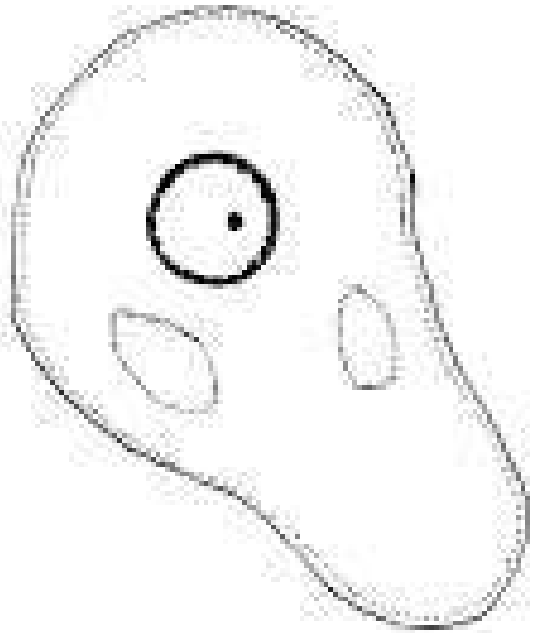
- The trophozoite, usually **20-30 m** in size
- The pseudopodia are short, blunt
- The nucleus is usually round to oval with thick membrane lined with irregular distributed coarse chromatin granules



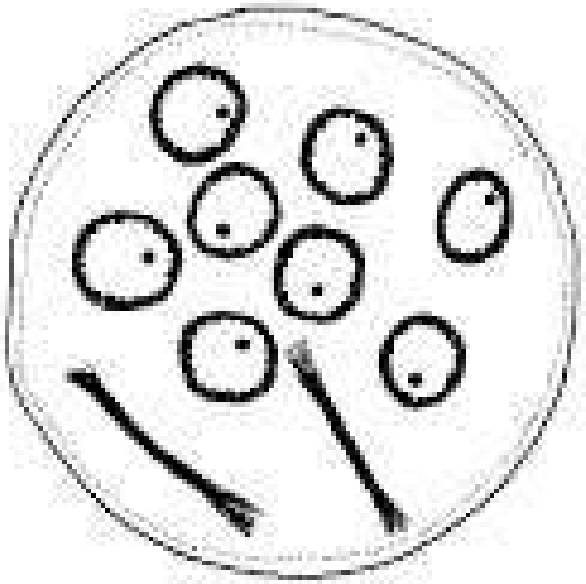
- The karyosome is large, irregular, eccentric.
- Occasionally the cytoplasm contains ingested bacteria.
- The spherical cysts are somewhat larger than those of **E.histolytica**.



- The mature cysts have **8 nuclei**
- Chromatoidal bodies, only observed in immature cysts, are slender rods, with sharp ends
- Does not invade tissues



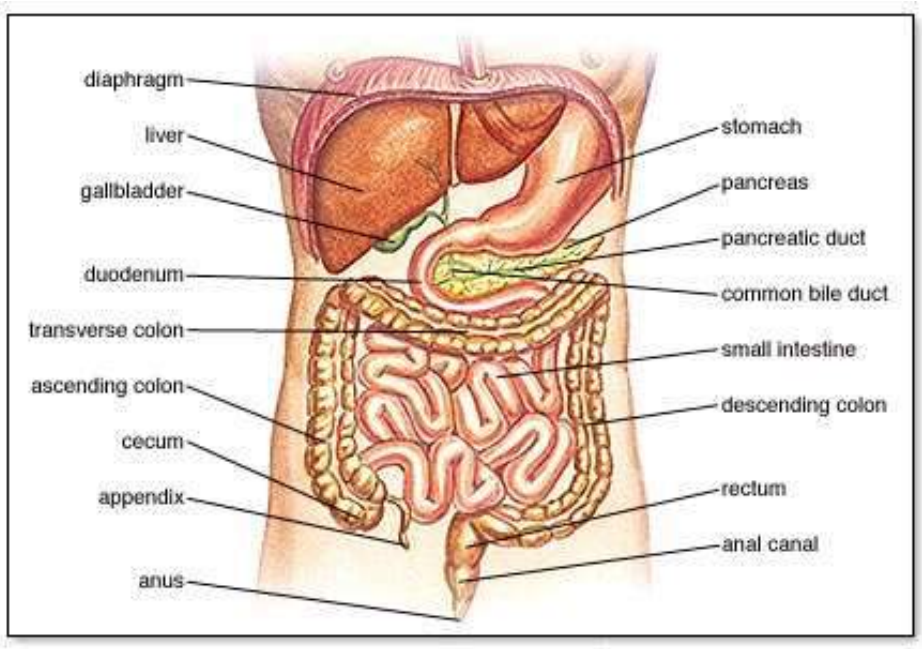
trophozoite



cyst

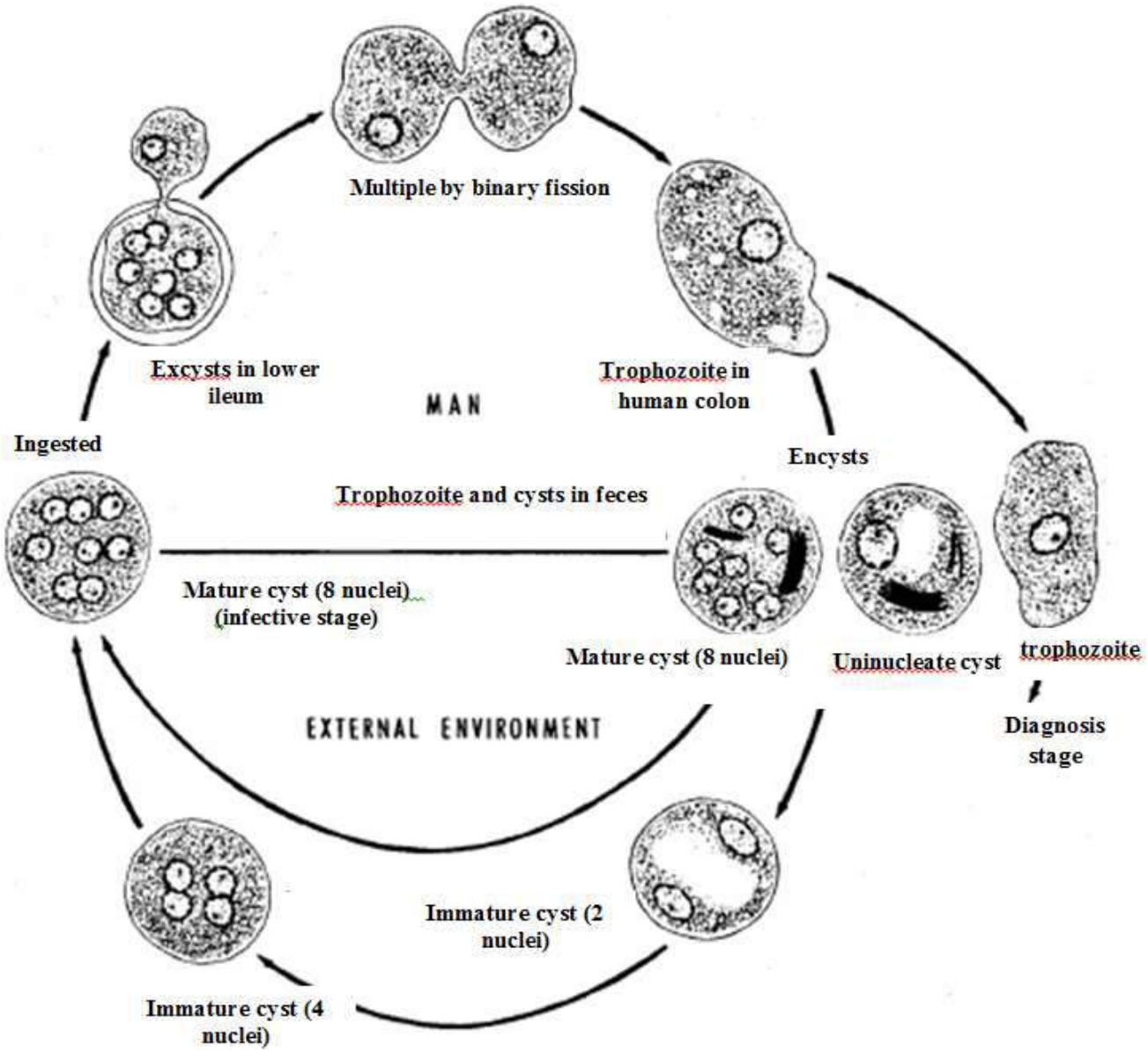
Life cycle:

- Cysts form in the large intestine and are passed in the feces
- The nucleus dividing to form eight small nuclei in the mature cysts.
- The cysts are carried to the small intestine of man in contaminated food or drink



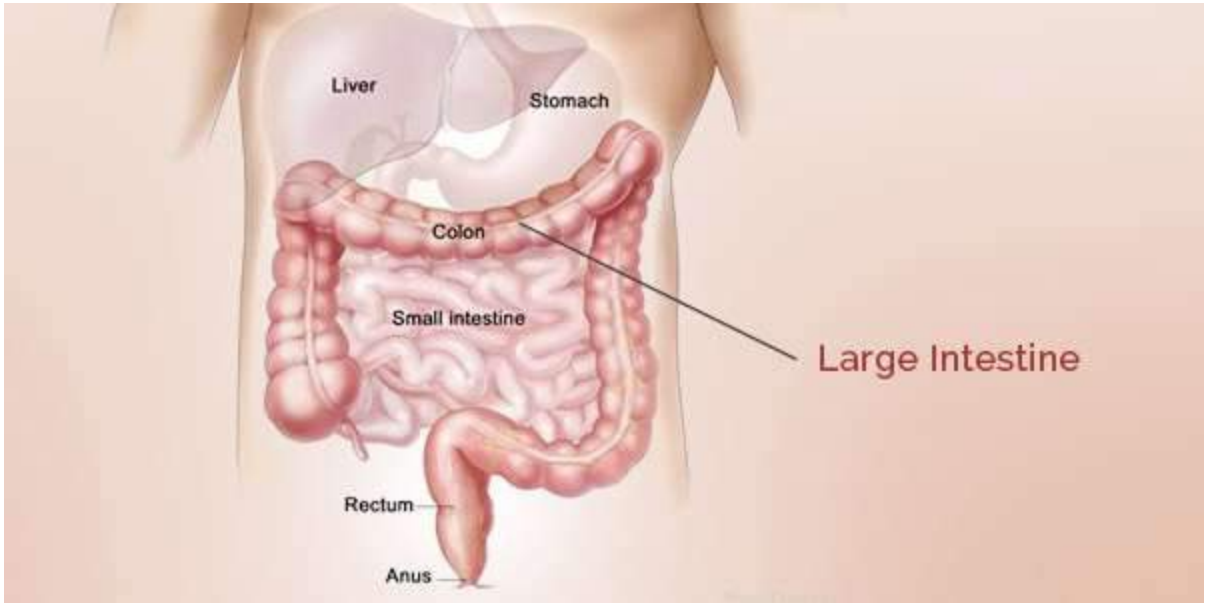
- The multinucleate ameba excysts and divides
- These young Amebae pass down to the cecum where they grow and multiply
- Man is the chief host

Life cycle of Entamoeba.coli



Physiology…

- **E.coli** is a nonpathogenic commensal of the large intestinal of man
- **Pathogenicity**
- Entamoeba coli is not pathogenic

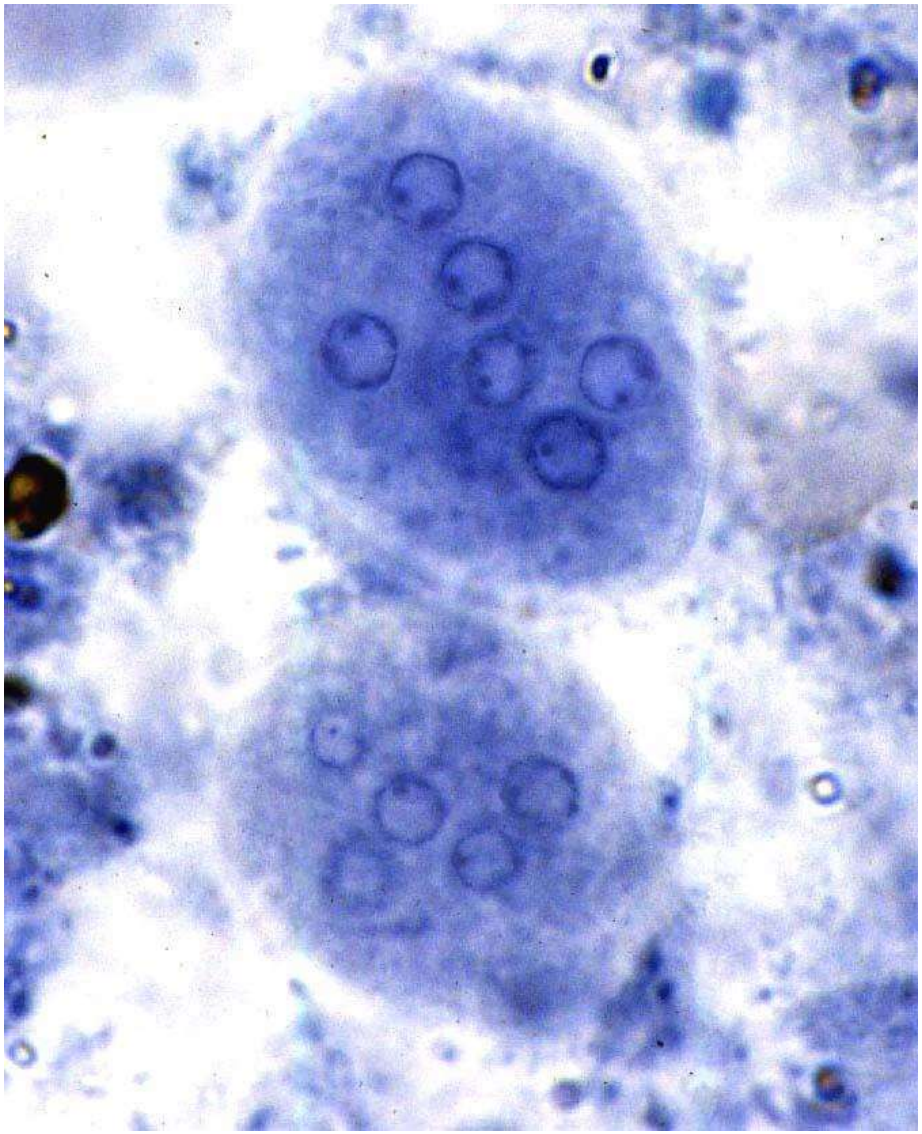


Diagnosis

- Microscopic stool examination- for trophozoites and cysts

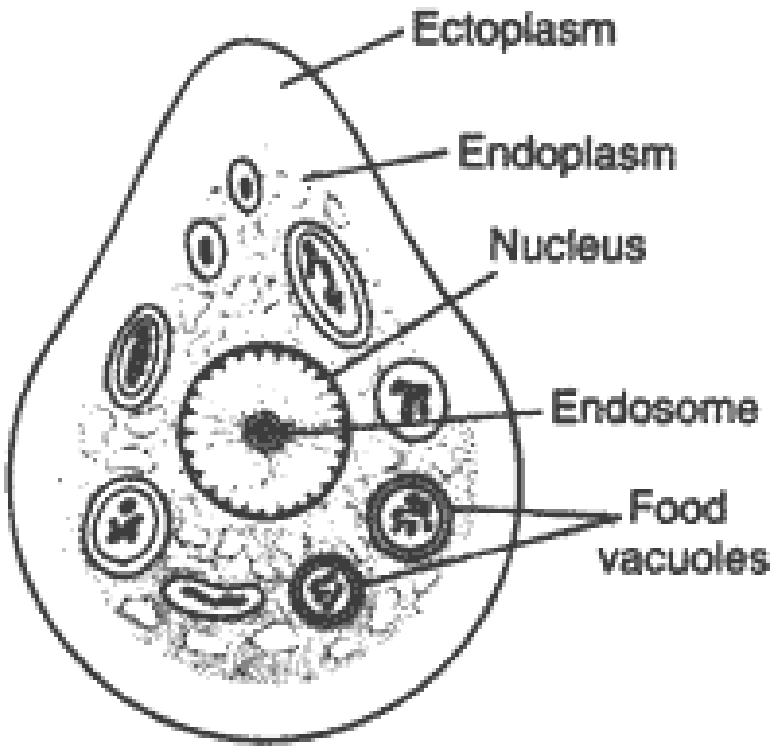
Treatment

- Treatment for **Entamoeba.coli** is unnecessary



Entamoeba gingivalis

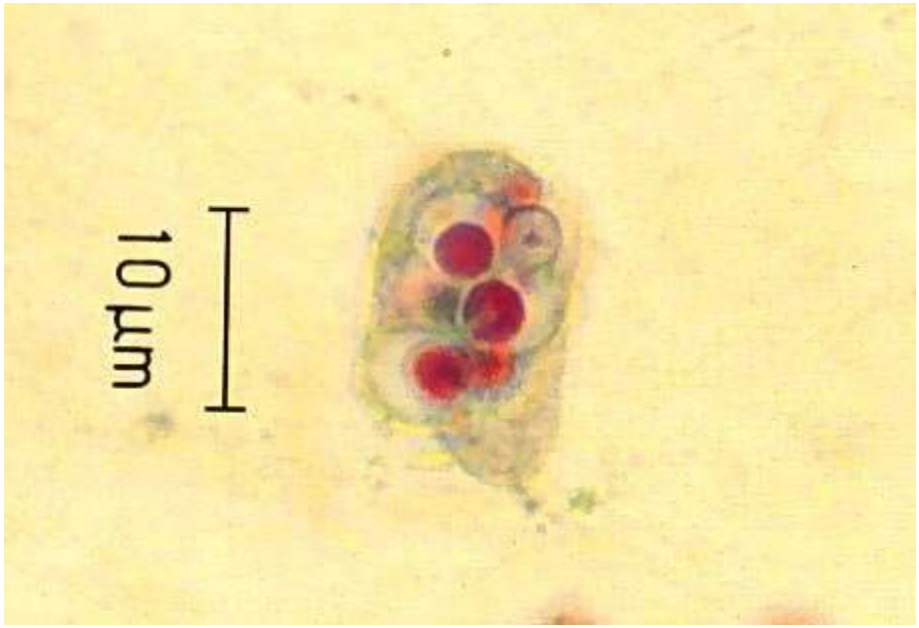
- **Synonymes…** Amoebae gingivali, Entamoeba coli gingivalis
- **Geographic distribution…**
- cosmopolitan



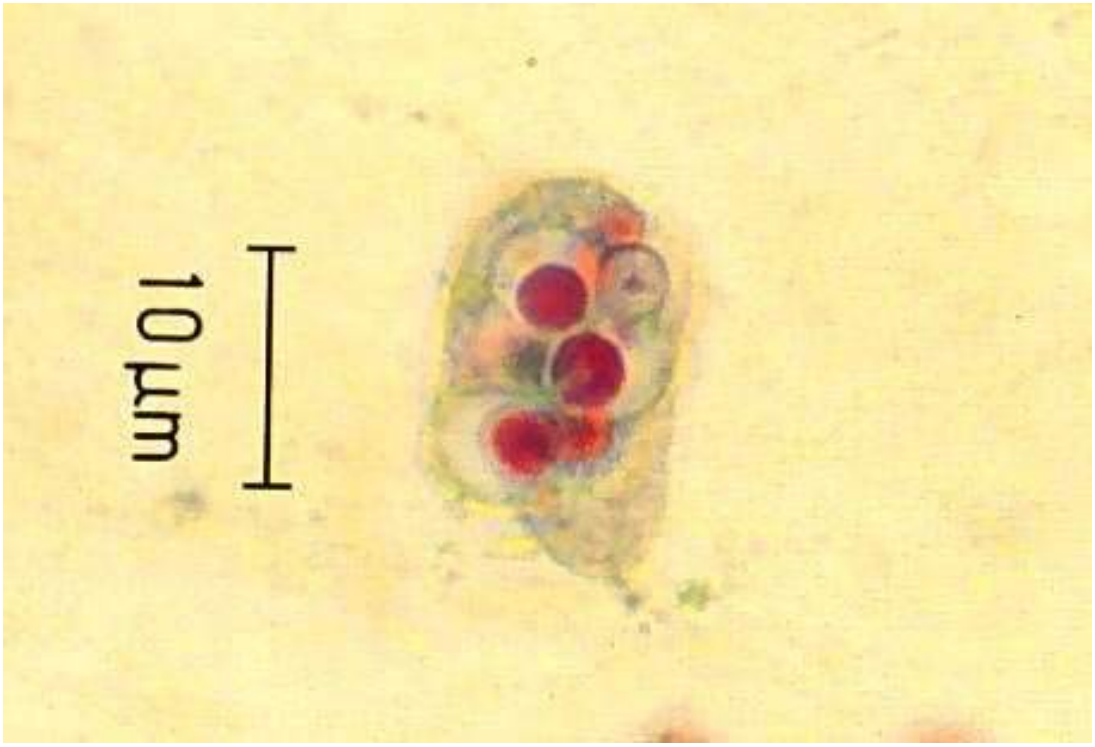
Entamoeba gingivalis

Morphology…

- The trophozoite is the only known form (no cysts formation)
- The usual size of trophozoite is from 10-**20m**
- The cytoplasm contains many food vacuoles that contain cellular debris, blood cells and bacteria

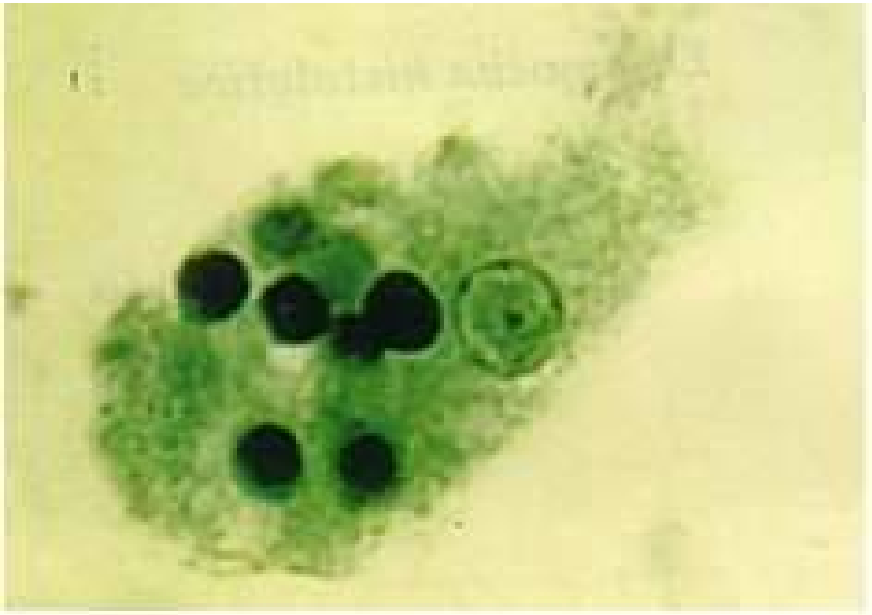


- The spherical nucleus is smaller than that of E.histolytica
- The nuclear membrane is lined with a ring of irregular small granules
- The karyosome position is centric or eccentric

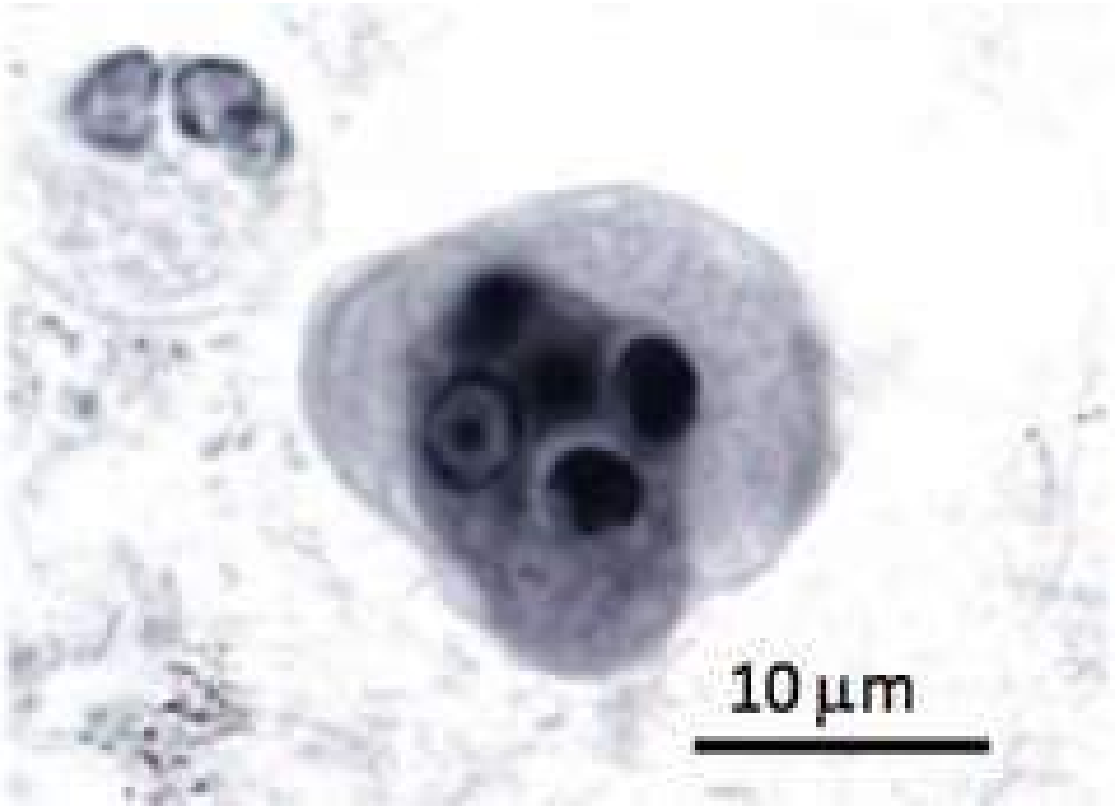


Life cycle:

- It is found in the mouth between the gingival pockets and near the base of the teeth. **Entamoeba gingivalis** is found in 95% of people with gum disease and in 50% of people with healthy gums



- The cyst formation is not present, therefore transmission is direct from one person to another by kissing, or by sharing eating utensils

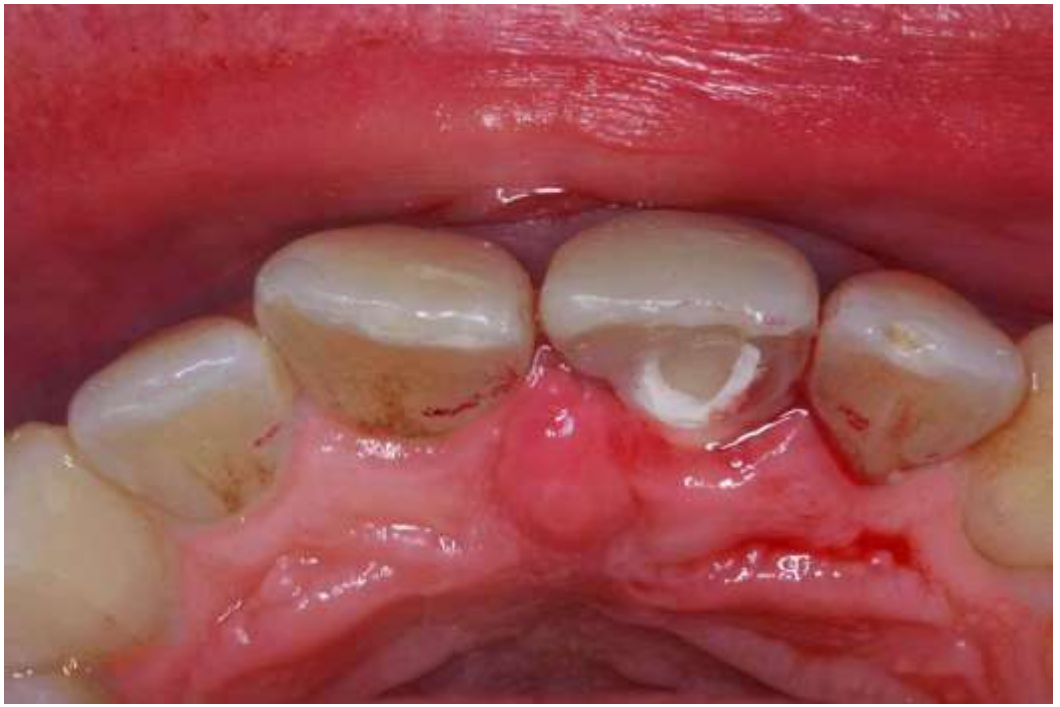


Pathogenicity:

- Entamoeba gingivalis is a non pathogenic protozoa
- It is a scavenger of disease tissues rather than a pathogenic invader

Diagnosis:

- Scraping for examination are taken from the tarter of the teeth or from pockets near the teeth. Diagnosis is easy, since this ameba is the only one found in the mouth.



Treatment

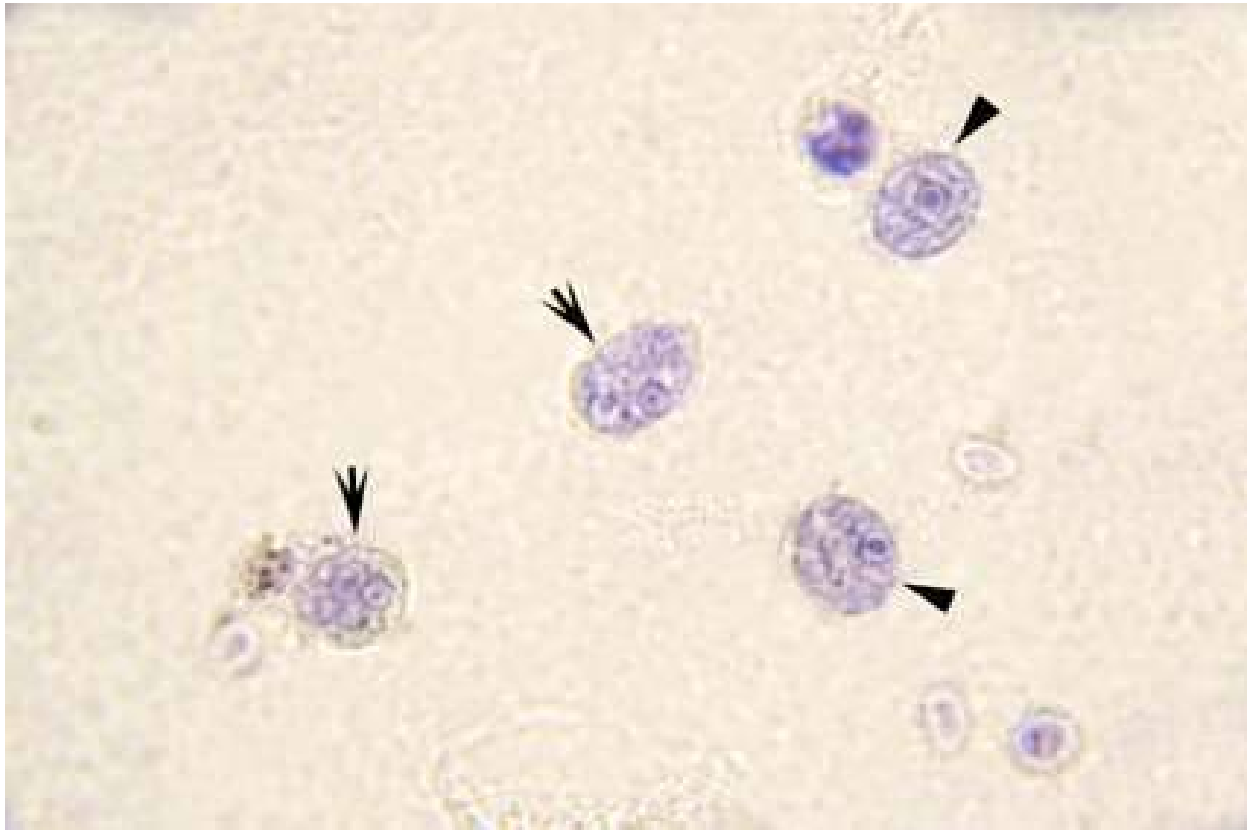
- Treatment of abnormal oral conditions is the best way to eliminate the parasite.

Prevention

- Correct oral hygiene should reduce the incidence of **Entamoeba gingivalis**

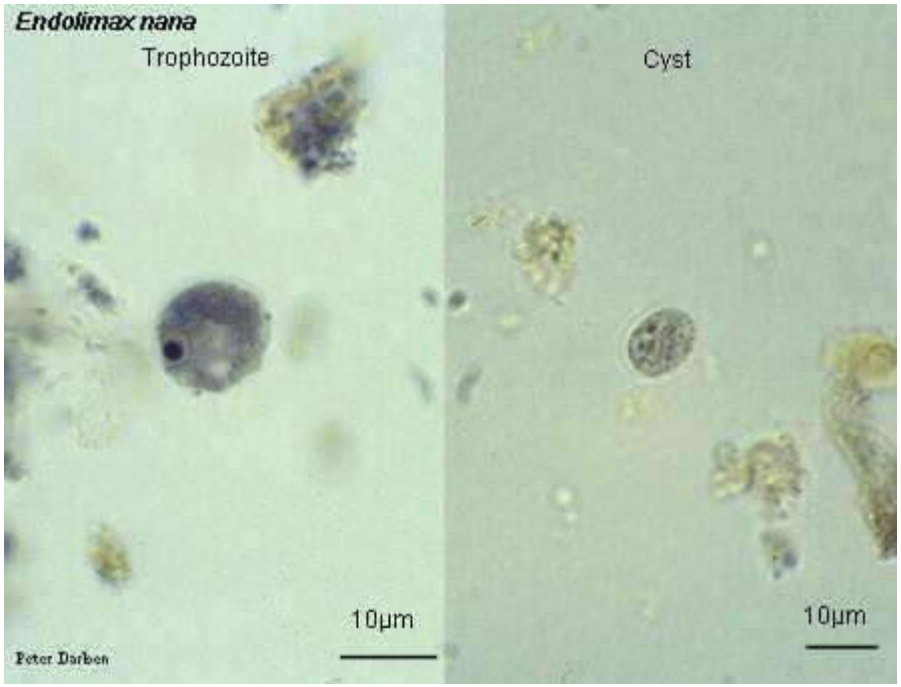
Endolimax nana

- **Synonymes…** Endolimax intestinalis
- **Geographic distribution…**cosmopolitan



Morphology…

- The trophozoite measures from **6-15m**
- The spherical nucleus shows a large central or eccentric pleomorphic karyosome something split into several masses connected with slender threads
- The thin nuclear membrane usually does not show attached chromatin granules

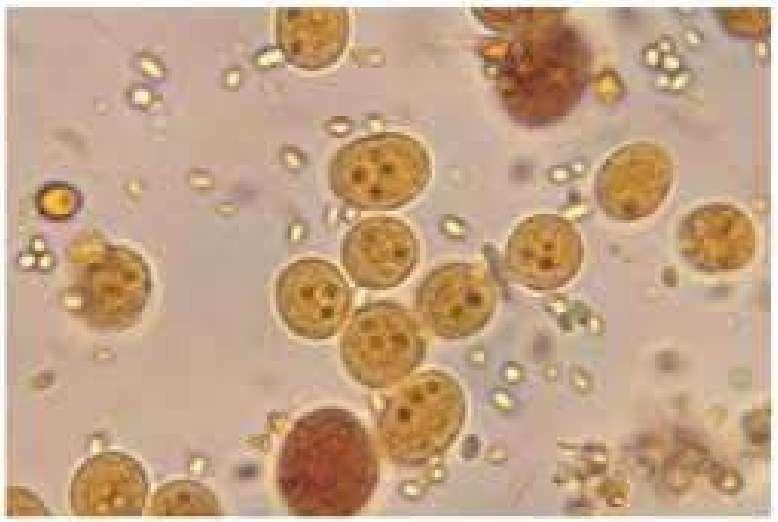


Life cycle

- Entamoeba nana resemble, Entamoeba coli in its life cycle and mode of transmission except the mature cyst probably produces four new amebae after excystation.

Physiology

- Endolimax nana inhibit the lumen of large intestine
- **Pathogenicity**
- It is a non pathogenic parasite of the large intestine of man

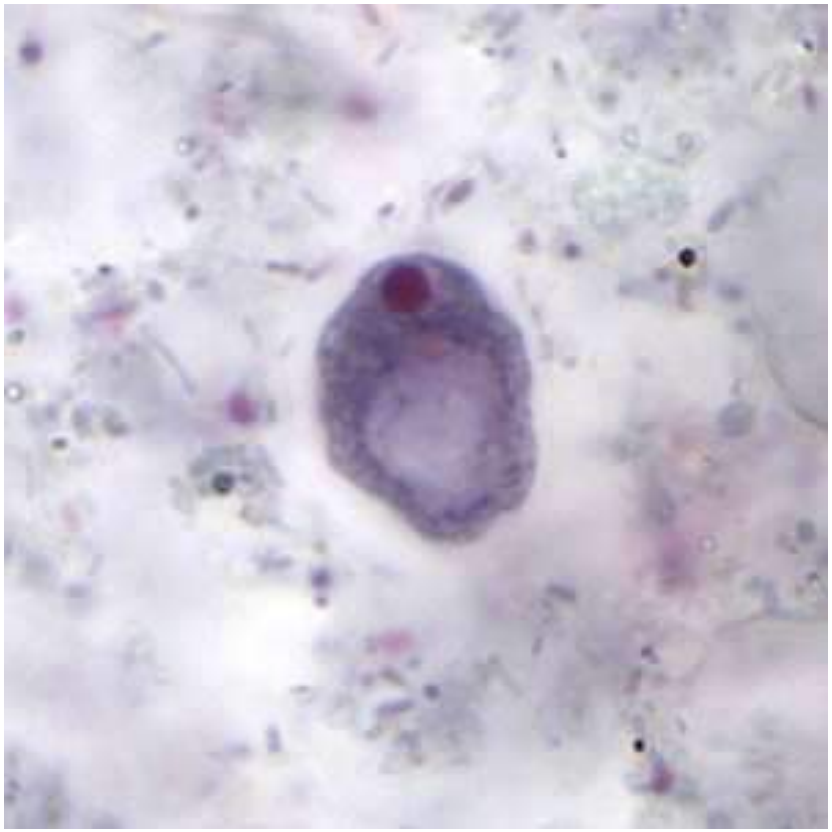


Diagnosis:

- The Amoebae identified by its small size, characteristic nucleus, and its quadrinucleate cyst
- **Treatment**
- It is resistant to chemotherapy

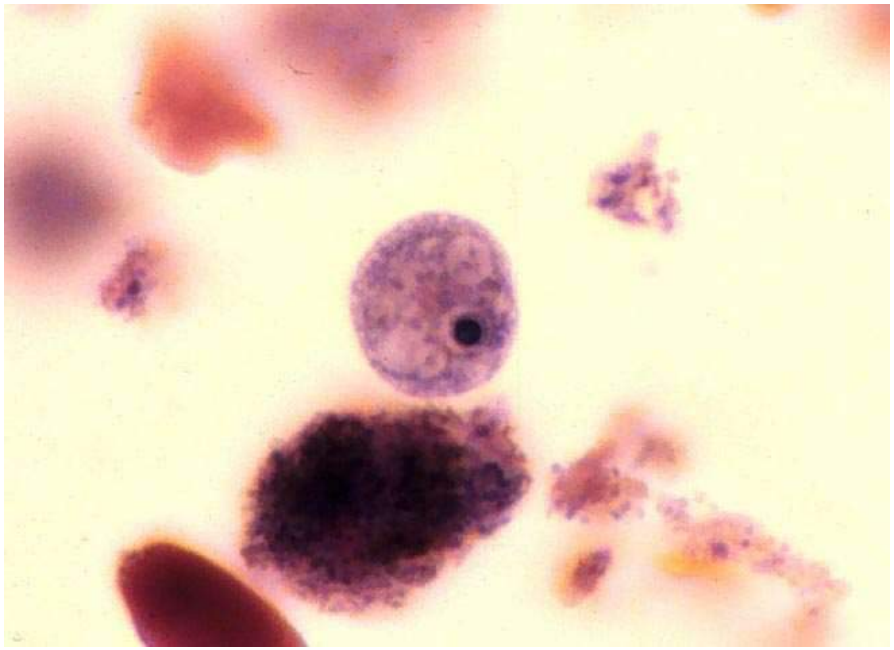
Iodomoeba butshlii

- **Synonyms…** iodomoeba williamsi
- **Geographic distribution…**cosmopolitan

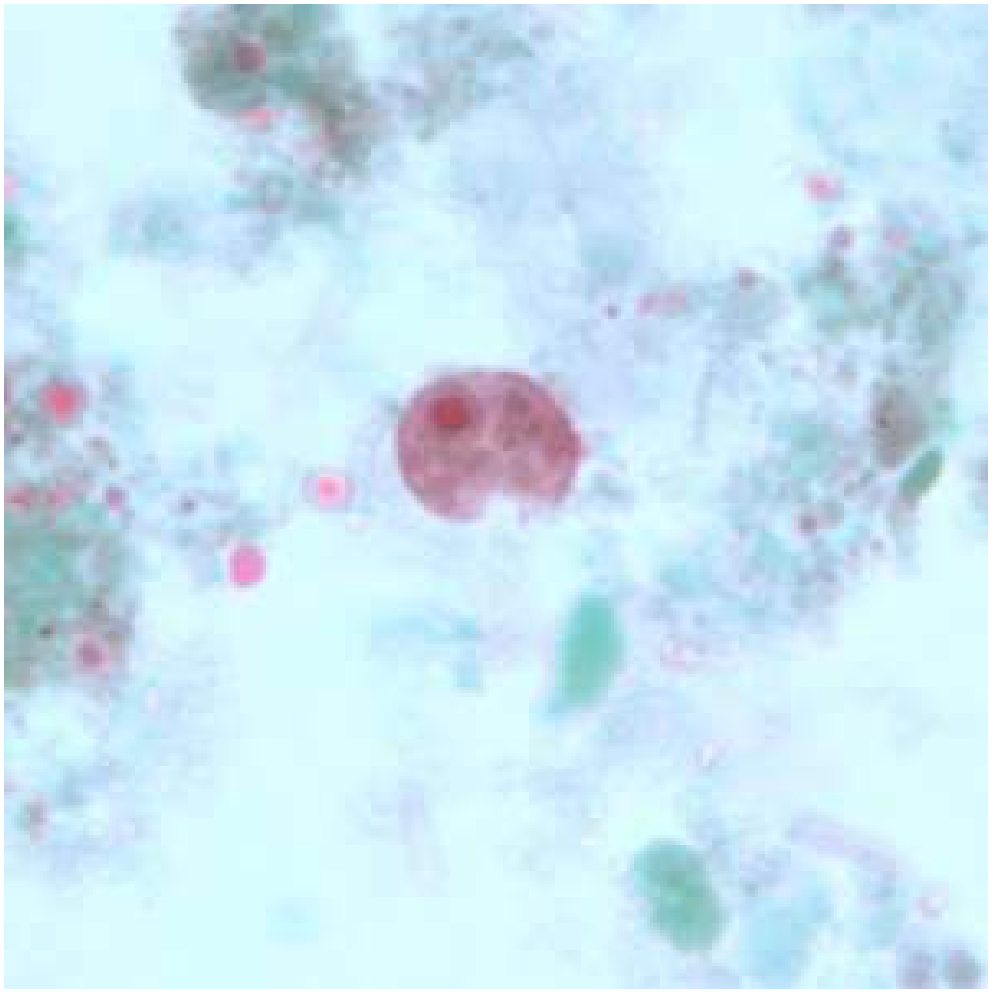


Morphology…

- The trophozoite is rarely in the feces, is usually from **9-14m** in size
- The nucleus is characterized by central karyosome one third to one half of its diameter
- The nuclear membrane shows no chromatin granules

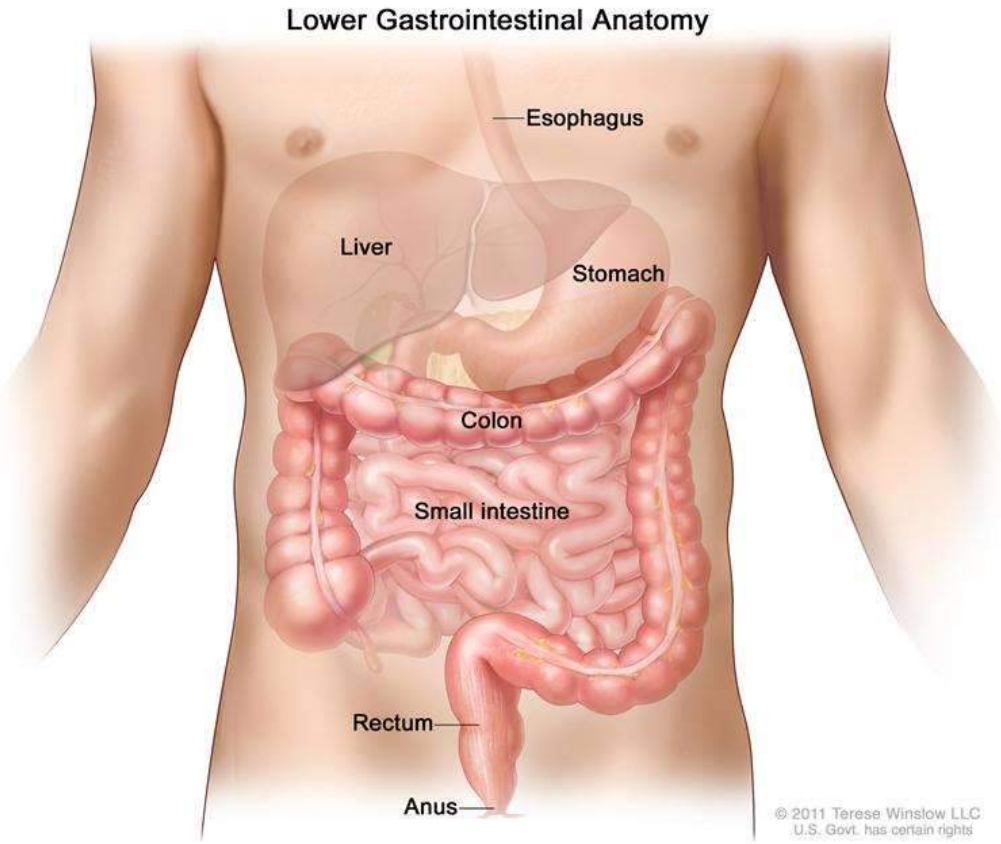


- The uninucleate cyst is characterized by a thick cyst wall
- The cyst contains a large compact glycogen vacuole.



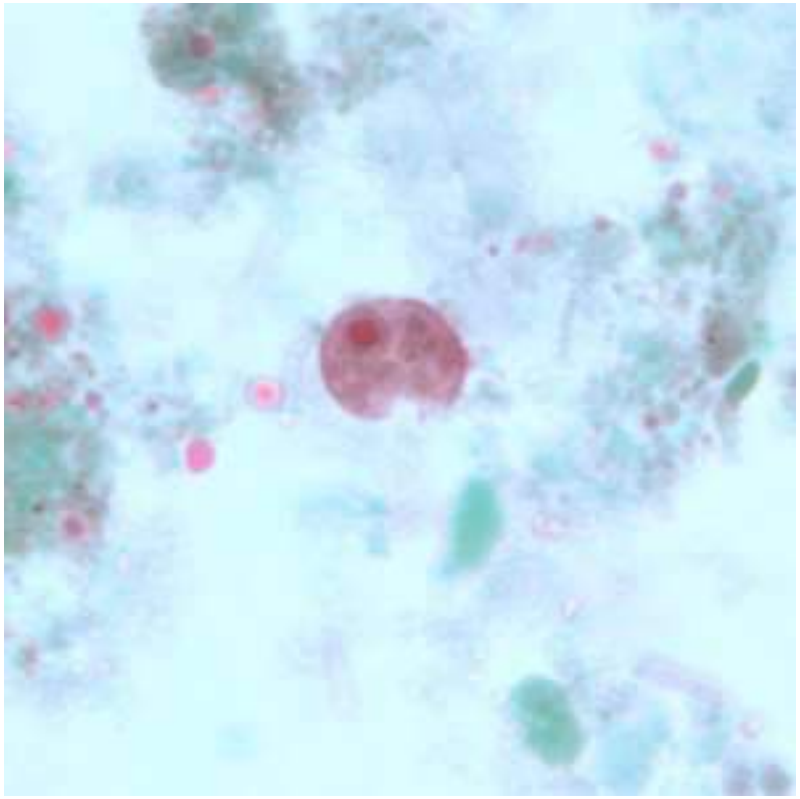
Life cycle

- Its resembles that of other cyst-forming intestinal amebae
- **Physiology: I. butchlii** inhabits the lumen of intestine
- **Pathogenicity**
- It is a non pathogenic parasite of the large intestine of man



Diagnosis:

- By its characteristic nucleus, and by large glycogen vacuole in the cyst

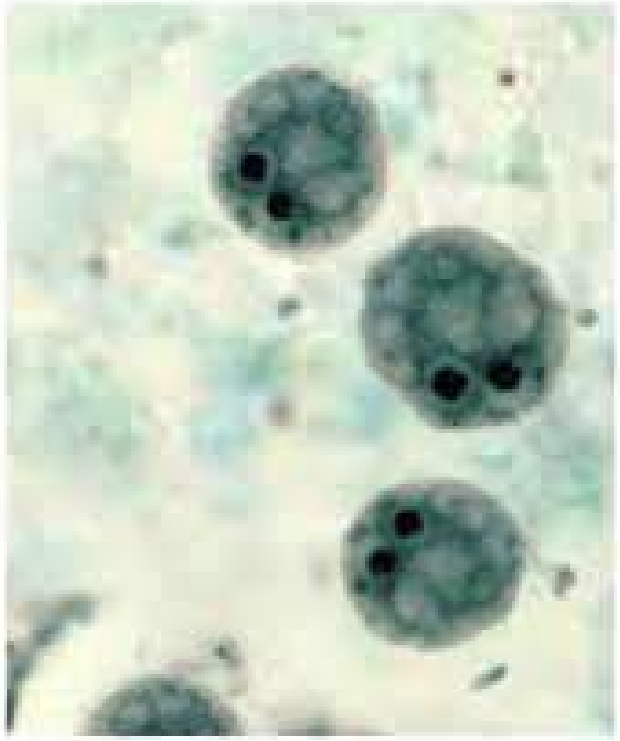


Diebtamoeba fragilis

- **Geographic distribution**…cosmopolitan

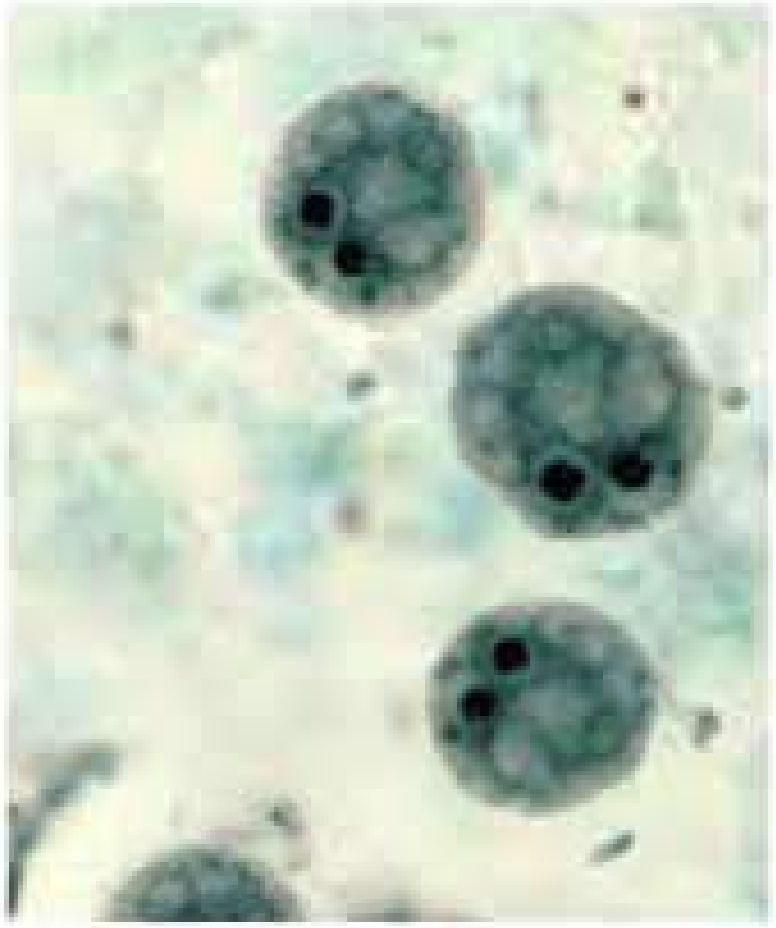
Morphology…

- Is only known in the trophozoite form
- The trophozoite is usually from **5-12m** in diameter
- **D. fragilis** differs from other intestinal amoebae in that about **80%** of specimens have two nuclei



Dientamoeba fragilis ~ eencellige parasiet, wordt bij 33% van de mensen met PDS klachten aangetroffen.

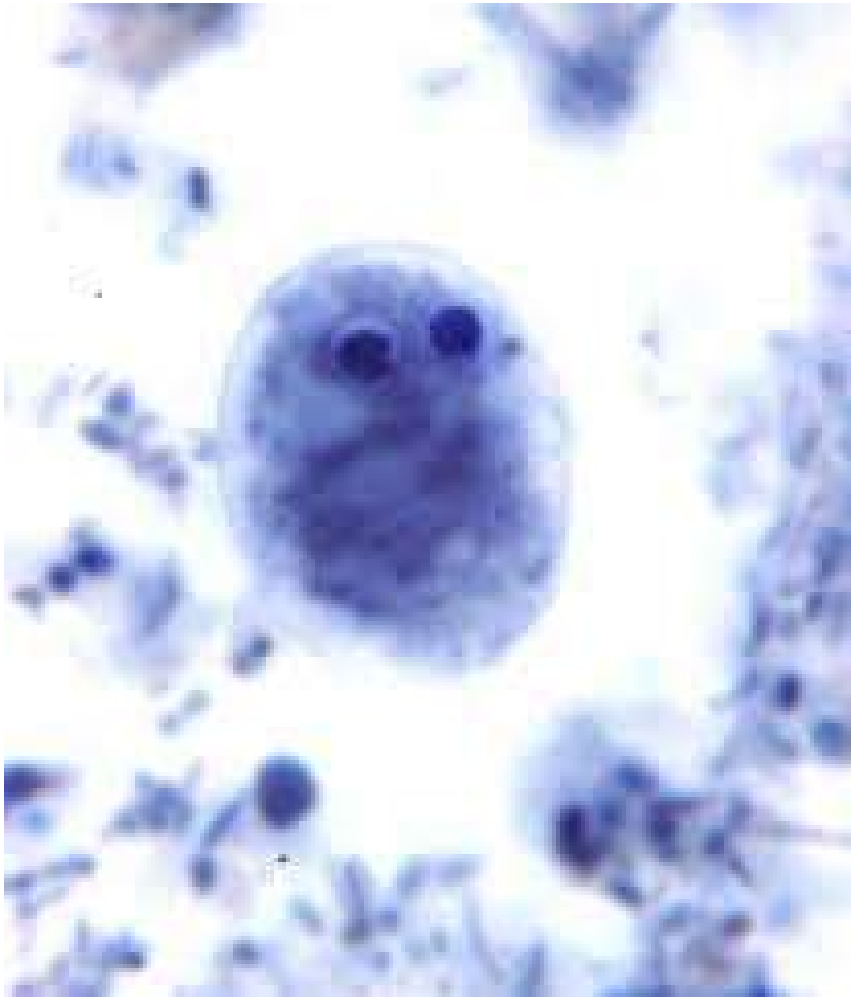
- The vesicular nucleus without chromatin granules on the nuclear membrane
- The central karyosome has **4-8** granules surrounded by a clear zone



Dientamoeba fragilis ~ eencellige parasiet, wordt bij 33% van de mensen met PDS klachten aangetroffen.

Life cycle:

- Man is the principle host
- Since cysts are unknown, the completion of its life cycle appears to be dependent on the transmission of the trophozoite from host to host.

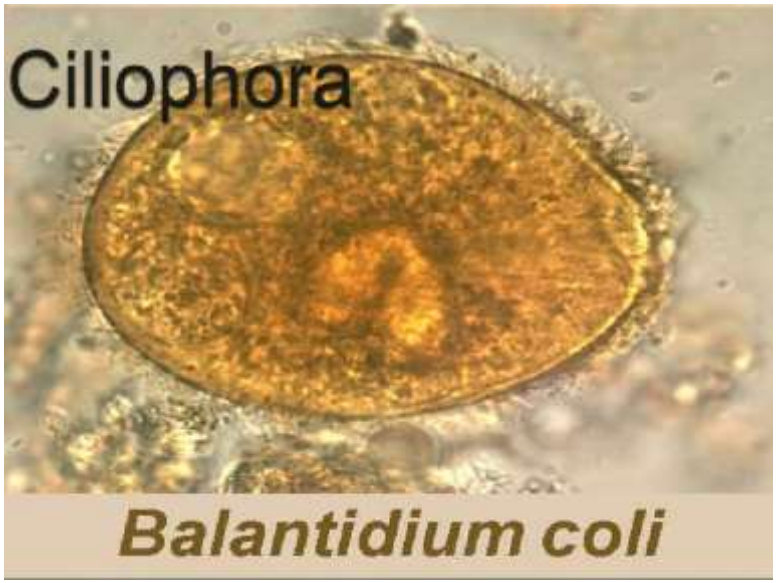


Pathogenicity

- It is a non pathogenic parasite of the large intestine
- **Diagnosis:**
- The trophozoite can be identified only in fresh liquid or soft stools

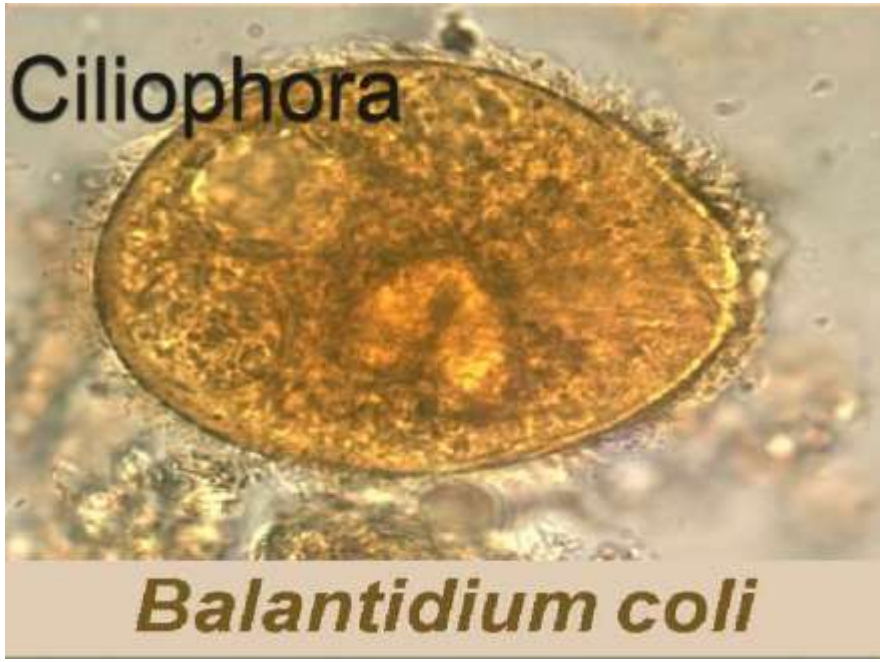
Sub phylum: ciliophora

- **Balantidium coli**
- Synonyms… paramecium coli
- Disease: balantidiasis, balantidiosis, balantidial dysentery
- Geographic distribution…cosmopolitan

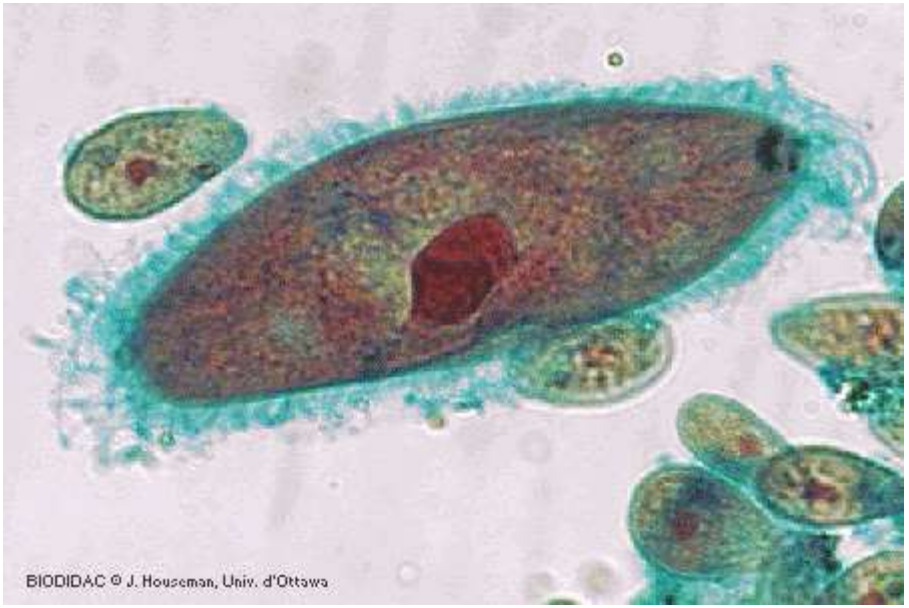


Morphology…

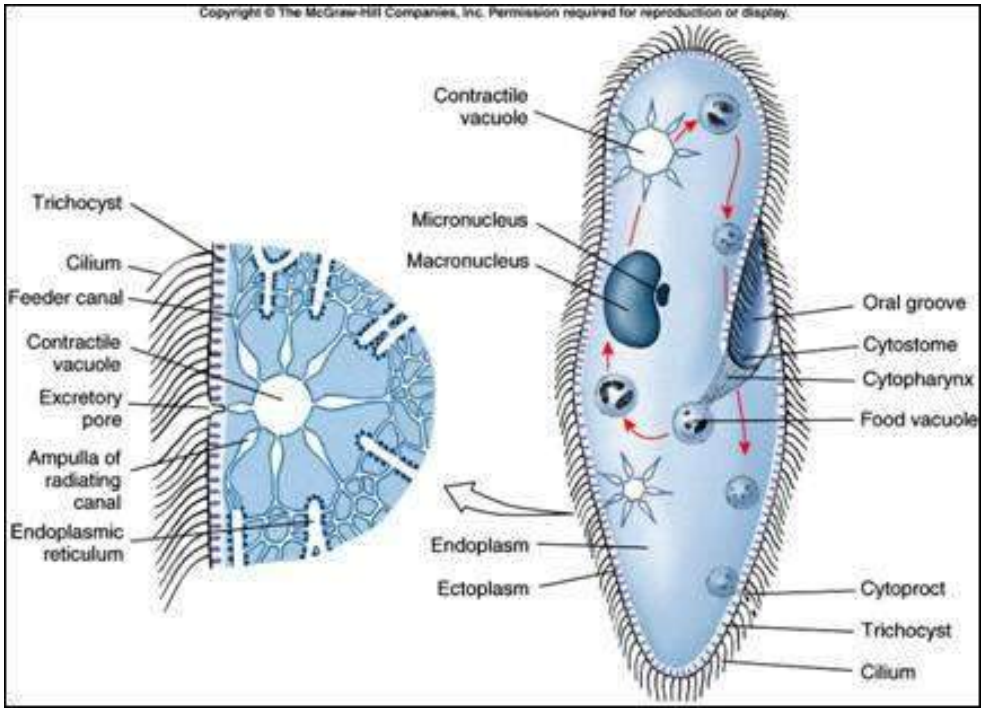
- **B.coli** is the largest intestinal protozoan in man
- The trophozoite usually measures from **50-70m** in breadth
- The grayish green un stained trophozoite is ovoid with a narrow anterior end and is shaped like **a sac (balantidium- little bag)**



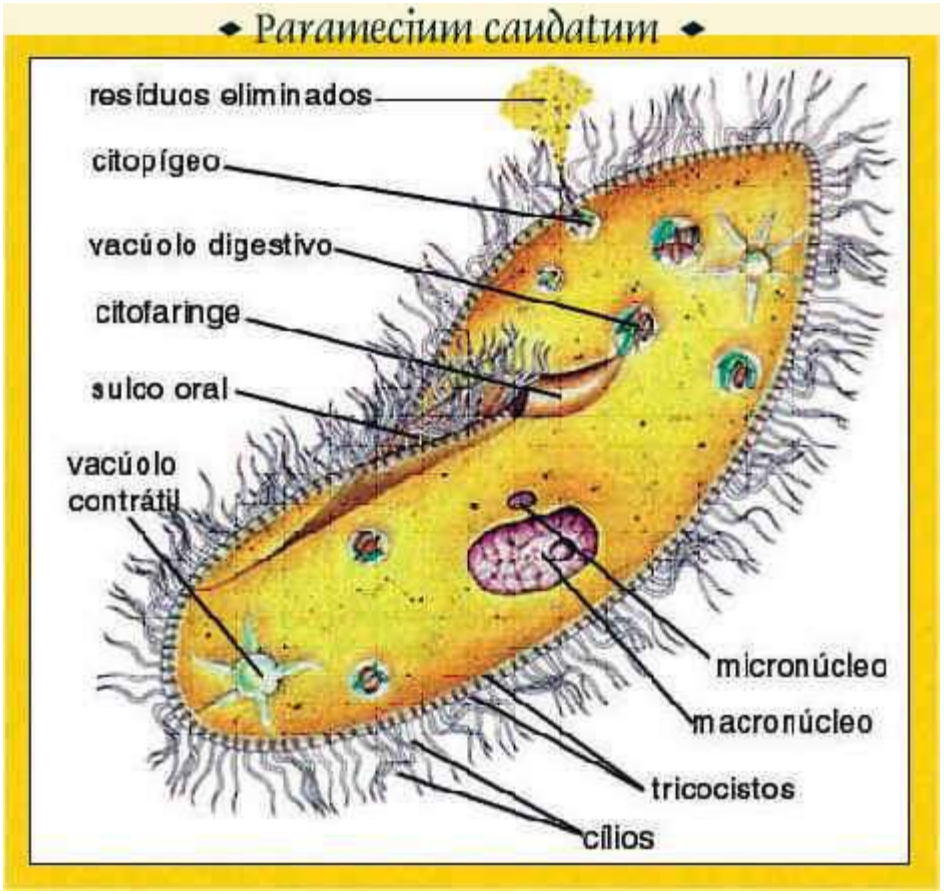
- The body is enclosed in a delicate protective pellicle beneath which is a narrow zone of ectoplasm
- The surface is covered by spiral longitudinal rows of cilia
- At the anterior end, is the narrow triangular peristome, which opens into the cytosome leading to the cytopharynx



- At the posterior end the cytopyge (excretory opening)
- Within the granular endoplasm are two contractile vacuoles, one near the anterior end and the other in the posterior third of the organism
- A large, elongated, kidney shaped macronucleus located obliquely near the middle, a small sub spherical micronucleus lying close to the concavity of the macronucleus



- Numerous vacuoles containing solid and partly digested food particles distributed throughout the endoplasm
- Cyst formation appears to be a protective rather than a reproductive function



- The encysted organism at first retains its cilia and revolves slowly within the double wall, but later the cilia degenerate and the animal becomes quiescent
- The average size of the cyst is about **55 by 52m**
- The macronucleus and contractile vacuole remain, but the food vacuoles and other structures disappear

Nutrition:

- **B.coli** consumes both tissues and intestinal contents. It ingests RBCs, leukocytes, bacteria, protozoa, yeast, starch, mucus.

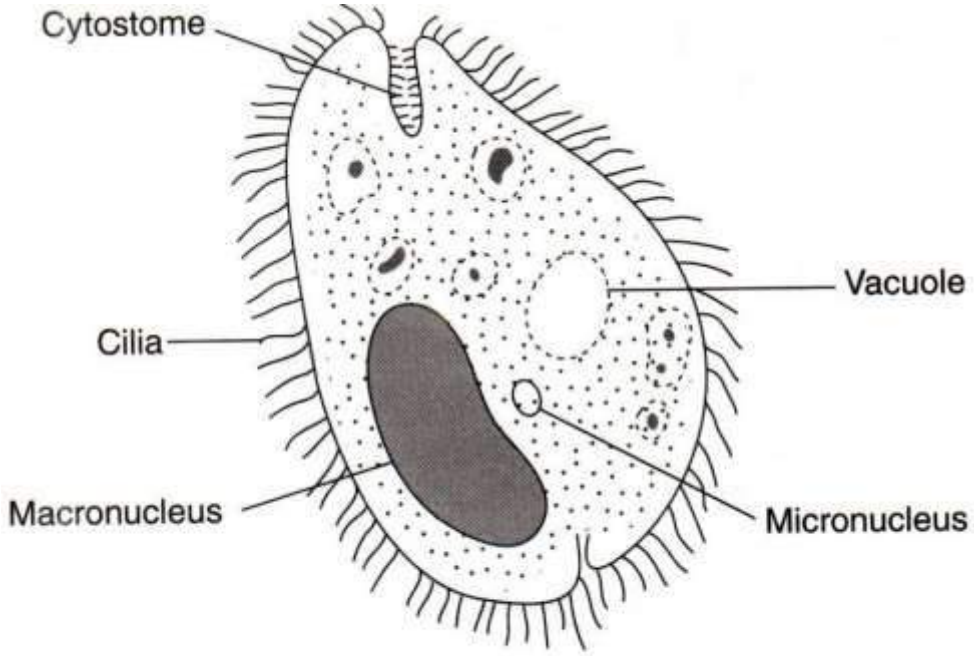
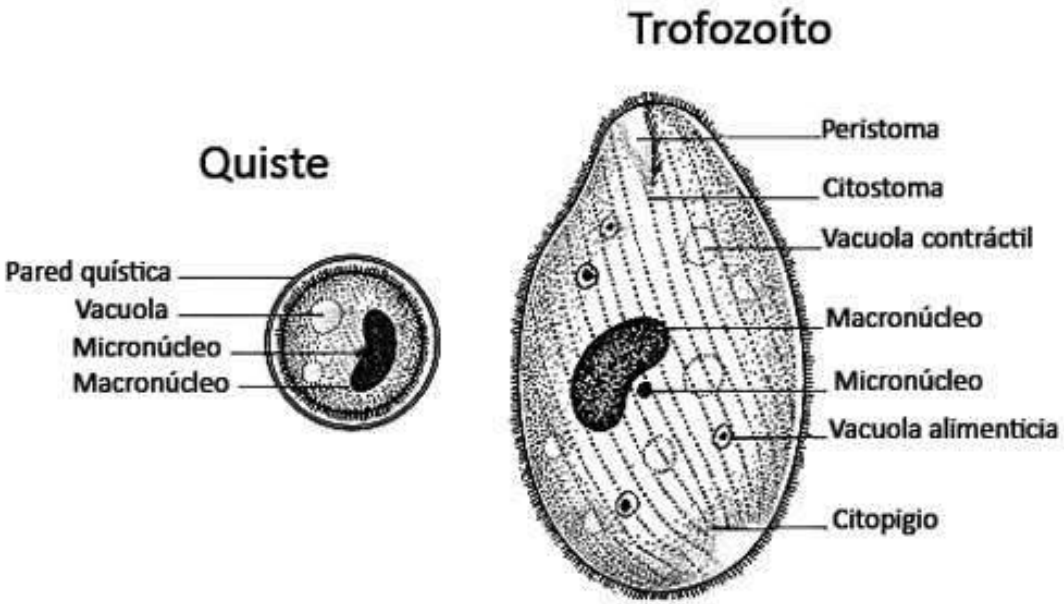


FIG 6–15 *Balantidium coli* trophozoite

Reproduction:

- The trophozoite reproduces by transverse binary fission
- Micronucleus divides mitotically, while the micronucleus undergoes mitotic division



Life cycle:

- Cysts are found in the large intestine and are found in greater numbers in rectum
- When ingested by a new host, the cyst wall dissolves, and the liberated trophozoite invades the intestinal wall to multiply in the tissues

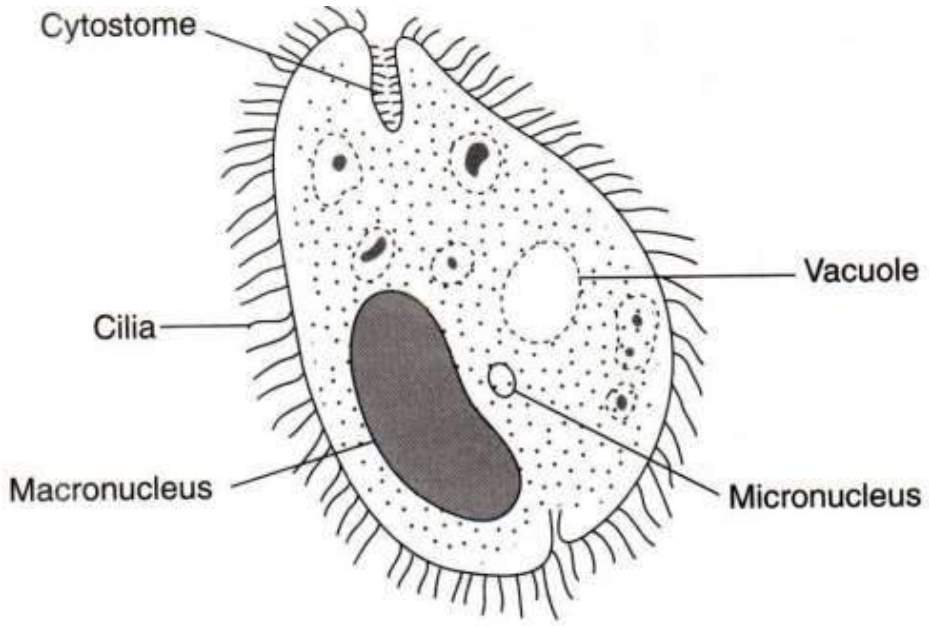


FIG 6–15 *Balantidium coli* trophozoite

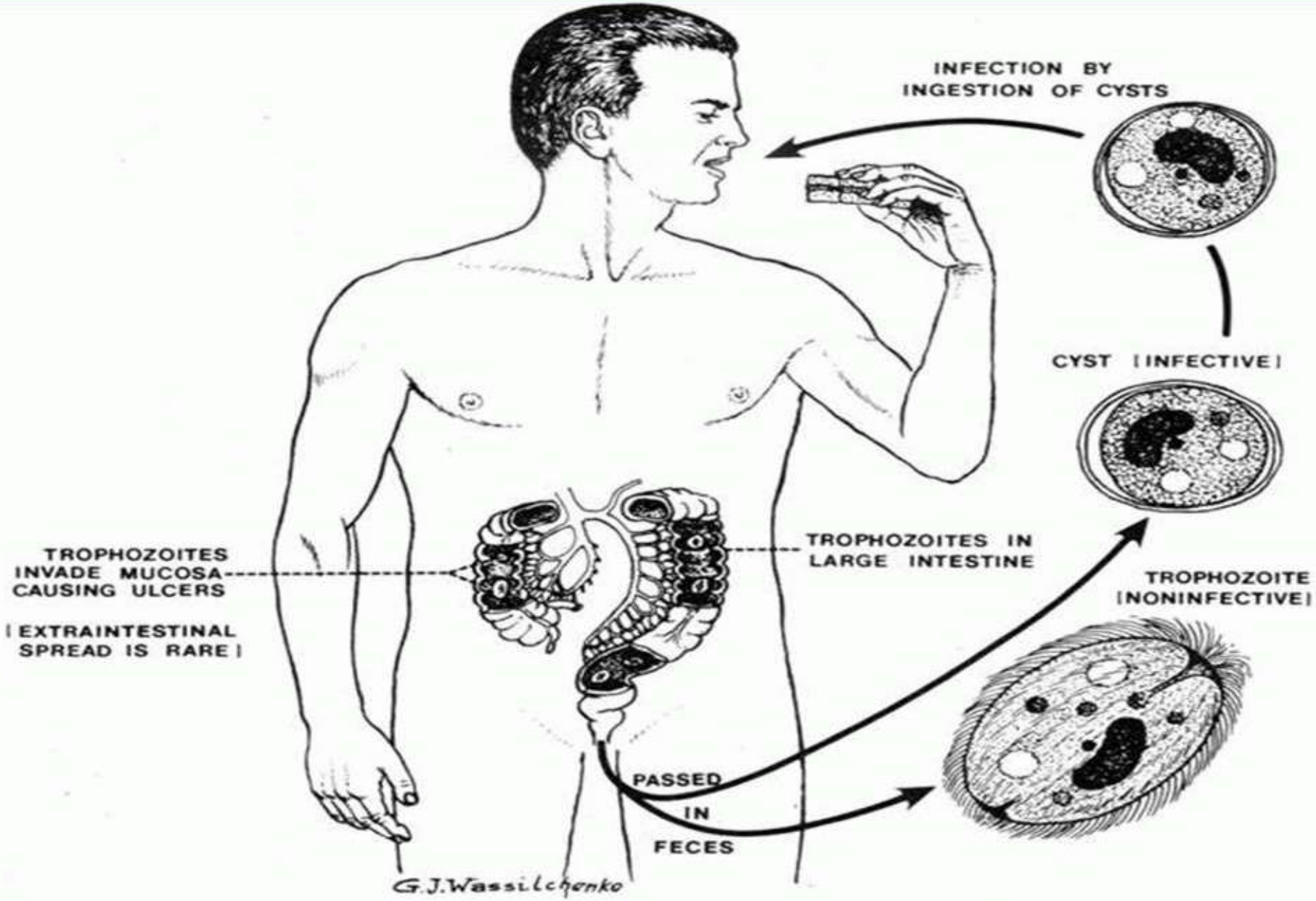
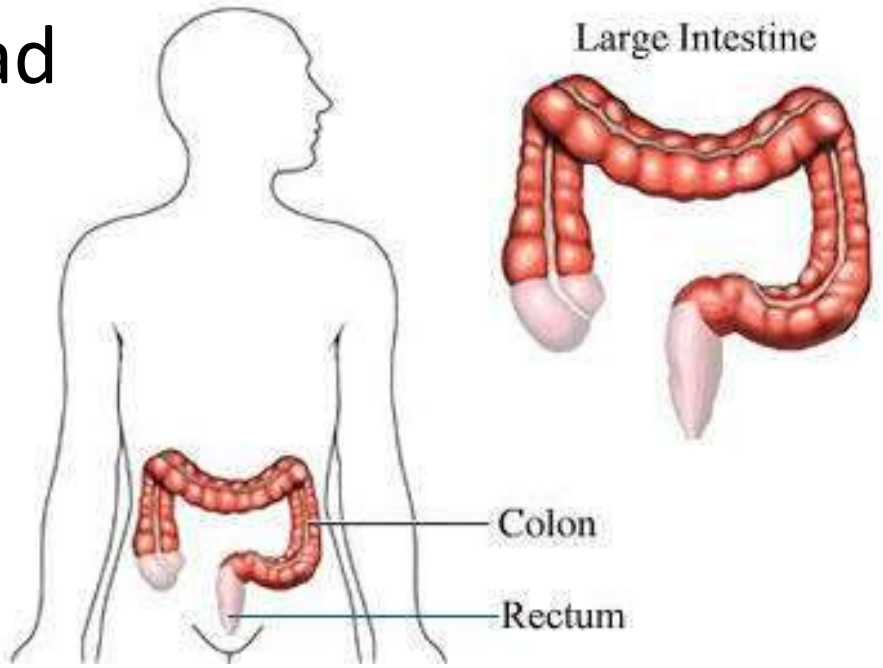


FIGURE 3–36

Life cycle of *Balantidium coli*.

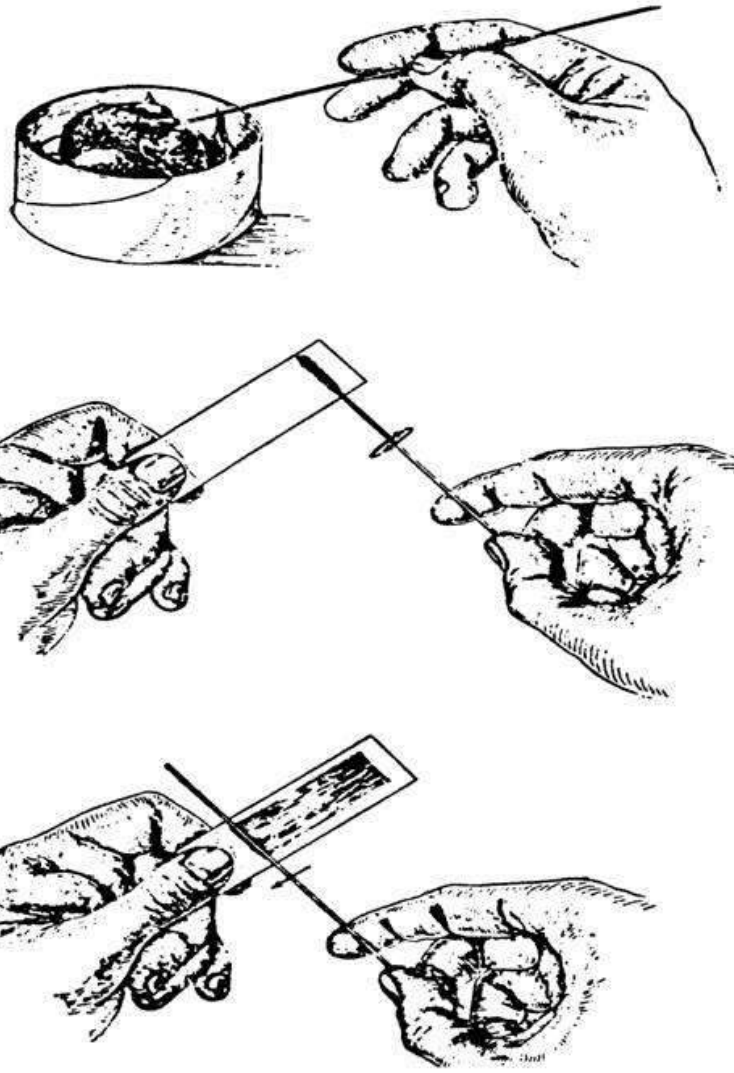
Pathogenicity:

- Problems occur in the ileum, colon, and rectum
- No extra-intestinal spread
- It causes dysentery
- Secondary infection is frequent
- Main complication is intestinal perforation



Diagnosis:

- Microscopic examination of diarrhea fresh specimen shown trophozoite active rotational movement
- Cysts are found in semi-formed and formed stool



Treatment:

- Tetracyclin and Humatin have been used

Parasitology

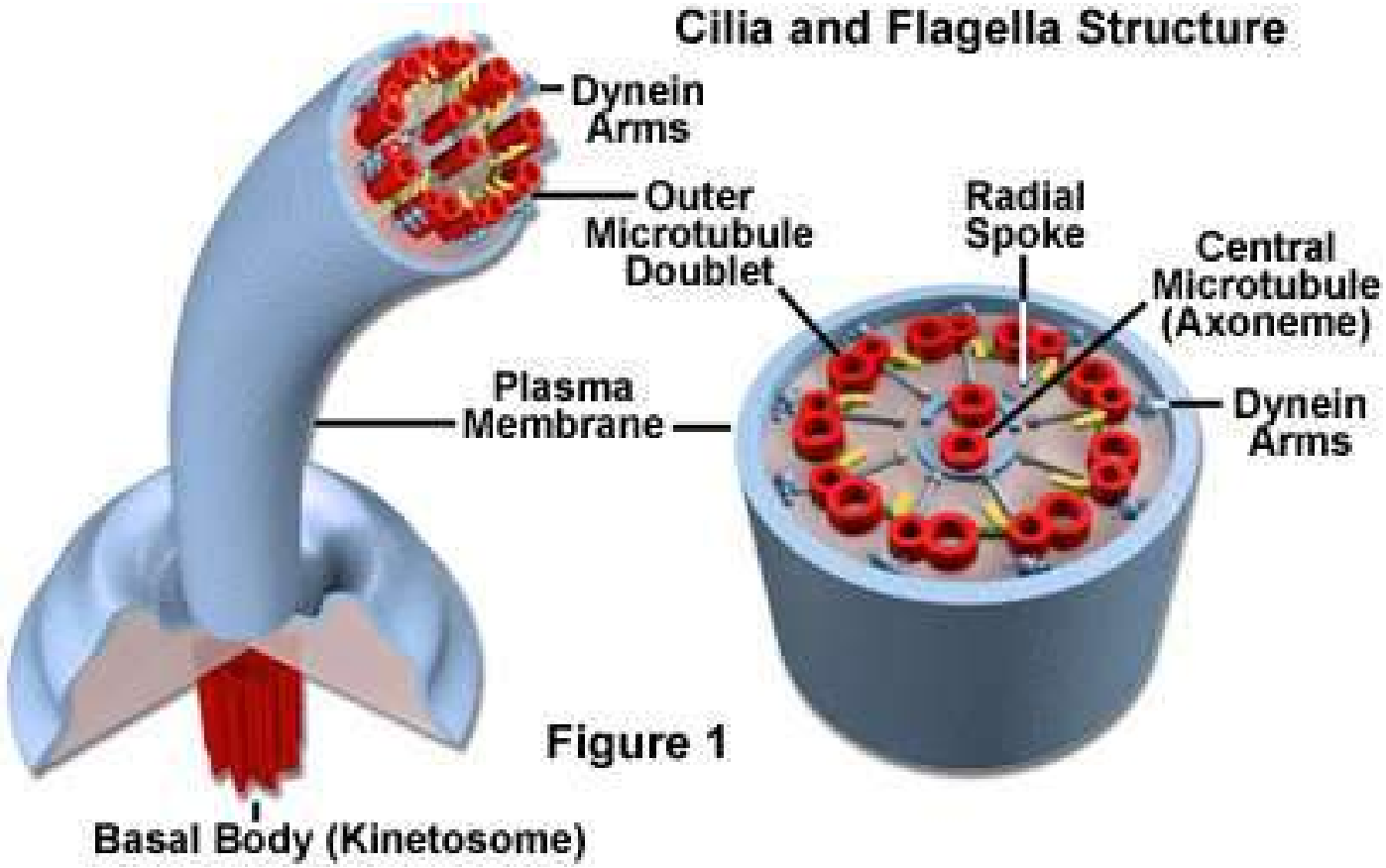


Figure 1

Lec.Dr.Ruwaidah F. Khaleel

Superclass: Mastigophora

Mastigophora (Flagellata) perform locomotion or movement by flagella . Flagella are extremely *fine* fibers having a central axoneme

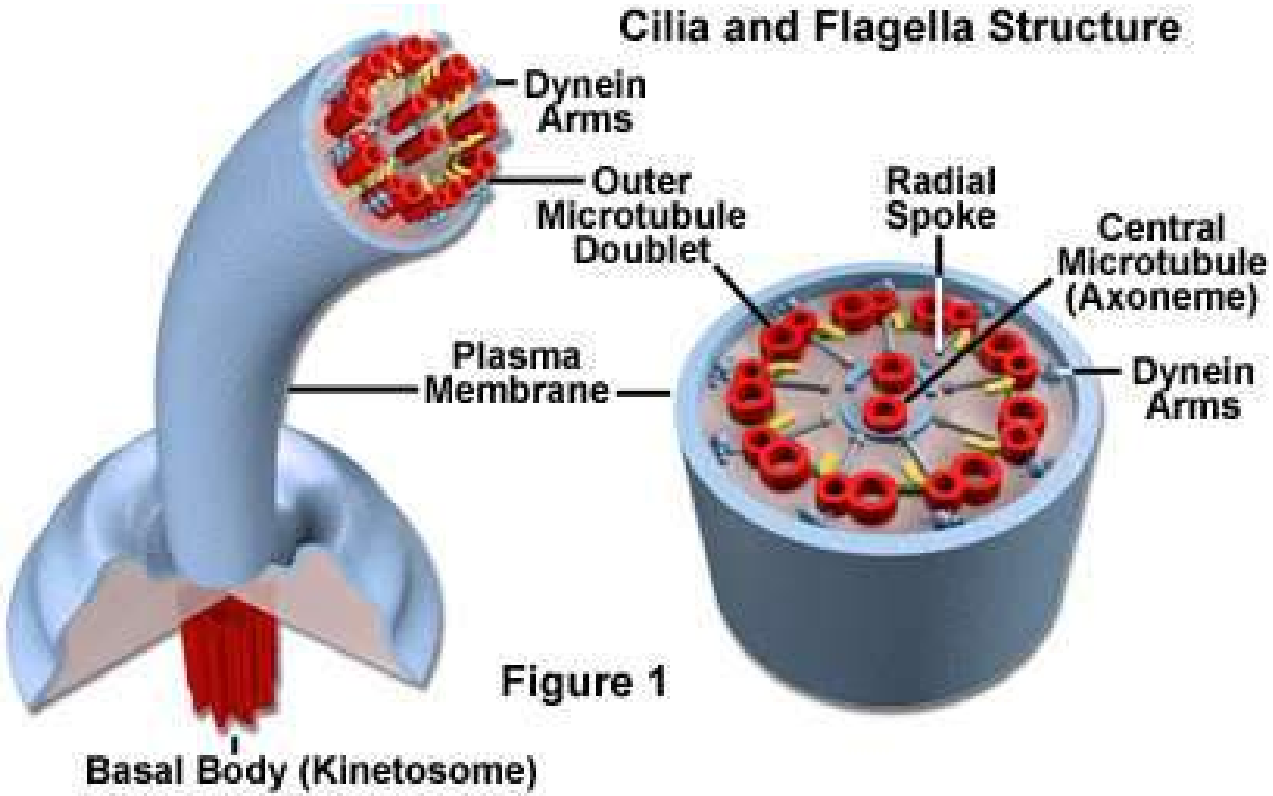
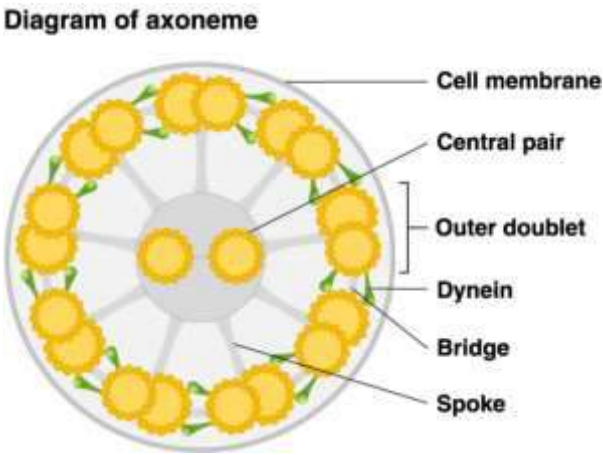
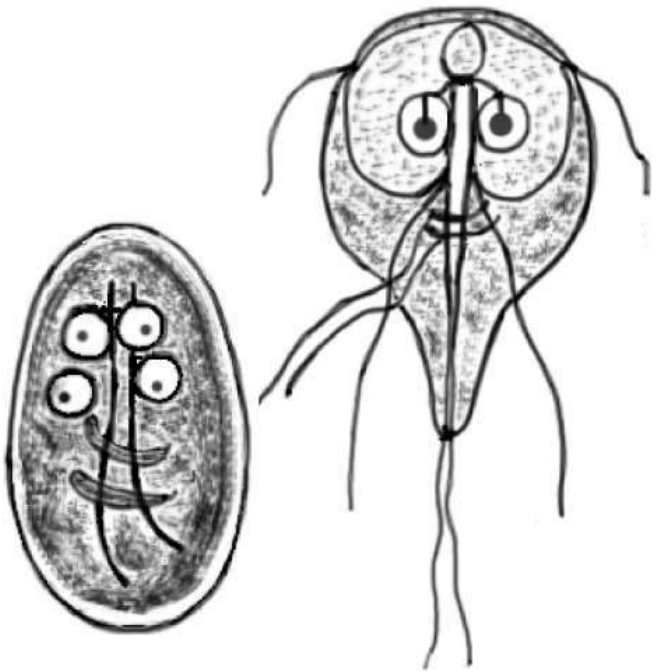


Figure 1



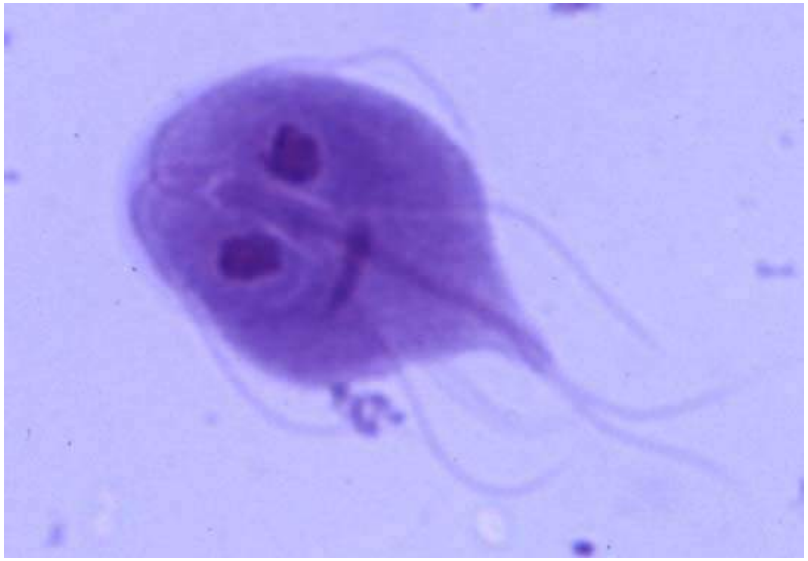
Intestinal Flagellata
Giardia lamblia

- ***Giardia lamblia*** (synonymous with ***Lamblia intestinalis*** and ***Giardia duodenalis)*** is a flagellated protozoan parasite that colonizes, and reproduces ***in*** the small intestine, causing **giardiasis.**
- The ***Giardia*** parasite attaches to the epithelium by a **ventral adhesive disc,** and reproduces via binary fission. Giardiasis does not spread via the bloodstream, nor does it spread to other parts of the gastro-intestinal tract, but remains confined to the lumen of the small intestine.

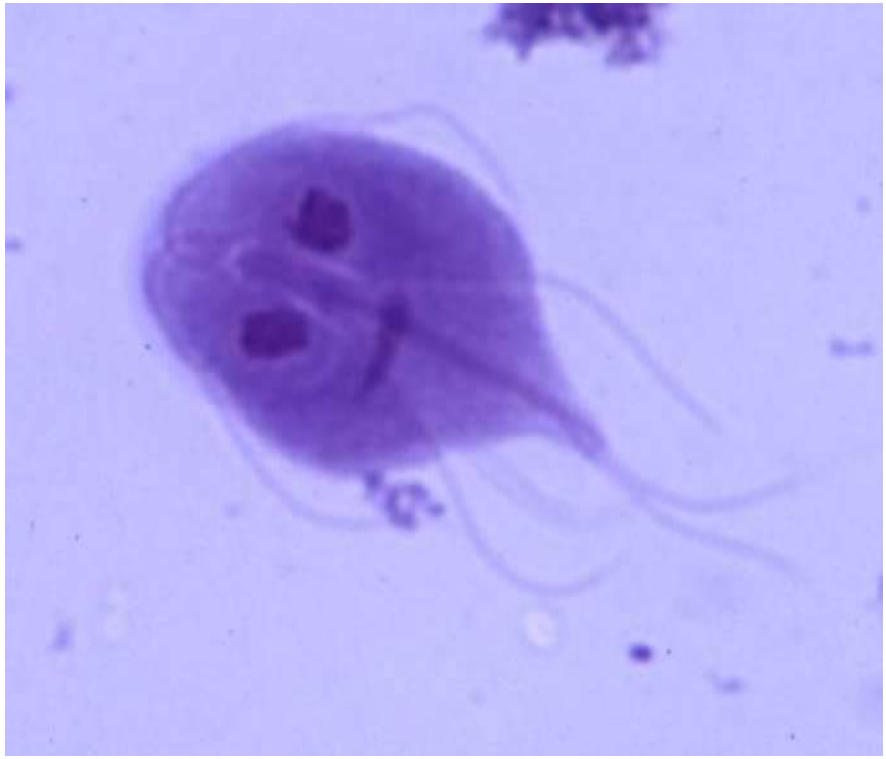


Chief pathways of human infection include:

- ingestion of untreated sewage, a phenomenon particularly common in many developing countries.
- Contamination of natural waters also occurs in watersheds where intensive grazing occurs

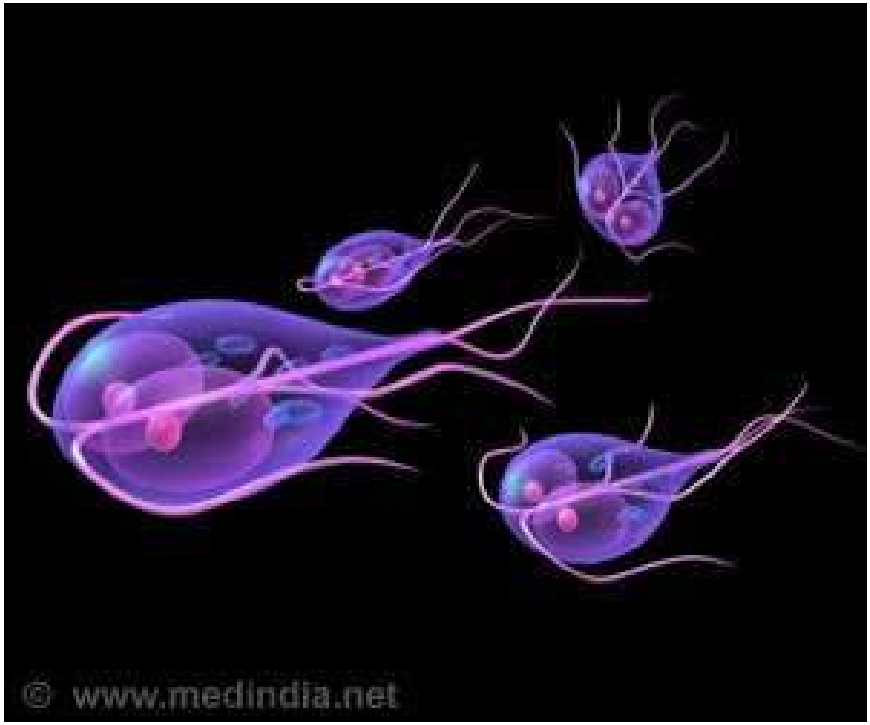


- Infects humans, but is also one of the most common parasites infecting cats, dogs and birds.
- Mammalian hosts also include cows, beavers, deer, and sheep.



Giardiasis

- **Giardiasis or beaver fever** in humans is a diarrheal infection of the small intestine .
- ***Giardia*** has a wide range of mammalian hosts besides humans, thus making it very difficult to eradicate. For people with **compromised immune systems, such as elderly or AIDS patients, giardiasis can be deadly.**



Transmission

Giardiasis is caused by the ingestion of infective cysts.

1. Person-to-person transmission
2. Water-borne transmission

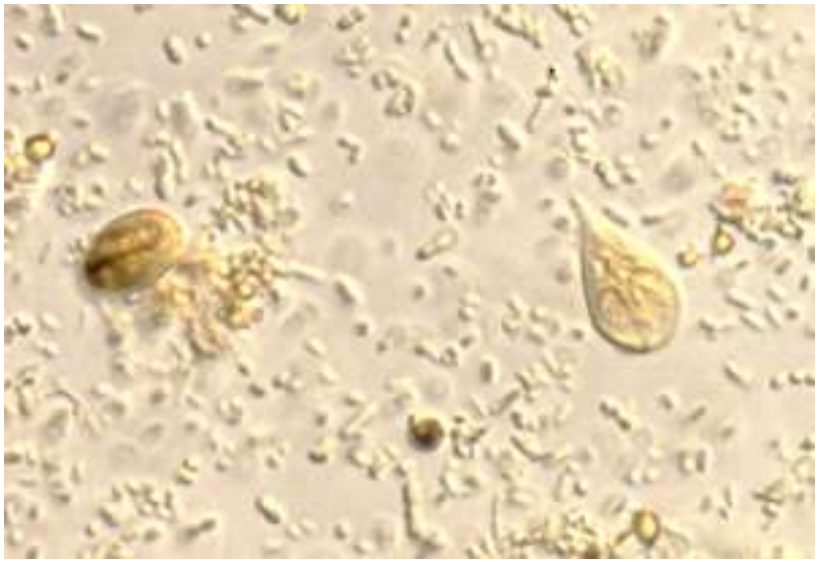
3. Venereal transmission happens through fecal-oral contamination.
4. Lastly, food-borne epidemics of Giardia have developed through the contamination of food by infected food-handlers.

- Giardiasis is spread during periods of heavy rains, such as monsoon runoff.

Morphology

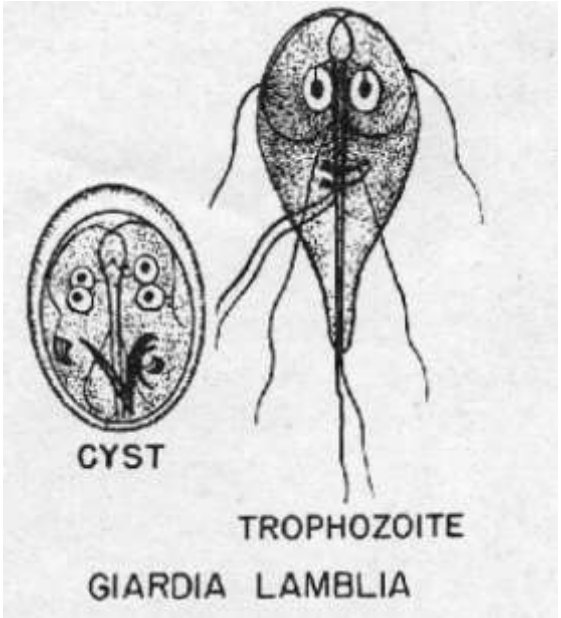
1) Cysts

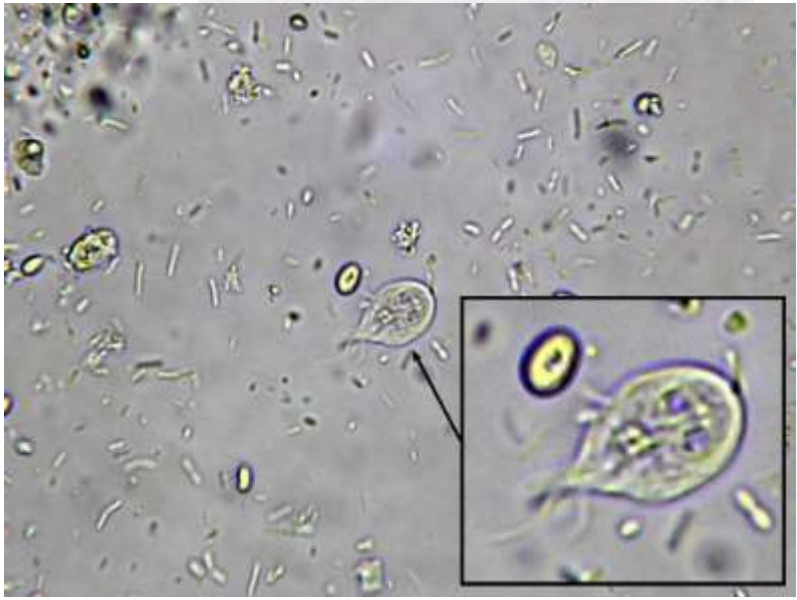
- The cysts are non-motile and egg-shaped.
- Newly formed cysts contain two genetically identical nuclei. However, each organelle duplicates so that in permanently stained mature cysts, **four prominent nuclei** and four median bodies are observed.
- The cysts are the **infective form** of the parasite and each cyst gives rise to two trophozoites.



Trophozoite

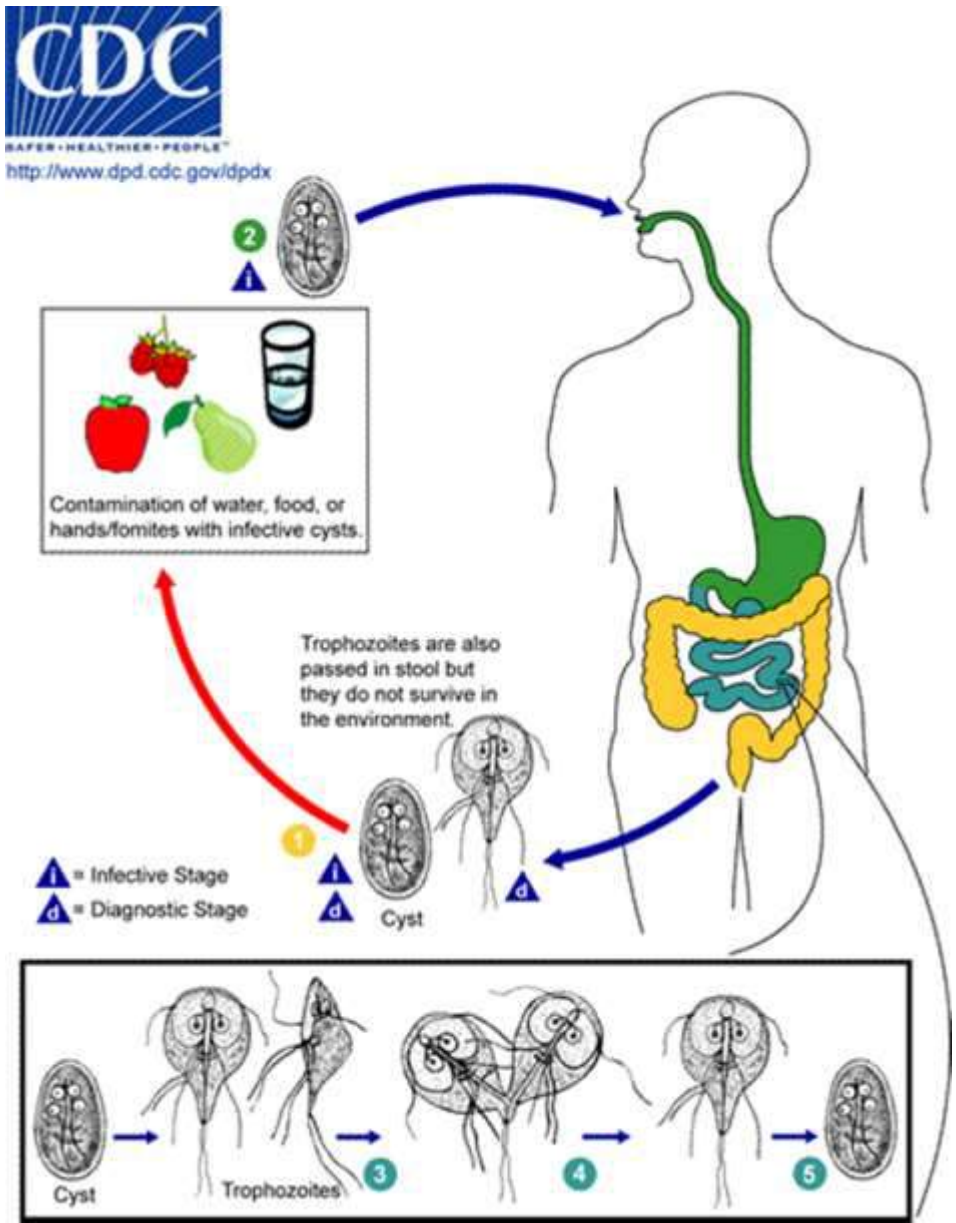
- Under a normal compound light microscope
  Giardia often looks like a **"clown face,"** with two nuclei outlined by adhesive discs
- There are 4 pairs of flagella, one anterior pair, two posterior pairs and a caudal pair.
- The parasites swim free and rapidly in a spiral motion in the intestinal lumen



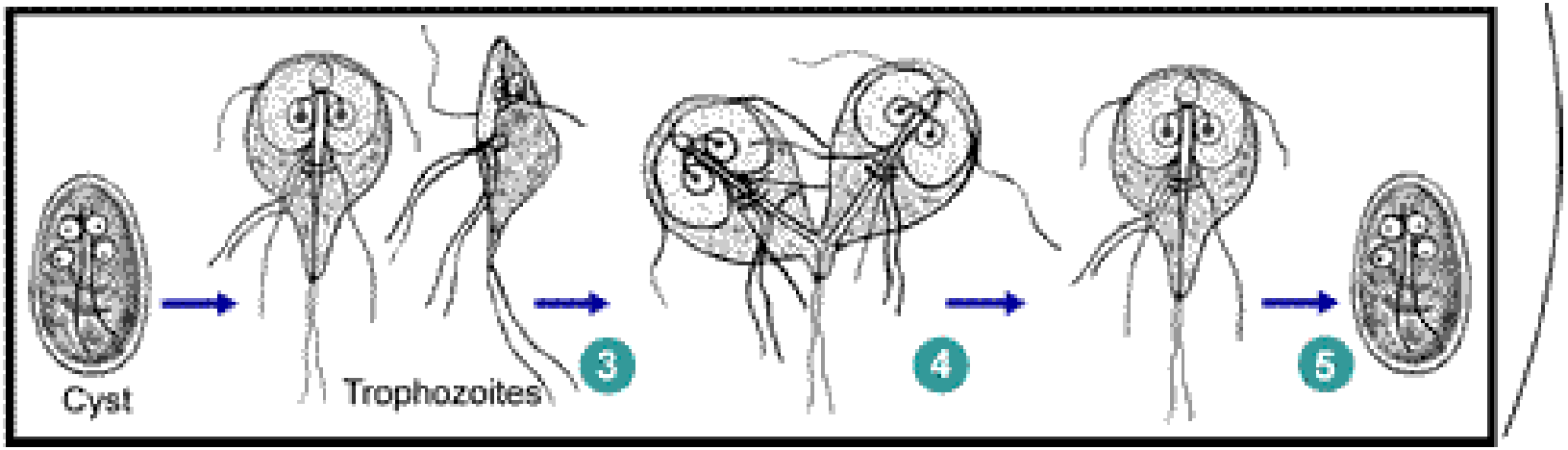


Life cycle

- The life cycle of *Giardia* alternates between the cyst and the trophozoite forms, and both forms are found in feces.
- Cysts are more often found in non-diarrheal feces, and they are the infectious stage of the parasite.
- The cysts are hardy and resistant to standard concentrations of chlorine used in water treatment and they can persist for several months in cold, moist environment.

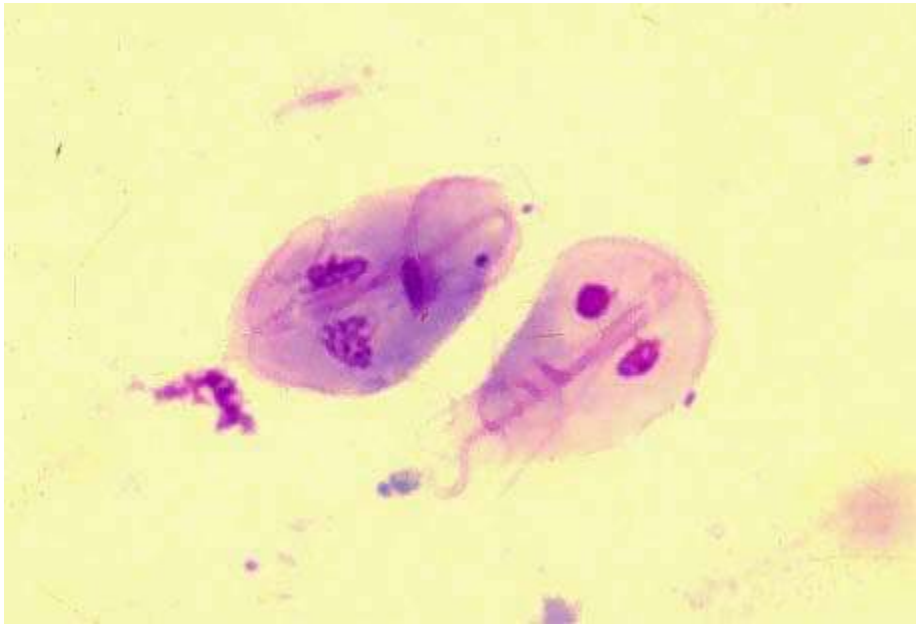


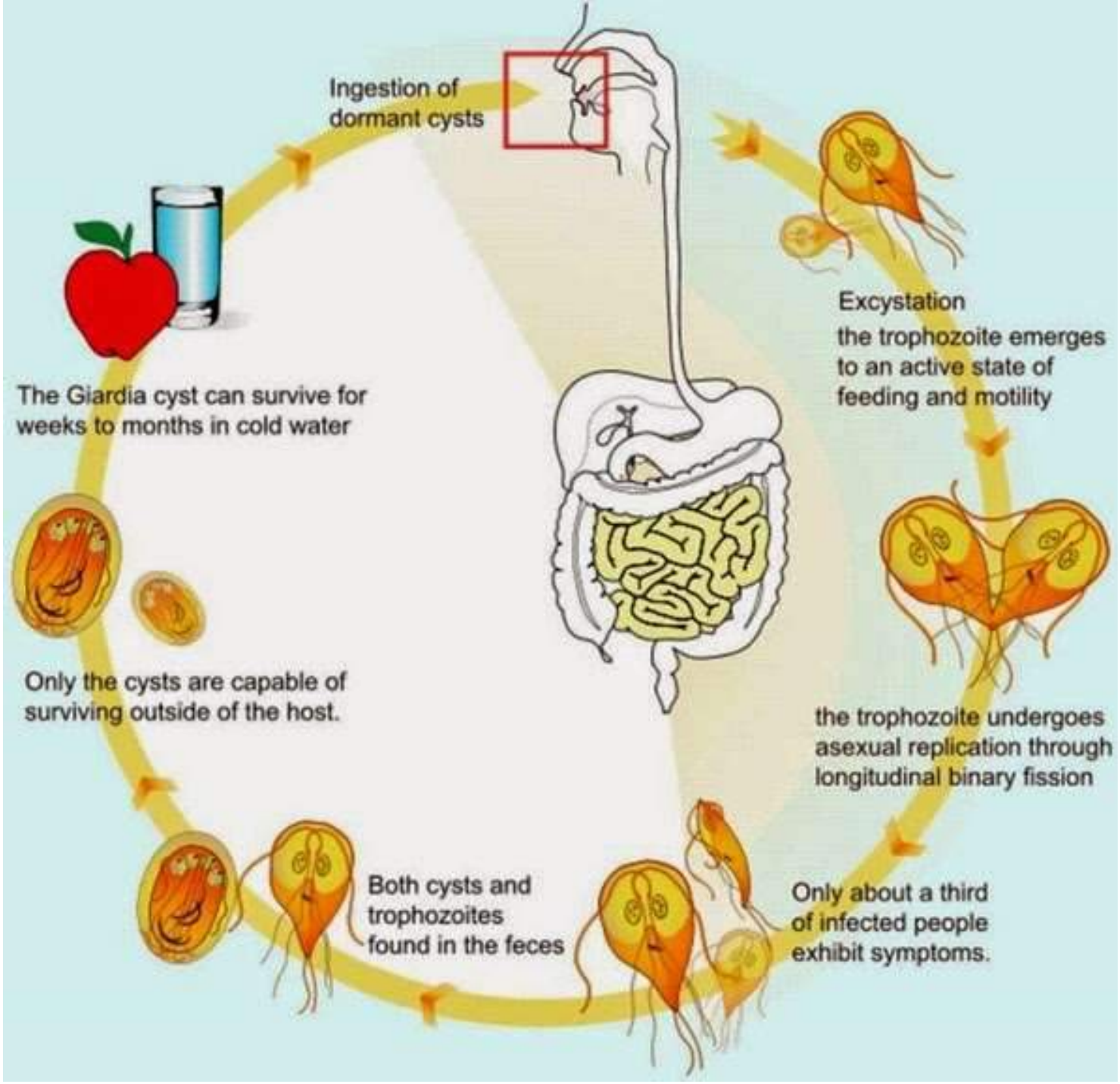
1. Mature cysts are able to survive the acidic environment of the stomach and migrate to the small intestine of the host. Exposure to stomach acid triggers a process called **excystation,** during which trophozoites are released from cysts-.Each quadrinuclear cyst gives rise to two binuclear trophozoites"
2. As trophozoites migrate toward the large intestine, they retreat into the cyst form in a process called **encystations.** Bile salts and intestinal mucous were found to enhance trophozoite multiplication and encystations.



Notes

- **Trophozoites** if excreted in feces, cannot survive long in the environment and are therefore noninfectious.
- **The cysts** in excrements will quickly become infectious and will begin a new cycle of infection if ingested by a naive host.




Ingestion of
dormant cysts
Excystation
the trophozoite emerges
to an active state of
feeding and motility
the trophozoite undergoes
asexual replication through
longitudinal binary fission
Only about a third
of infected people
exhibit symptoms.
Both cysts and
trophozoites
found in the feces
Only the cysts are capable of
surviving outside of the host.
The Giardia cyst can survive for
weeks to months in cold water

Symptoms

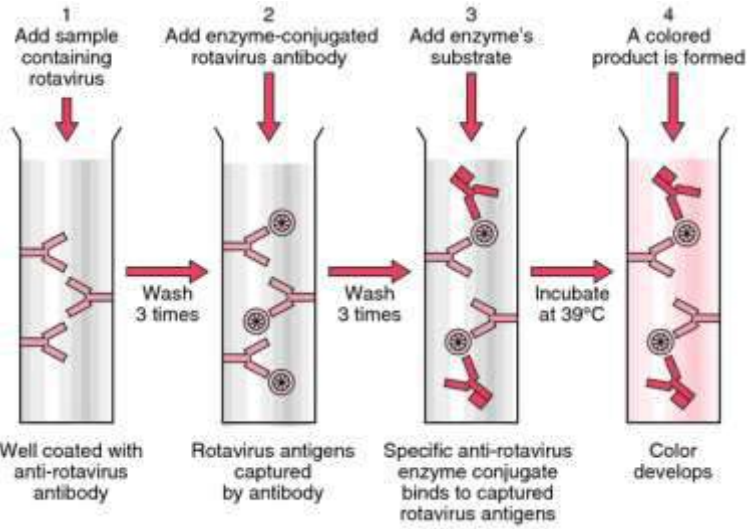
A range of clinical syndromes may occur, with gastrointestinal syndromes being the most prevalent.

- Abdominal cramps,
- Watery diarrhea,
- Vomiting,
- Foul flatus.
- Fever may last for 3-4 days.
- Stools become greasy and malodorous but do not contain blood or pus because giardiasis does not involve dysenteric symptoms. Watery diarrhea may cycle with soft stools and constipation.

- Due to long term effect of inflammation and scarring occurring commonly with giardiasis, possible effects including **weight loss, malnutrition, failure to thrive, lactose intolerance,**
- Chronic inflammation of intestinal lining leading to various forms of **cancer.** For immunocompromised individuals, these effects can be and often are **deadly.**

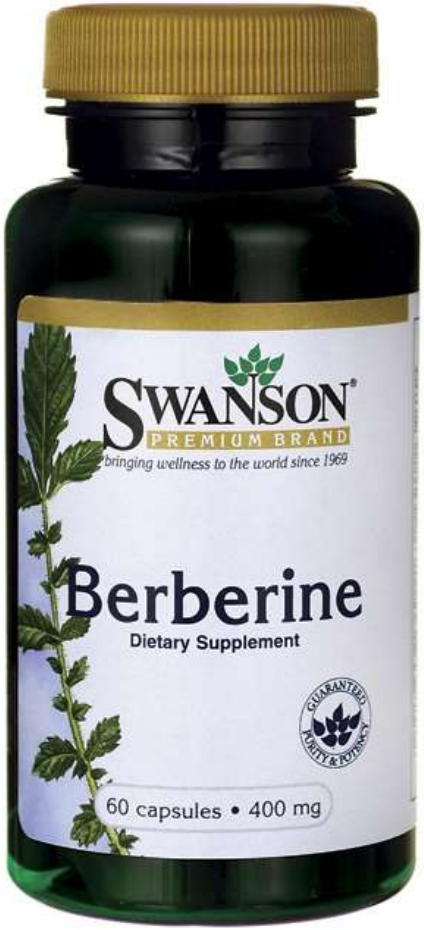
Diagnosis

- Accurate diagnosis requires an **antigen test** or, if that is unavailable,
- An ova and parasite **examination of stool.**
- Multiple stool examinations are recommended,
- Treating based on symptoms.



Treatment

- Human infection is conventionally treated with **metronidazole, tinidazole or nitazoxanide.** One of file most common alternative treatments is **berberine sulfate** (found in Oregon grape root, goldenseal, yellow root, and various other plants.



Trichomonas

- ***Trichomonas*** is a genus of an anaerobic, flagellated protozoan, that are parasites of vertebrates

Species of *Trichomonas* found in human include:

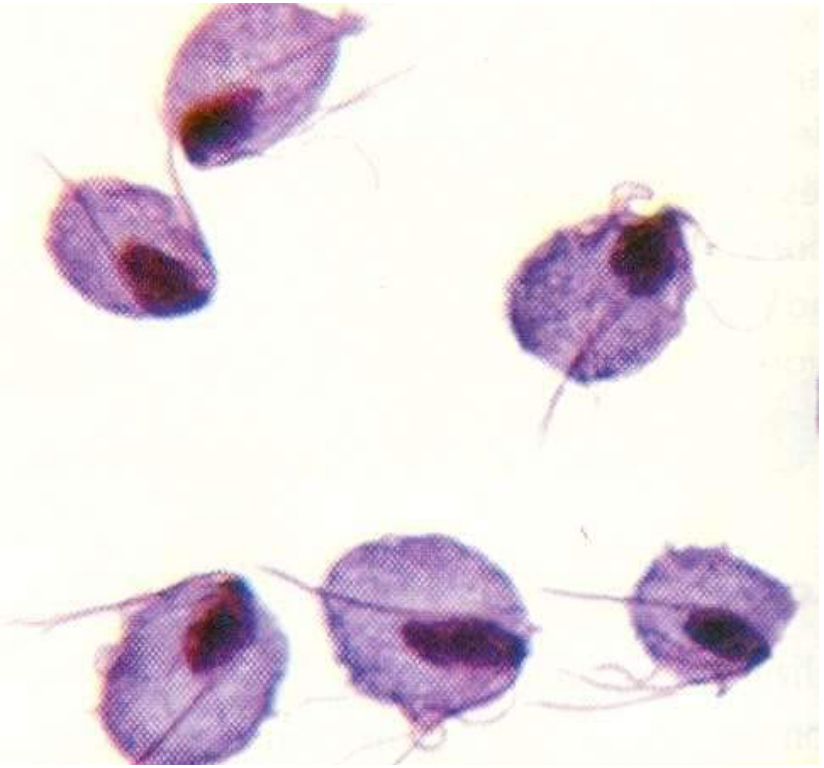
1. Harmful protozoa.

Trichomonas tenax in mouth.

Trichomonas hominis in large intestine.

2. Pathogenic protozoa.

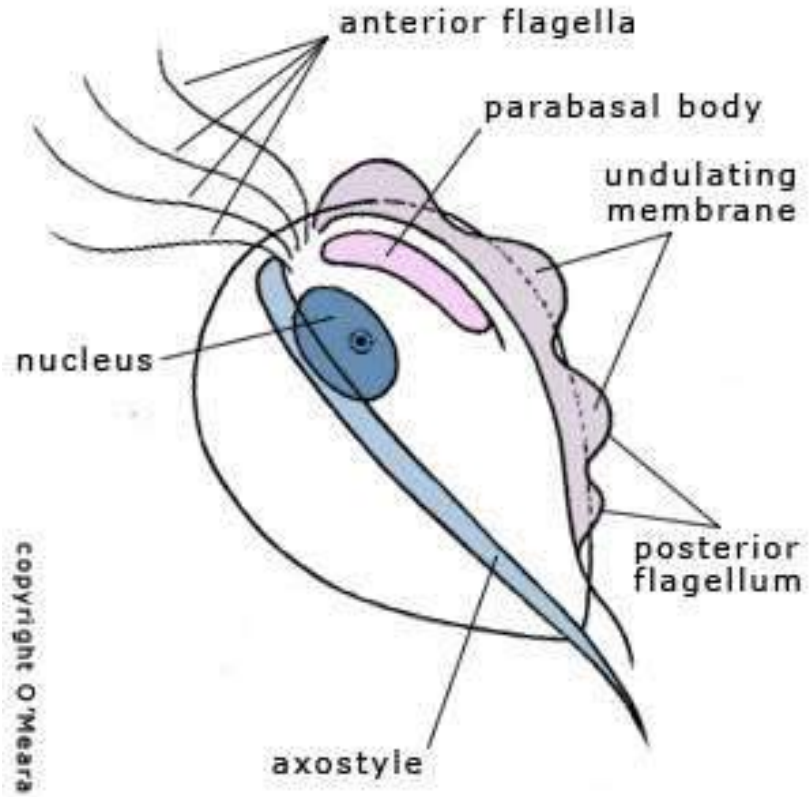
Trichomonas vaginalis in human reproductive tract.

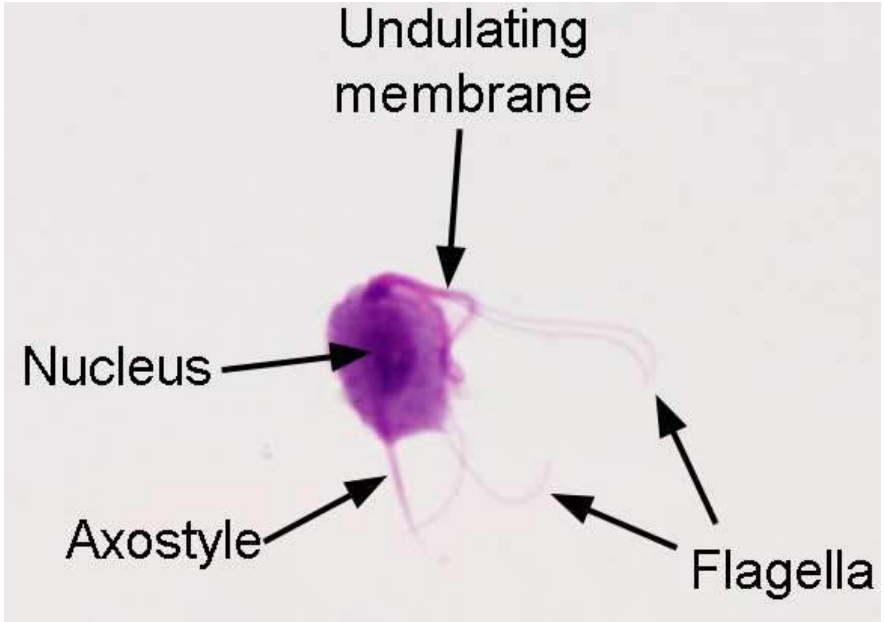


Trophozoite

- Is oval as well as flagellated, or "pear" shaped as seen on wet-mount slide.
- Five flagella arise near the cytostome; four of these immediately extend outside the cell together, while the fifth flagellum wraps backwards along the surface of the organism.

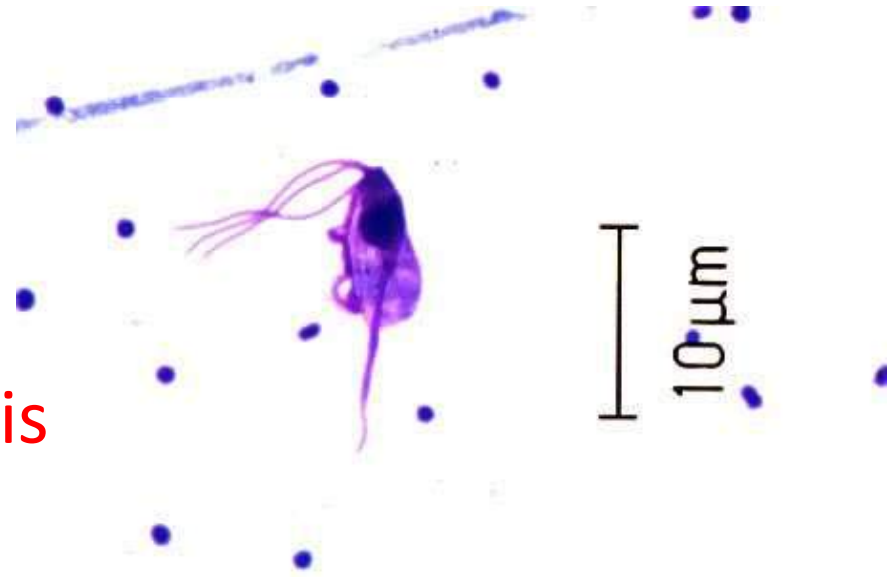
- **Axostyle** projects opposite the four-flagella bundle; the axostyle may be used for attachment to surfaces and may also cause the tissue damage noted in **trichomoniasis infections.**



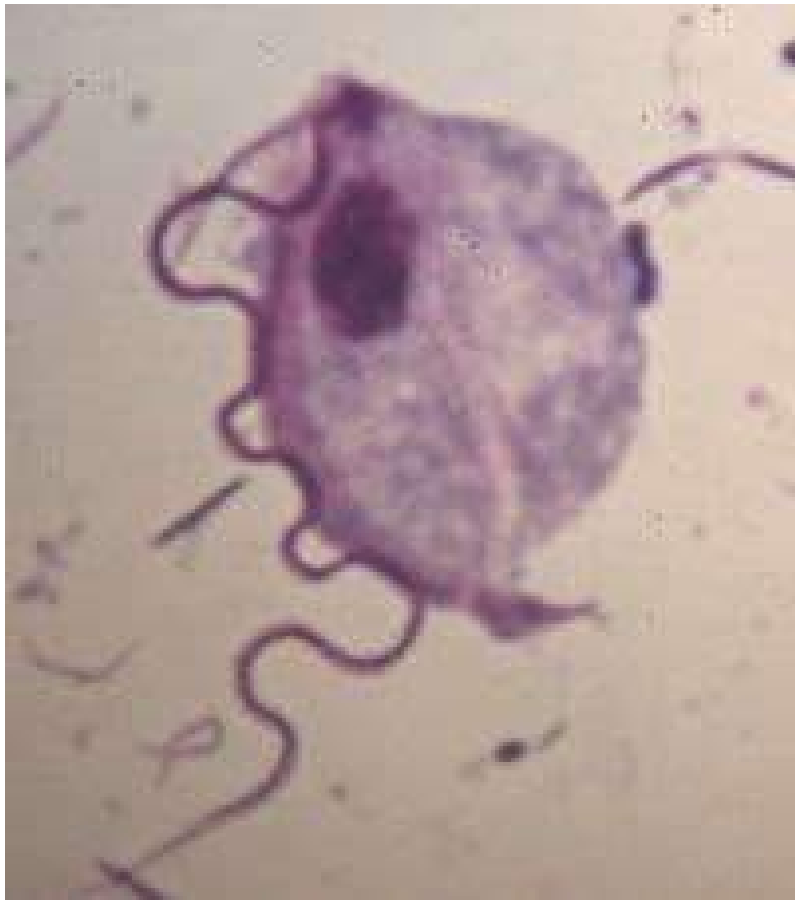


❖First: *Trichomonas tenax*

- A protozoan that lives as a **commensal in the mouth**. It is usually found around tartar in the teeth or in defects of carious teeth.
- *Trichomonas tenax* is a protozoan flagellate which is a parasite of the **mouth, tonsils** and, **lungs** of many vertebrates, including Homo sapiens



- *Trichomonas* from the mouth will not survive in the digestive tract, and vice-versa.
- The same is true relative to the mouth trichomonad, *Trichomonas tenax* in relation to *Trichomonas vaginalis.* The latter is a parasite of male and female reproductive tracts.



- Although these trichomonads have a very similar appearance under the microscope, the two species may be distinguished since ***Trichomonas vaginalis*** is 2-3 times larger than ***Trichomonas tenax***. The latter is normally 6-10 microns in length as against the 15 to 30 micron size attained by ***Trichomonas vaginalis***. Surprisingly Trichomonads are found in the mouths of household cats.
- Strangely enough these cats are infected with the same species of ***Trichomonas*** as humans. There is the chance that both the owner and the pet may share the same germs.

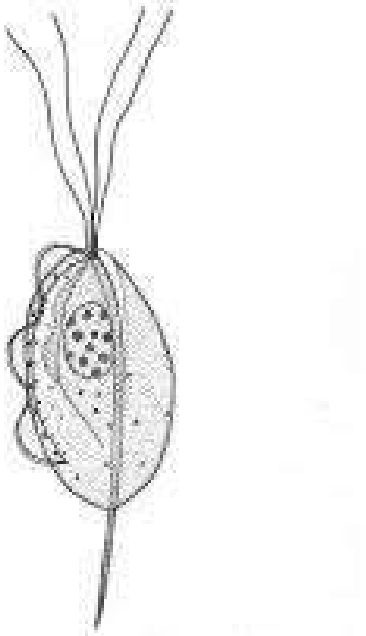


Fig. 9-3.. *Trichomonas tenax.* (Unos 1.600 aumentos.) (Según Wenrich, Am. Jour. Trop. Med.; por cortesía de Williams and Wilkins Co.)

Second: *Trichomonas hominis*

- It has a relatively wide host range and is generally a harmless **commensal found in the caecum and colon of man,** other primates, dogs and cats.
- increased prevalence is usually directly associated with poor hygiene since the parasite is transmitted by the oral-fecal route via contaminated food, water and flies etc.

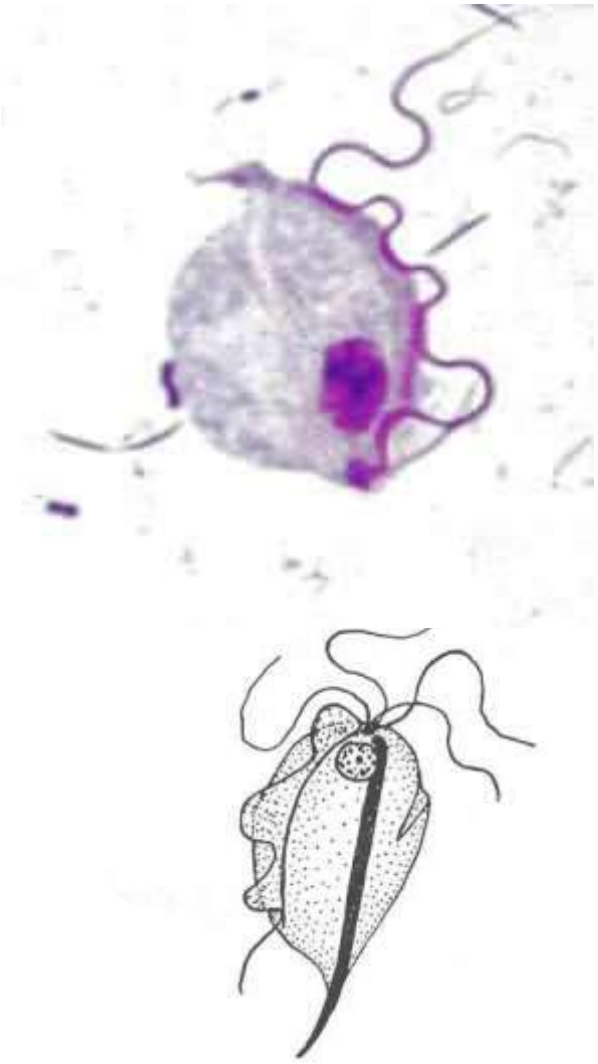
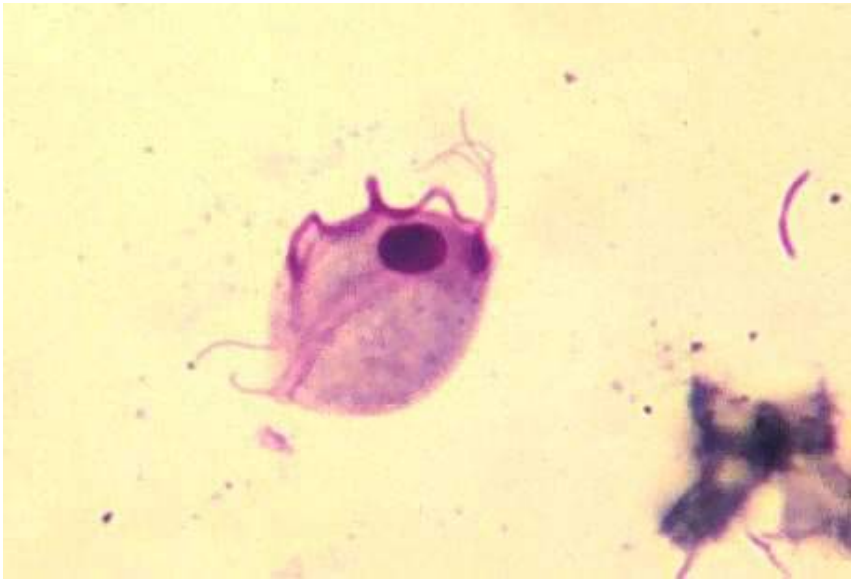


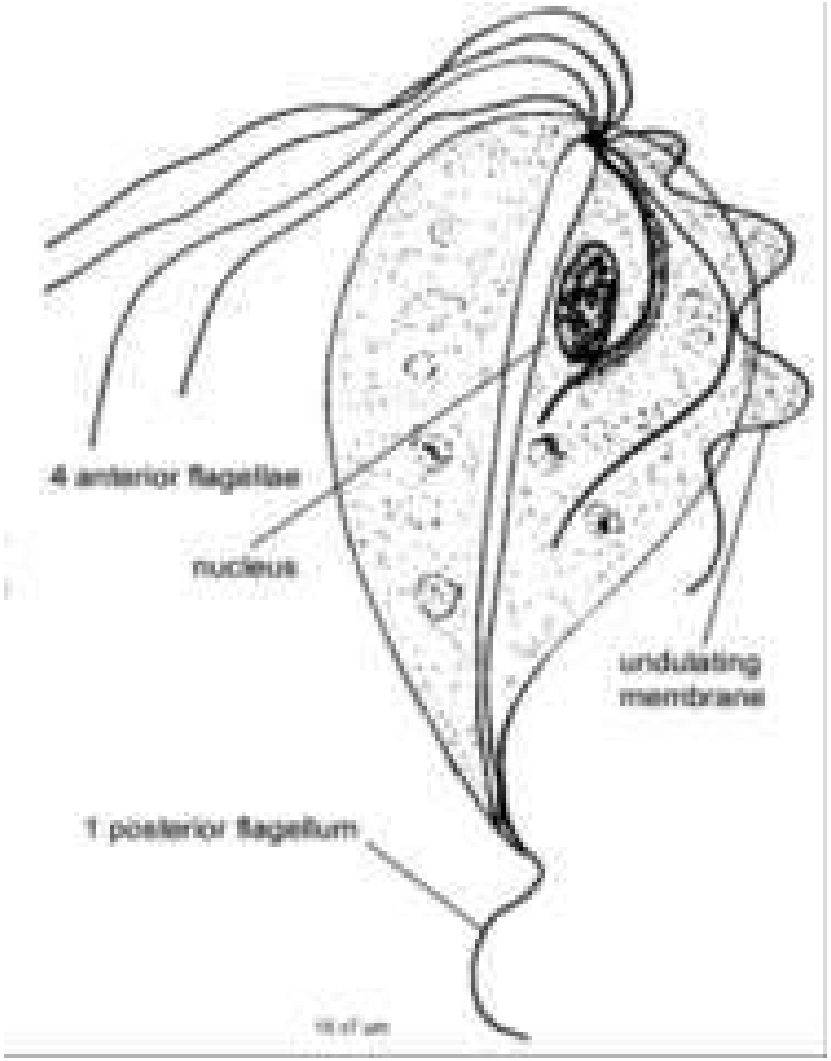
Fig. 9-6. *Trichomonas hominis* en materias fecales diarreicas. (1.600 aumentos.) (Original de Faust.)

- Infections with ***T. hominis*** are easily distinguished from the other two species since there is a strict habitat restriction and this species will not survive in either the oral cavity or the genitourinary tract. Although not proved to be pathogenic infections are often associated with other protozoal gut parasites such as ***Entamoeba histolytica*** but their presence is probably coincidental and secondary to the primary pathogen.

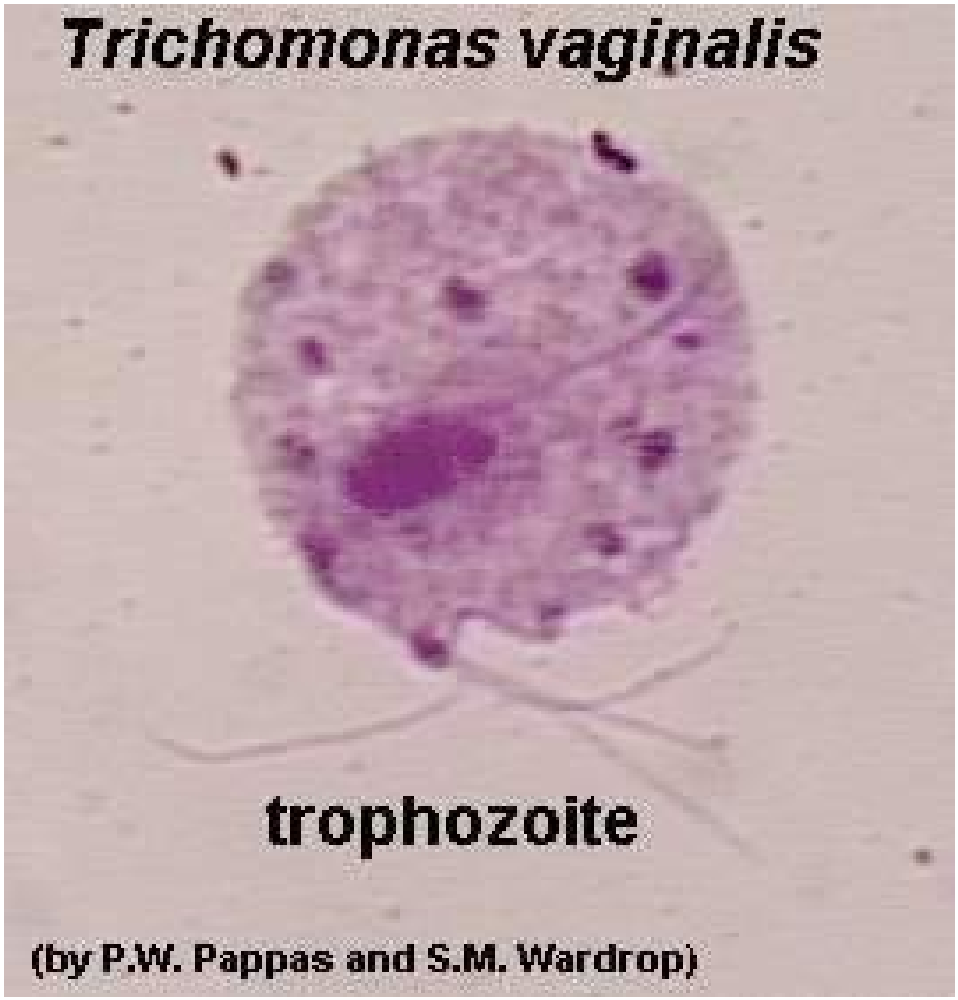


Trichomonas vaginalis

- is the causative agent of trichomoniasis, and is the most common pathogenic protozoan infection of humans in industrialized countries. Infection rates between men and women are the same with women showing symptoms while infections in men are usually asymptomatic.

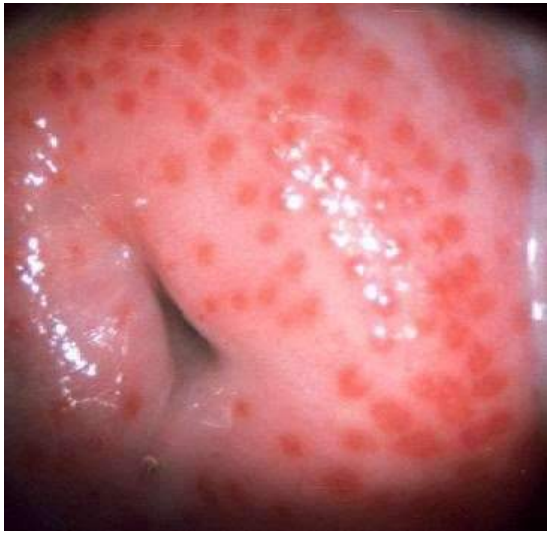


- Transmission takes place directly because the trophozoite **does not have a cyst.**

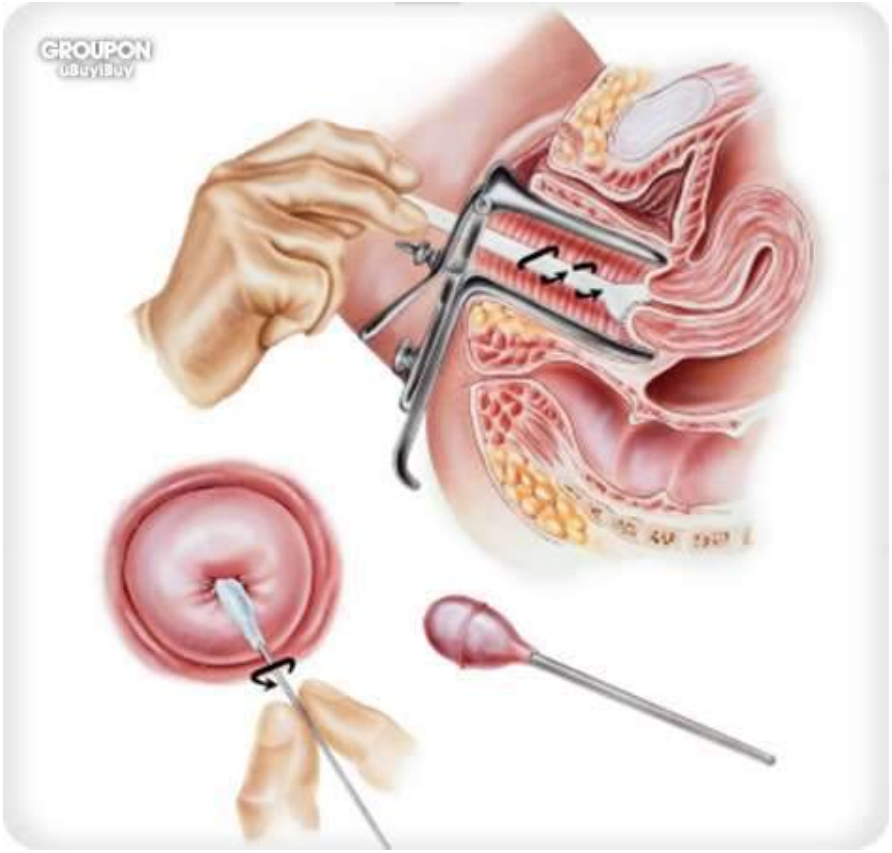


Trichomoniasis:

- **Trichomoniasis,** sometimes referred to as "**trich**" **or** "**fishy fanny syndrome**" because of the smell associated therewith, is a common cause of vaginitis. It is a sexually transmitted disease, The human genital tract is die only reservoir for this species
- Trichomoniasis is primarily an infection of the urogenital tract; the most common site of infection is the urethra and the vagina in women.

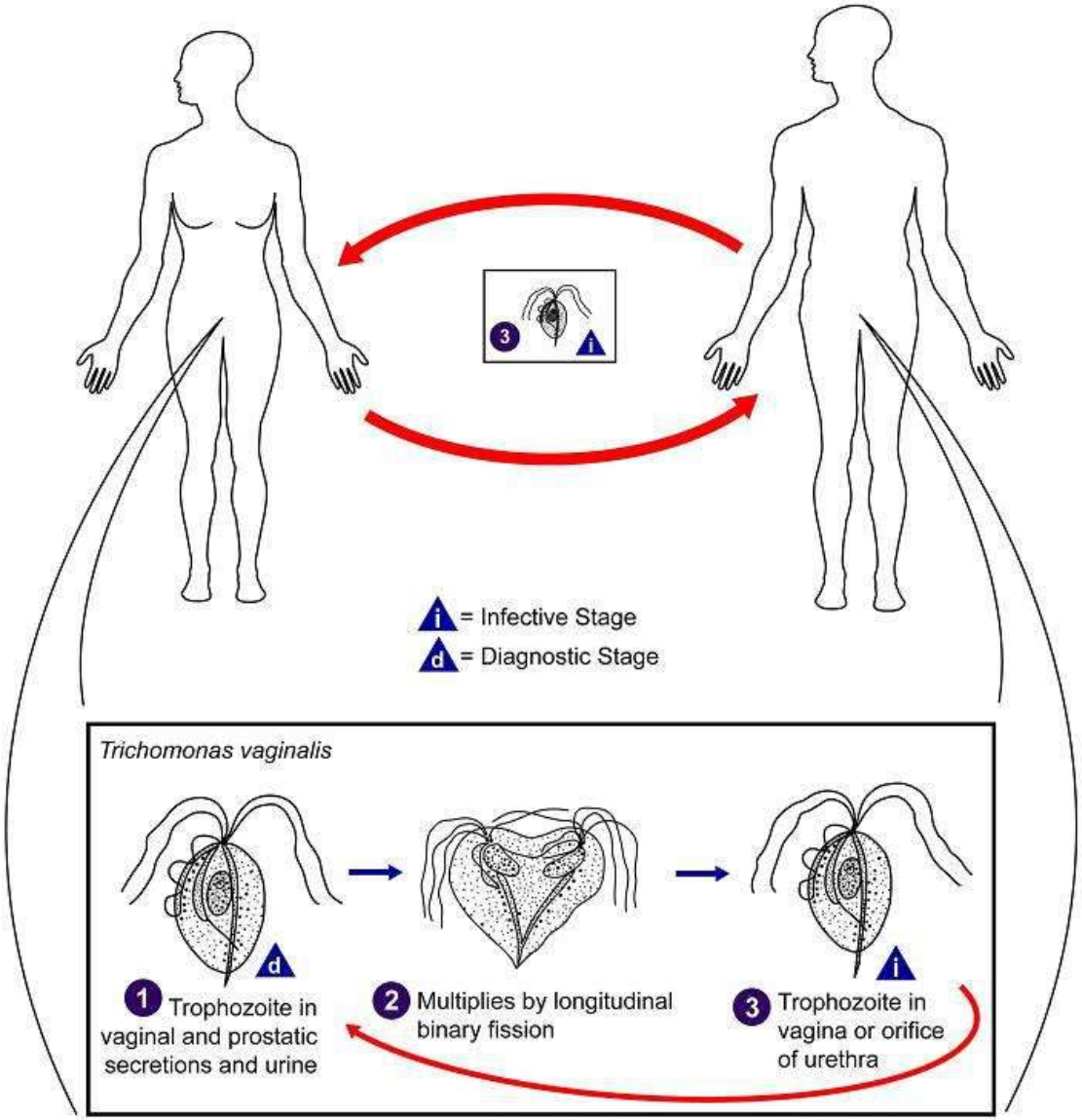


- The infection can occur in females if the normal acidity of the vagina is shifted from a healthy, semi-acidic pH (3.8 - 4.2) to a much more basic or alkaline one (5 - 6) that is conducive to ***T. vaginalis*** growth. Males rarely exhibit symptoms of a ***T. vaginalis*** infection.



Trichomoniasis

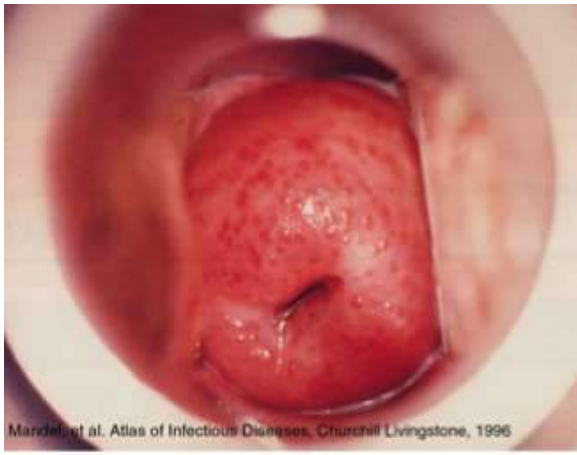
(Trichomonas vaginalis)

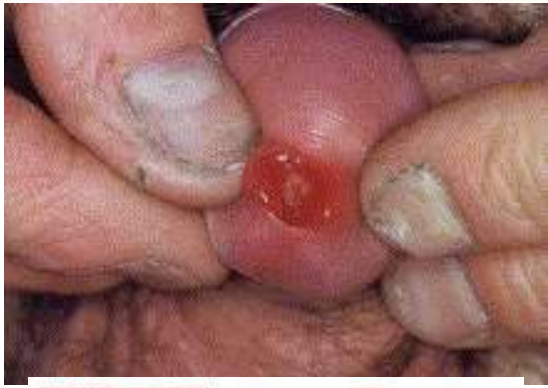


Symptoms:

Typically, only women experience symptoms associated with *Trichomonas* infection. Symptoms include inflammation of the cervix **(cervieitis),** urethra **(urethritis),** and vagina **(vaginitis)** which produces:

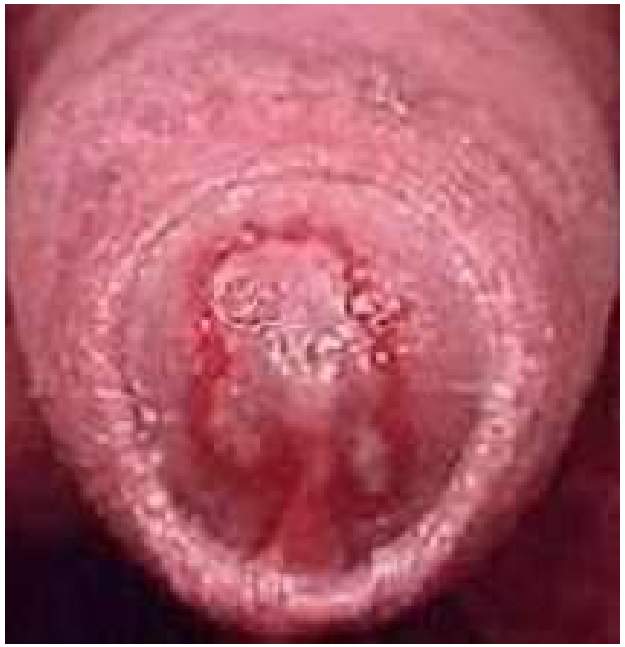
1. An itching or burning sensation.
2. Discomfort may increase during intercourse and urination.
3. A yellow-green, itchy, frothy foul-smelling vaginal discharge.
4. In rare cases, lower abdominal pain can occur.





Symptoms usually appear in women within 5 to 28 days of exposure. In many cases, men may hold the parasite for some years without any signs (dormant). While symptoms are most common in women, some men may temporarily exhibit symptoms such as:

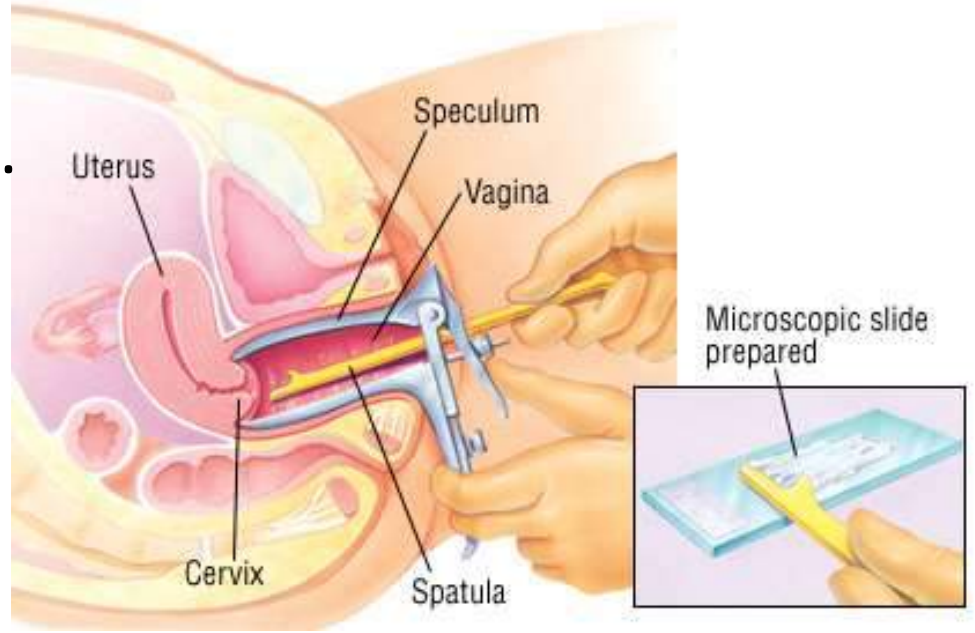
1. An irritation inside the penis.
2. Mild discharge.
3. Slight burning after urination or ejaculation



Diagnosis:

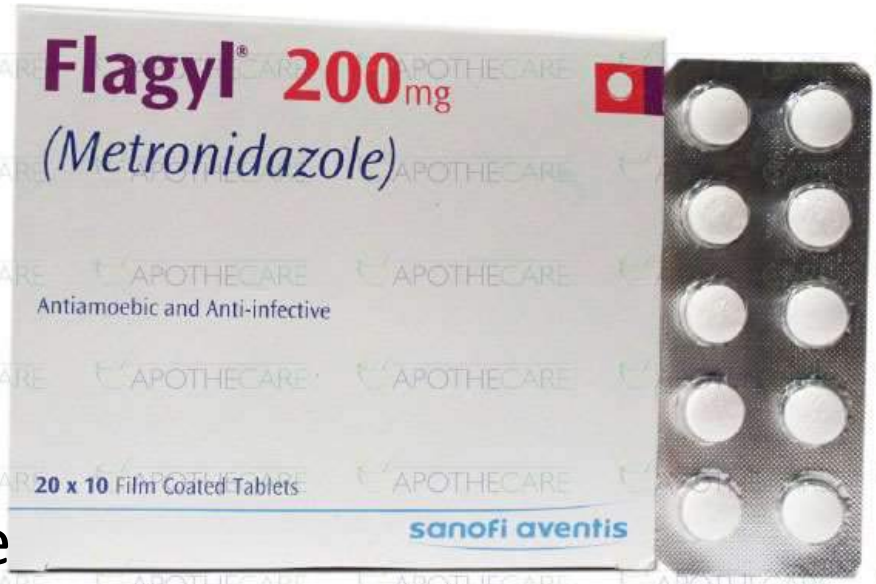
Trichomoniasis is diagnosed by:

- Visually observing the trichomonads via a microscope.
- In women, the doctor collects the specimen during a pelvic examination. The sample is then placed into a microscopic slide and sent to a laboratory to be analyzed.
- An examination in the presence of trichomoniasis ptaulas may also reveal small red ulcerations on the vaginal wall or cervix.

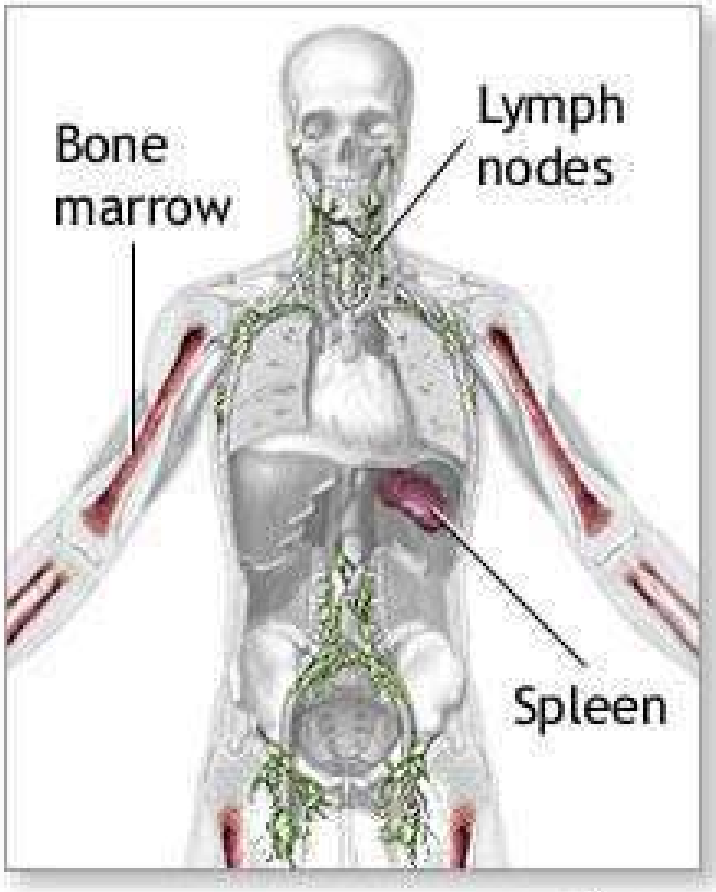


Treatment:

- Treatment for both pregnant and non-pregnant patients usually utilizes **Metronidazole (Flagyl)** by mouth at once. Sexual partners, even if asymptomatic, should be concurrently treated.
- Although both men and women are susceptible to suffer the infection, it is suspected that more than one half of men who are infected will naturally expel the parasite within 14 days, while in women it will persist unless treated.



Blood and Tissue Flagellata

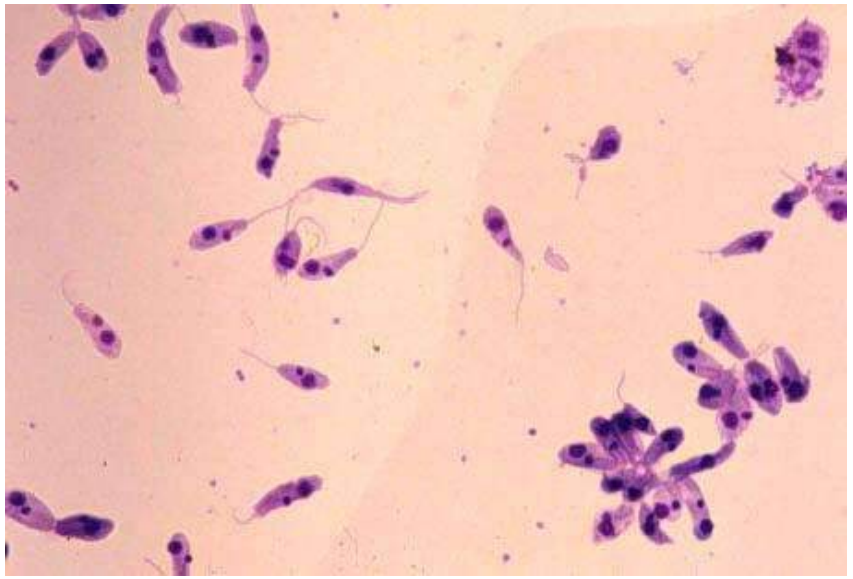


A.D.A.M.

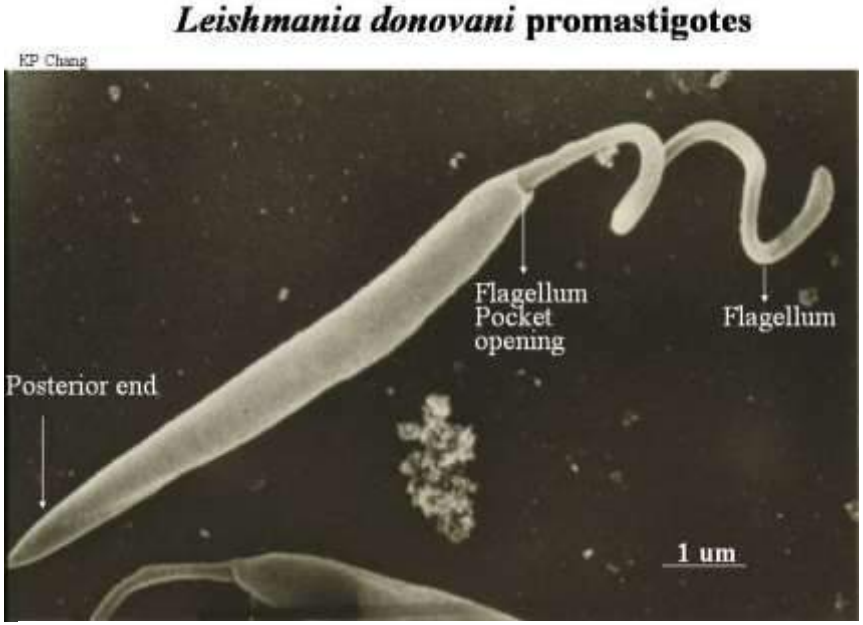
Lec.Dr.Ruwaidah F. Khaleel

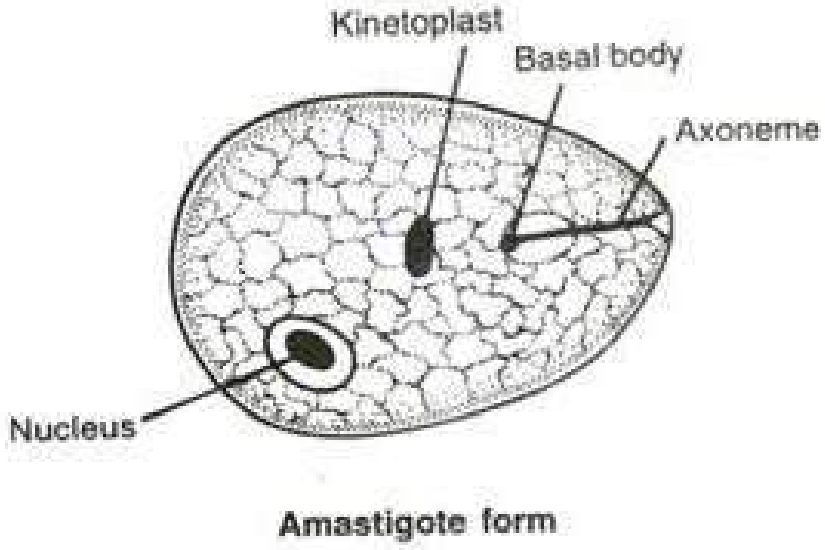
Blood and Tissue Flagellata

- ***Leishmania***
- *Leishmania* is a genus of Trypanosomatid protozoa, and is the parasite responsible for the disease leishmaniasis.
- It is spread through **sandflies of the genus *Phlebotomus*** in the Old World, and of the genus *Lutzomyia* in the New World.
- Their primary hosts are vertebrates; *Leishmania* commonly infects **hyraxes, canids, rodents, and humans**.



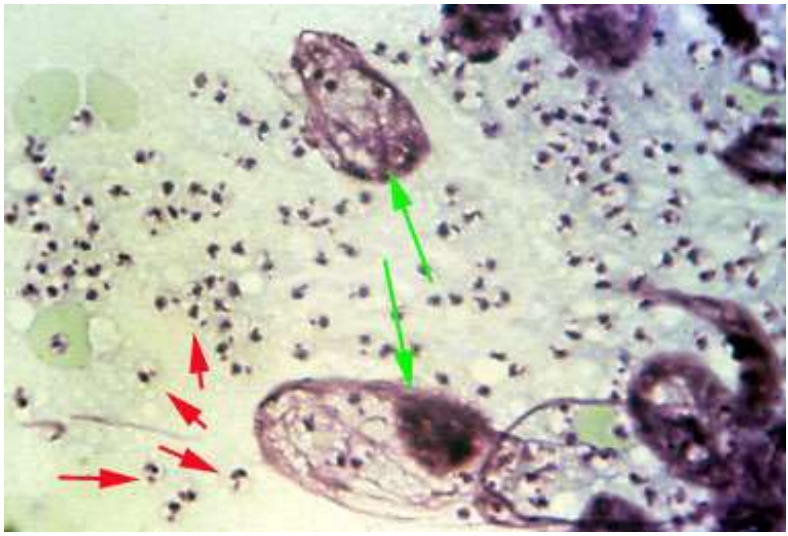
- *Leishmania* currently affects *12* million people in 88 countries.
- ***Leishmania* cells have two morphological forms:**
- ***promastigote* (with an anterior flagellum)** in the insect host
- **Amastigote (without flagella)** in the vertebrate host.
- Infections are regarded as **cutaneous, mucocutaneous, or visceral.**





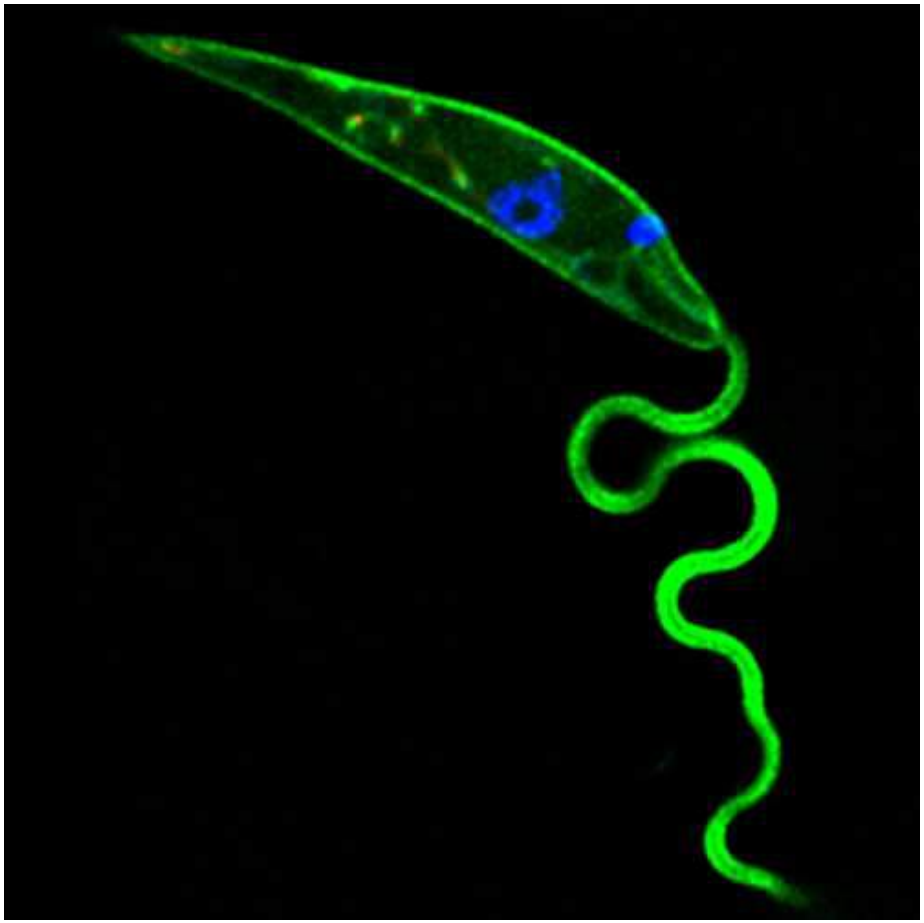
Morphology:

- **Amastigote**
- The amastigote, literally "**without a flagellum**,"
- Is the intracellular,
- Non- motile form in the vertebrate host,
- It divides by longitudinal binary fission at **37°C**.
- Intracellular amastigotes are **3-6 mm** in length and **1.5-3.0 mm** in width.
- The amastigote is also called the **Leishman-Donovan (LD)** body.
- The amastigote is not really devoid of a flagellum, it is simply that the flagellum does not protrude beyond the-body surface and by light microscopy cannot be seen.

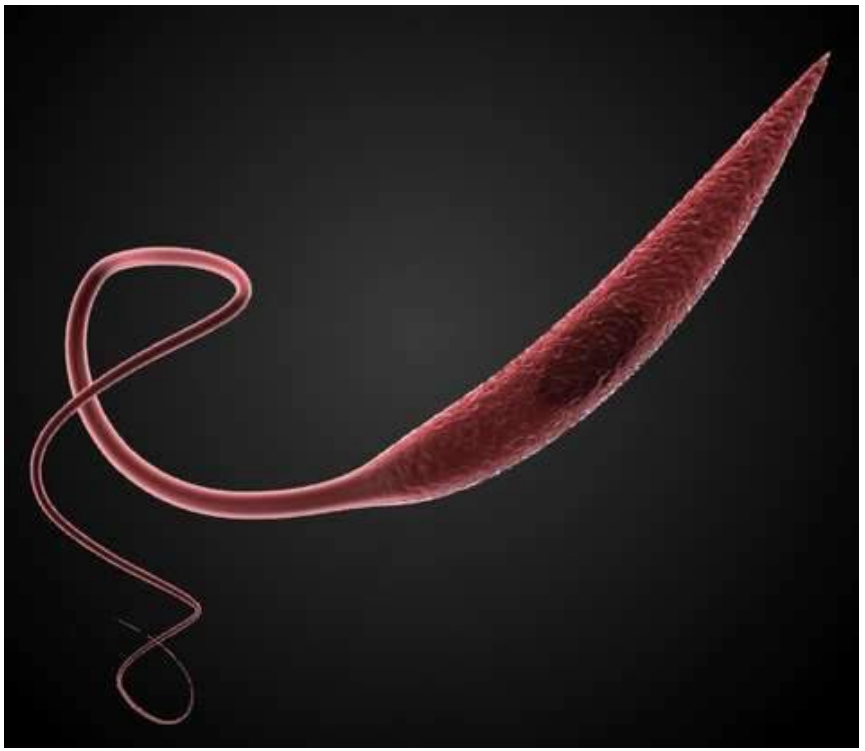


Promastigote

- **Promastigote, characterized by :**
- A free anterior flagellum, the kinetoplast and axoneme at the anterior end of the body.
- There is no undulating membrane.
- They are found in arthropods.
- The promastigote, is 15-30 mm in length and 5mm in width; it is extracellular, motile, and grows and divides by longitudinal binary fission at 27°C in the sand fly.



- Promastigotes can be grown in vitro at 25°C temperature on **NNN medium (Novy-MacNeal-Nicolle medium)**, which has a solid phase of blood agar and a liquid phase containing a physiologic salt solution.
- Liquid media that support promastigote growth are also available.
- Amastigotes usually are grown inside tissue culture cells and can also be grown extracellularly at 37°C under special conditions.

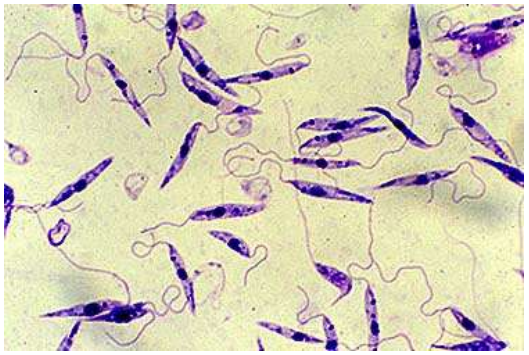


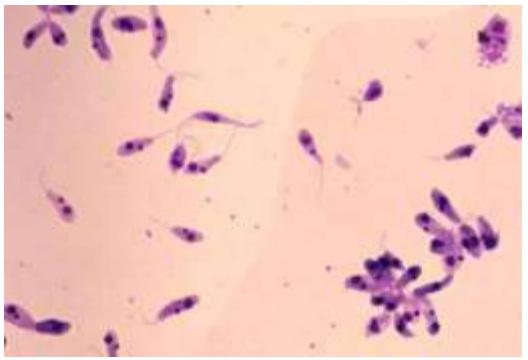
Leishmaniasis

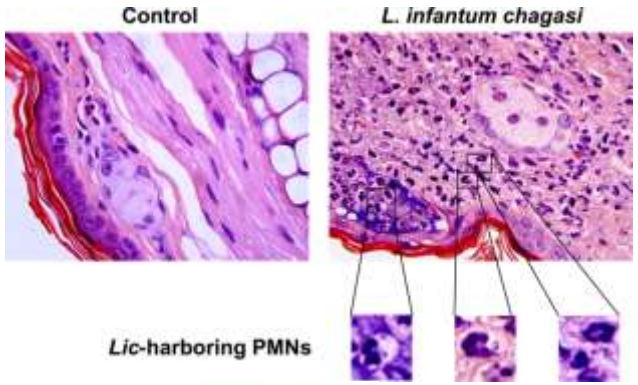
- **Leishmaniasis** is a disease caused by protozoan parasites that belong to the genus *Leishmania* and is transmitted by the bite of certain species of sand fly (subfamily **Phlebotominae**).
- Most forms of the disease are transmissible only from animals (zoonosis), but some can be spread between humans

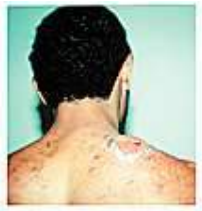
Human infection is caused by about 21 of 30 species that infect mammals. Some of them include:

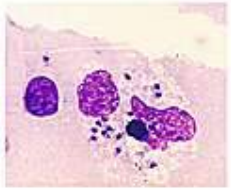
- The *L. donovani* complex with three species *(L. donovani, L. infantum,* and ***L. chagasi****).*
- The *L. **mexicana*** complex with four main species *(L. mexicana, L. amazonensis*, and *L. venezuelensis*); *L. tropica; L. major; L. aethiopica.*
- The subgenus *Viannia* with four main species *(L. (V'.) braziliensis, L. (V.) guyanensis, L. (V) panamensis,* andZ. *(V.) peruviana).*



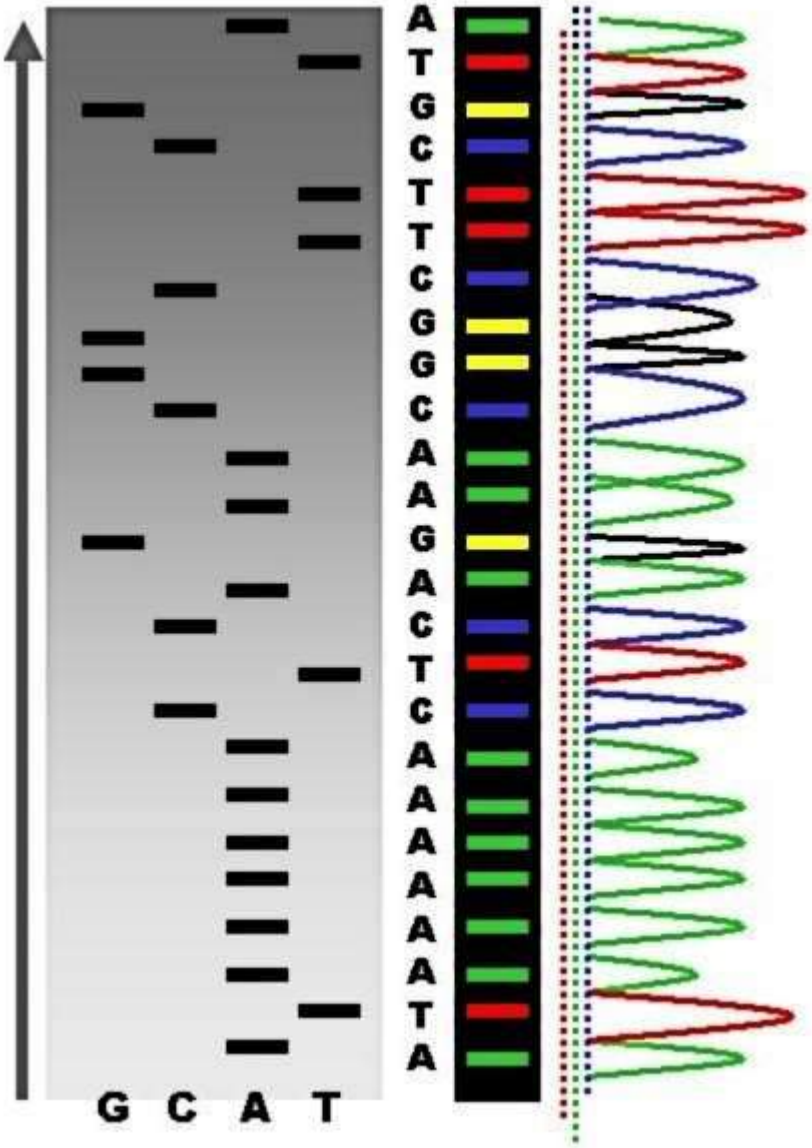






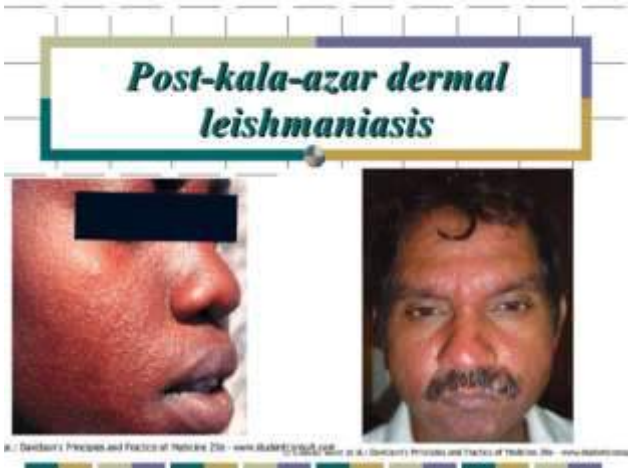


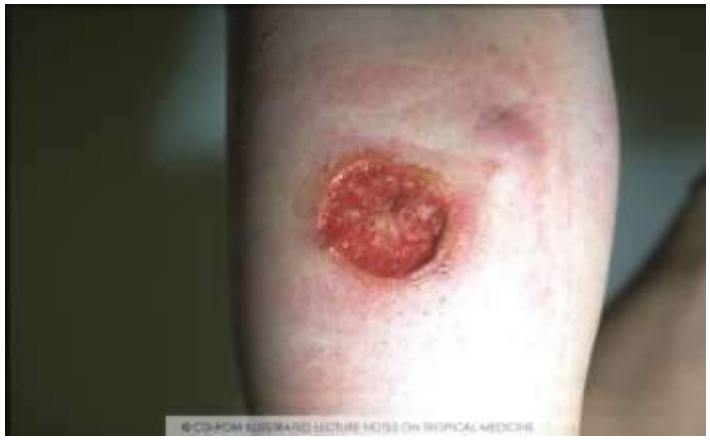
- **The different species are morphologically indistinguishable, but th can be differentiated by:**
- **Isoenzyme analysis**
- **DNA sequence analysis, or**
- **Monoclonal antibodies.**

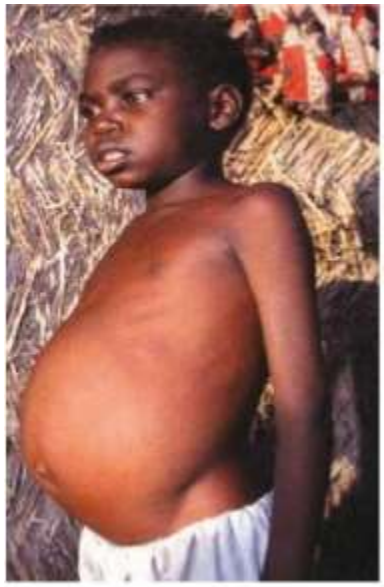


Leishmaniasis may be divided into the following types:

1. Cutaneous leishmaniasis.
2. Visceral leishmaniasis.
3. Post-kala-azar dermal leishmaniasis.
4. Mucocutaneous leishmaniasis.

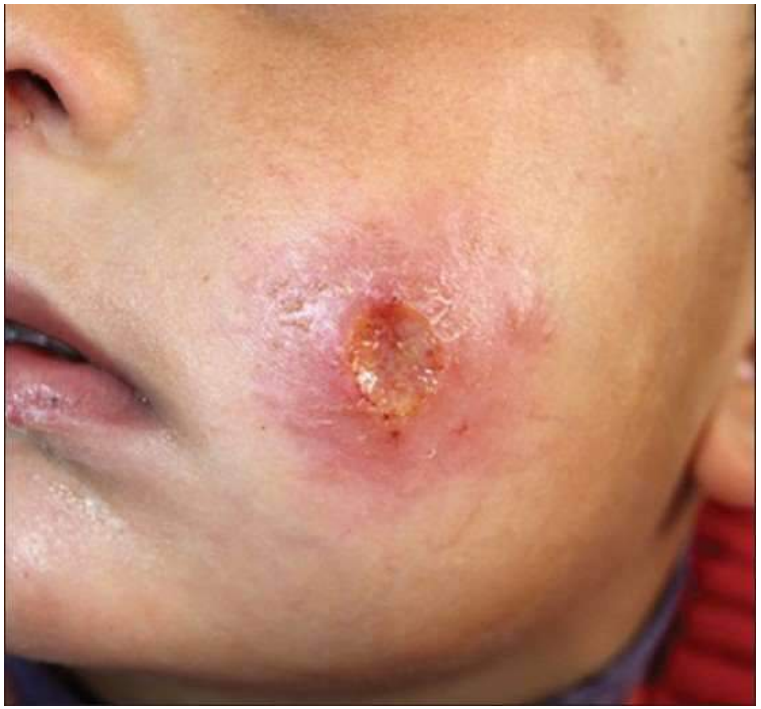


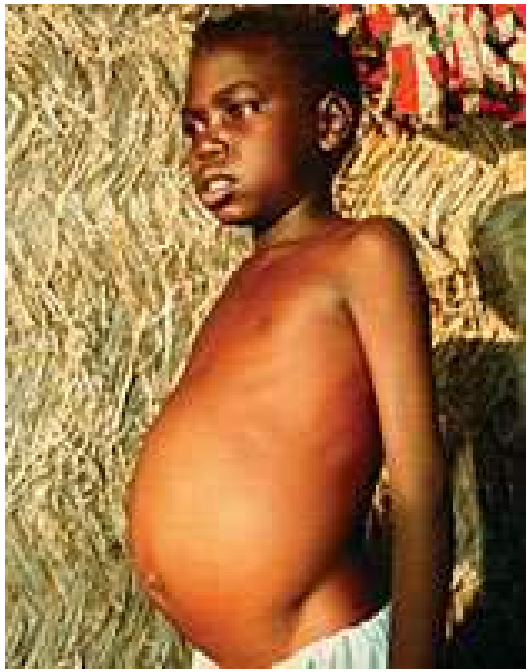




Spleenomegaly

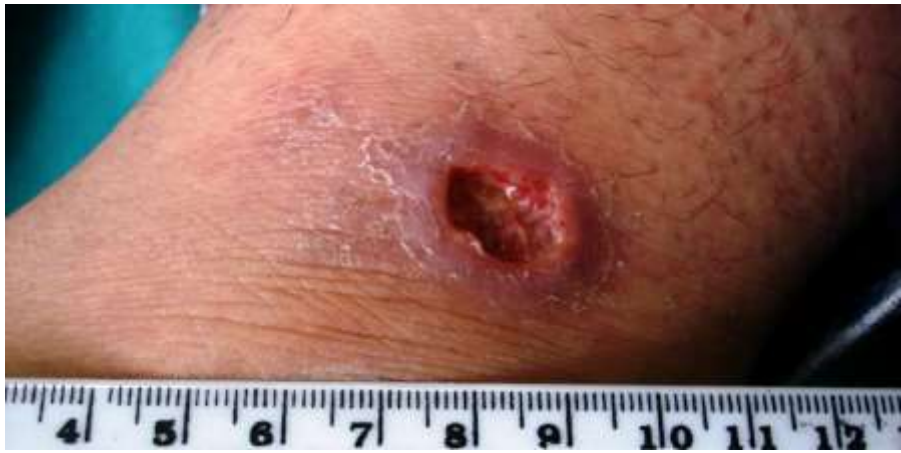
- **Cutaneous leishmaniasis** is the most common form of leishmaniasis.
- **Visceral leishmaniasis** is a severe form in which the parasites have migrated to the vital organs.





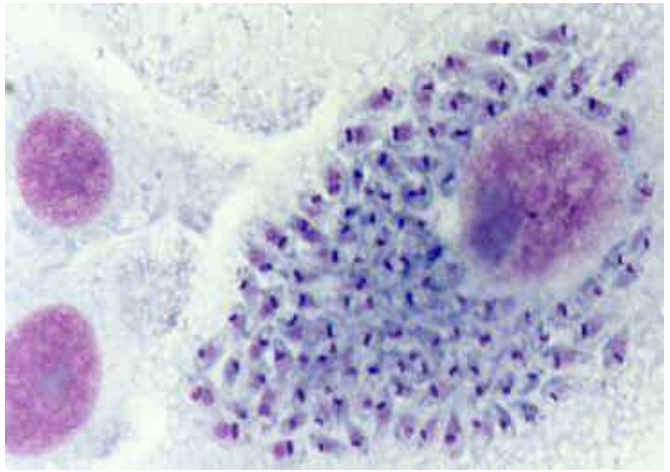
1) Cutaneous leishmaniasis

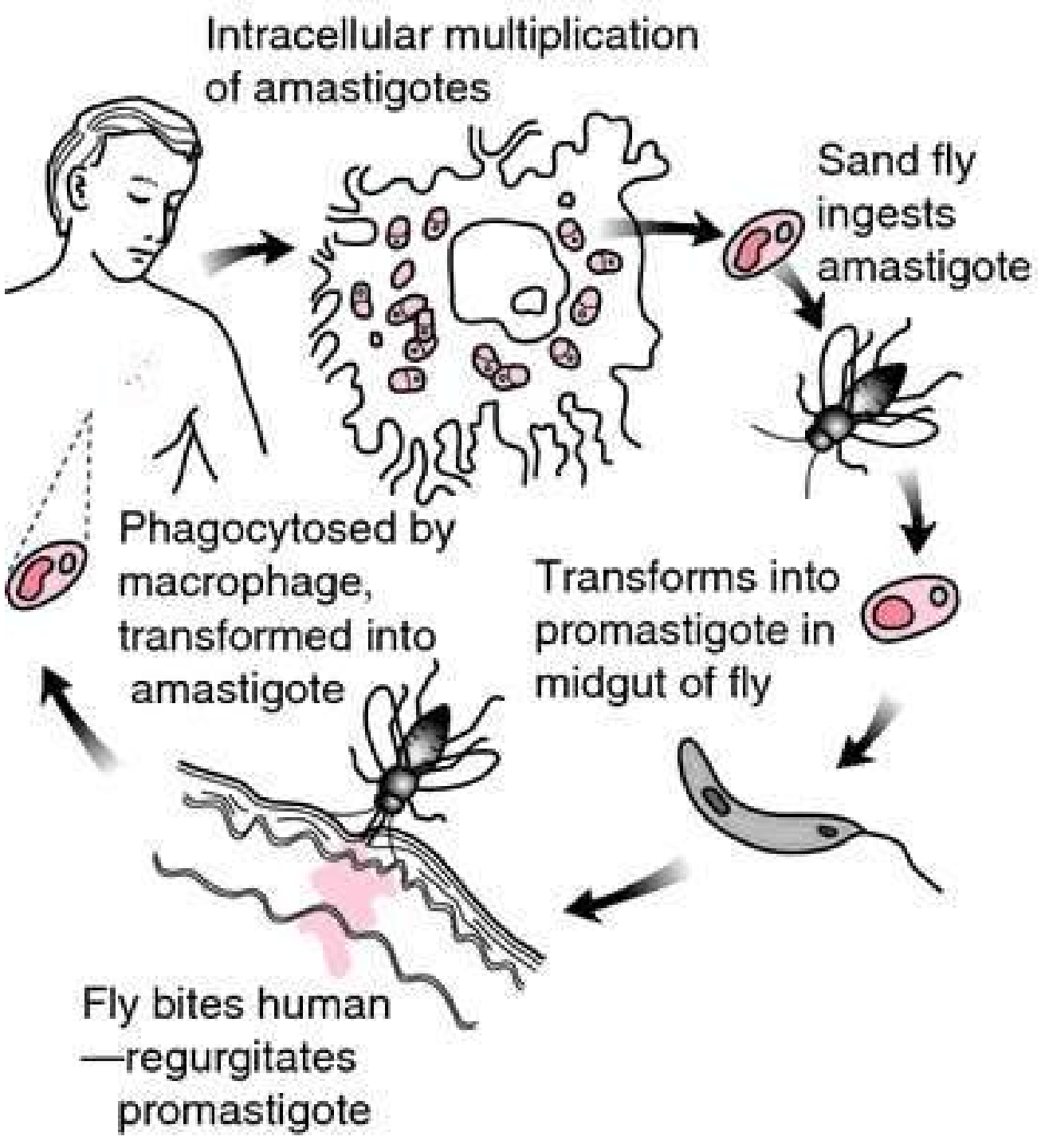
- Cutaneous leishmaniasis (also known as **"Aleppo boil," "Baghdad boil," "Bay sore," "Biskra button," "Chiclero ulcer," "Delhi boil," "Kandahar sore," 'Lahore sore," "Oriental sore," "Pian bois," and "Uta")** is the most common form of leishmaniasis.
- It is a skin infection caused by a single-celled parasite that is transmitted by sand fly bites. There are about 20 Species of *Leishmania* that may cause cutaneous leishmaniasis.



Life cycle

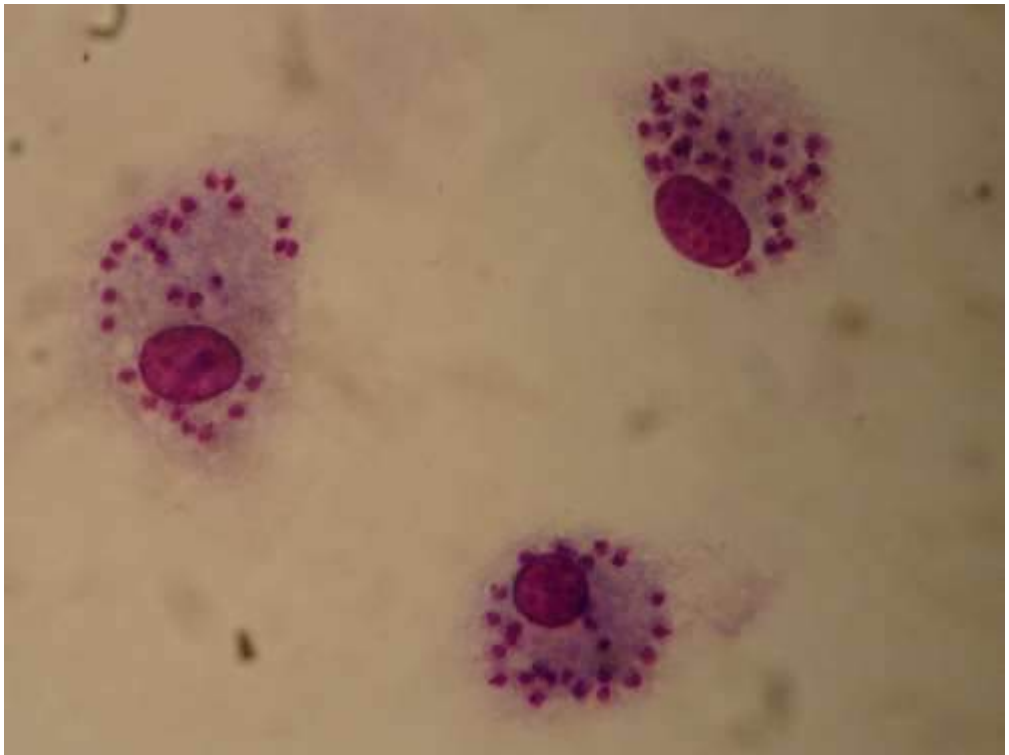
- Leishmaniasis is transmitted by the bite of infected female **phlebotomine sandflies**. The life cycle of *Leishmania Tropica* is identical to that of other related parasites of the same genus and includes both an amastigote and a promastigote stage.
- The sand flies inject the infective stage of promastigote, in which a sand fly infects a host with the parasite through feeding.
- The amastigote is part of the tissue stage in which the parasite transforms after being engulfed by a macrophage.



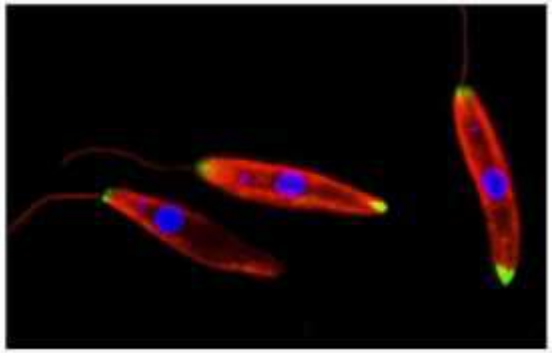

Intracellular multiplication
of amastigotes
Sand fly
ingests
amastigote
Phagocytosed by
macrophage,
transformed into
amastigote
Transforms into
promastigote in
midgut of fly
Fly bites human
—regurgitates
promastigote

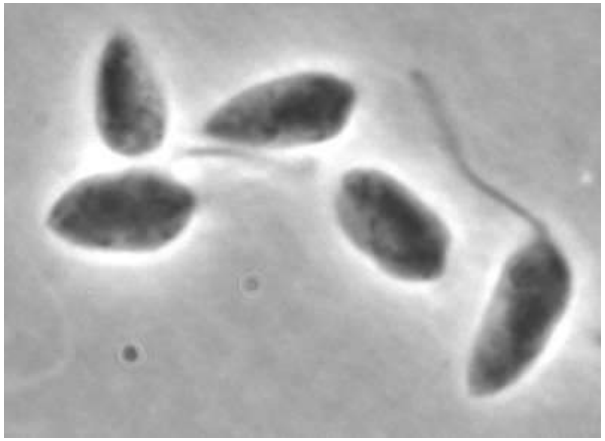
Pathology

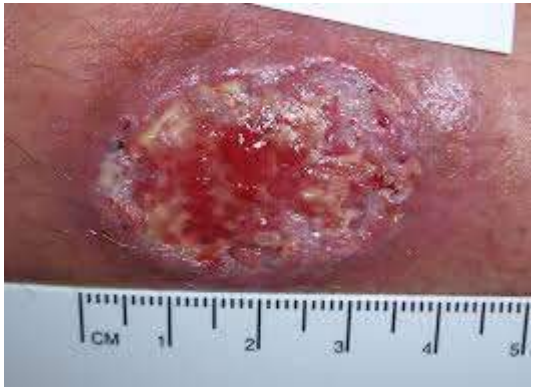
- Amastigotes replicate intracellular inside macrophages of human skin. A raised, red lesion develops at the site of the bite (often weeks or sometimes years afterwards).
- The lesion then ulcerates and may become secondarily infected with bacteria.



- In many species (for example, ***L. major***) the lesion often spontaneously heals with atrophic scarring.
- In some species (for example, *L. viannia braziliensis*)' the lesion may spontaneously heal with scarring, but then re-appear elsewhere (especially as destructive mucocutaneous lesions),
- Lesions of other *leishmania* species may spontaneously heal and then re-appear as satellite lesions around the site of the original lesion, or along the route of lymphatic drainage.



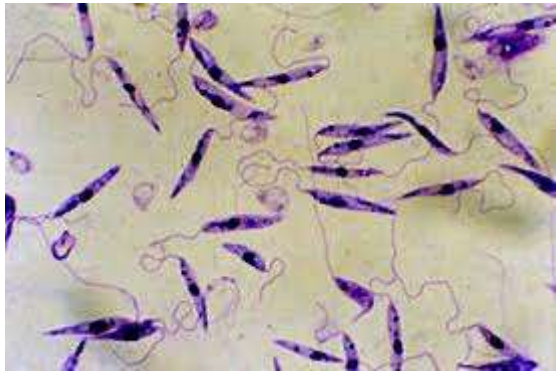


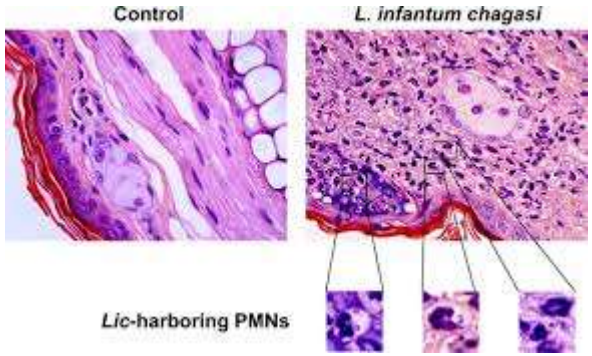


2) Visceral leishmaniasis

- Visceral leishmaniasis (VL), also known as **kala-azar, black fever, and Dumdum fever**, is the most severe form of leishmaniasis.
- This disease is the second-largest parasitic killer in the world (after malaria), responsible for an estimated 500,000 eases each year worldwide.

- Several species of *Leishmania* are known to give rise to the visceral form of the disease.
- The "Old World" (Africa, Asia, Europe) species are ***L. donovani*** and L. ***infantum*** and the "New World" (South America) species is ***L. chagasi***

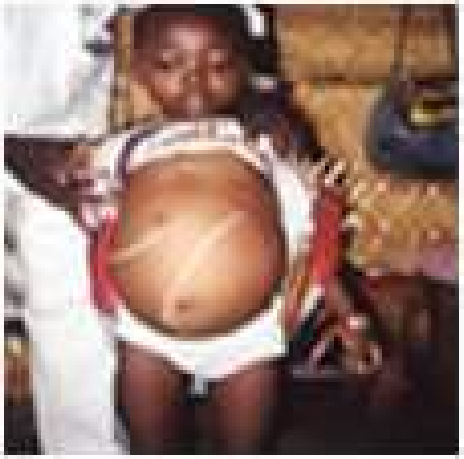




- The parasite migrates to the internal organs such as liver, spleen and bone marrow, and, if left untreated, will almost always result in the death of the host.

Sandfly.
The vector for leishmaniasis

Termite hill.
Breeding site for sandfly

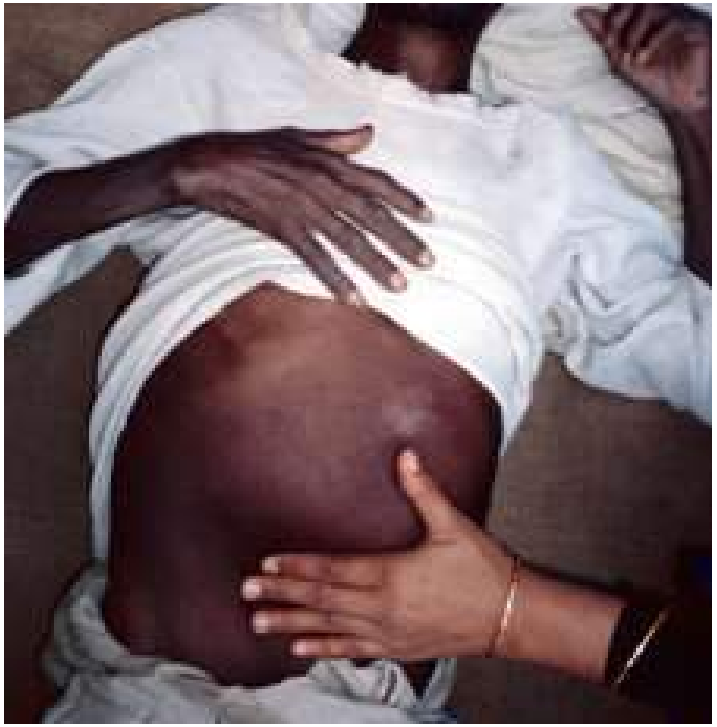


A child with large spleen a sign of leishmaniasis

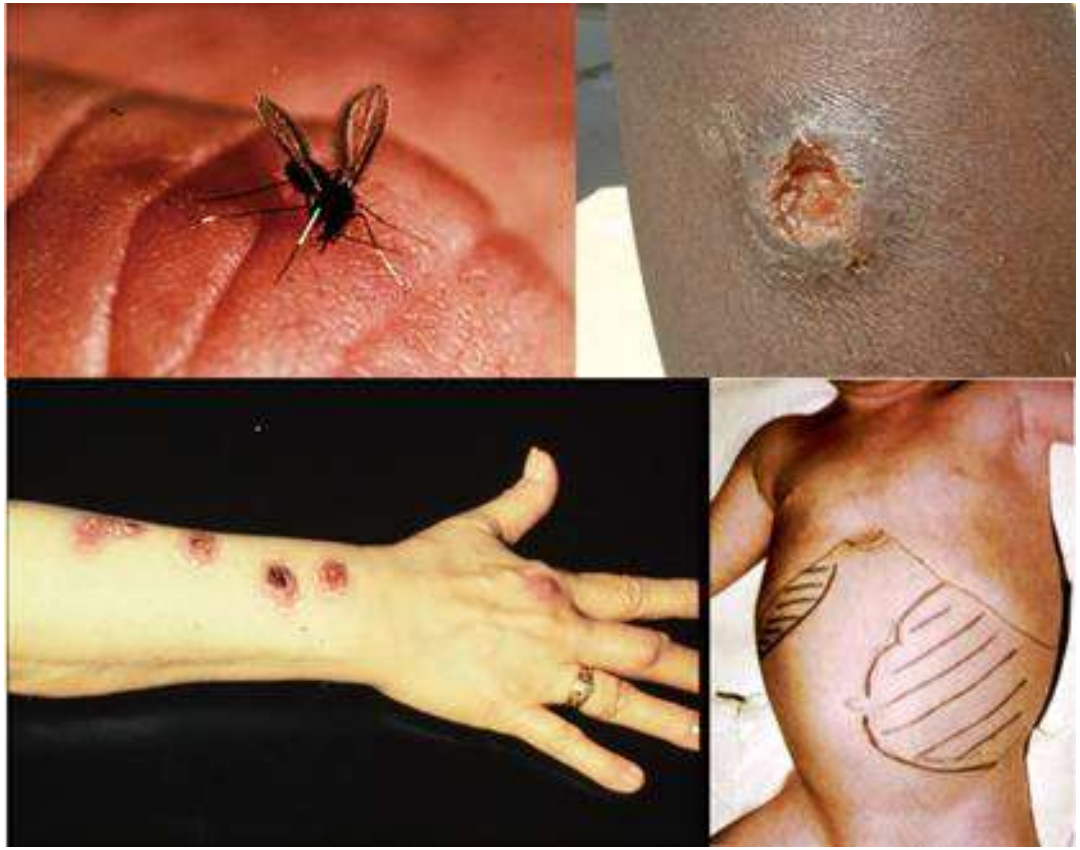
Signs and symptoms include

- Fever.
- Weight loss.
- Mucosal ulcers.
- Fatigue.
- Anemia.
- Substantial swelling of the liver and spleen.

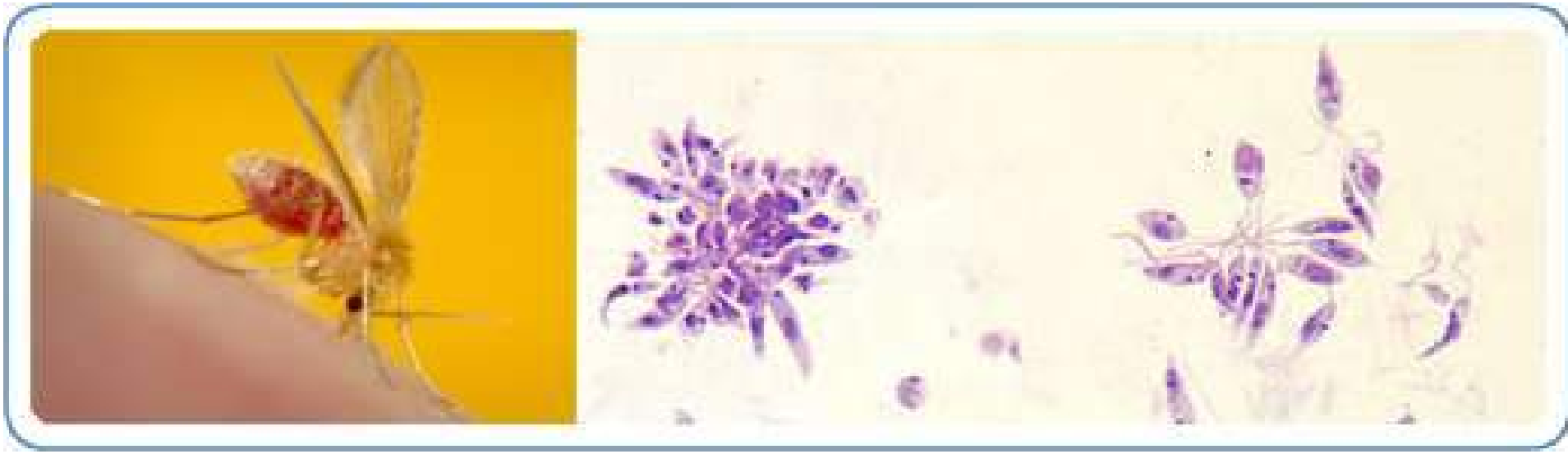
Of particular concern, according to the World Health Organization (WHO), is the emerging problem of HIV/VL, co-infection.



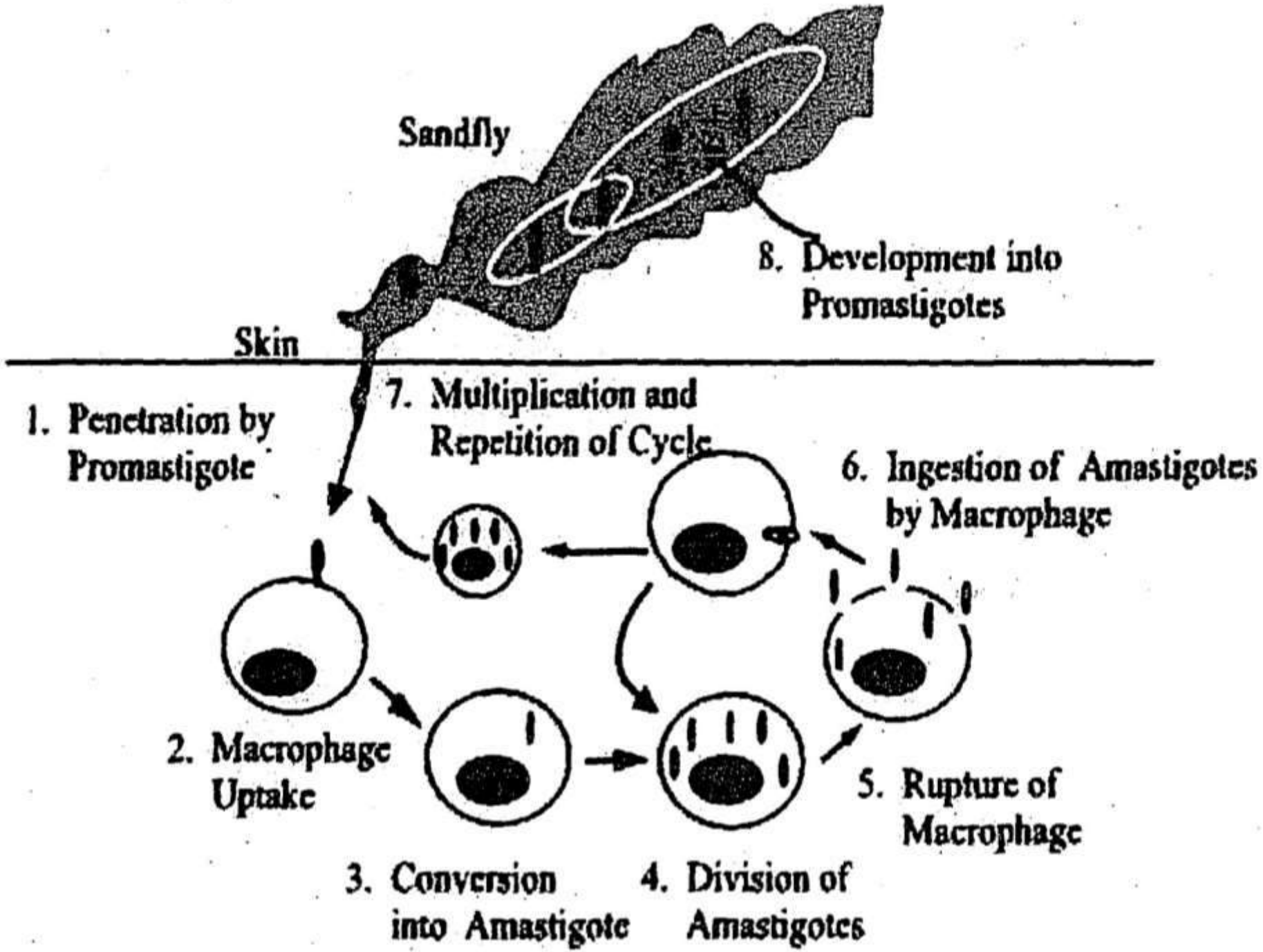
- The adult female sand fly is a bloodsucker, usually feeding at night on sleeping prey.
- When the fly bites an animal infected with *L. donovani,* the pathogen is ingested along with the prey's blood. At this point the protozoan is in the amastigote form.



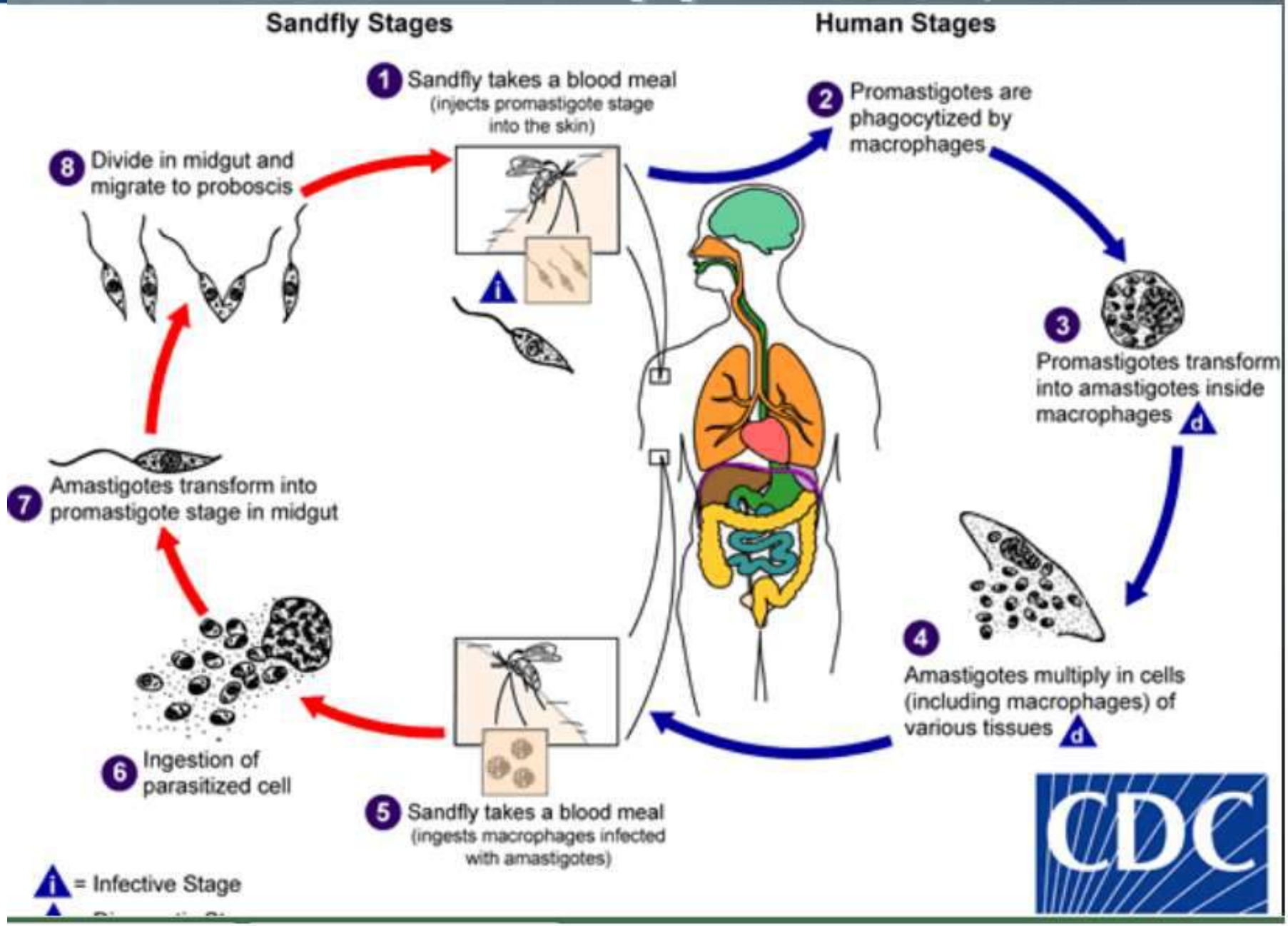
- Inside the stomach of the sandfly, the amastigotes quickly transform to promastigotes.
- This form is spindle-shaped, triples the size of the amastigote, and has a single flagellum that allows for motility.
- The promastigotes live extracellularly in the sandfly's alimentary canal, reproducing asexually.



- Promastigotes migrate to the proximal end of the gut where they become poised for a regurgitation transmission. This is their means of transmission back into a mammalian host.
- As the fly injects its saliva into prey when it bites. The, promastigotes are introduced locally at the bite site along with the fly's saliva. Once inside the new host, promastigotes invade macrophages.
- Promastigotes transform back into the smaller amastigote form inside the macrophages.


Sandfly
8. Development into Promastigotes
Skin
1. Penetration by Promastigote
7. Multiplication and Repetition of Cycle
6. Ingestion of Amastigotes by Macrophage
2. Macrophage Uptake
5. Rupture of Macrophage
3. Conversion into Amastigote
4. Division of Amastigotes

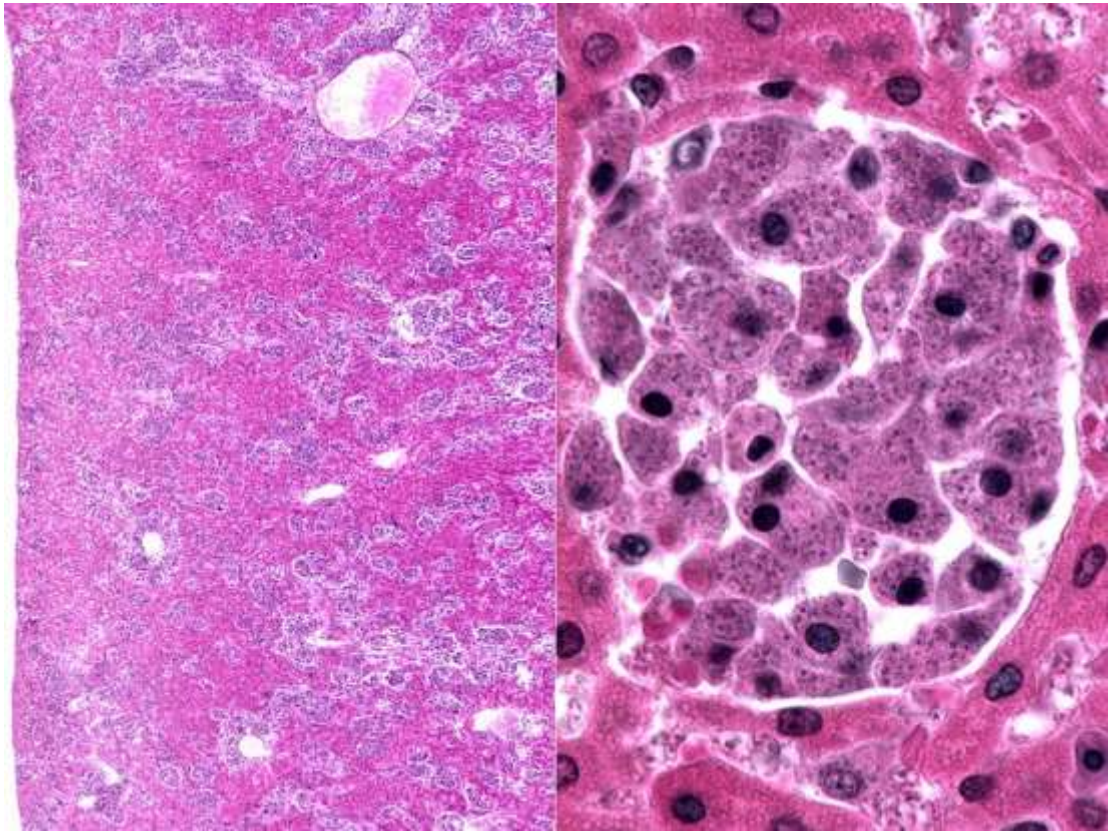
Life cycle of leishmania



NOTE:

- As an amastigote, *L. donovani* can only reproduce intracellularly and the amastigotes replicate in the most hostile part of the macrophage cell, inside the phagolysosome, whose normal defensive response they are able to prevent.

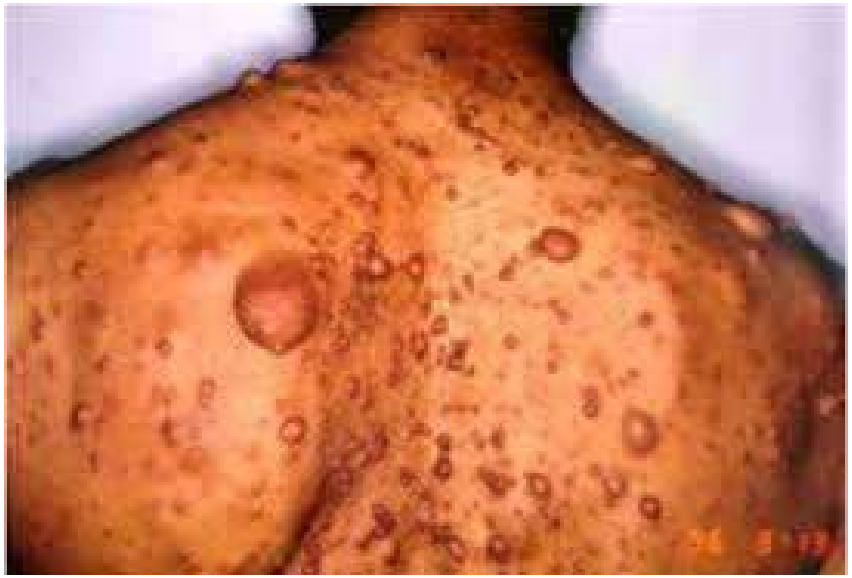
- After they have reproduced to a certain extent, the *L. donovani* lyses their host cell by sheer pressure of mass.
- The daughter cell amastigotes then migrate through the bloodstream to find new macrophage hosts.
- L. donovani becomes a systemic infection, spreading to all the host's organs, particularly the spleen and liver.



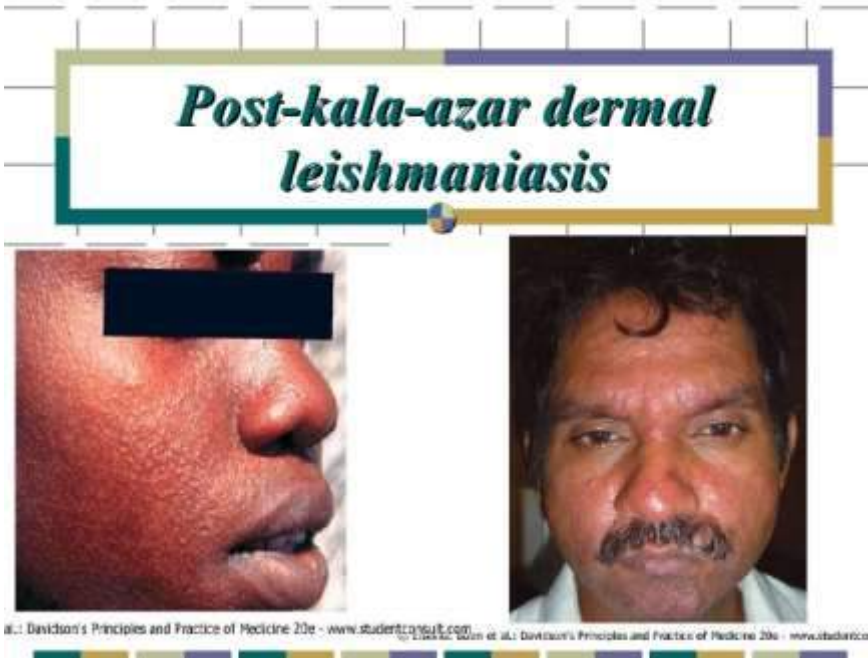
When a human patient does develop visceral leishmaniasis, the most typical symptoms are:

1. Fever.
2. Enlargement of spleen and liver (splenomegaly & hepatomegaly).
3. The blackening of the skin that gave the disease its common name; in India does not appear in most strains of the disease, and the other symptoms are very easy to mistake for those of malaria.

- Mis-diagnosis is dangerous, as without proper treatment the mortality rate for kala-azar is close to 100%. X. *donovani* itself is not usually the direct cause of death in kala-azar sufferers, however. Progress of the disease is extremely variable, taking anywhere from one to twenty weeks, but a typical duration for the Sudanese strain of the disease is narrower, between twelve and sixteen weeks

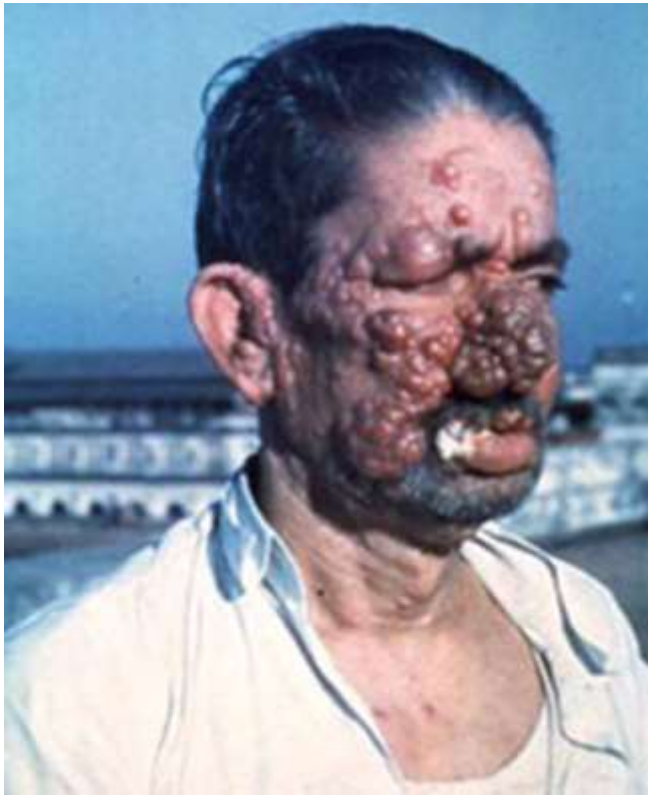


- 4. Even with recovery, kala-azar does not always leave its hosts unmarked. Sometime after successful treatment—generally a few months with **African kala-azar,** or as much as several years with the Indian strain—a secondary form of the disease may set in, called **post kala-azar dermal leishmaniasis, or** PKDL.



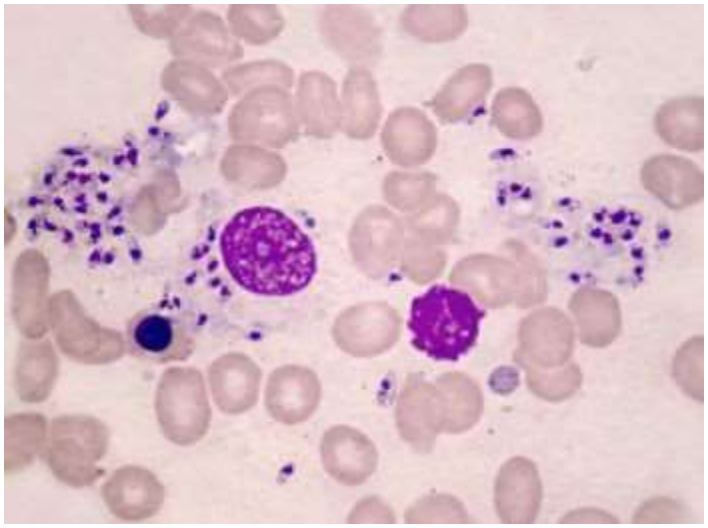
- This condition manifests first as **small, measle-like skin lesions on the face**, which gradually increase in size and spread over the body.
- Eventually the lesions may coalesce to form disfiguring, swollen structures resembling leprosy, and occasionally causing blindness if they spread to the eyes.

- (This disease is not the same as cutaneous leishmaniasis, a milder disease caused by another protozoan of the Leishmania genus which also causes skin lesions.)



Diagnosis:

- The gold standard for diagnosis is visualization of the **amastigotes in splenic aspirate or bone marrow aspirate.**
- This is a technically challenging procedure that is frequently unavailable in areas of the world where visceral leishmaniasis is endemic.
- **Serological testing** is much more frequently used in areas where leishmaniasis is endemic.



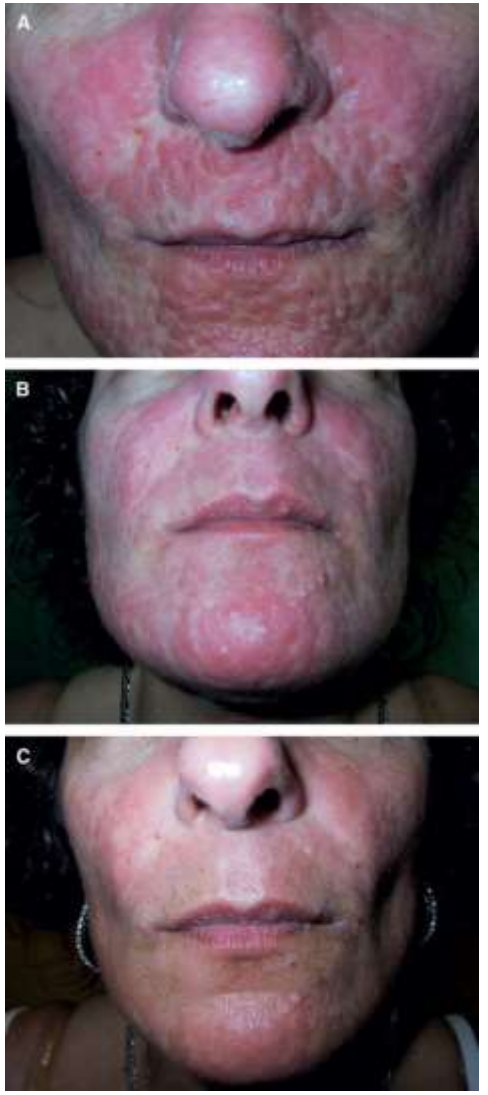
Treatments:

- The traditional treatment is with **pentavalent antimonials** such as **sodium stibogluconate** and **meglumine antimoniate.**
- Resistance is now common in India, and rates of resistance have been shown to be as high as 60% imparts of Bihar, India.

- The Indian medical practitioner, Upendra Nath Brahmachari, was nominated for the Nobel Prize in Physiology or Medicine in 1929 for his discovery of ureastibamine (an antimonial compound for the treatment of kala-azar) and, post kala-azar dermal leishmaniasis.

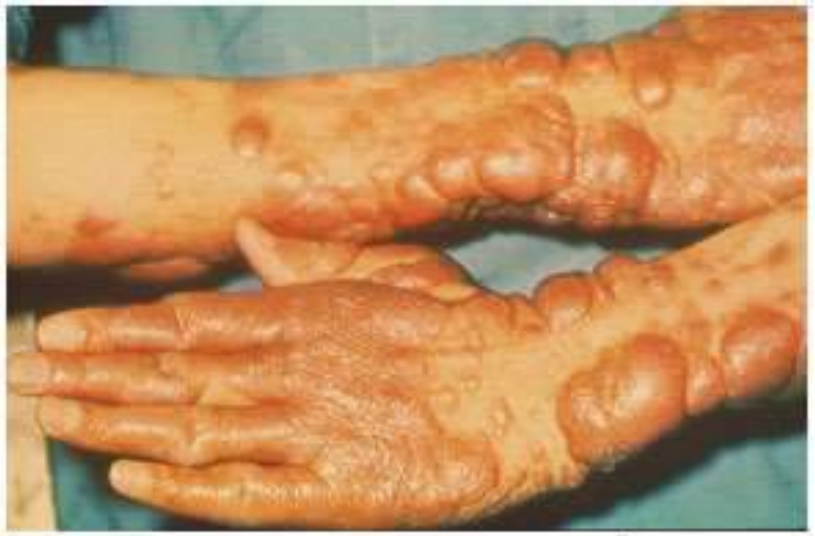
3) Post kala-azar dermal leishmaniasis

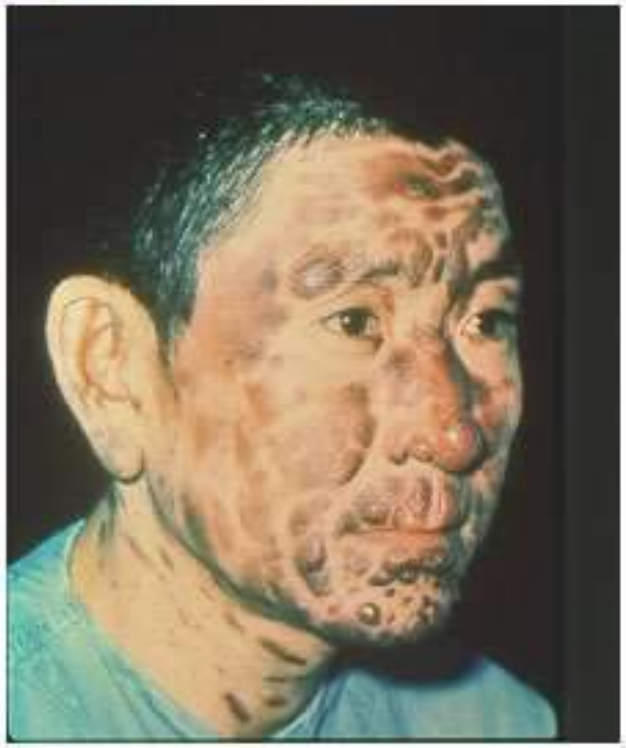
- **Post kala-azar dermal leishmaniasis** (PKDL) is a recurrence of Kala- azar that may appear on the skin of affected individuals up to **20 years** after being partially treated, untreated or even in those considered adequately treated.
- In Sudan they can be demonstrated in up to **60%** of treated cases.



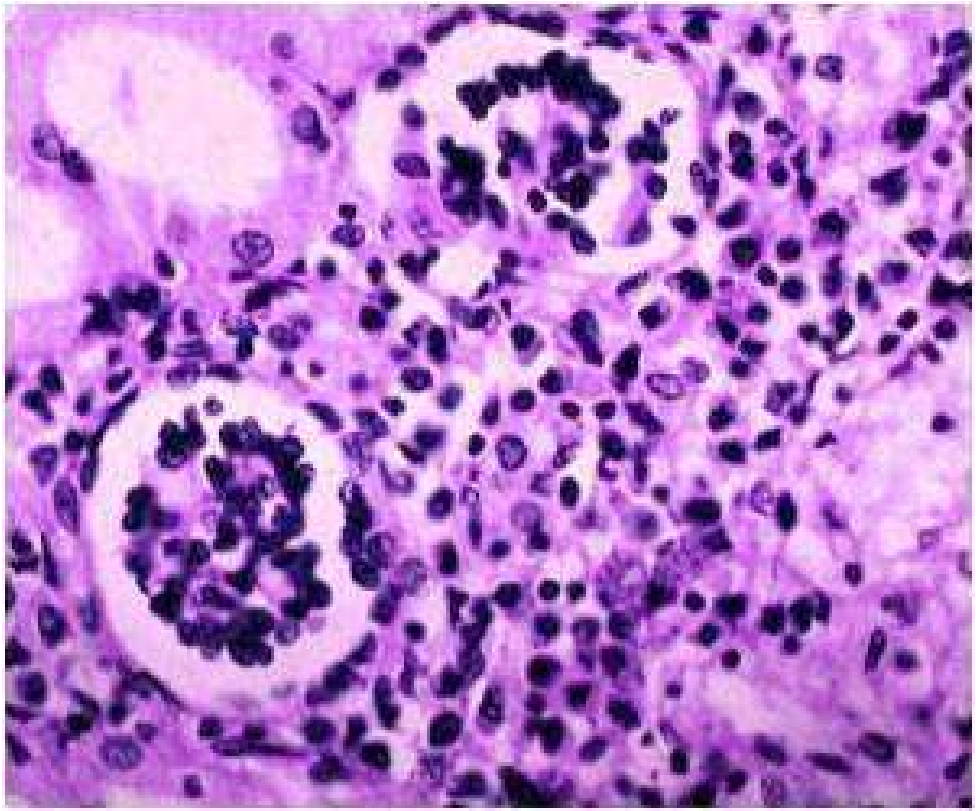
- They manifest as hypo-pigmented macules, papules, nodules, or facial erythema.
- Though any organism causing Kala-azar can lead to PKDL, it is commonly associated with *L. donovani* which gives different disease patterns in India and Sudan.

- In the Indian variant, nodules enlarge with time and form plaques but rarely ulcerate, but nodules from the African variety often ulcerate as they progress.





- Histology demonstrates a mixture of chronic inflammatory cells; there can be macrophage or epithelial granuloma.
- Parasite concentration is not consistent among studies, perhaps reflecting low sensitivity of diagnostic methods used in earlier entries.



Diagnosis & Treatment:

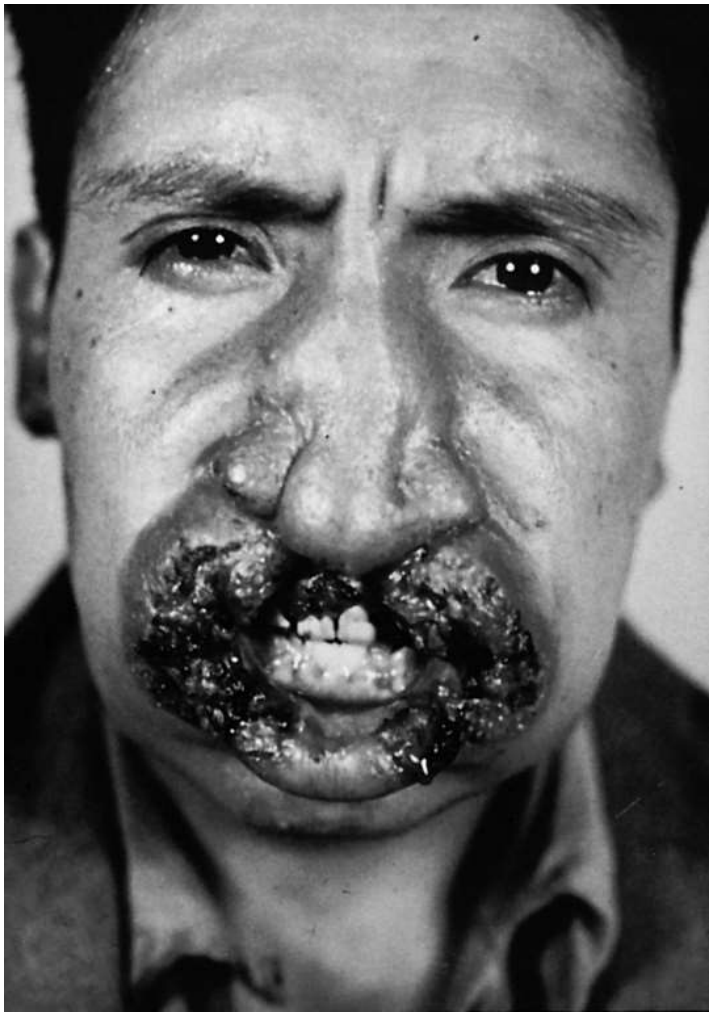
- **Current approach to diagnosis involves**

1. Demonstration of parasite by microscopy, *in vitro* culture or animal inoculation.
2. immune-diagnosis of parasite antigen.
3. Detection of parasite DNA in tissue. Newer PCR based tools have higher sensitivity and specificity.

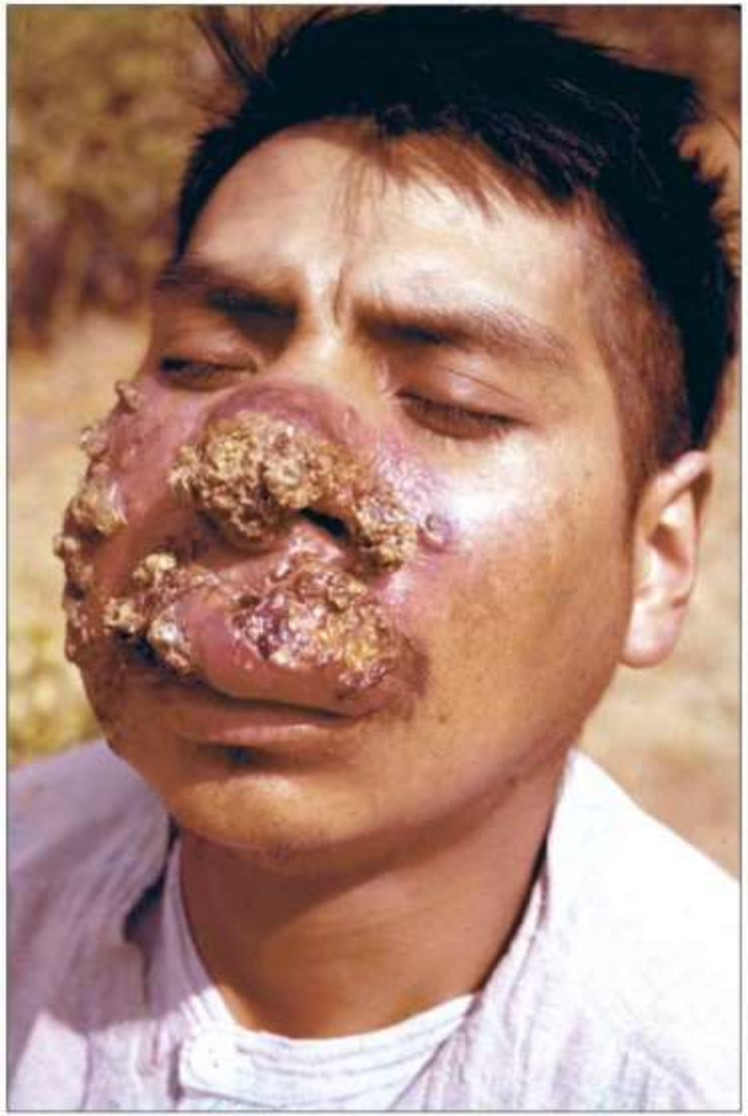
- **Sodium stibogluconate** alone or in combination with **rifampicin** is used for the treatment of PKLD for a long course of up to 4 months. Compliance can be an issue for such a long course.

4) Mucocutaneous leishmaniasis:

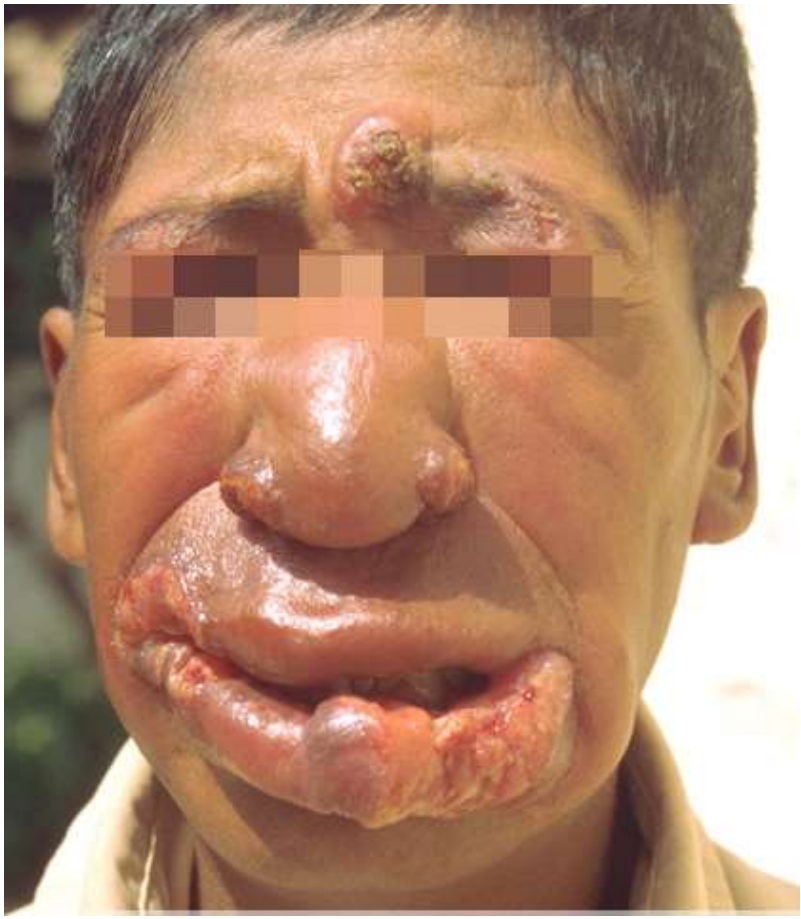
- A serious disease caused by ***Leishmania braziliensis,*** and ***L. braziliensis panamensis*** endemic in southern Mexico and Central and South America, except for northern Chile; the parasite does not invade the viscera, and the disease is limited to the skin and mucous membranes;



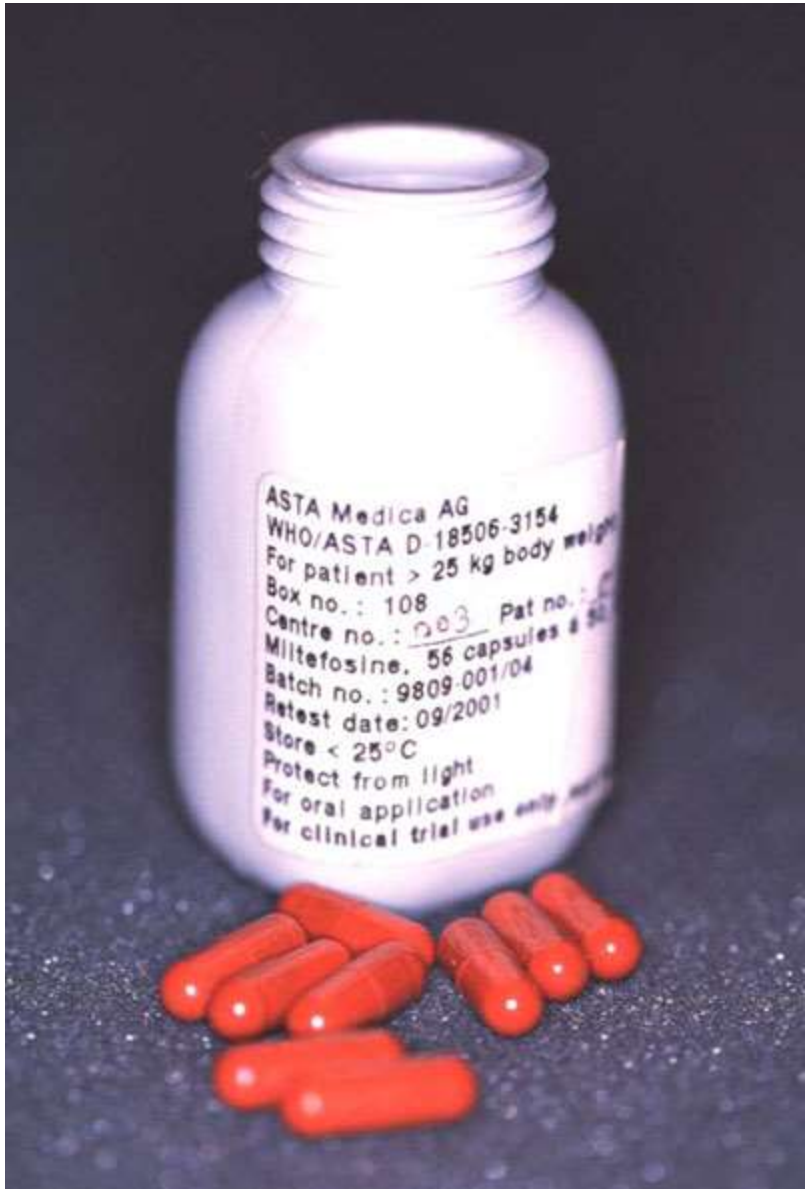
- the lesions resemble the sores of cutaneous leishmaniasis caused by *L. mexicana* or *L. tropica;*
- the **chancrous sores** heal after a time, but some months or years later, fungating and eroding forms of ulceration may appear on the tongue, buccal or nasal mucosa, and pharynx.



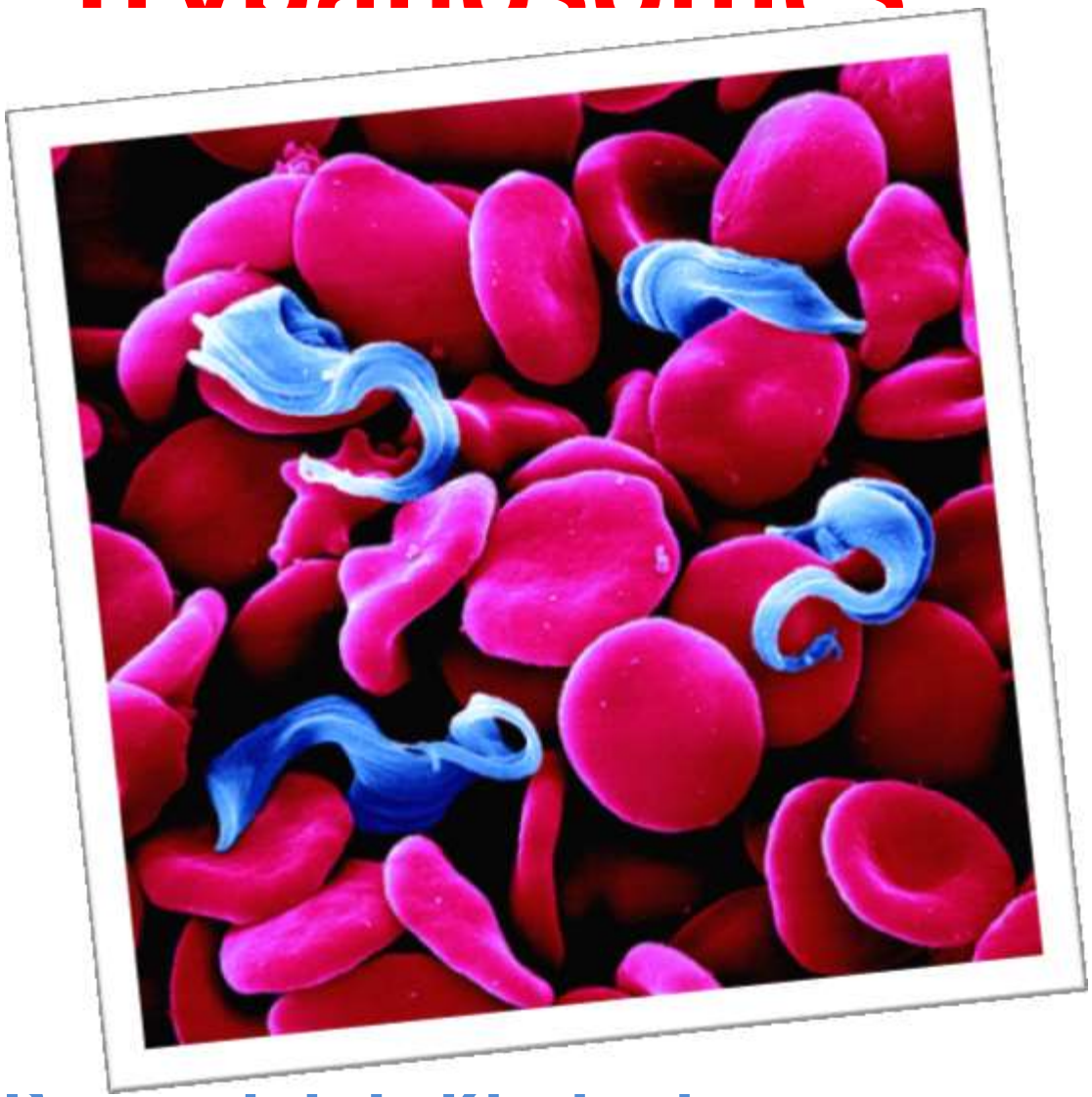
- Many variants of the disease exist, marked by differences in distribution, vector, epidemiology, and pathology, which suggest that it may in fact be caused by a number of closely related etiologic agents.
- *Espundia,* American leishmaniasis, leishmaniasis americana, bubas, nasopharyngeal leishmaniasis, New World leishmaniasis.



- Mucocutaneous leishmaniasis is **treated** with long courses (e.g. 30 days) of pentavalent antimonials in a high dose (20 mg/kg). This may fail to cure up to 42% of patients.
- Even in those patients who achieve an apparent cure, as many as 19% will relapse.



Trypanosomes



Lec.Dr.Ruwaidah F. Khaleel

Trypanosomes

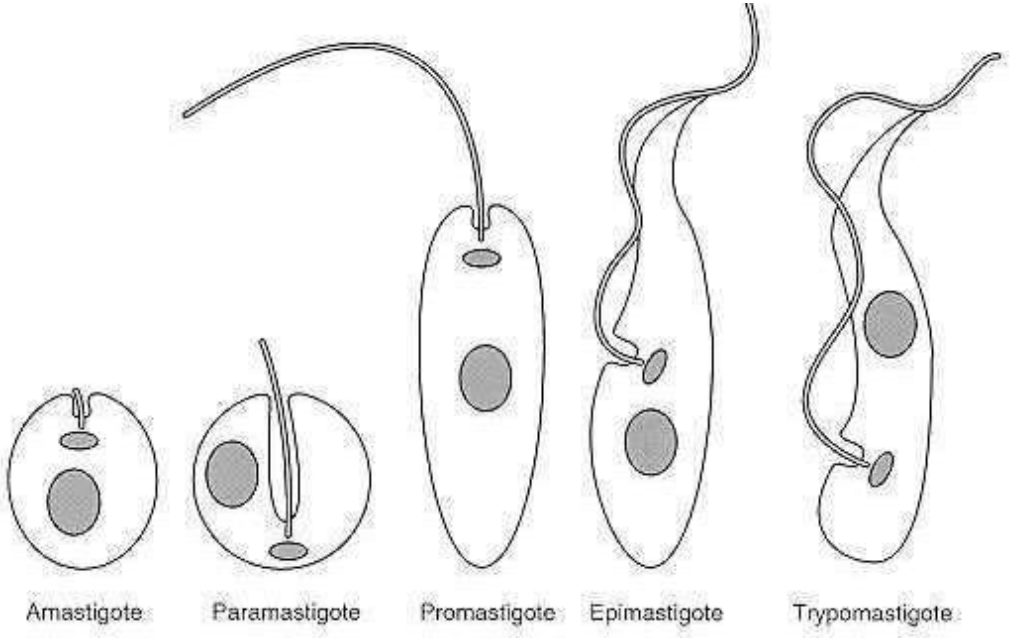
- Trypanosomes are hemoflagellates, spend part of their life cycle in the blood and tissues of vertebrates and part in invertebrates.
- Numerous variation in body shapes (pleiomorphic)are found in the host

A. **Trypanomastigote....** **Kinetoplast** posterior to nucleus, **undulating membrane** runs length of organism.

B. **Epimastigote....** **Kinetoplast** anterior to nucleus, **undulating membrane** running a portion of the body.

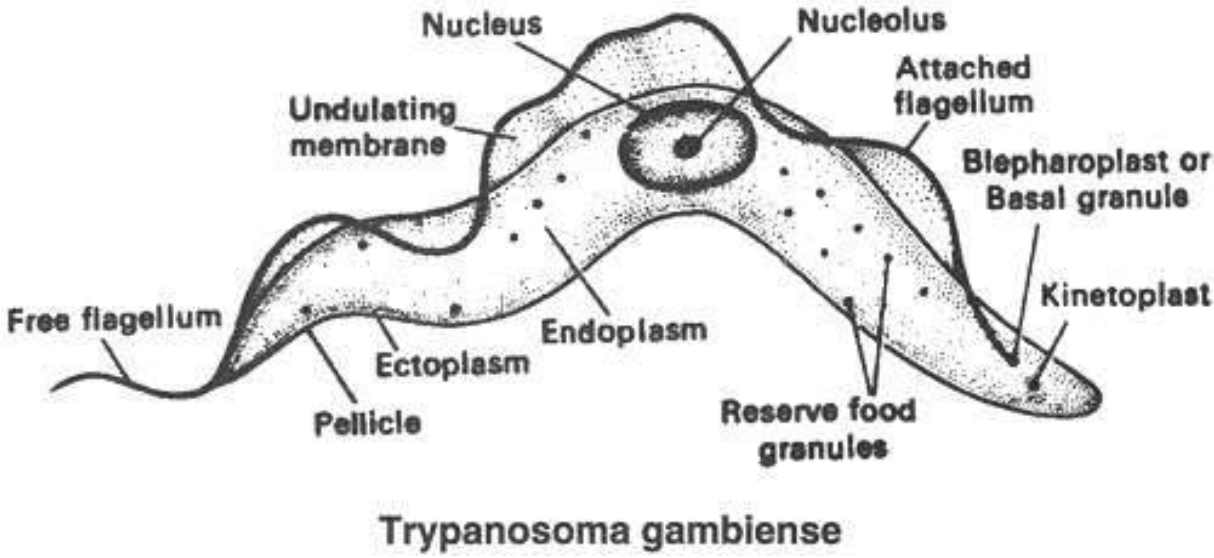
C. **promastigote....** **Kinetoplast** anterior to nucleus, no **undulating membrane**, with free flagellum.

D. **Amastigote....** **Kinetoplast** anterior to nucleus, no **free flagellum**, usually spheroid .



Trypanosome gambiense

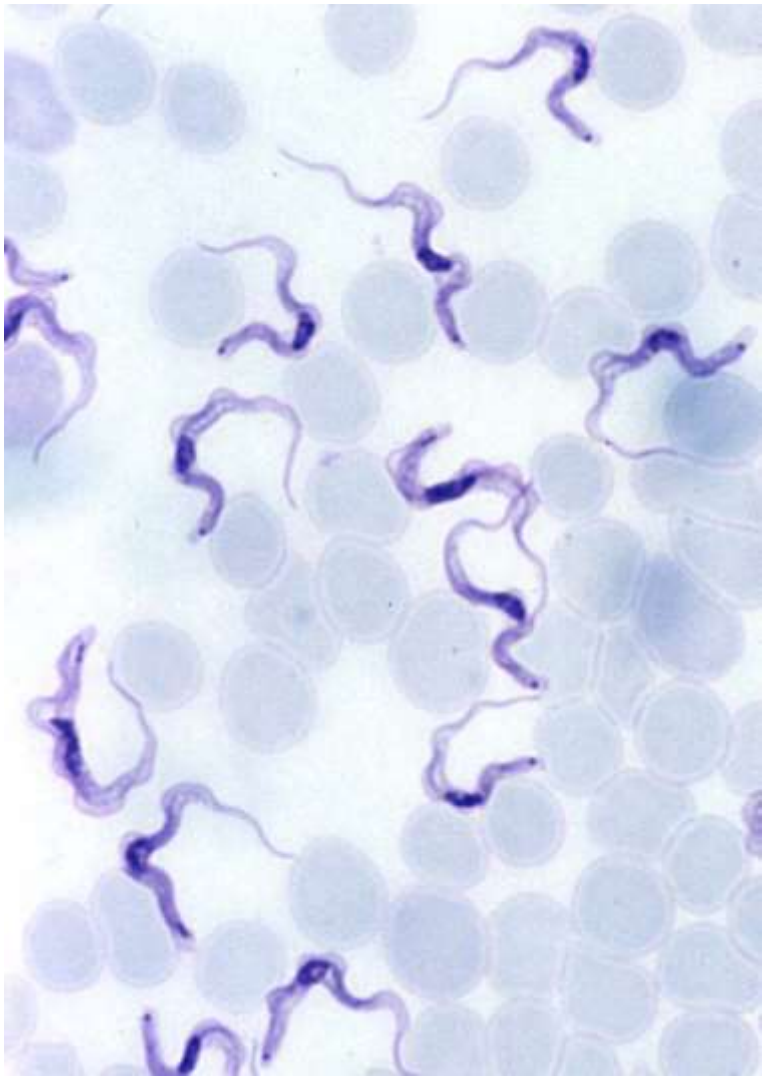
- ***Disease ...*** Gambian trypanosomiasis, West and Mid-African sleeping sickness(in human), and Nagana (in cattle).
- ***Geographic distribusion....*** Tropical West and Central Africa.
- It is transmitted to humans through the bite of a **tsetse fly** of the genus ***Glossina***
- Human trypanosomiasis is therefore a **vector-borne parasitic disease**



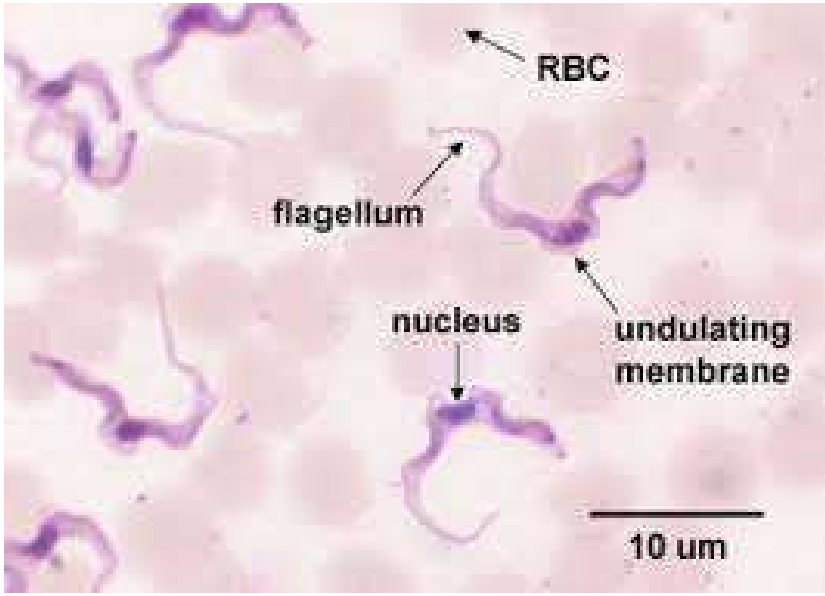
Trypanosoma gambiense

Morphology

- In human blood the polymorphic *T.gambiense* range from long, slender trypanosomal, to short, blunt forms without free flagella.
- In severe parasitemias the proportion of slender forms is highest and that of the stumpy forms lowest.
- In the spinal fluid all sizes and shapes occur, including multiple forms and even round.



- The length of *T.gambiense* range from **15-30 m.**
- The cytoplasm is granular and sometimes contains volutin granules and vacuoles,
- The nucleus is centric in its position with large central karyosome.
- At the posterior end of the trypanosome is a dark red mass consisting of the almost parabasal body and blepharoplast.
- The broad undulating membrane, which originates near the blepharoplast and passes wavily forward to the anterior end of the trypanosome to project as a free flagellum



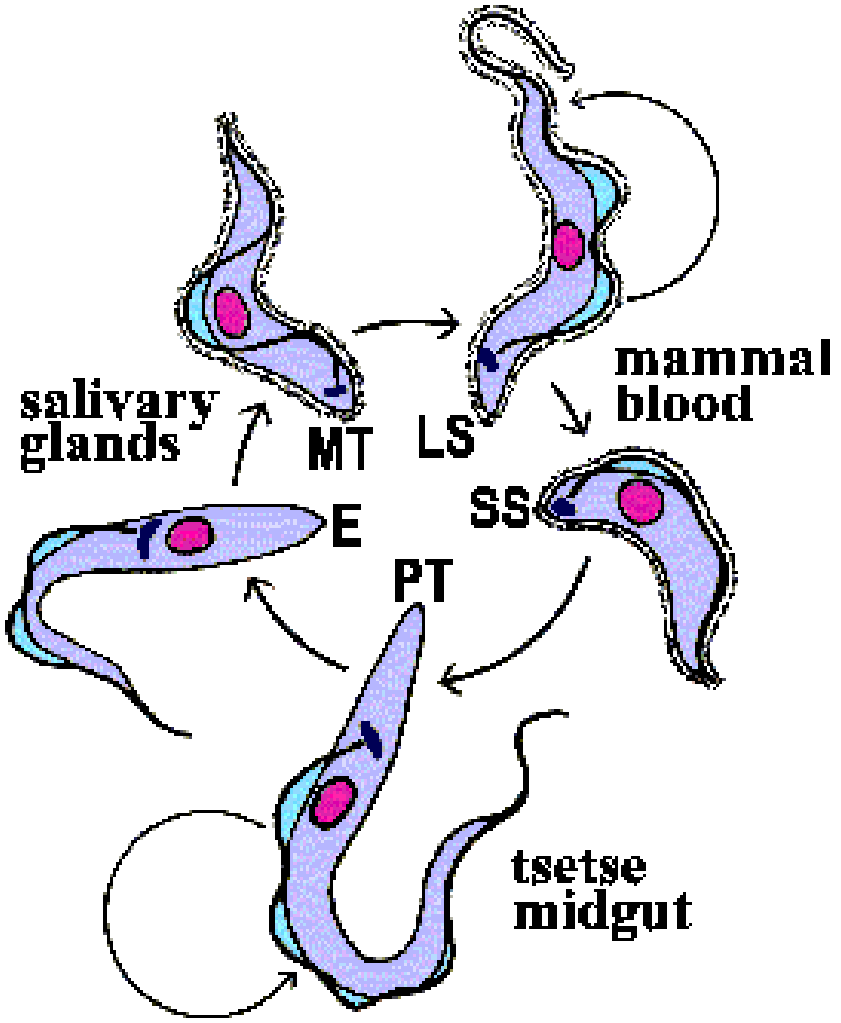
- ***Nutrition…*** *T.gambiense* absorbs nourishment from the **blood plasma, lymph, cerebrospinal fluid, or products of cellular disintegration.**
- ***Reproduction ….***The typanosomes divided by binary longitudinal fission.
- **In spinal fluid**, several division of nucleus and kinetoplast may occur before the cytoplasm separates

LIFE CYCLE

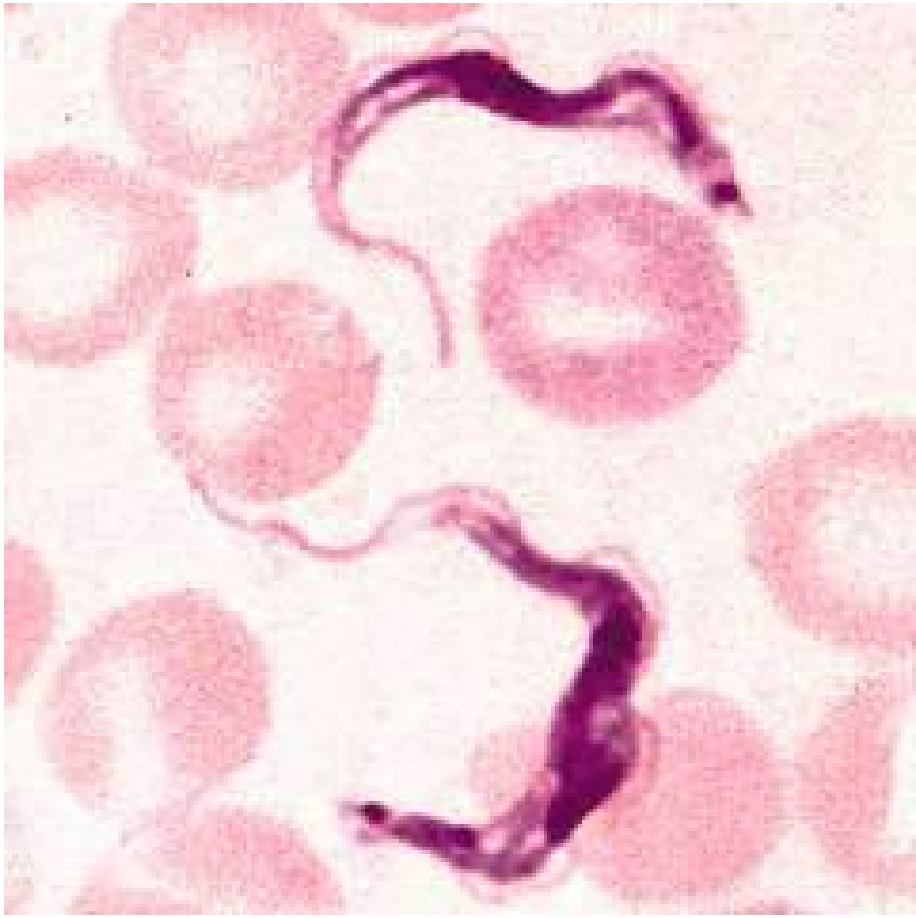
- The life cycle of most mammalian trypanosomes involves an alternate existence in vertebrate (man) and invertebrate (***Glossina* tsetse flies**) hosts, the latter serving as the transmitting agent.

Man...

- The infective trypandsomes (metacyclic trypomastigotes, small and stumpy without free flagellum) gain access to man through the bite of the tsetse flies (salivarian = anterior station, the parasites developed in anterior portion of the insect gut).



- After entry into man, parasites remains in the local tissues at the site of the bite for several days and then multiplies in the blood **(slender, medium-sized, and stumpy forms).**
- The trypanosomes proliferate and gradually invade all the organs of the host **(the blood, lymph, and in the intercellular spaces of the lymph glands, spleen, liver, brain, and other organs).**
- Most of the parasites are effectively destroyed by the host's natural defenses, but some trypanosomes manage to evade the immune system by modifying their surface membrane, a process known as **antigenic variation.**



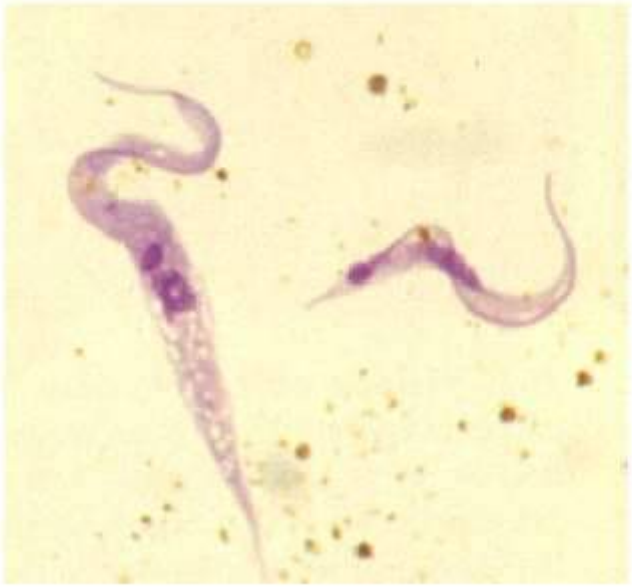
Tsetse flies...

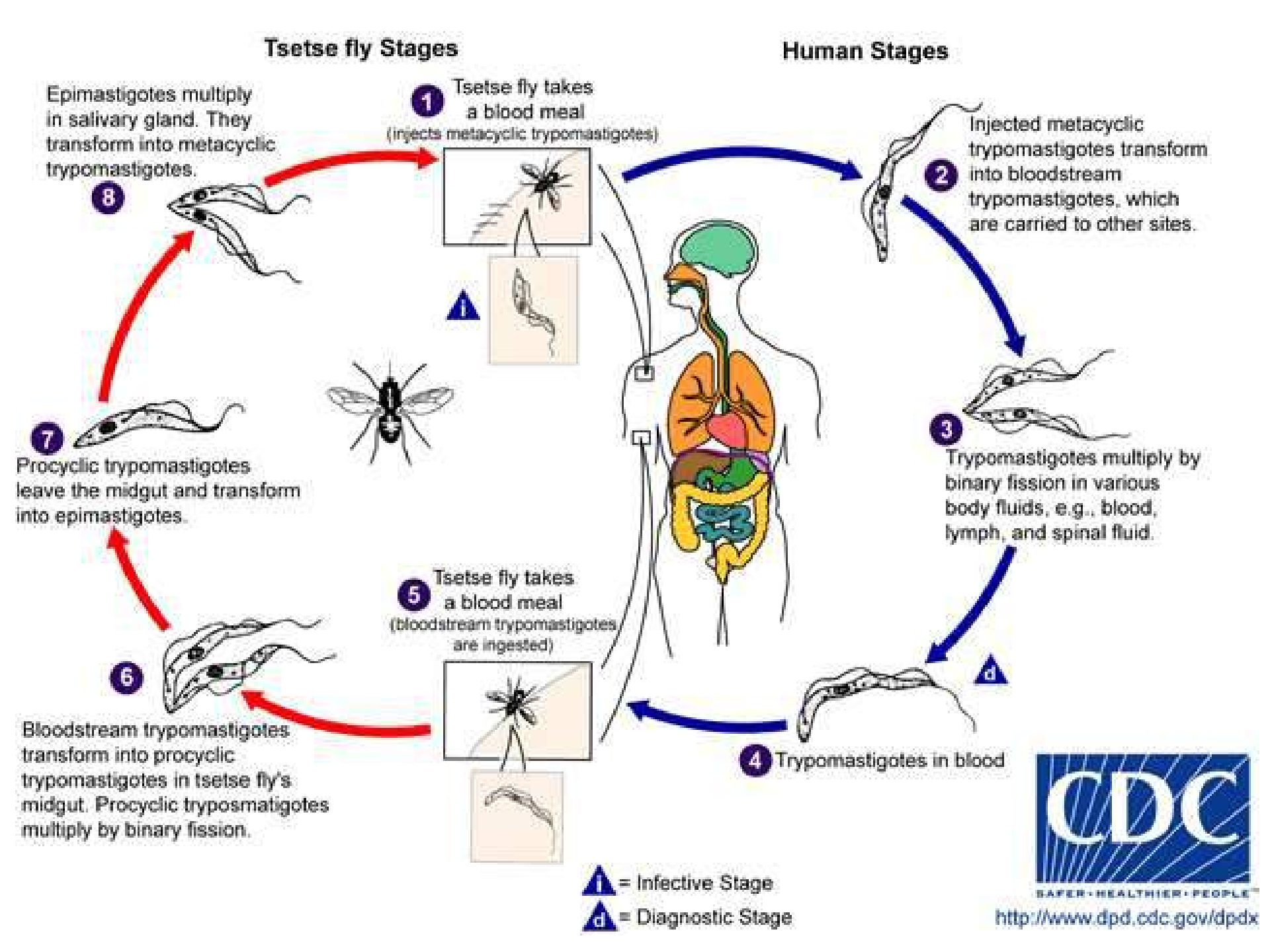
- Within two days after the fly has sucked infected blood contains **slender, medium-sized, and stumpy trypomastigotes,** only ***stumpy forms*** survive and being to multiply in the lumen of the mid-and hindgut, until by the tenth day a large number of broad forms are present, and transform into slender trypomastigotes.

- These forms migrate to foregut and remain for **12-20 days.**
- Migrate to salivary glands, and transform into epimastigotes, divide several times by longitudinal binary fission.
- Transform into metacyclic trypomastigotes
- During blood meal (feeding), metacyclic trypomastigotes injected into new host.

Trypanosoma cruzi

Epimastigote & Trypomastigote




Tsetse fly Stages
Human Stages
Epimastigotes multiply in salivary gland. They transform into metacyclic trypomastigotes.
8
1 Tsetse fly takes a blood meal
(injects metacyclic trypomastigotes)
i
2 Injected metacyclic trypomastigotes transform into bloodstream trypomastigotes, which are carried to other sites.
3 Trypomastigotes multiply by binary fission in various body fluids, e.g., blood, lymph, and spinal fluid.
d
4 Trypomastigotes in blood
5 Tsetse fly takes a blood meal
(bloodstream trypomastigotes are ingested)
6
Bloodstream trypomastigotes transform into procyclic trypomastigotes in tsetse fly's midgut. Procyclic tryposmatigotes multiply by binary fission.
7
Procyclic trypomastigotes leave the midgut and transform into epimastigotes.
i = Infective Stage
d = Diagnostic Stage
CDC
SAFER·HEALTHIER·PEOPLE™
http://www.dpd.cdc.gov/dpdx

Pathogenesity

- **At first, the main clinical signs of human trypanosomiasis are:**
- High fever,
- Weakness
- Headache,
- Joint pains and pruritus (itching).
- Gradually, the immune defense mechanisms and the patient's resistance are exhausted.

- As the parasite develops in the lymph and blood of the patient, the initial symptoms become more pronounced and other **manifestations such as :**
- Anaemia.
- Cardiovascular
- Endocrine disorders.
- Oedema
- Kidney disorders appear.

- In advanced stages of disease, the parasite **invades the central nervous system,**
- The patient's behavior changes.
- They can no longer concentrate
- Become indifferent to their environment.
- Sudden and unpredictable mood changes become increasingly frequent,
- Giving rise to lethargy with bouts of aggressiveness.

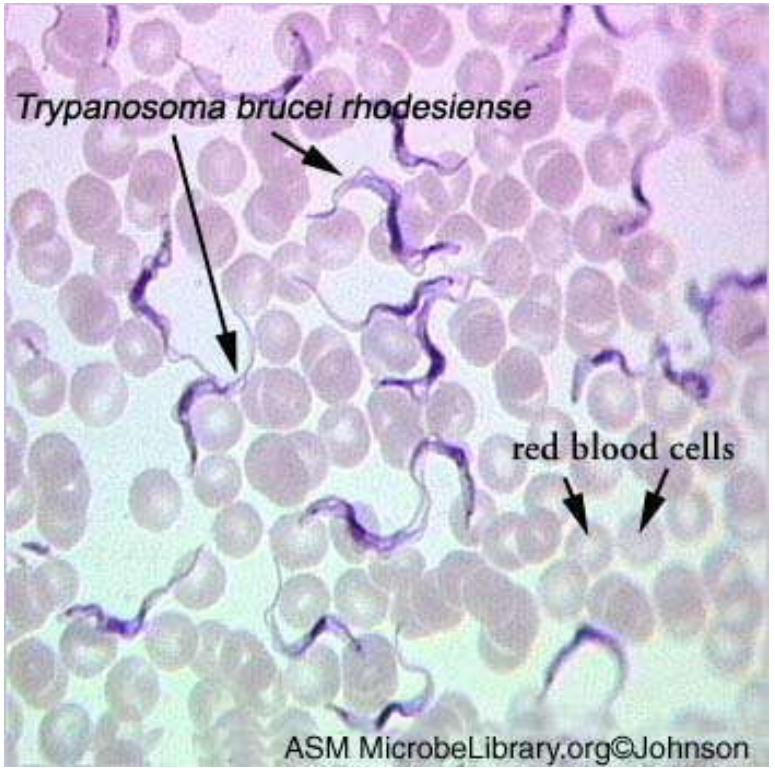
David Bruce

Brucella abortis ***Trypanosoma brucei***

- Patients are overcome by such extreme torpor that eating, speaking, walking or even opening the eyes call for an insurmountable effort.
- At night they suffer insomnia and during the day are exhausted by periods of sleep-like unconsciousness. Finally, patients fall into a deep coma and die.

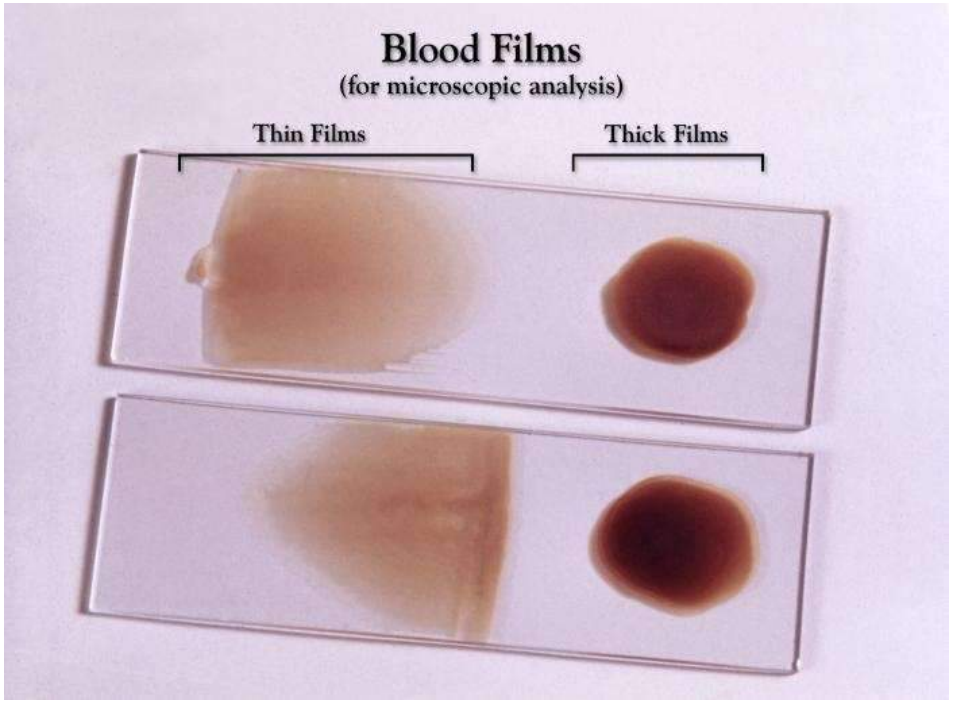
- There are two forms of African sleeping sickness, caused by two different parasites:
- ***Trypanosoma brucei gambiense ,*** which causes a chronic infection lasting years and affecting countries of western and central Africa

- ***Trypanosoma brucei rhodesiense*** , which causes acute illness lasting several weeks in countries of eastern and southern Africa
- ***T. rhodesiense*** **differs from** ***T. gambiense*** **in its greater virulence and in the production of a more acute and rapid disease.**
- The Rhodesian disease differs from Gambian in geographic distribution (central and east central Africa), low incidence, and fewer epidemics.

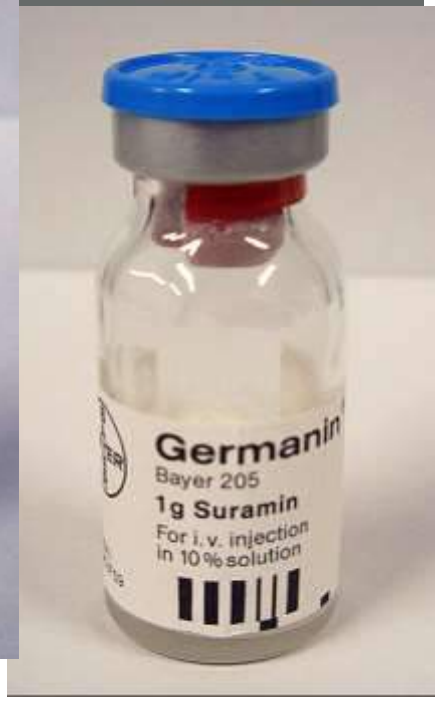


Diagnosis

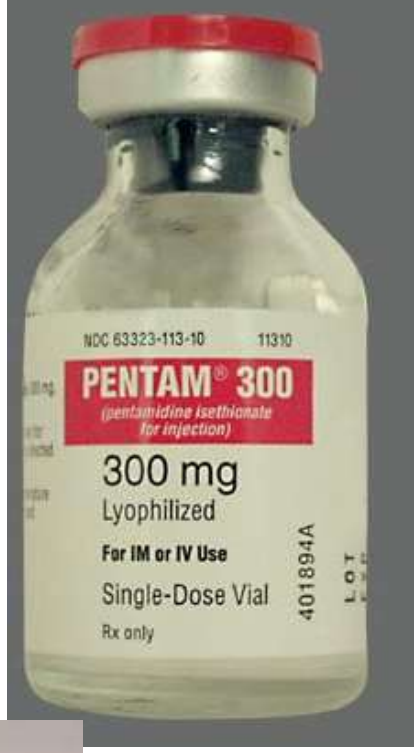
- Microscopic examination of thick and-thin blood films to investigate trypanosomes.
- Smears from bone marrow and CSF.
- ***Treatment***
- The type of treatment depends on the stage of the disease. The drugs used in the first stage of the disease are of lower toxicity and easier to administer.
- The earlier the disease is identified, the better the prospect of a cure.

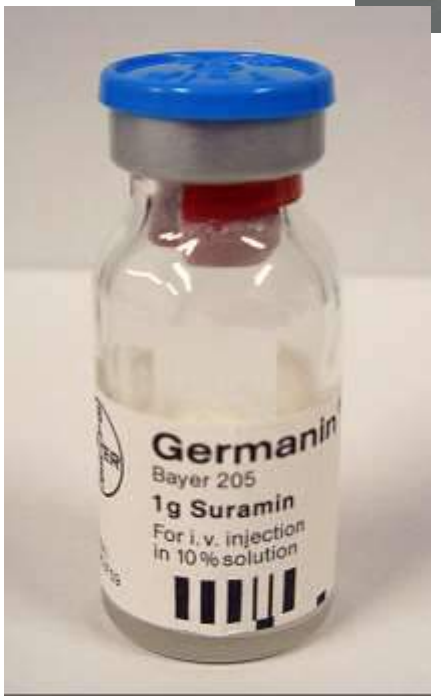


- Treatment success in the second stage depends on a drug that can cross the blood-brain barrier to reach the parasite. Such drugs are toxic and complicated to administer.
- Four drugs are-registered for the treatment-of sleeping sickness and provided free of charge to endemic countries.



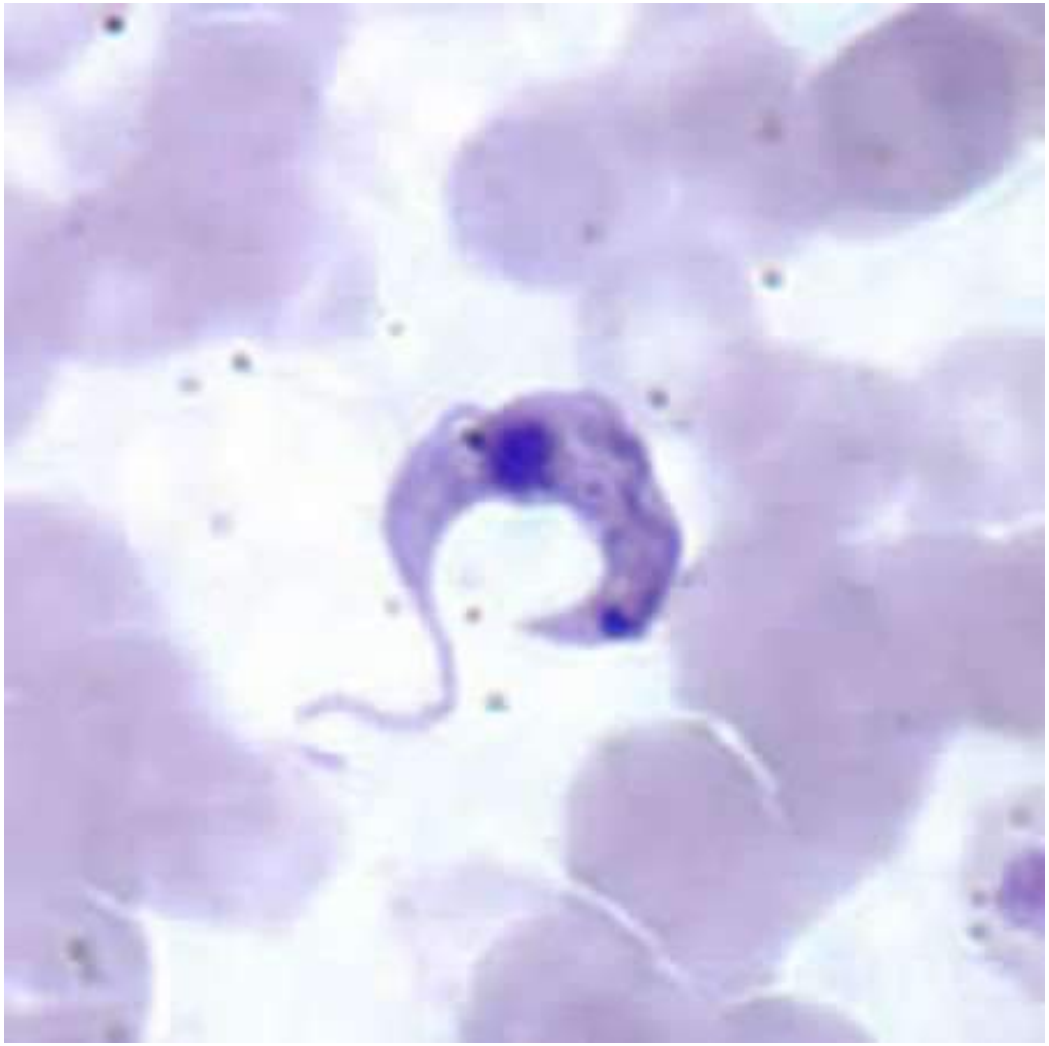
- First stage treatment:
- **Pentamidine.**
- **Suramin**
- Second stage treatment:
- **Meiarsoprol**





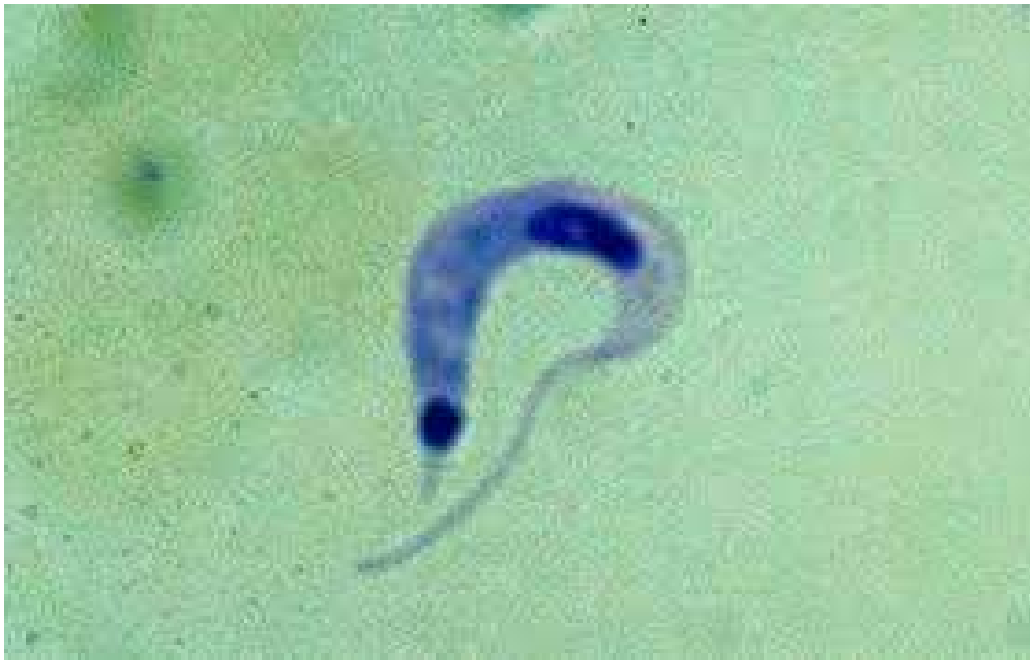
Trypanosome cruzi

- ***disease ….*** American trypanosomiasis, Chagas disease.
- ***geographic distribution….*** In North, Central and South America .



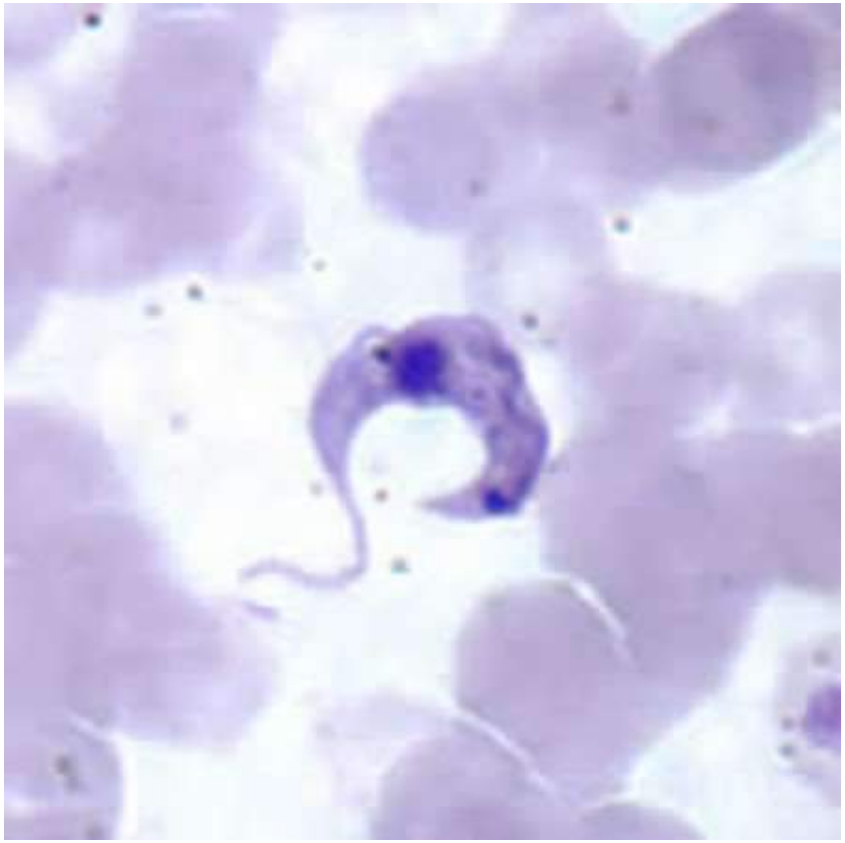
Morphology

- Trypomastigote in the blood are 16-20 in length and generally Lie in a **C-shape** in stained blood film.
- The kinetoplast is large and sub-terminal.

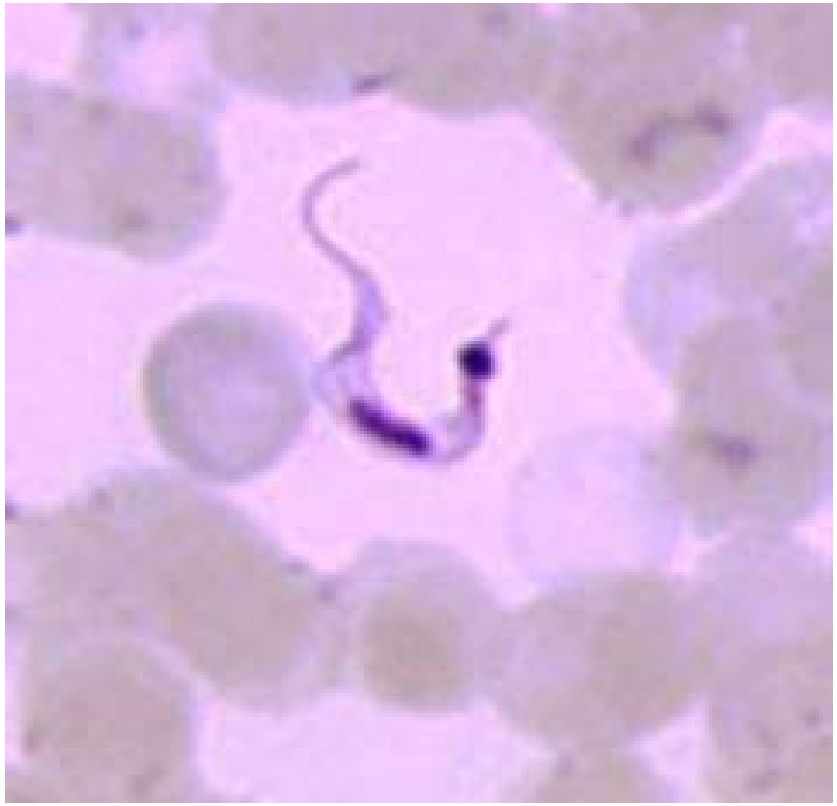


Life cycle

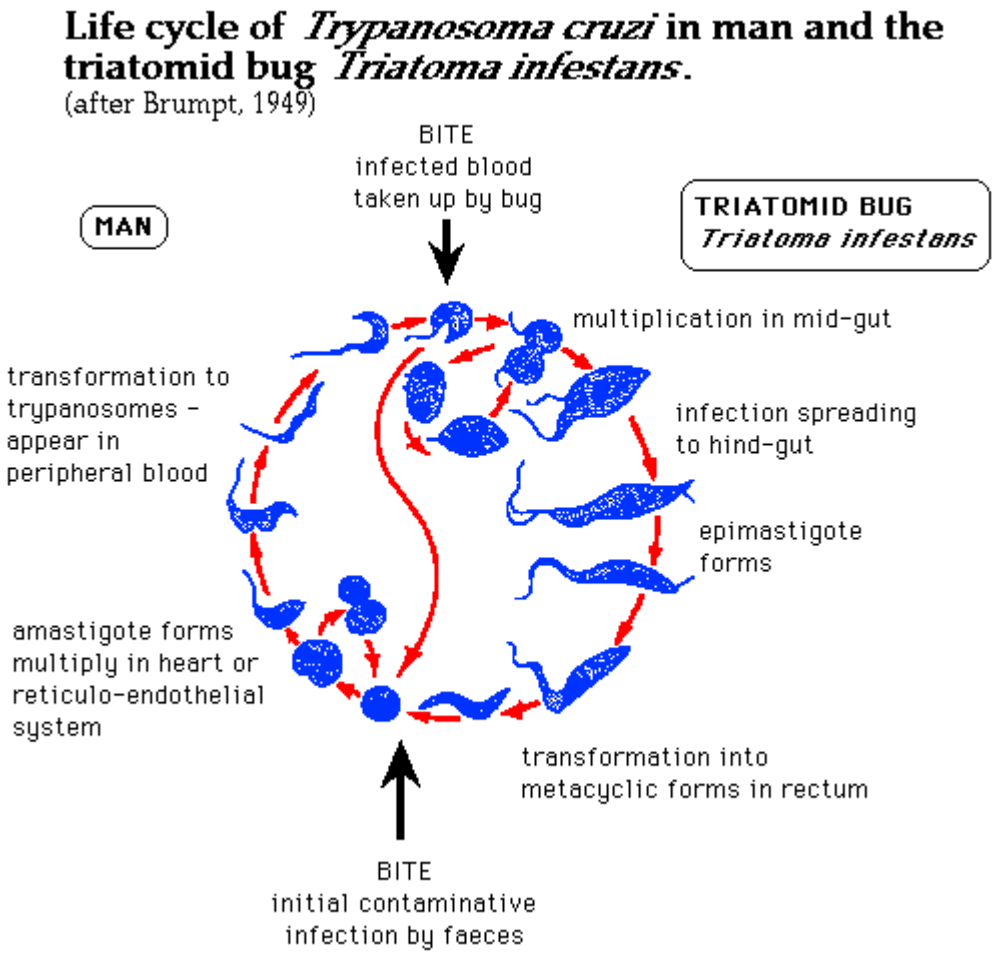
- Small numbers of trypomastigotes circulate in the blood of the mammalian host.
- The organisms are picked up by kissing bugs during blood feeding
- The organisms multiply as epimastigotesin the midgut and hindgut of the insect.
- One to two weeks after infection, depending on temerature, metacyclic trypomastigotes appear in the hindgut and the infection can then be transmitted back to another mammal.



- T. *cruzi* *is* ***stereorarian*** trypanosome and transmitted through fecal contamination.
- The fecal droplet of an Infected bug may contain thousands of metacyclic trypomastigotes.
- The trypomastigotes gain entry into the mammal when it scratches or rubs them into the skin, or they enter the mucous membranes of the eye, mouth, or nose.

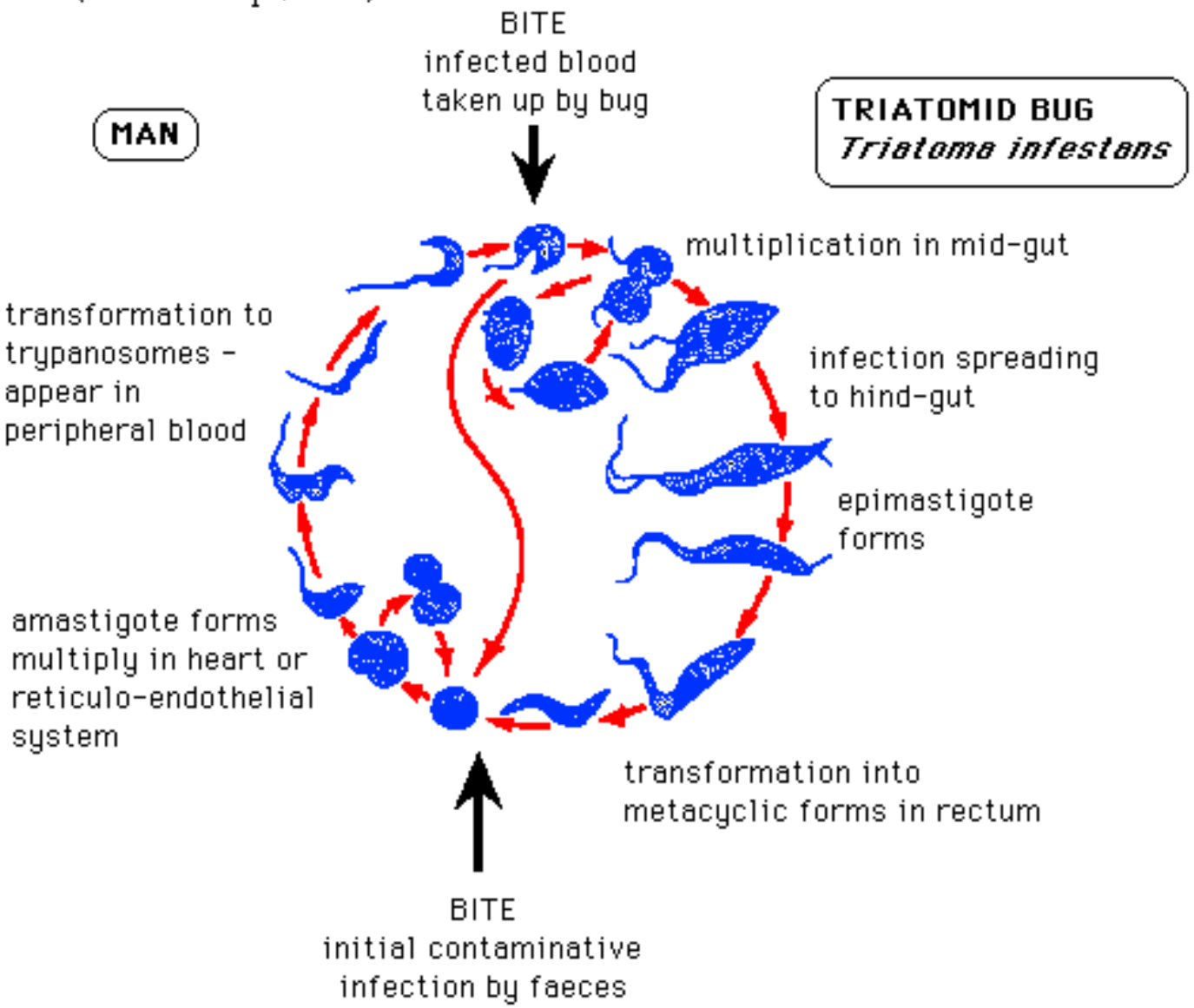


- Upon regaining a mammalian host, the trypomastigotes may remain in the bloodstream for a time, but they do not multiply there.
- They invade cells of the reticuloendothelial system a skeletal and cardiac muscle among other tissues.
- Multiplication is by binary fi the amastigote form.
- As host cells break down, so organisms are released into bloodstream.
- These free organisms transf into trypomastigotes in the bloodstream and are availab picked up when a kissing bu on the host.
- Trypomastigotes do not multiply in the bloodstream



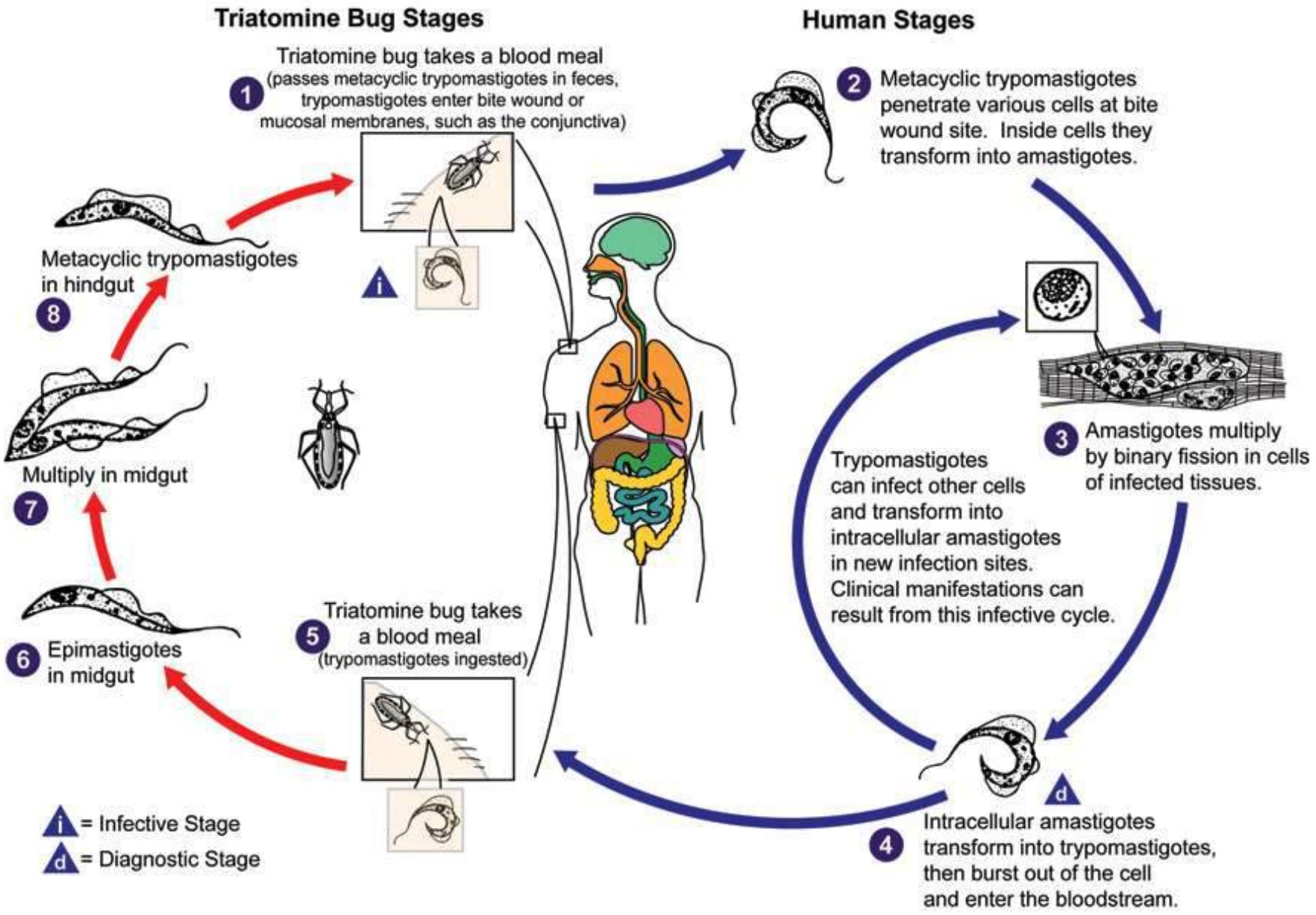
Life cycle of *Trypanosoma cruzi* in man and the triatomid bug *Triatoma infestans*.

(after Brumpt, 1949)



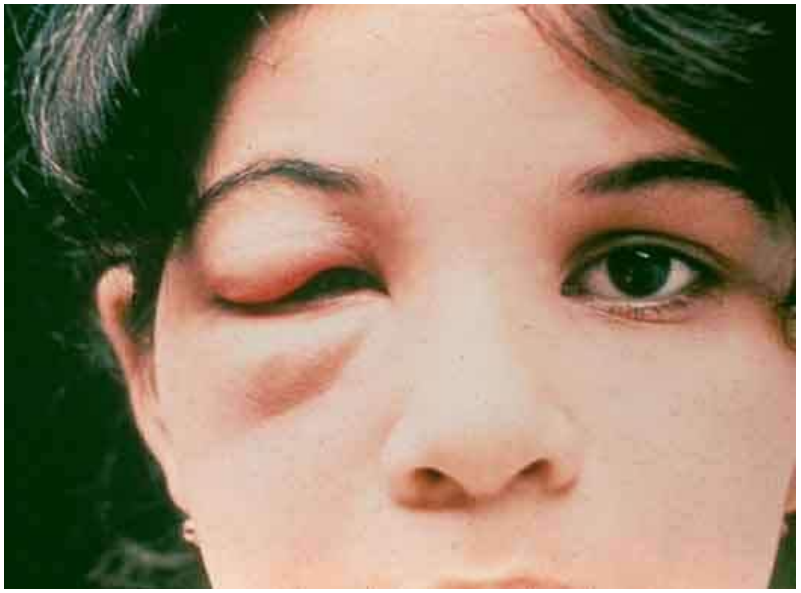
Trypanosomiasis, American (Chagas disease)

(Trypanosoma cruzi)



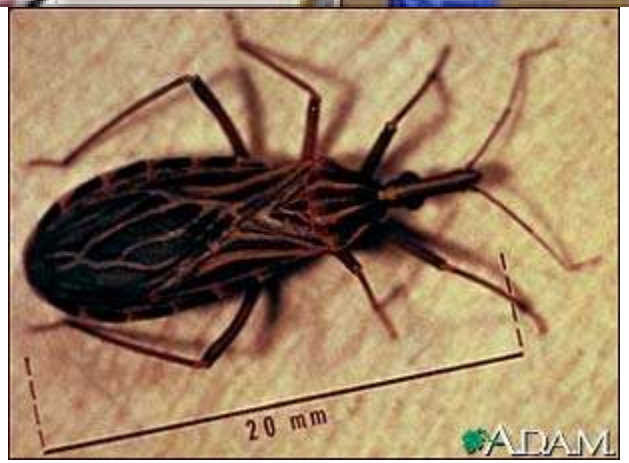
Pathogenicity

1. local acute inflammation at bite site (chagoma) if this occurs near eye there is a swelling of eyelid area, termed Romanas sign
2. Dissemination of parasites throughout body, resulting in destruction of parasitized cell.
3. Gradual degeneration of tissues throughout body, the most sever of which are muscles cells which cannot be replaced.
4. Edema, chills, fever, muscle pain and weakness, megaesophagus, megacolon, heart failure, death.
5. The disease manifestations of this parasite are collectively termed (Chagas disease).

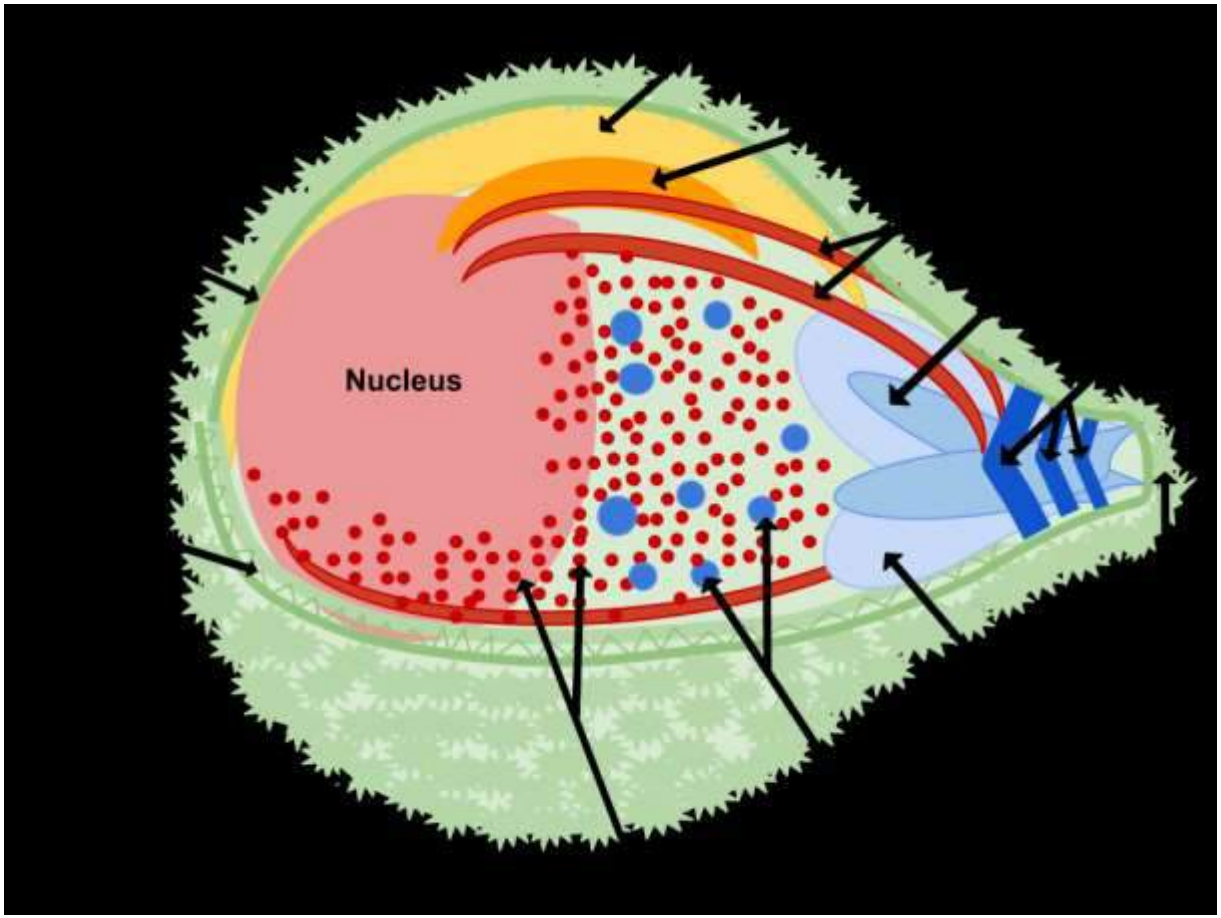


Diagnosis:

1. Microscopic examination of stained peripheral blood film show trypanosomal forms in C Shape.
2. Stained film of lymph node fluid show amastigotes
3. Serological tests
4. Xenodiagnosis (triatomid bugs fed on the patients blood develop trypanosomes in the gut).



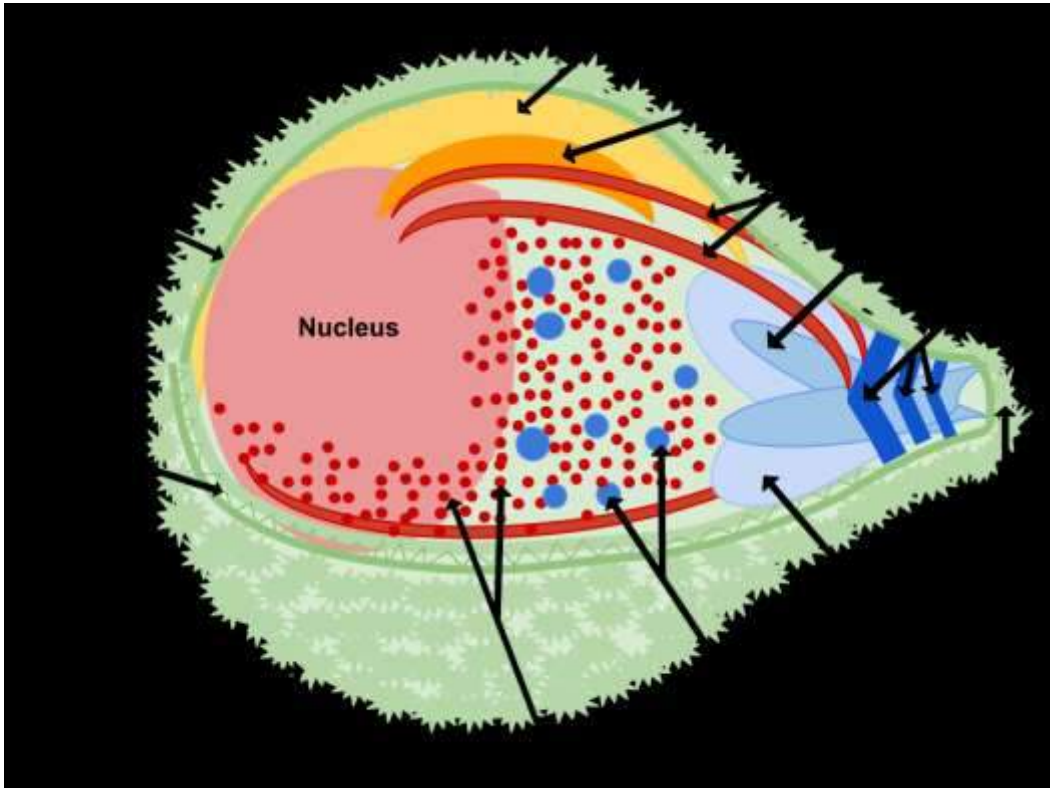
Superclass Sporozoa



Lec.Dr.Ruwaidah F. Khaleel

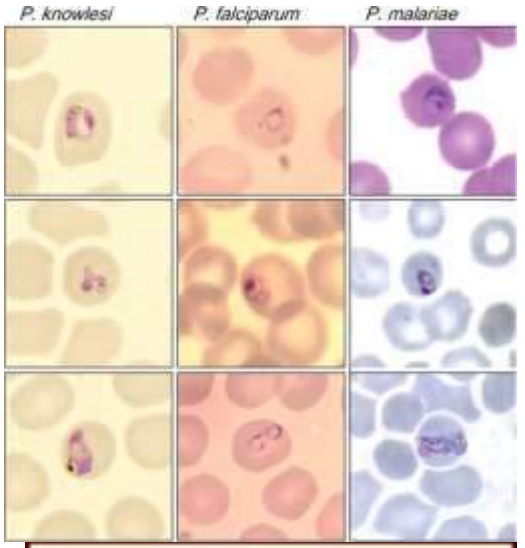
Superclass Sporozoa
Plasmodium

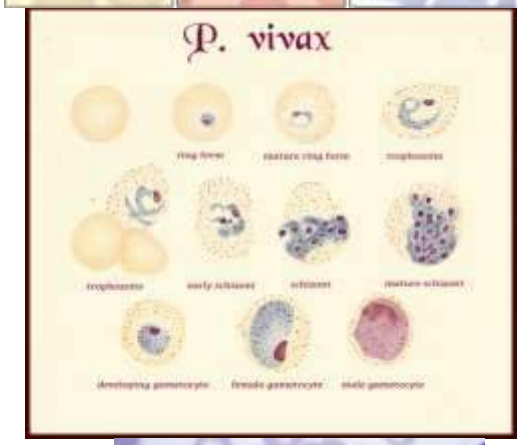
- ***Plasmodium*** is a genus of parasitic **protists**. Infection by these organisms is known as **malaria.** Currently over **200** species of this genus are recognized and new species continue to be described.
- **Classification:**
- **Phylum**: Apicomplexa
- **Class:** Aconoidasida
- **Order:** Haemosporida
- **Family:** Plasmodiidae
- **Genus** *:Plasmodium*

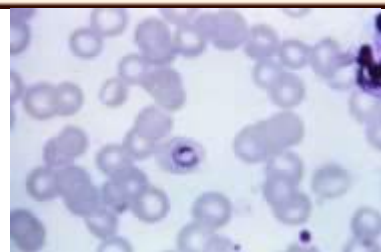


Five species of the plasmodium parasite can infect humans:

- *Plasmodium falciparum* cause **(malignant tertian malaria)** is the most widespread and dangerous of the four, untreated it can lead to fatal **cerebral malaria.**
- *Plasmodium vivax* cause **(benign tertian malaria).**
- *Plasmodium ovale* cause **(ovale tertian malaria).**
- *Plasmodium malariae* cause **(quartan malaria),**
- *Plasmodium knowlesi,* is a **zoonosis** that causes malaria in macaques but can also infect humans.

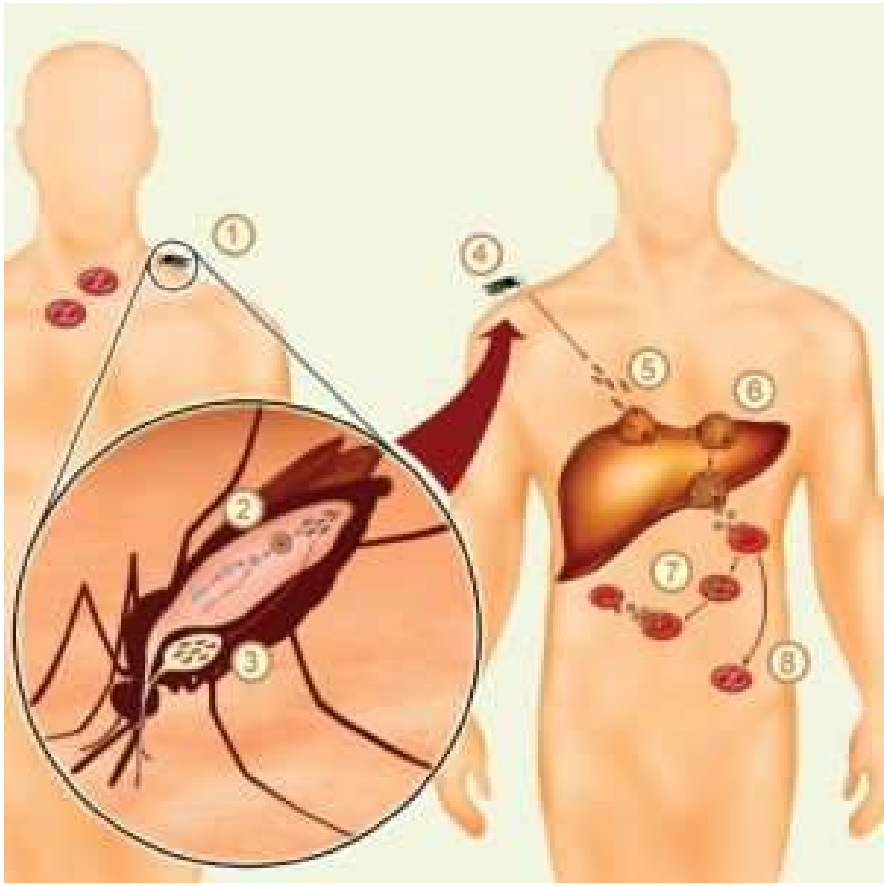




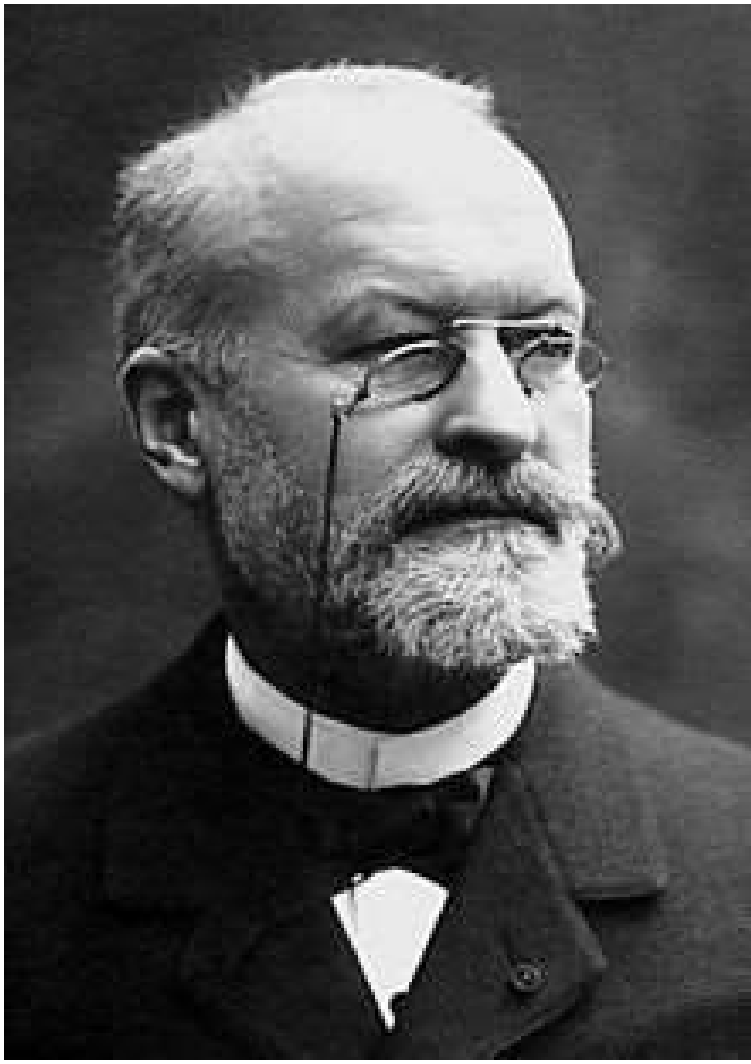


Disease: Malaria

- Approximately **300** million people worldwide are affected by malaria and between **1-1.5 million** people die; from it every year mainly in **Africa**, **Asia** and **Latin America** because of **inadequate health structures** and **poor socioeconomic conditions,** The situation has become more complex in the last few years with die increase in resistance to the drugs.

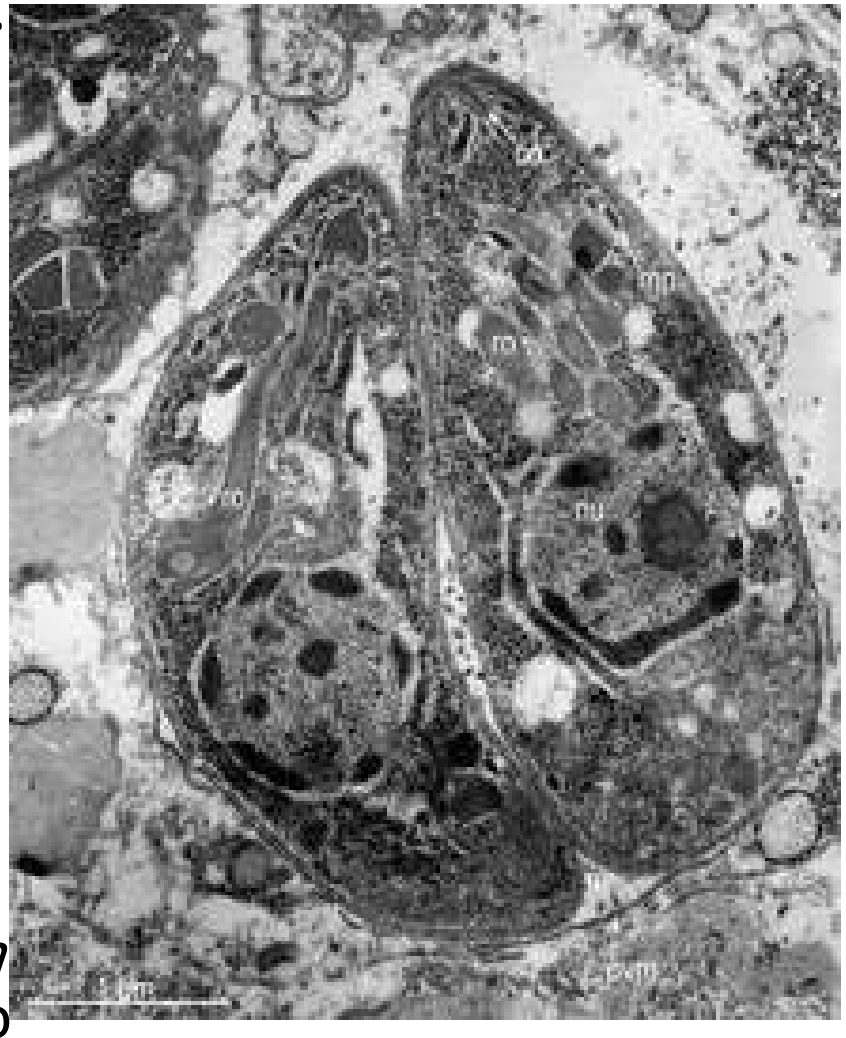


- Systematic control of malaria started after the discovery malaria parasite by **Laveran in 1889** (for which he received the **Nobel Prize for medicine in 1907).**
- Malaria parasites are transmitted from one person to another by the female **anopheline mosquito.** The males do not transmit the disease as they feed only on plant juices.



Characteristics:

- ***Plasmodium*** genus is parasites undergo **merogony** in erythrocytes.
- **{Merogony:** (multiple divisions of the nucleus followed by segmentation of the cytoplasm producing daughter cells called merozoites)}.
- ***Plasmodium*** produces **hemozoin** pigment, from hemoglobin digestion.
- ***Plasmodium*** has a **sexual** life stage in a definitive host which is a blood feeding insect mosquito's of the *genus* ***Anopheles*** (Species of the mosquito genera ***Aedes, Culex, Culiseta, Mansonia*** and *Theobaldia* can also transmit malaria but not to humans).



Life cycle:

- ***FIRST: Life cycle in mosquito host:***

1. A mosquito becomes infected when it takes a blood meal from an infected human at night.
2. In the mosquito's midgut, the gametocytes develop into **gametes** by **a** process known as **exflagellation** of the microgametocyte, then **eight** mobile microgametes are formed.
3. After fertilization of the macrogamete, motile zygote called **ookinete** is formed.
4. The ookinetes penetrate and escape the midgut, then embed themselves onto the exterior of the gut membrane and develop to **oocyst.** Here they divide many times to produce large numbers of tiny elongated **sporozoites.**
5. These **sporozoites** migrate to the salivary glands of the mosquito where they are injected into the blood and subcutaneous tissue of the next host the mosquito bites.

(*Plasmodium* life cycle in mosquito host)

Amastigote (infected) When takes blood meal from infected humen at night

Midgut (gametocytes) gametes(exflagellation)

Microgametocyte (8) mobile microgametes are formed

After fertilization (macrogamete)

Motile zygote (ookinete)

Penetrate and escape the mid gut

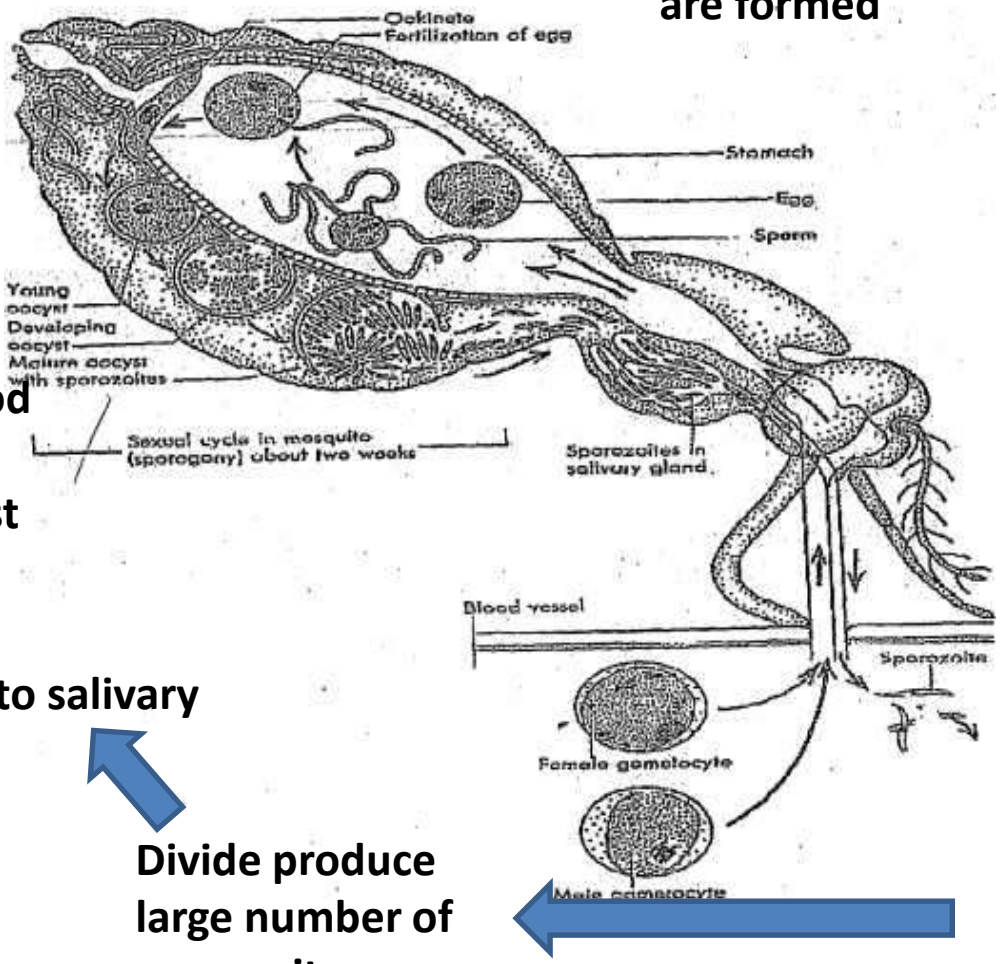
Embed onto exterior of gut membrane

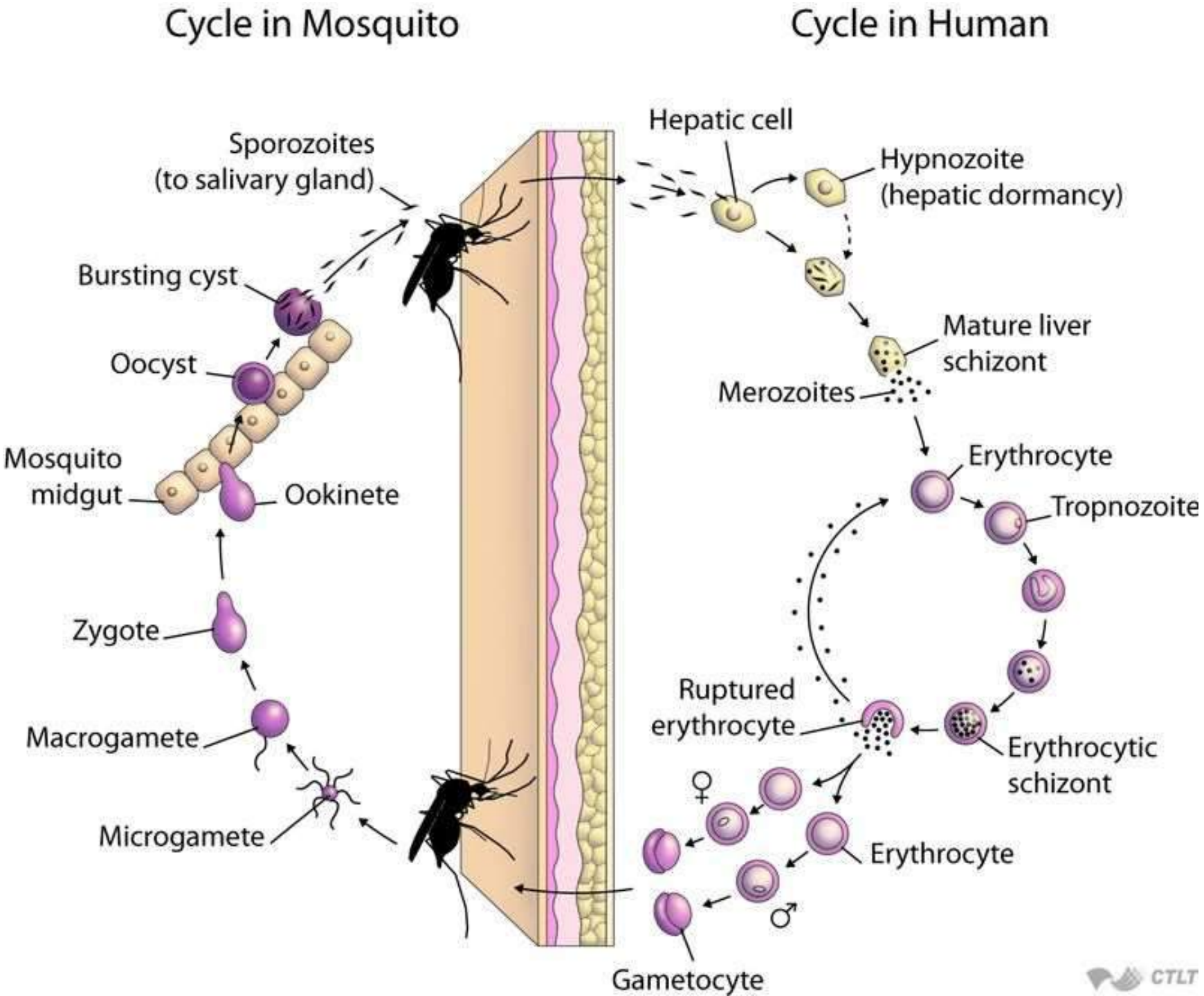
Develop oocyte

Divide produce large number of sporozoites

Migrate to salivary gland

Injected into blood and subcutanous tissue of next host

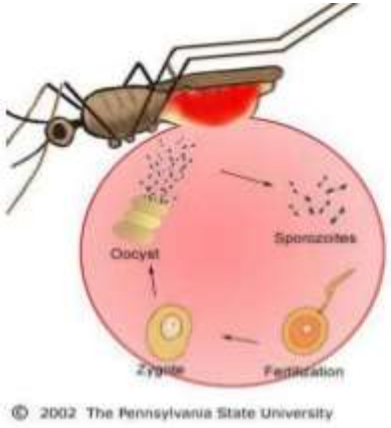


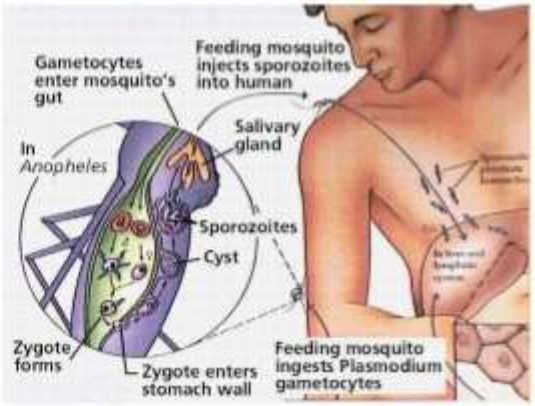

Cycle in Mosquito
Cycle in Human
Sporozoites
(to salivary gland)
Bursting cyst
Oocyst
Mosquito
midgut
Ookinete
Zygote
Macrogamete
Microgamete
Hepatic cell
Hypnozoite
(hepatic dormancy)
Mature liver
schizont
Merozoites
Erythrocyte
Tropnozoite
Ruptured
erythrocyte
Erythrocytic
schizont
♀
Erythrocyte
♂
Gametocyte
CTLT

SECOND: Life cycle in human host:

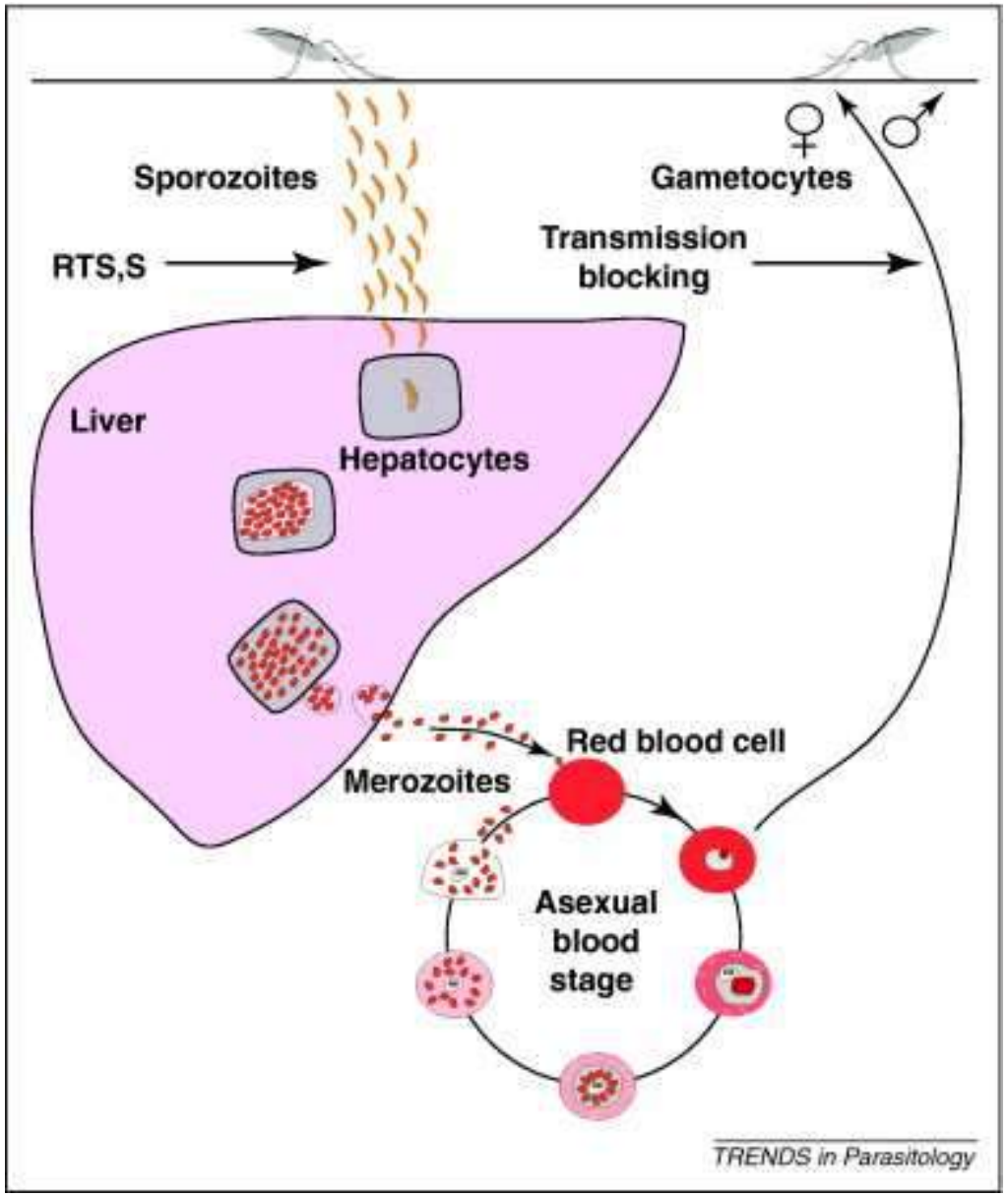
In human host malaria develops via two phases:

- **An exoerythrocytic phase** (in the hepatic system or liver).
- **An erythrocytic phase** (in the erythrocytes, or red blood cells).

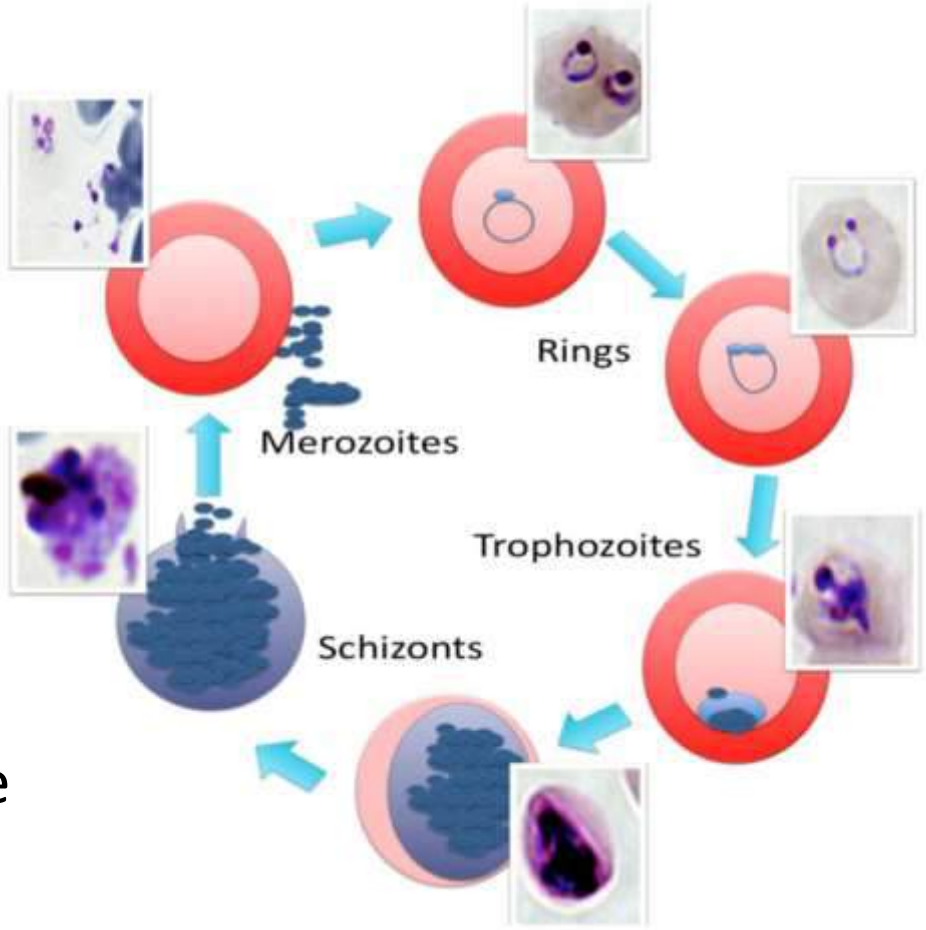




An exoerythrocytic phase

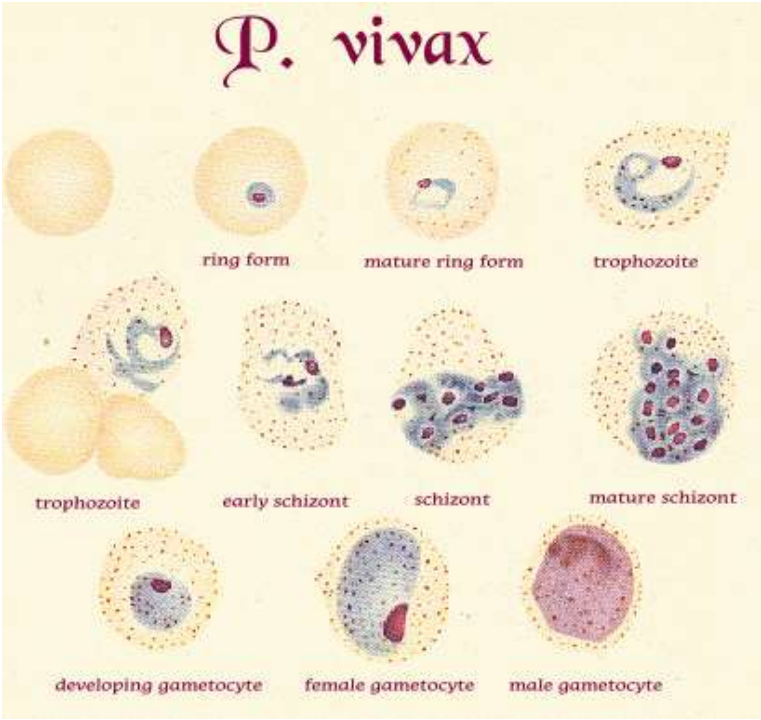


1. When the mosquito pierces a person's skin to take a blood meal, sporozoites in the insect saliva enters the bloodstream and migrates to the liver.
2. The sporozoites infect hepatocytes, multiplying asexually and asymptomatically for a period of **8-30 days.** These organisms differentiate to yield thousands of merozoites, which, following rupture of their host cells, escape into the blood and infect red blood cells. The parasite escapes from the liver undetected by wrapping itself in the cell membrane of the infected host liver cell.

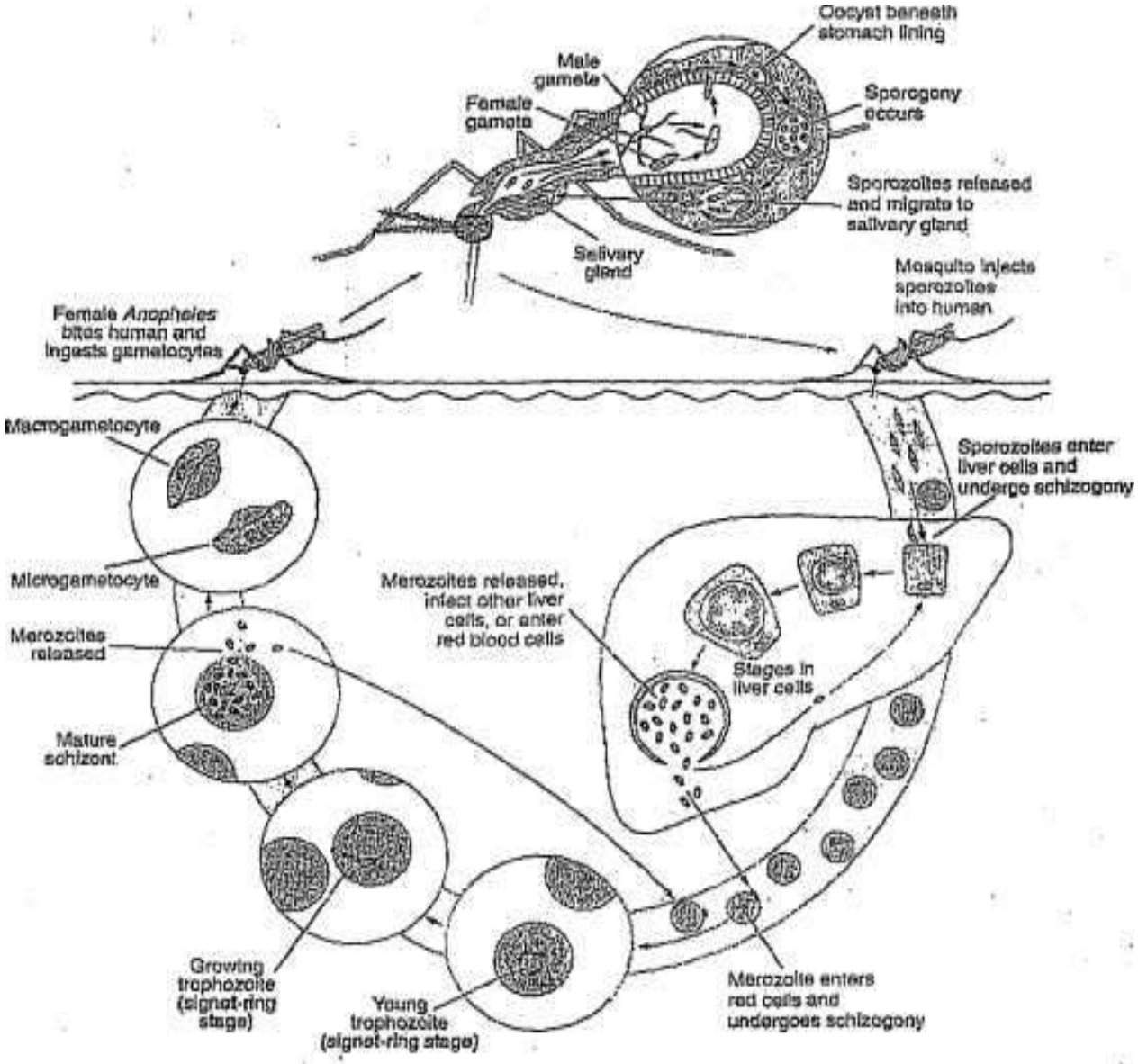


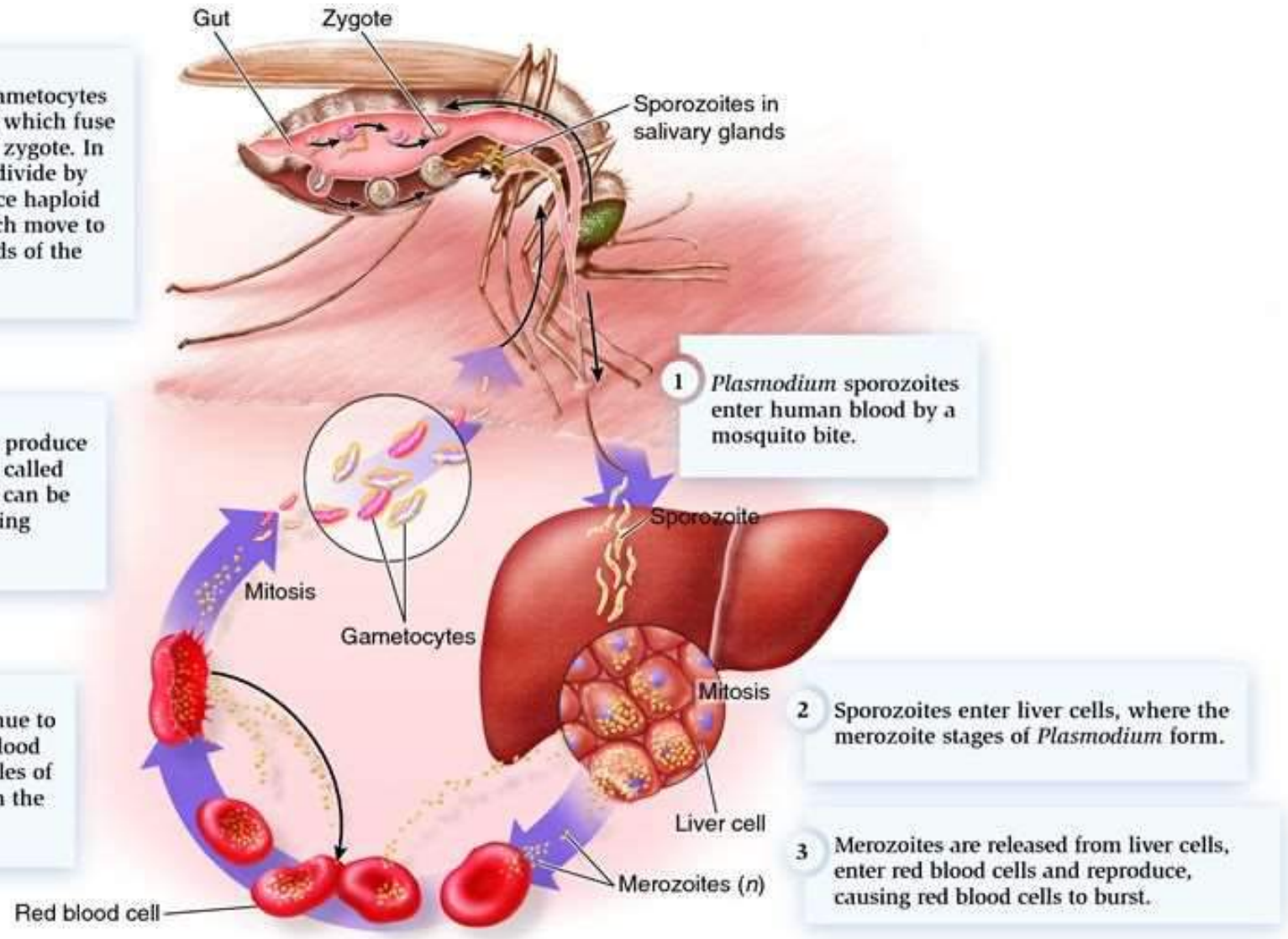
Note:

- Some ***P. vivax*** **and** ***P. ovale*** sporozoites do not immediately develop into exoerythrocytic-phase merozoites, but instead produce hypnozoites that remain dormant for periods ranging from several months (6-12 months is typical) to as long as three years.
- After a period of dormancy, they reactivate and produce **merozoites.**
- **Hypnozoites** are responsible for long incubation and late relapses in these two species of malaria.



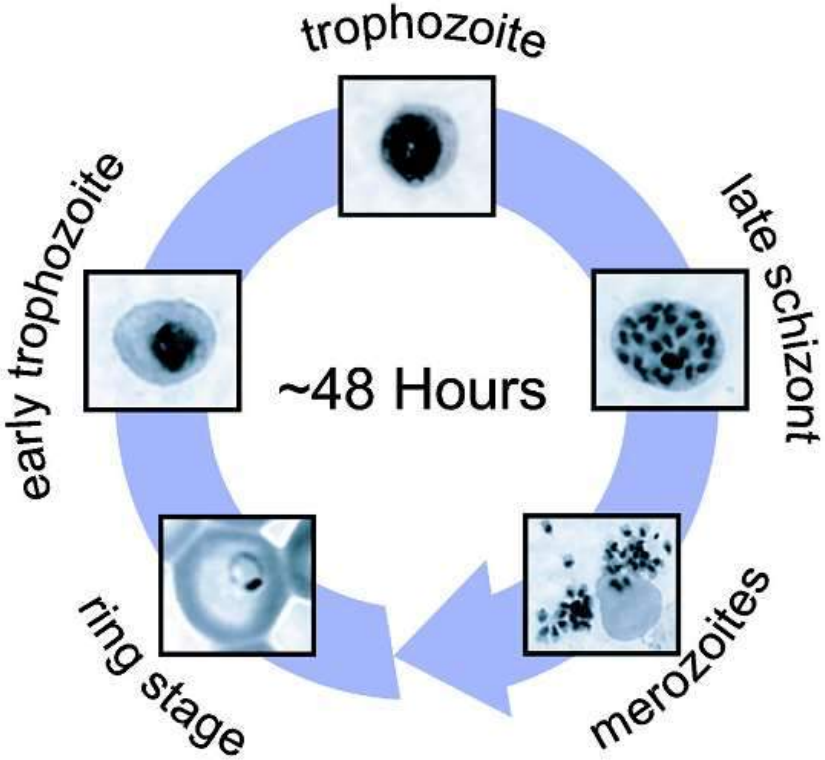
(Plasmodium life cycle in human host)




Gut
Zygote
Sporozoites in salivary glands
1 *Plasmodium* sporozoites enter human blood by a mosquito bite.
Sporozoite
2 Sporozoites enter liver cells, where the merozoite stages of *Plasmodium* form.
Mitosis
Liver cell
3 Merozoites are released from liver cells, enter red blood cells and reproduce, causing red blood cells to burst.
Merozoites (*n*)
Red blood cell
4 Merozoites continue to infect more red blood cells, causing cycles of chills and fever in the infected person.
Mitosis
Gametocytes
5 Some merozoites produce sexual structures called gametocytes that can be taken up by a biting mosquito.
6 In mosquitoes, gametocytes produce gametes which fuse to form a diploid zygote. In the gut, zygotes divide by meiosis to produce haploid sporozoites, which move to the salivary glands of the mosquito.

An erythrocytic phase:

1. **The merozoite** inside RBCs multiply asexually first to a ring-shaped form **(ring stage)** and then to a larger **trophozoite** form.
2. **Trophozoite** enlargement is accompanied by an active metabolism including the ingestion of host cytoplasm and the proteolysis of hemoglobin into amino acids. Multiple nuclear divisions without cytokinesis resulting is **a schizont.**
3. **Schizont stage** divides several times to produce new **merozoites,** which leave the RBCs and travel within the blood stream to invade new RBCs.

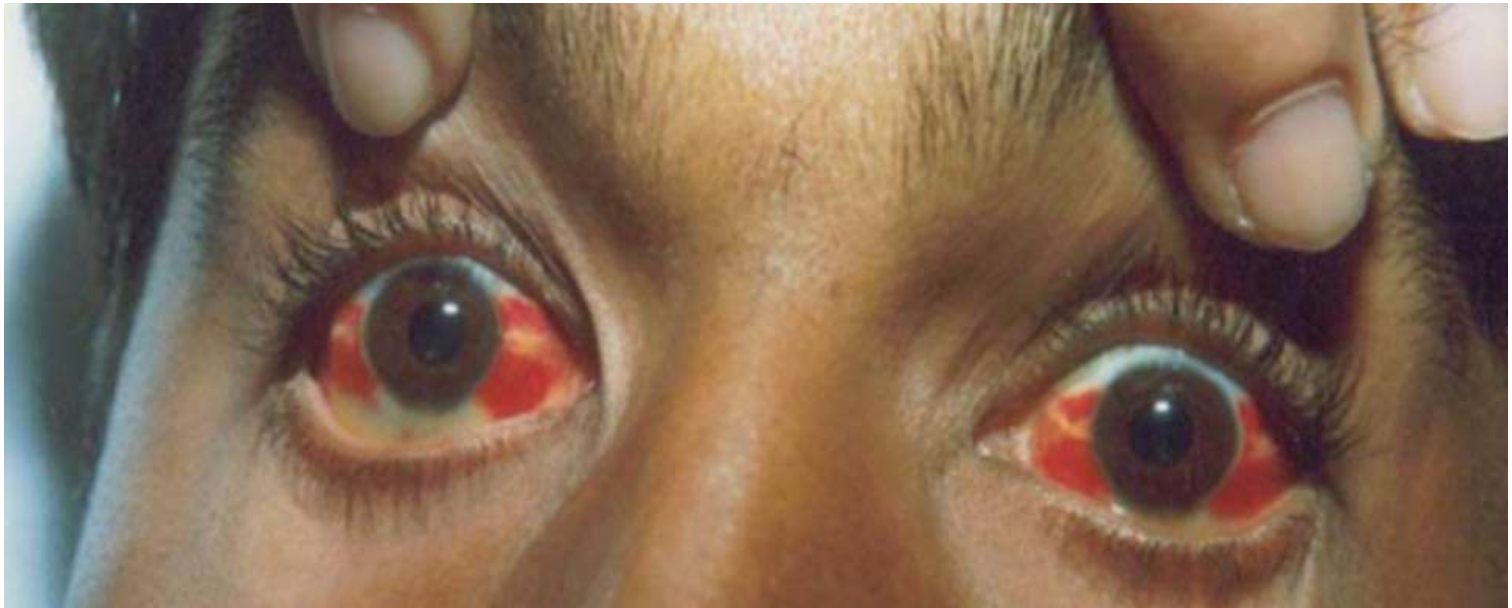


Note:

- The parasite feeds by ingesting hemoglobin and other materials from RBCs and damages them.
- The destruction of RBCs and the release of the parasites' waste products produce the episodic **chills and fever** that characterize the disease.

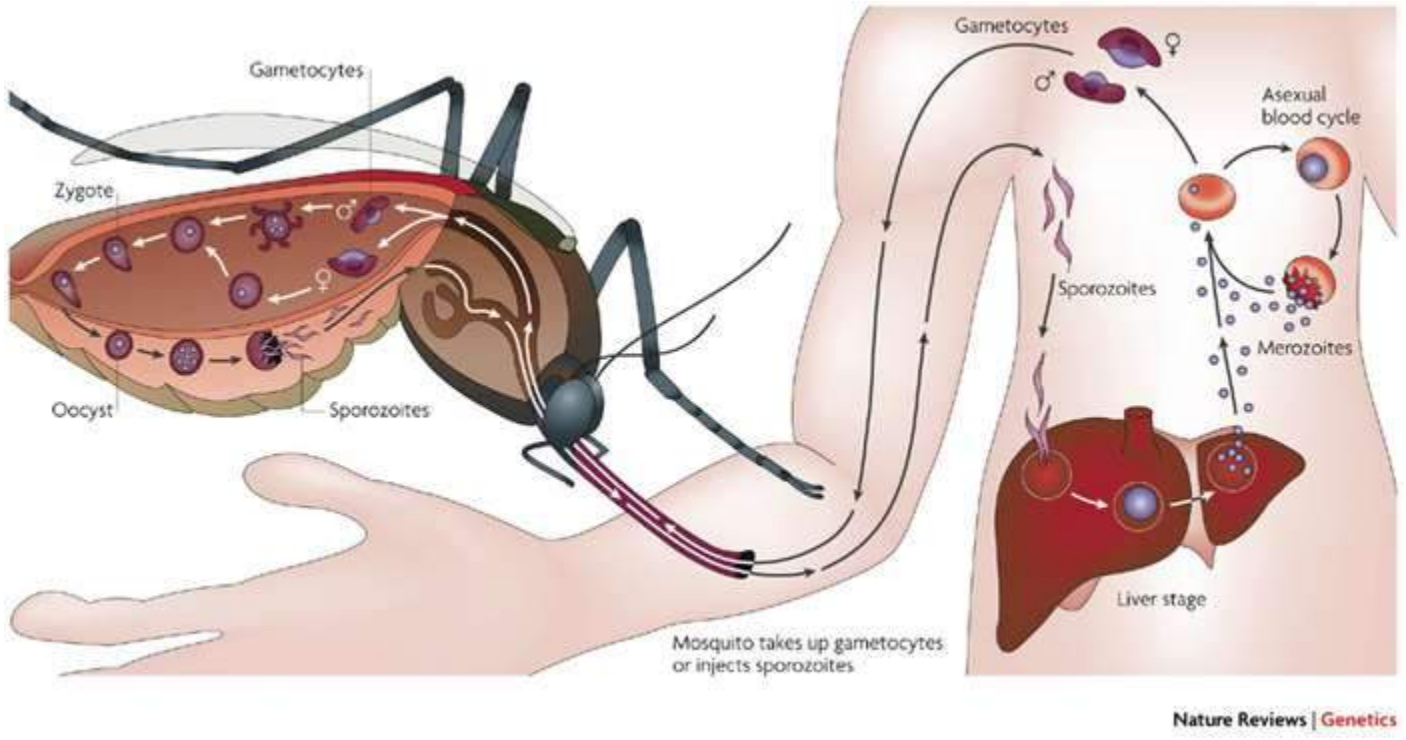
Note:

- The parasite resides within the liver and blood cells thus it is protected from attack, by the body's immune system. However, circulating infected RBCs are destroyed in the spleen, the ***P. falciparum*** parasite displays adhesive proteins on the surface of the infected RBCs, causing the blood cells to stick to the walls of small blood vessels, then giving rise to **hemorrhagic** complications of malaria.



4. Some merozoites turn into **male and female gametocytes.** If a mosquito pierces the skin of an infected person, it potentially picks up gametocytes within the blood.

- **Note:** In chronic infections in humans the gametocytes are often the only forms found in the blood.



Symptoms:

❖ **Symptoms of malaria include:**

- Fever,
- Shivering,
- Arthralgia (joint pain),
- Vomiting.
- Anemia (caused by hemolysis),
- Hemoglobinuria,
- Retinal damage, and convulsions. '

- Malaria is characterized by **episodic** attacks **of** chills and fever that coincide with mass destruction of blood cells:

I. Sudden coldness.

II. Rigor.

III. Fever and sweating lasting four to six hours.

- Occurring every **two days (48hr)** in *P. vivax* and *P. ovale* infections, while every **three days (72hr)** for *P. malariae. P. falciparum* can have recurrent fever every **36-48 hours.**

Severe malaria

- ✓ **is almost exclusively caused by *P. falciparum* infection, and usually arises 6-14 days after infection. Consequences include:**

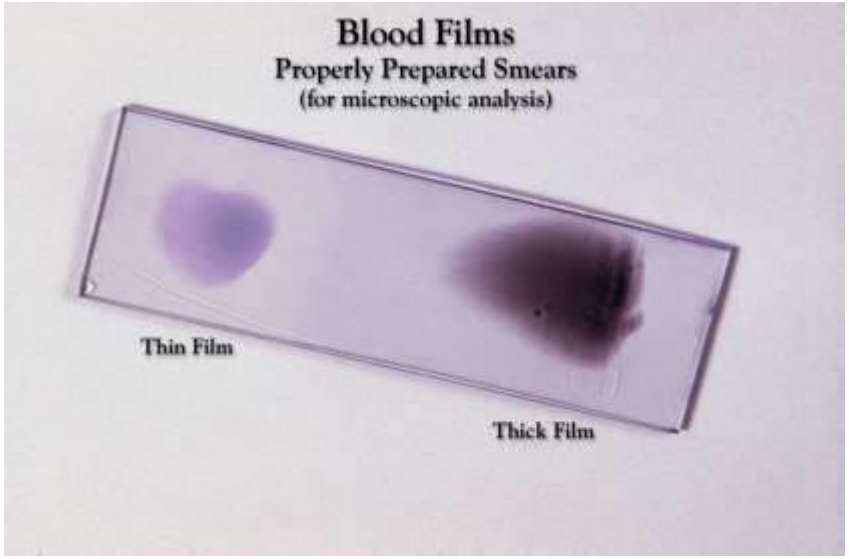
- ❑ Coma and death if untreated.
- ❑ Hepatomegaly & Splenomegaly.
- ❑ Severe headache.
- ❑ Cerebral ischemia.
- ❑ Hypoglycemia.
- ❑ Hemoglobinuria.
- ❑ Severe malaria can progress extremely rapidly and cause **death** within hours or days.

Chronic malaria

- is seen in both P. ***vivax*** and P. ***ovale,*** but not in *P.falciparum.* Here, the disease can relapse months or years after exposure, due to the presence of latent parasites **(hypnozoites)** in the liver.

Diagnosis:

- **1) Microscopic examination *of blood* film** (both saliva and urine have been investigated as alternative, less invasive specimens).
- **Thin blood films** allow species identification because the parasite's appearance is best preserved in this preparation.
- **Thick blood films** allow to screen a larger volume of blood and are about eleven times more sensitive than the thin film, so picking up low levels of infection is easier on the thick film,



2. Areas that cannot afford even simple laboratory diagnostic tests .often use only a **history of subjective fever** as the indication to treat for malaria.
3. ***Antigen detection tests.***
4. Molecular ' methods such as **real-time PCR** assays are being developed with the hope of being able to deploy them in endemic areas.

Treatment:

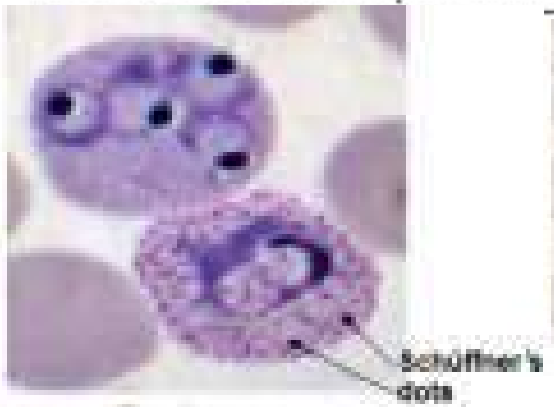
- **Quinine** was used historically; however the development of more effective alternatives such as :
- **Quinacrine,**
- **Chloroquine,**
- **Primaquine** in the **20th** century reduced its use.

❖ **Modem drugs used include**

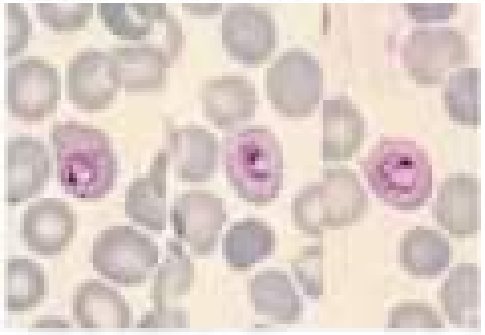
- **Mefloquine** (*Lariam*),
- **Doxycycline,**
- The combination of **atovaquone** and **proguanil hydrochloride** (*Malarone*).

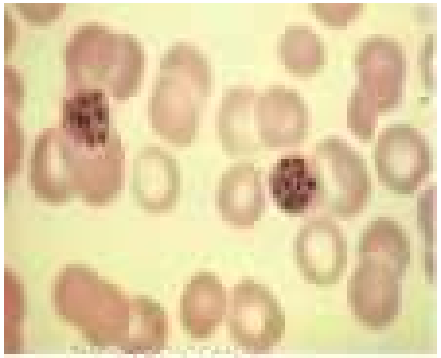
Key Morphological Differences Between Human *Plasmodium* Species in Blood Smears

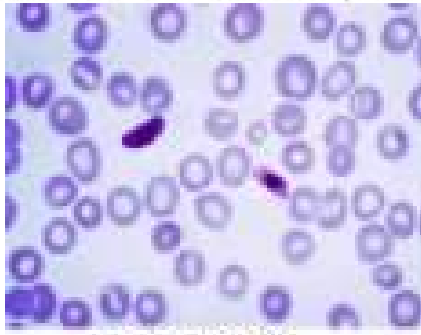
vivax	ovale	malariae	falciparum
• Enlarged erythrocyte.	• Similar to *P. vivax*.	• Compact parasite.	• Numerous rings.
			• Smaller rings.
• Schüffner's dots.	• Compact trophozoite.	• Merozoites in rosette	
	• Fewer merozoites in schizont.		• No trophozoites or schizonts.
• 'Ameboid' trophozoite.	• Elongated erythrocyte.		• Crescent.-shaped gametocytes.



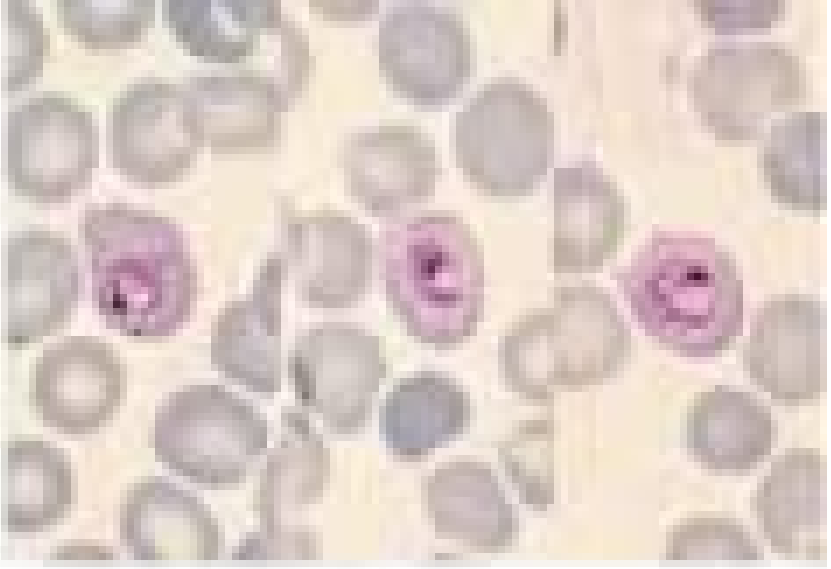
P. vivax


P. ovale

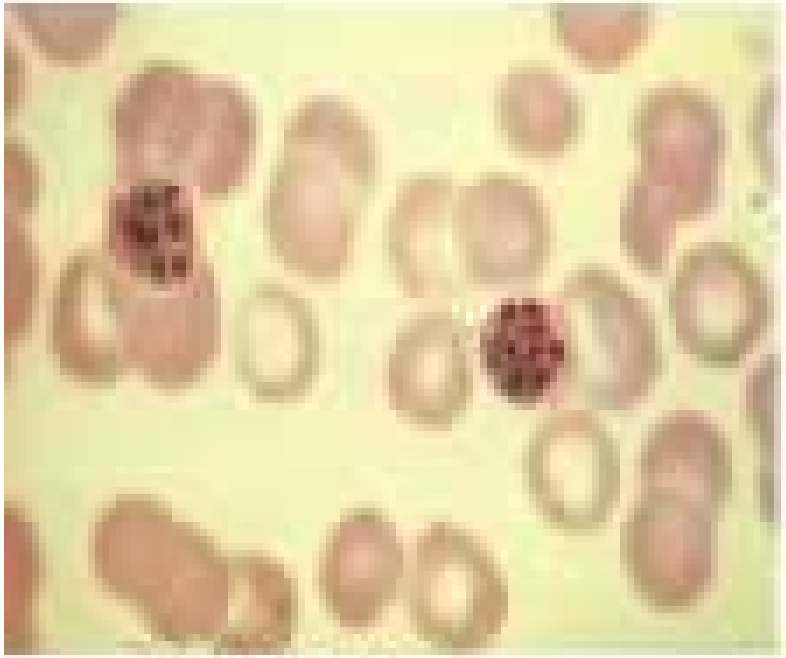

P. malariae


P. falciparum

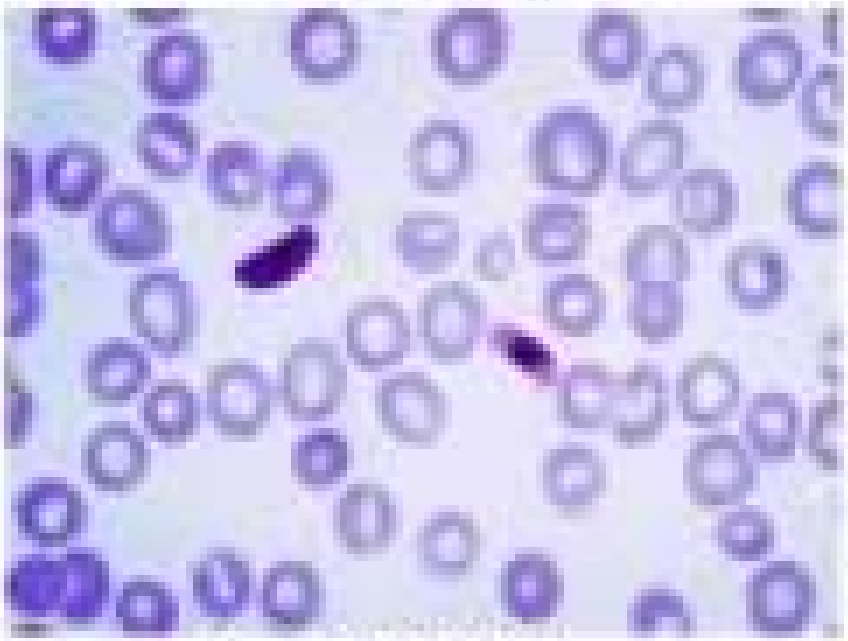
P. vivax



P. ovale



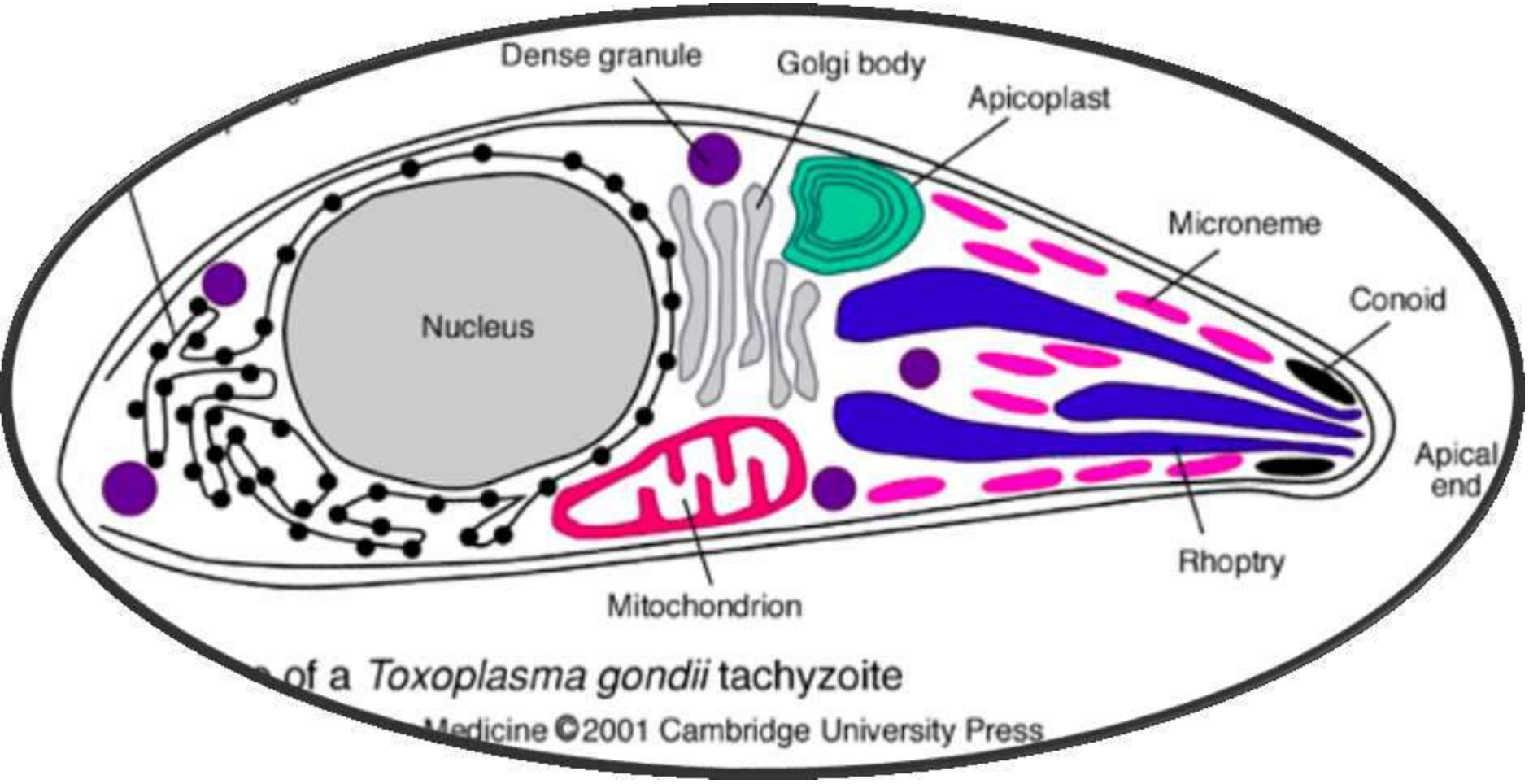
P. malariae



P. falciparum


Thank You

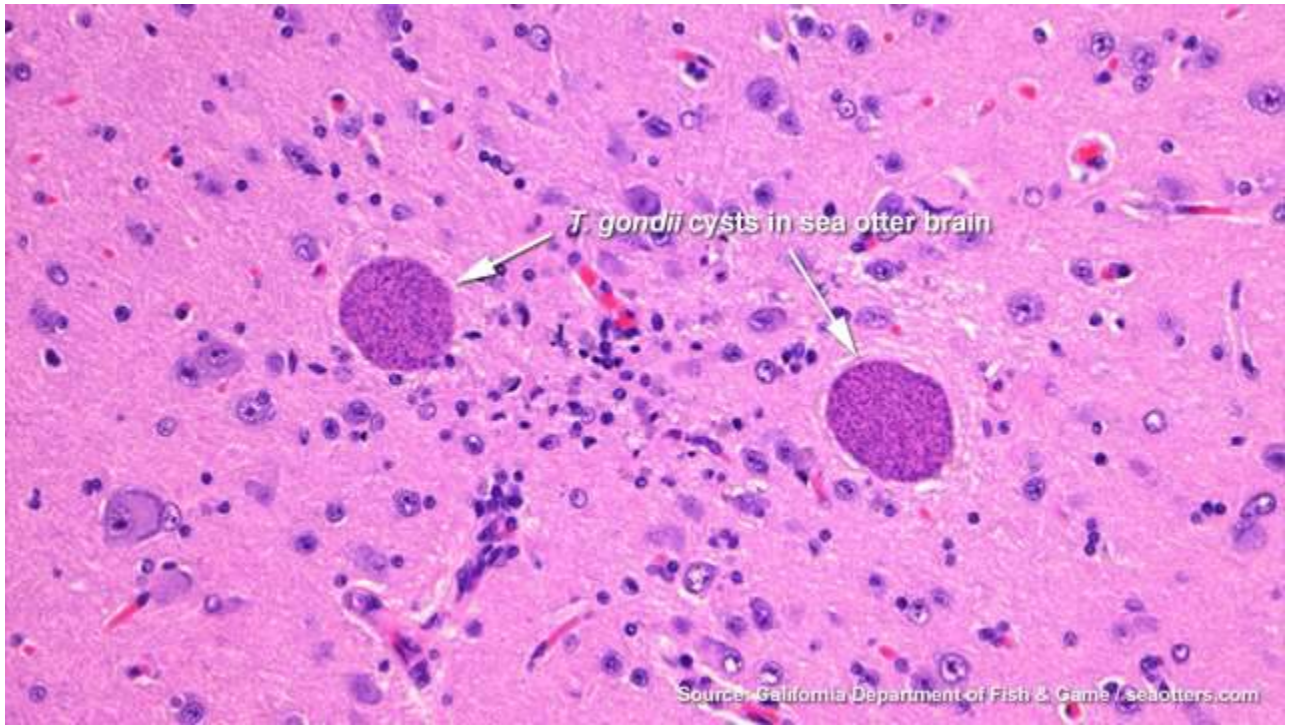
Toxoplasma



Lec.Dr.Ruwaidah F. Khaleel

Toxoplasma gondii

- is a species of parasitic protozoa in the Genus *Toxoplasma* belong to the **Phylum Apicomplexa**.
- **The definitive host of *T. gondii* is the cat**, but the parasite can be carried by many **warm-blooded animals (birds or mammals, including humans).**



Toxoplasmosis

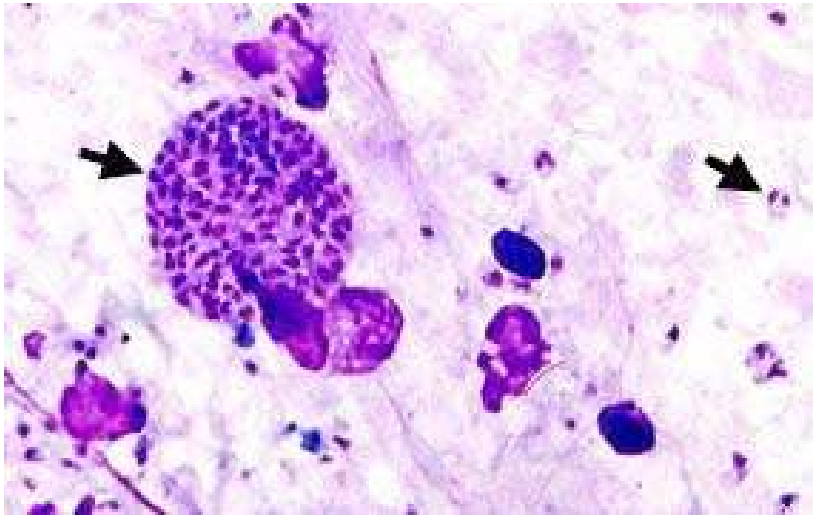
- It is the disease of which ***T. gondii*** is the causative agent, is usually *minor* and self-limiting but can have serious or even fatal effects on a fetus whose mother first contracts the disease during pregnancy or on an **immunocompromised** human or cat.

Geographic distribution ....

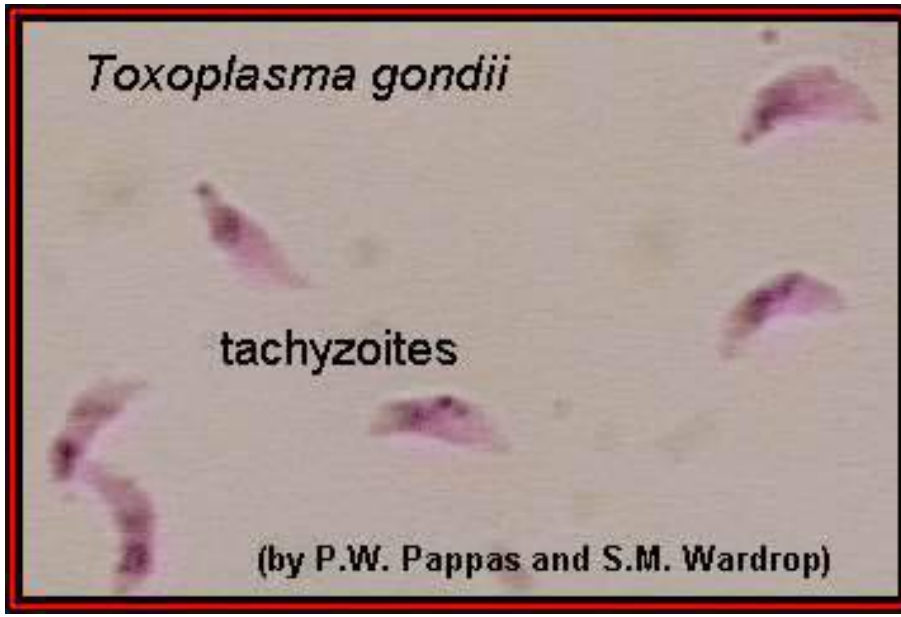
- Probably cosmopolitan.
- The organism is widespread **in mammals and birds,** and infections **in man have been reported in the America, Asia, Africa, Europe, and Australia**.

Morphology....

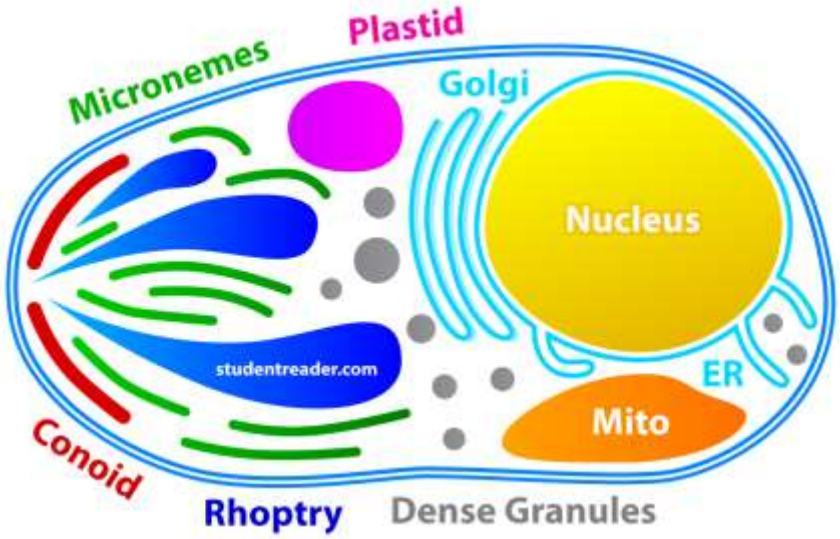
- The parasite is most frequently found **intracellularly.**
- It has an affinity for **reticuloendothelial cells, monocytes, macrophages.**
- Within the cells it occurs singly, in pairs, or in **rosette-like clusters**, and ultimately divides into **10-128** pyriform or rounded daughter parasites, **4-7×2-4 µ**
- The crescentic, almond-shaped extracellular parasites measure **6- 10µ×3-4µ.**



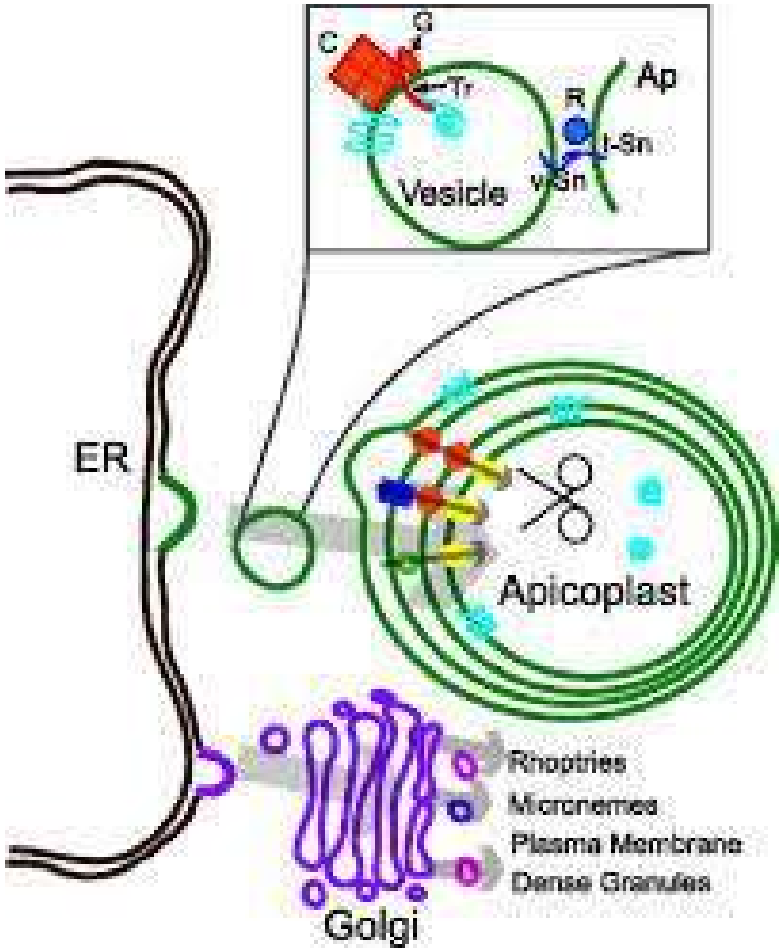
- Both ends are somewhat **pointed,** but the posterior is more rounded than the anterior
- The parasite is enclosed in smooth cell membrane, there are no flagella or cilia.
- At the pointed anterior end is the **conoid** Structure which is used to penetrate the hosts cells.
- The parasite has a number of regulated secretory organelles, these include the **apical micronemes** and **rhoptries (club-shaped)** and the more generally localized **dense granules.**



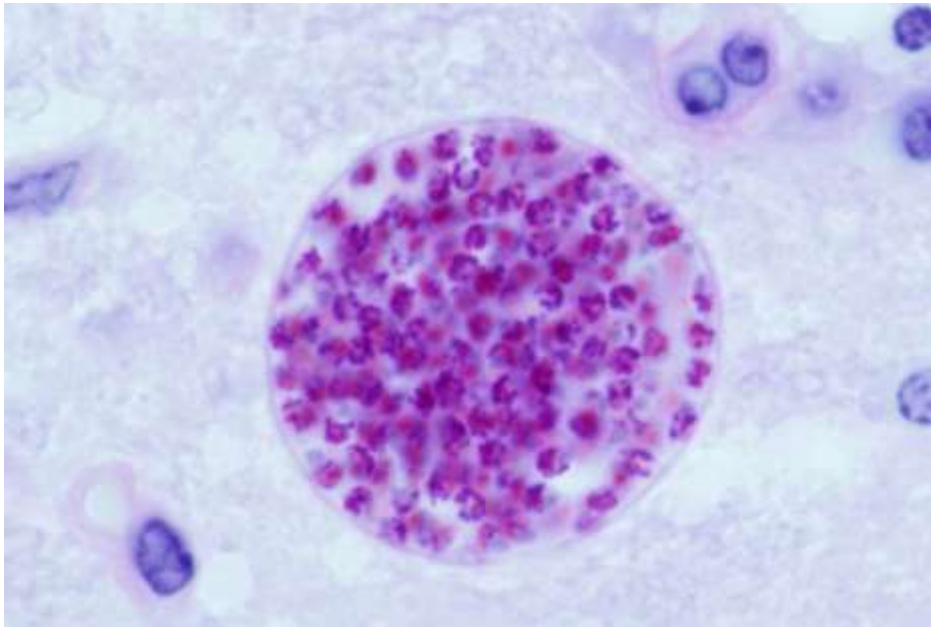
- ***Micronemes, rhoptries*** and ***dense granules*** are the three major secretory organelles, found predominately at the apical end of the parasite.
- **Microneme proteins** are released very early in the invasion process, facilitating host-cell binding and gliding motility.
- **Rhoptry proteins** are also released during invasion, and can be detected within the lumen and membrane of the newly generated ***parasitophorous vacuole*** **(PV).**
- Dense-granule proteins are released during and after the formation of the PV, modifying the PV environment for intracellular survival and replication of the parasite.



- In the middle third of the parasite usually toward the blunt end there is a **non vesicular nucleus with large nucleolus**.
- The ***apicopiast*** is a plastid-like four-membrane organelle containing a **35 circular DNA.**
- Most of the proteins functioning within the organelle are encoded by the nucleus, and are specifically targeted to the apicopiast.
- This targeting involves the secretory pathway, including the **rough *endoplasmic* reticulum (ER)** and a ***Golgi body*** situated immediately apical to the nucleus.
- *T.gondii* cells have a single nucleus and a single mitochondrion).



- ***Motility....***
- The extracellular parasites are capable of independent motion and progress with a gliding movement.

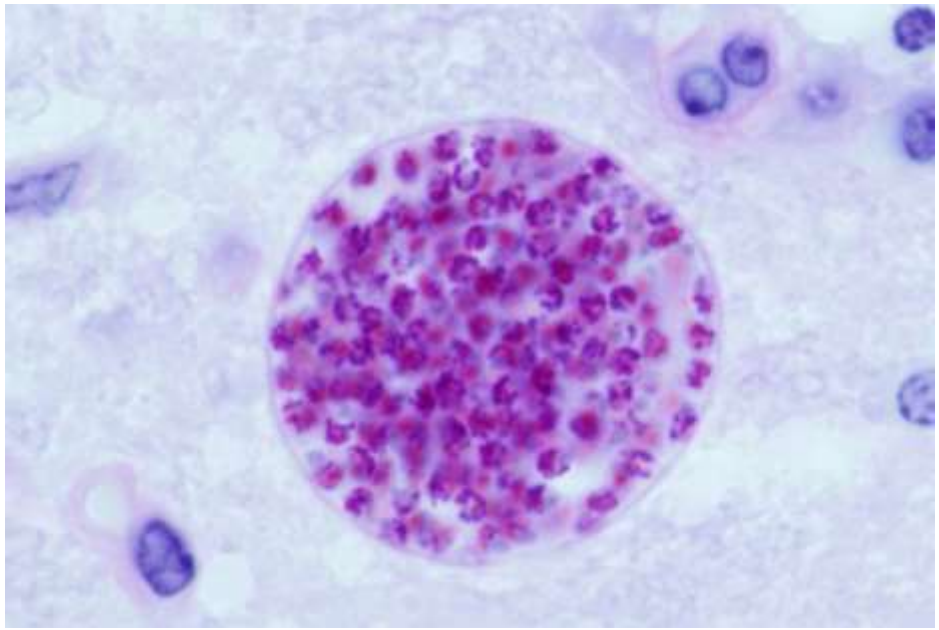


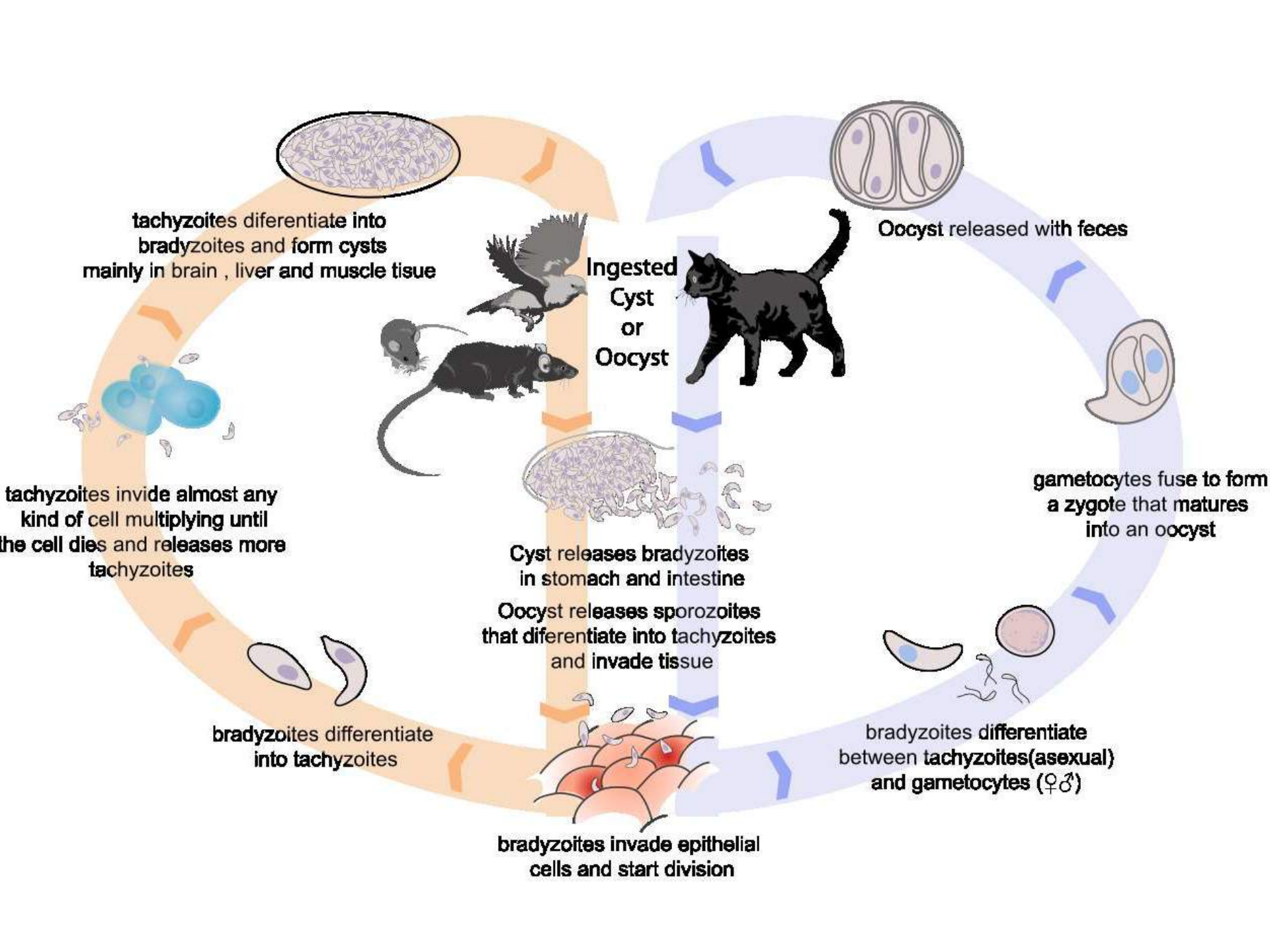
- **Nutrition....**
- T. *gondii* aquires its nourishment by absorption from the tissues of the host.

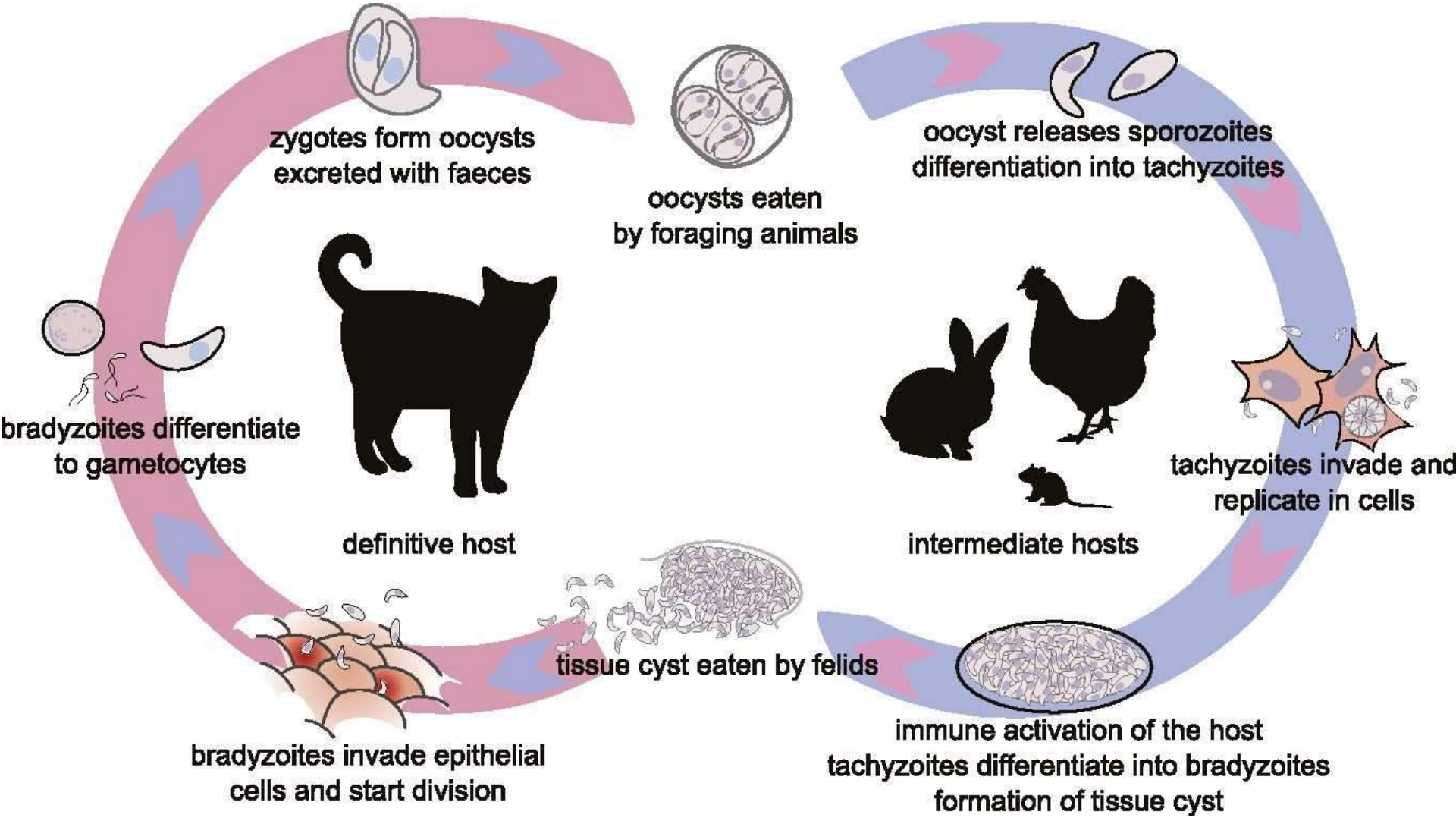
Life cycle

- **The life cycle of *T. gondii* has two phases.**

1. The **sexual part** of the life cycle (coccidia like) takes place only in members of the **Felidae** family (domestic and wild cats), which makes these animals the parasite's primary host.
2. The **asexual part** of the life cycle can take place in any warm-blooded animal, like other **mammals** (including felines) and **birds.**




tachyzoites diferentiate into bradyzoites and form cysts mainly in brain , liver and muscle tisue
Ingested Cyst or Oocyst
Oocyst released with feces
tachyzoites invide almost any kind of cell multiplying until the cell dies and releases more tachyzoites
Cyst releases bradyzoites in stomach and intestine
gametocytes fuse to form a zygote that matures into an oocyst
Oocyst releases sporozoites that diferentiate into tachyzoites and invade tissue
bradyzoites differentiate into tachyzoites
bradyzoites differentiate between tachyzoites(asexual) and gametocytes (♀♂)
bradyzoites invade epithelial cells and start division


zygotes form oocysts
excreted with faeces
oocysts eaten
by foraging animals
oocyst releases sporozoites
differentiation into tachyzoites
bradyzoites differentiate
to gametocytes
definitive host
intermediate hosts
tachyzoites invade and
replicate in cells
tissue cyst eaten by felids
bradyzoites invade epithelial
cells and start division
immune activation of the host
tachyzoites differentiate into bradyzoites
formation of tissue cyst

- The life cycle of toxoplasma begins as **unsporoulated oocyst,** which is non-active egg cell.

- These cells sporulate **(mature oocyst with eight sporozoites)**

- make sporozoites in the environment to become **infective.** These are found in the feces of infected cats.

- Water, surrounding soil, and food can become contaminated with these infective cells. When these are ingested by a mouse, or another intermediate host,

- the sporozoites invade almost every kind of tissue in the host's body.

- They then begin to multiply **asexually** by endodyogeny.

- At this point the sporozoites are called **tachyzoites.**

- They multiply very quickly forming **pseudocysts** inside host cells, which burst open to release more tachyzoites.

- After the first few days of infection, the tachyzoites are called **bradyzoites.**

- They multiply more slowly and stop bursting out of **pseudocysts.**

- They form **shelled cysts** to hide in the host's tissues until the second and final host, the cat, eats the intermediate host

Tissue cysts

- are ingested by a cat (e.g., by feeding on an infected mouse).
- Inside the cat, the cysts passage through the stomach of the cat and the parasites infect epithelial cells of the small intestine
- where they undergo sexual reproduction and oocyst formation by **gametogony**
- then **sporonts**
- then **sporoblasts,**
- and finally **sporozoites** to complete the sexual life cycle Oocysts are shed with the feces.

- *T. gondii* **also infects humans as an intermediate host,** causing Toxoplasmosis. This can happen if a human ingests **undercooked or contaminated meat.**

- A human can also be infected by coming in close contact with the contents of **cat litter boxes,** which is one reason the percentage of infection among humans is so high.

- Once inside a human, *T. gondii* undergoes the same rapid multiplication in which flu like symptoms might be had, then settles into cyst formation and hiding, however it is unlikely that the human will ever be eaten by the secondary host, the cat.

- If women are infected after pregnancy there is a chance that their infants will be infected as well.
- This can lead to birth defects or early abortion.

Pathogenicity ....

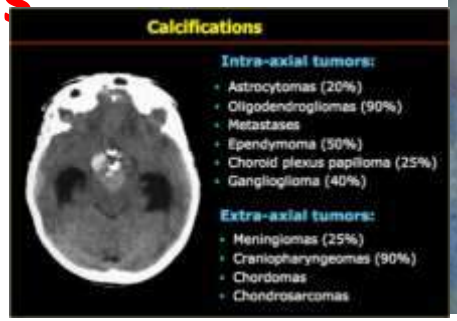
- **There are three stages of infection, and clinical illness may be evidenced.**
- During the **acute phase** the proliferation of the parasites results in lysis of the invaded cells so that eventually small necrotic foci.
- Acute stage *Toxoplasma* infections can be asymptomatic, but often give **flu-like symptoms** in the early acute stages, and like flu can become, in very rare cases, fatal.

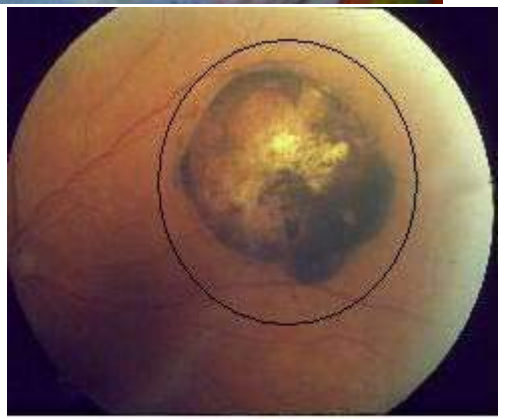
The acute stage fades in a few days to months, leading to:

- **Sub-acute stage** where the parasites decrease in number with the development of antibodies.
- **then the chronic stage** where the parasites are mostly contained in resistant cysts in the **brain, skeletal muscles,** and **heart.**

- **Latent infection (chronic)** is normally asymptomatic; however, in the case of immunocompromised patients (such as those infected with HIV or transplant recipients on immunosuppressive therapy), toxoplasmosis can develop.
- The most notable manifestation of toxoplasmosis in immunocompromised patients is toxoplasmic encephalitis, which can be deadly.

- If infection with *T. gondii* occurs for the first time during **pregnancy,** the parasite can cross the placenta, possibly leading to **congenital infections** that may leads **to hydrocephalus** or **microcephaly, intracranial calcification, and chorioretinitis, with the possibility of spontaneous abortion (miscarriage) or intrauterine death.**





Chorioretinitis as a result of a Toxoplasmosis infection

Diagnosis....

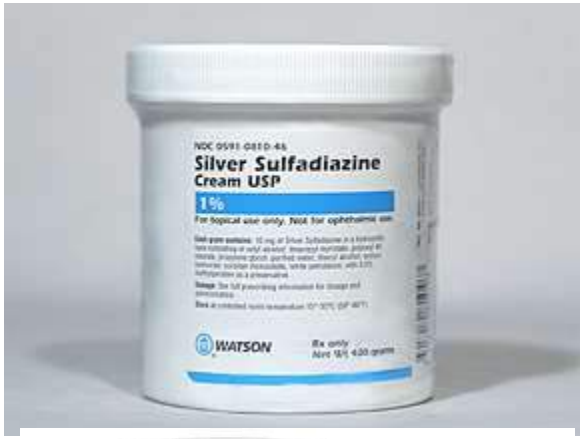
1. Demonstrating the organism in the tissue or body fluid by stained preparation.
2. Detecting specific antibodies by serological tests.

Treatment

- Treatment is often only recommended for people with serious health problems, because the disease is most serious when one's immune system is weak.

Acute

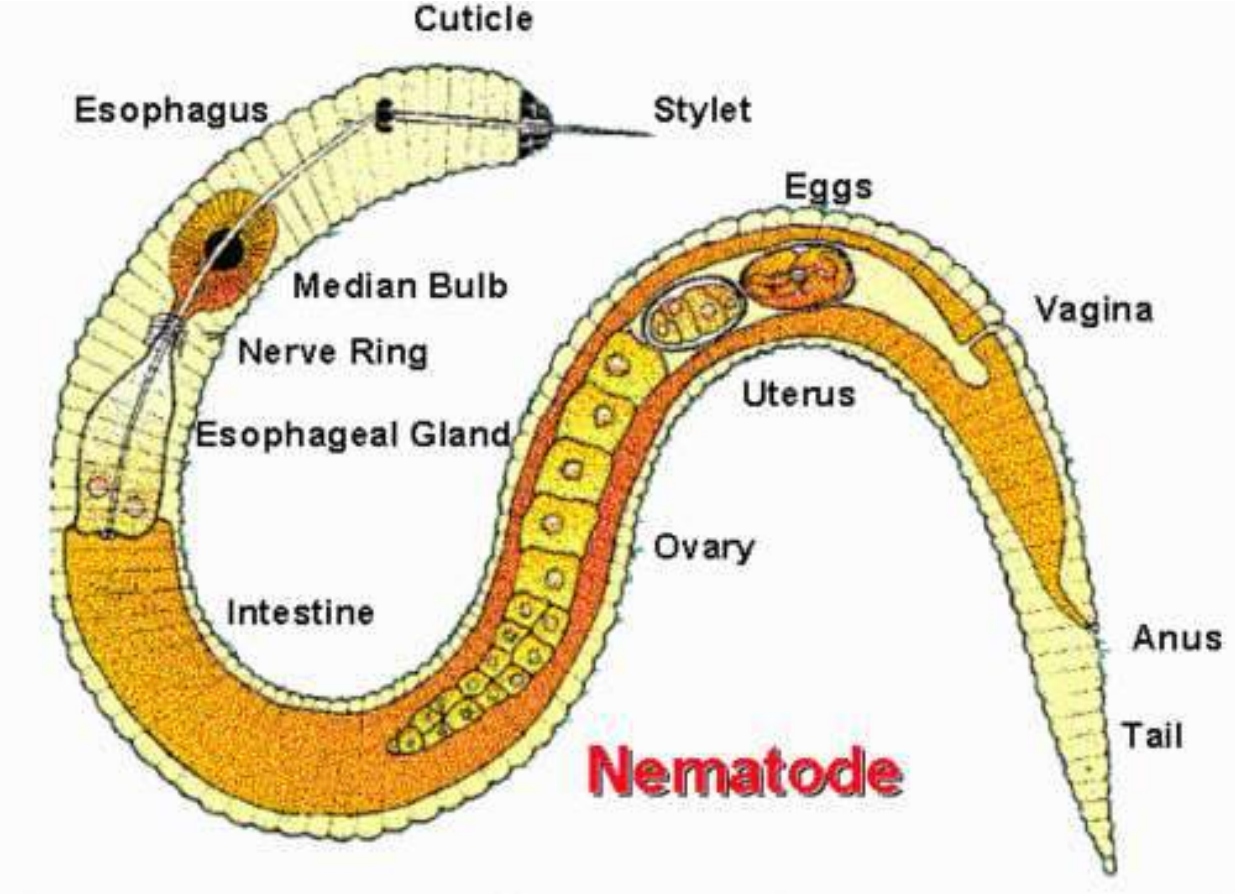
- **Medications that are prescribed for acute toxoplasmosis are:**
- **Pyrimethamine** — an **antimalarial medication.**
- **Sulfadiazine** — an **antibiotic** used in combination with pyrimethamine to treat toxoplasmosis,
- **clindamycin** — an antibiotic used most often for people with HIV/AIDS.
- **spiramycin** — an antibiotic used most often for pregnant women to prevent the infection of their child.



Latent

- In people with latent toxoplasmosis, the cysts are immune to these treatments, as the antibiotics do not reach the bradyzoites in sufficient concentration.
- **Medications that are prescribed for latent toxoplasmosis are:**
- Atovaquone — an antibiotic that has been used to kill *Toxoplasma* cysts inside AIDS patients.
- Clindamycin — an antibiotic which, in combination with atovaquone, seemed to optimally kill cysts in mice.

Phylum Nematodes

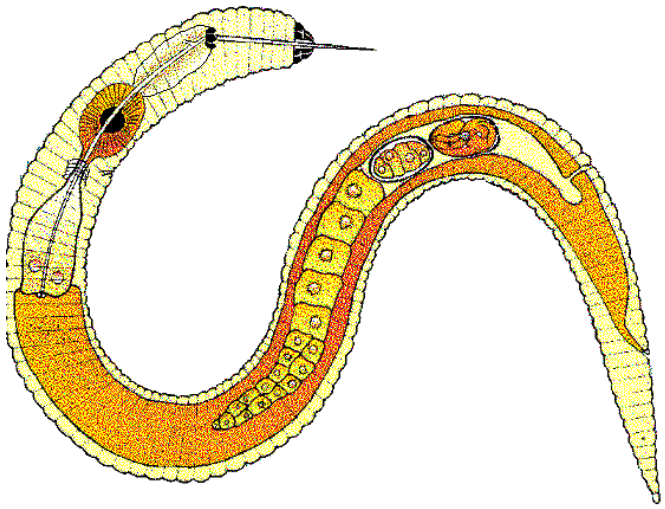


Lec.Dr.Ruwaidah F. Khaleel

Phylum Nematodes

- The word nematode means "thread" in Greek, about 25.000 species.
- **General characteristics of nematodes:**

1. They are commonly called **"roundworms"** because they have cylindrical body and look round in cross section, the worm is taper at both ends.
2. Thread like non-segmented and have bilateral symmetry.
3. Nematodes possess protective cuticle and pseudocoelmate.
4. Usually diecious and few are hermaphrodite, male is smaller with curved end.
5. Female is either viviparous, oviparous or ovo-viviparous.
6. Nematodes reside in small intestine or tissues.
7. Length ranging from 2-40 cm.

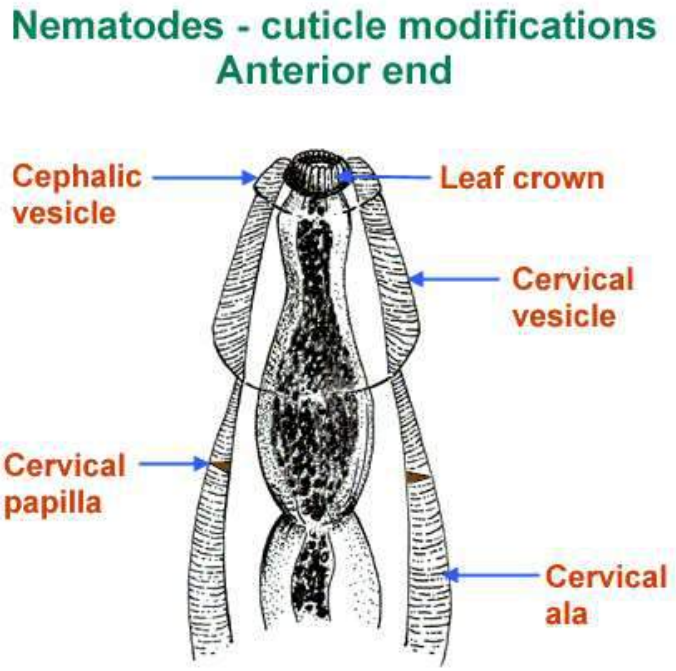


Nematodes cuticle modifications:

- **A- Anterior end**
- Leaf crowns which are rows of finger-like projections surrounding the buccal cavity **(A).**

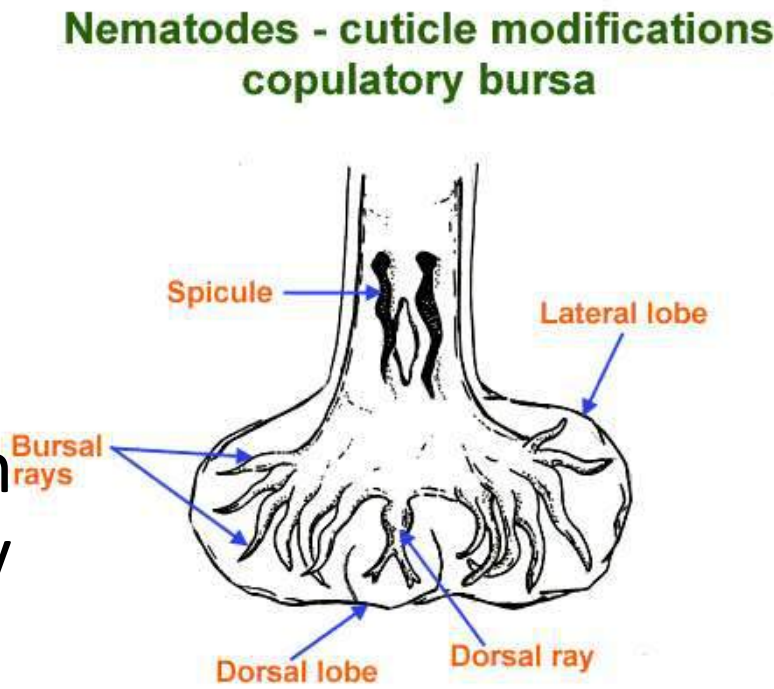
1. Vesicles which are inflations of the cuticle, wing-like, around the mouth **(B)** or esophagus **(C).**
2. Alae, also wing-like expansions of the cuticle and it is of two kinds,

- **1. first are cervical alae (D)** located in terminal half of esophagus region, they are found when the vesicles are absent,
- **2. second are cervical papillae (E),** they are paired spine-like projections found in the esophageal region, their function is tactile or sensory.



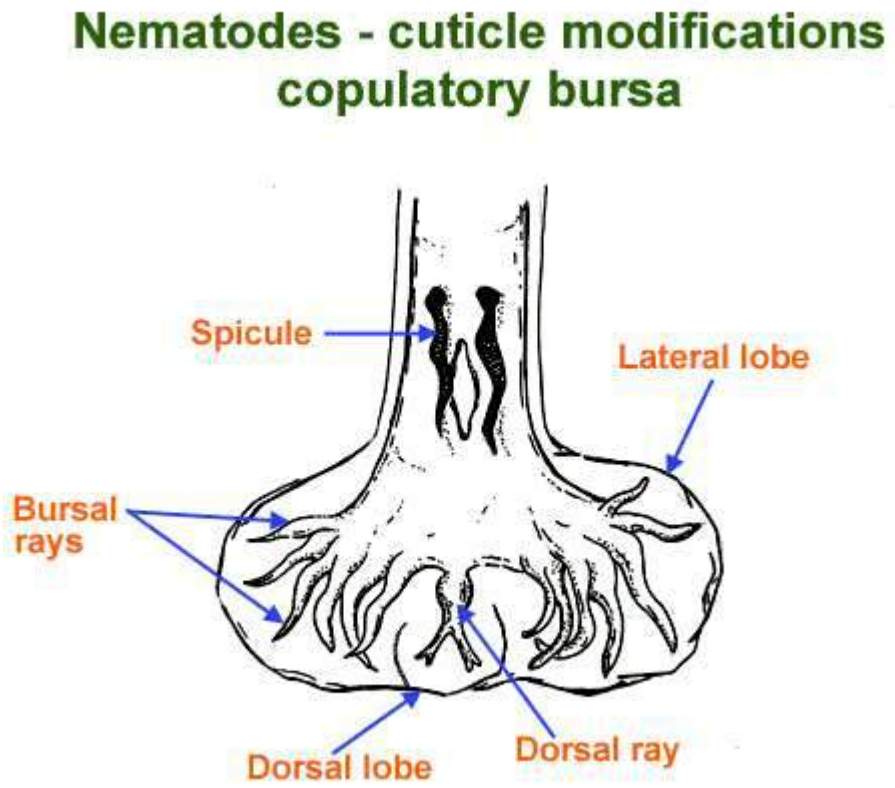
B- Posterior end which includes caudal papillae and caudal alae:

1. **Caudal papillae:** cuticular projections of sensory function.
2. **Caudal alae:** wing-like expansions found in the tail.
3. **Copulatory bursa:** found only in male of some nematodes in some species, they are greatly expanded in which the male grasps the female during copulation.



Structure of Copulatory Bursa

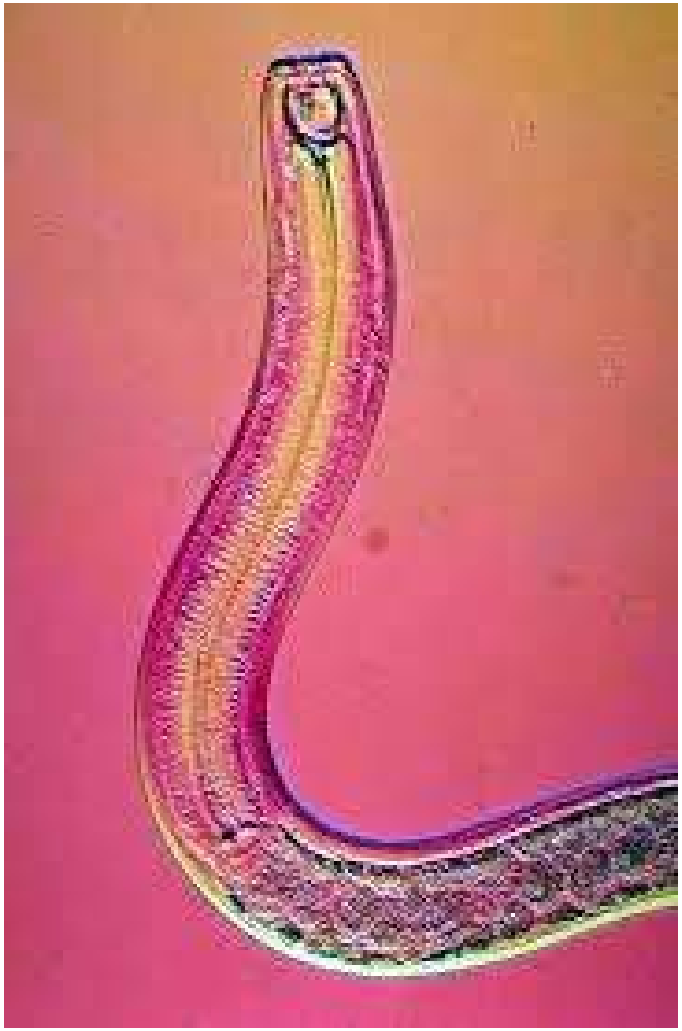
- A bursa has two lateral lobes and in some species, a dorsal lobe. The bursa is supported by finger-like structures called rays.
- When resting, the bursa looks like a relaxed, folded hand but during copulation it is greatly expanded and used to grasp the female.



Nematodes classification:

- **According to the presence or absence of sensory chemoreceptor organs near the posterior end, nematodes can be divided into:**
- **A- Class Adenophora (Aphasmida):**

1. Do not have phasmids.
2. Amphids (sensory structures) located to the posterior of head region.
3. Male and female with one single gonad and male with one spicule and no caudal papillae.
4. Simple spindle-shaped worm with simple excretory organs.
5. Most are free living, few are parasitic.

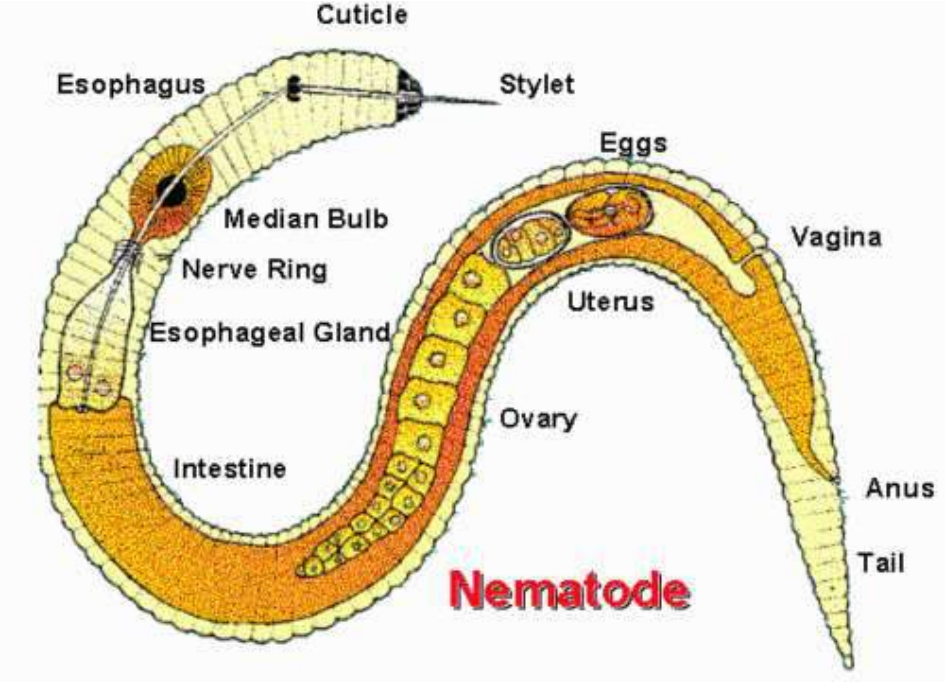


B- Class Secernentea (Phasmida):

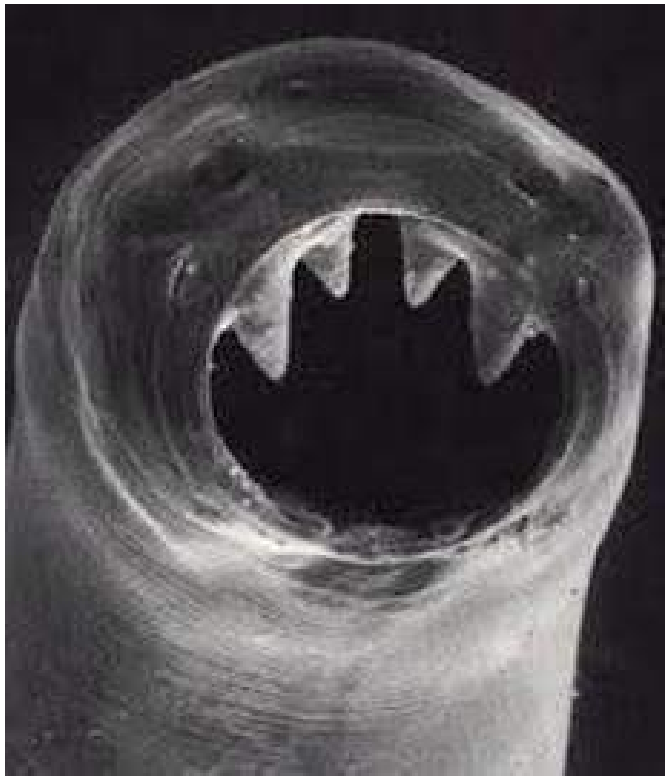
1. They have phasmids.
2. Amphids located to the anterior of head region.
3. Female reproductive system is double, male with two spicules and has caudal papillae.
4. Excretory system is more developed with ducts.
5. Most are parasitic (destructive).

Digestive system of Nematodes:

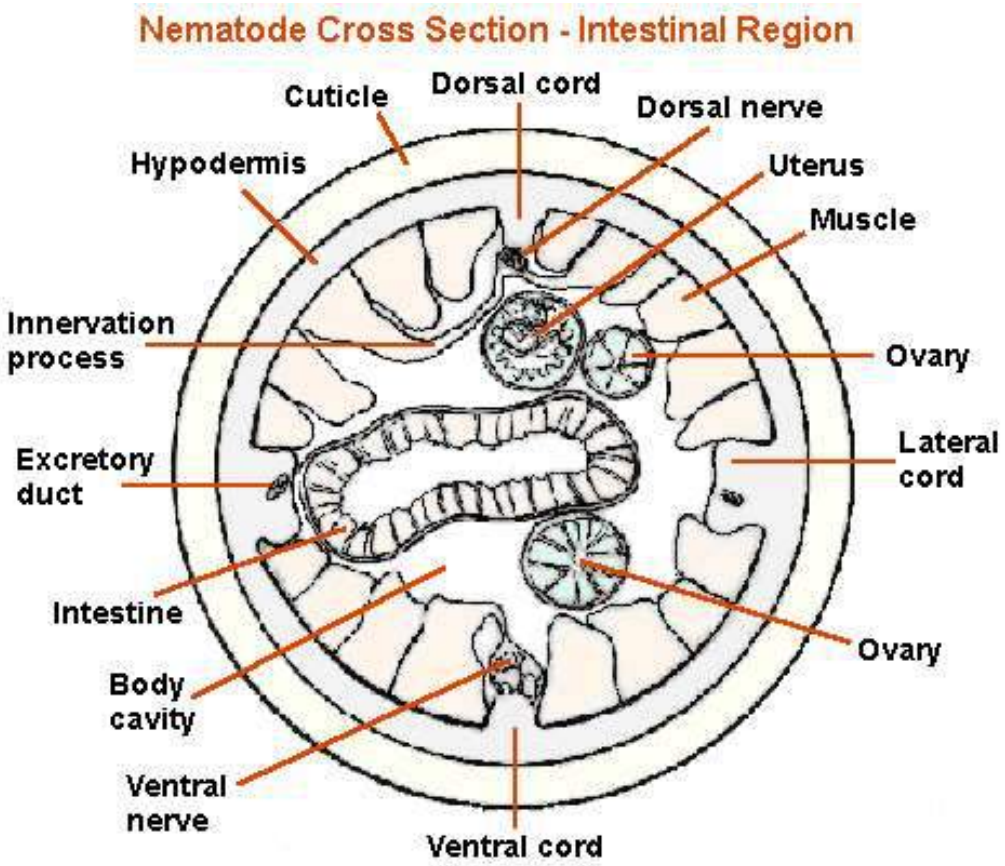
- The digestive system of nematodes is usually a simple tube with the majority of variations occurring in the sizes of the mouth opening,
- buccal capsule
- esophagus.
- The mouth may be a simple opening with a tiny buccal cavity leading directly to the esophagus. Mouth opening may be quite large leading to a prominent buccal cavity with thick walls and often containing teeth.



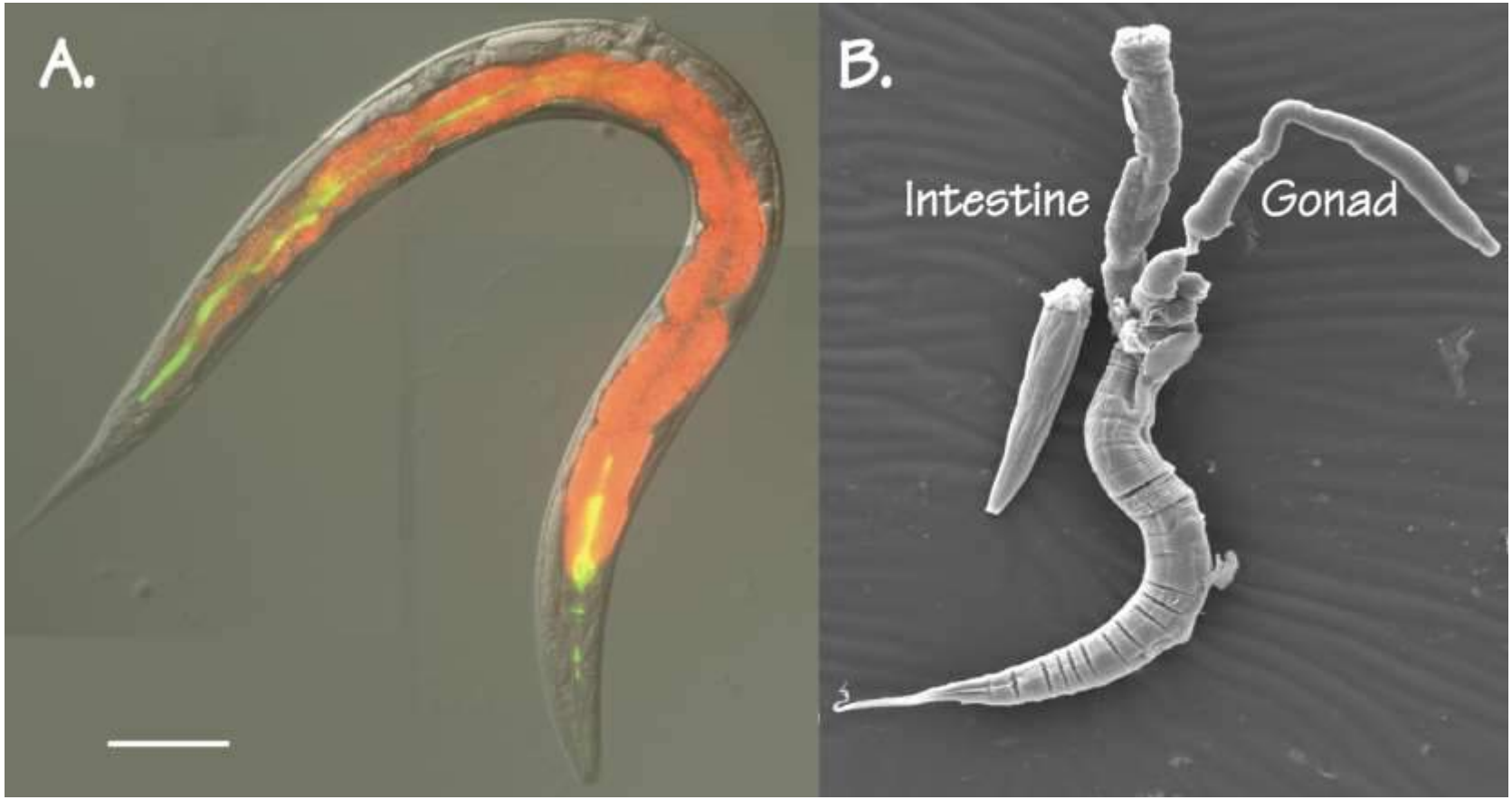
- A large mouth opening and prominent buccal cavity is found in those nematodes that feed by taking a bite of mucosa, and drawing it into the buccal cavity where it is digested.
- These nematodes are often called "plug feeders".
- In nematodes that feed by simple ingestion of host fluids, the mouth opening and buccal cavity are generally quite small.
- In nematodes that are called mucosal grazers, rather than plug feeders, the mouth opening and its buccal cavity are usually intermediate in size.



- In most nematodes, the esophagus is muscular and it is used to pass food into the intestine by a pumping action.
- The intestine is a straight tube, the lumen surfaces consist of number of projecting microvilli used in absorption and because of their big numbers provide an immense absorptive capacity.

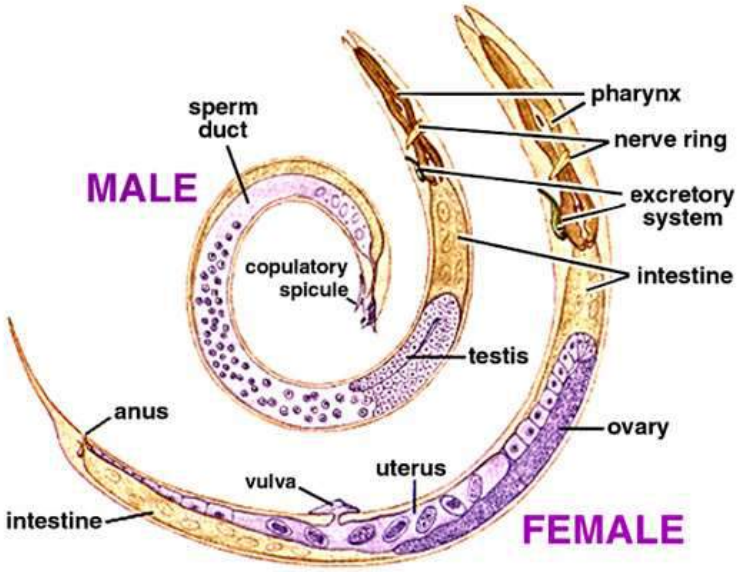


- The intestine terminates in a rectum in females and a cloaca in males.
- The cloaca is a common termination for the intestinal tract and the vas deferens in adult males. In either case, the cloaca (males) and rectum (females) leads to an anus which usually opens to the outside at the posterior end of the body.



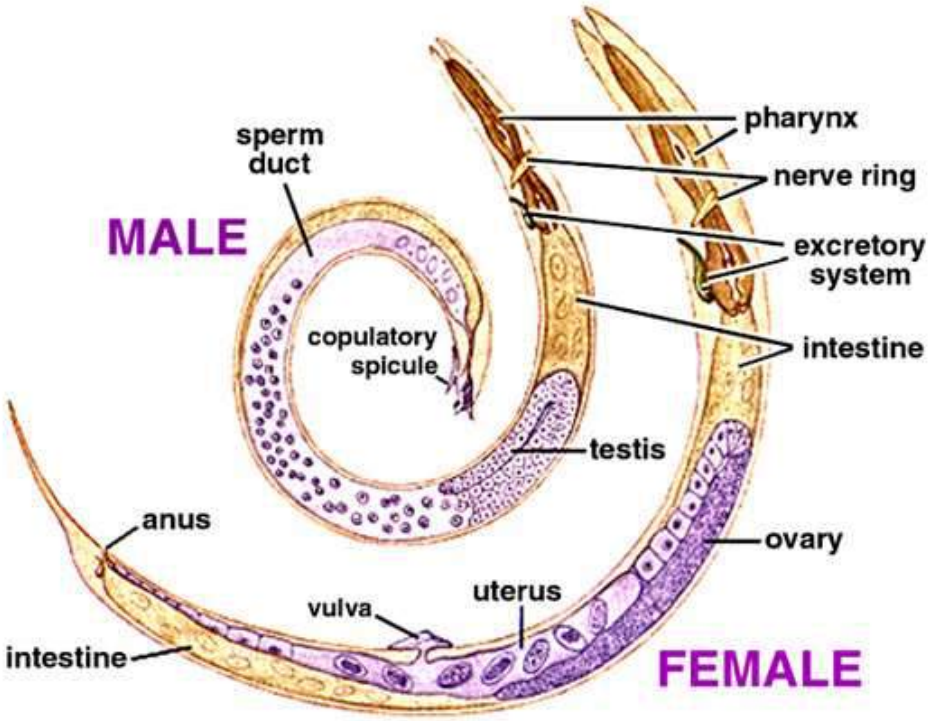
Reproductive system:

- The sexes are separate in most species of nematodes and males are always smaller than females because females need to accommodate the production of large quantities of eggs.
- **Female reproductive system:** The female reproductive system is tubular and in most nematodes of veterinary importance consists of two ovaries, each of which connects to an oviduct, and a uterus. The two uteri end in a common vagina which opens to the outside by a vulva which is often covered by a protective flap of cuticle, the vulva flap. In female members of the order Strongylida a muscular structure called an ovejector controls the exit of eggs from the uterus.



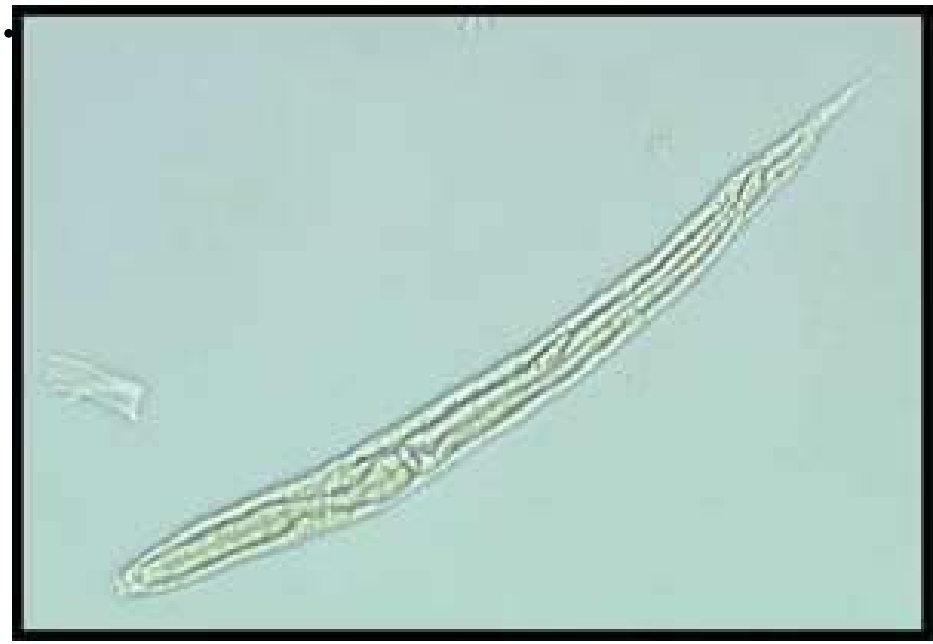
Male reproductive system

- The male reproductive system in nematodes of veterinary importance is a single tube differentiated into testis, seminal vesicle and vas deferens and terminating in a muscular ejaculatory duct which empties into the cloaca. Male usually has spicules, they are made of cuticle and are often paired and used in copulation to dilate the female vulva.



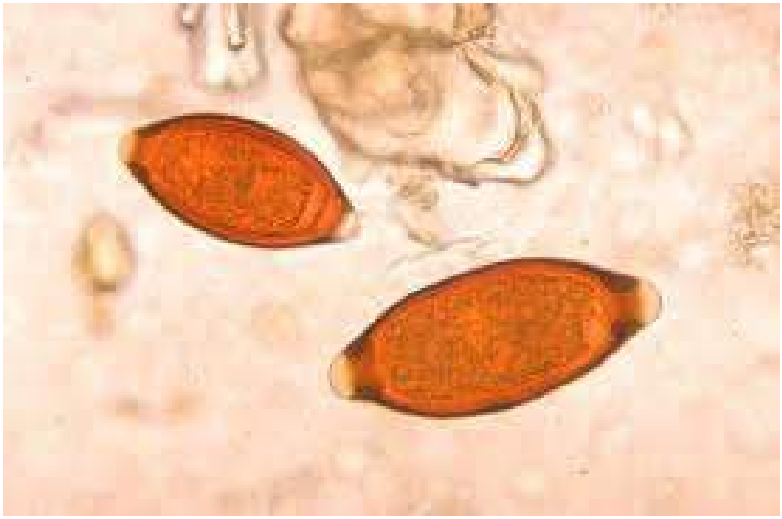
Note:

- In most nematodes life cycle, the stage that is passes from the definitive host is not the same that is infective for another definitive host.
- The egg that contains larva one (L1) passes from a definitive host most develops, by a process called molting, through a stage (usually L3) to be infective for another definitive host.



Class: Adenophora (Aphasmida), Order: Trichurida- *Trichuris trichiura,* whipworm (Infective stage: emryonated ova)

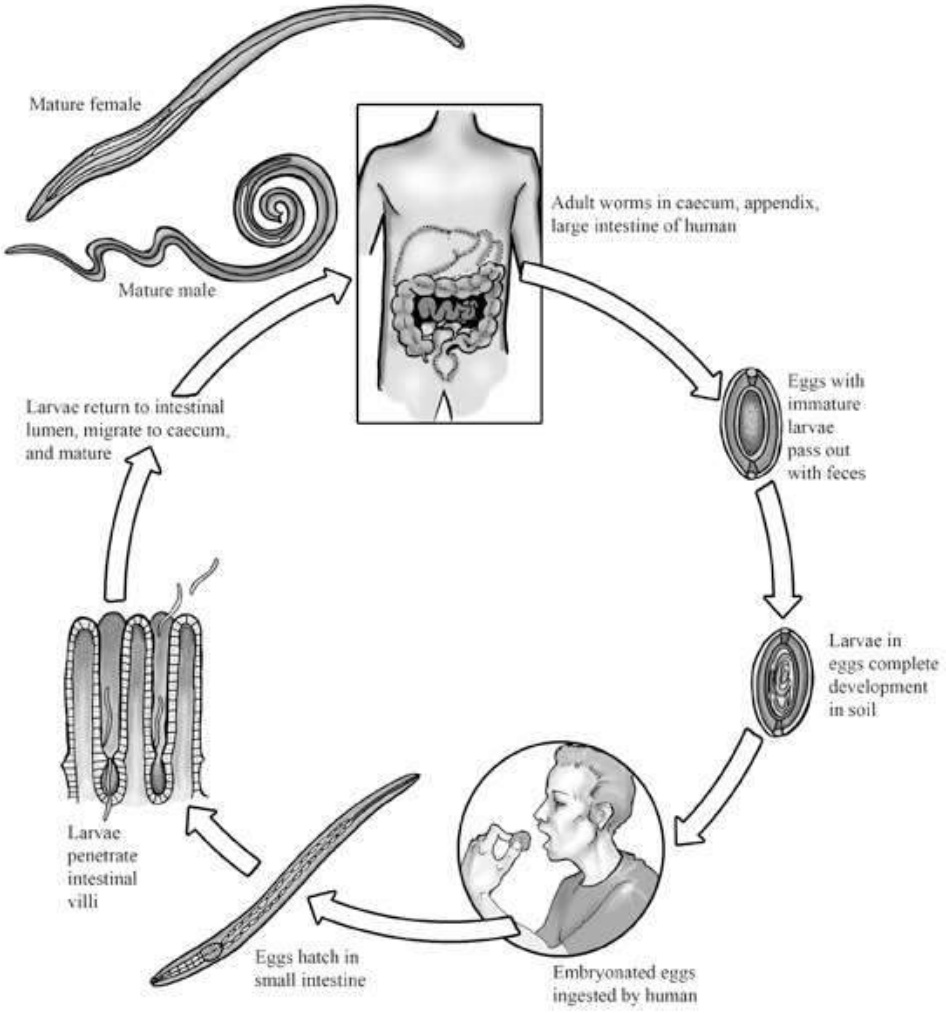
- Whip-like shape worms, adults found in the large intestine (cecum and colon) quarter of the world's population is thought to carry the parasite and only patients with heavy parasite burden become symptomatic;
- whipworm is very common in children due to poor hygiene.
- Adult length from 30-50mm, egg is 60 pm, barrel shape with two polar plugs,



- male and female are characterized by the slender anterior section and thickened posterior section;
- the infective form is the ovum and it is usually mature and un-embryonated.
- The juveniles buries its thin threadlike anterior half into the intestinal villi and mucosa and feeds on tissue secretions (not blood), this thin part contains the esophagus and the rest contains the intestine and reproductive organs.
- The tissue invasion of this worms leads to eosinophilia.

Life cycle:

- ova are deposited in the soil through human feces and it is transmitted via fecal-oral transmission, ova become infected in 10-14 days.
- Larvae hatch in the small intestine, where they grow and molt before residing in the large intestine with no tissue migratory phase (unlike *Ascaris*).
- The time between the ingestion of ovum to be mature adult takes 3 months, this worm lives about 5 years.



Symptoms and pathogenicity:

- The name of this disease is trichuriasis and symptoms appear when the parasite burden is more than 10 worms; normal symptoms are:

1. diarrhea,
2. abdominal pain,
3. weight loss and nausea
4. vitamin A deficiency is common.

- **Heavy infection leads to**

1. intestinal bleeding and anemia,
2. rectal prolapse is associated with the infection of this worm in which the rectum is protruded out of the anus,

- **a common case in children.**
- **Trichuriasis can be diagnosed by** stool examination and identification of the bipolar thick walled ova. Infection of this worm is commonly seen along with infection of *Ascaris* because of the similar mode of infection. This worm is resistant to some of treatments but mebendazole found to be effective.

Class: Secernentea (phasmida). Order: Ascaridida - *Ascaris lumbricoides* (common or giant roundworm) (Infective stage: egg with larva 2)

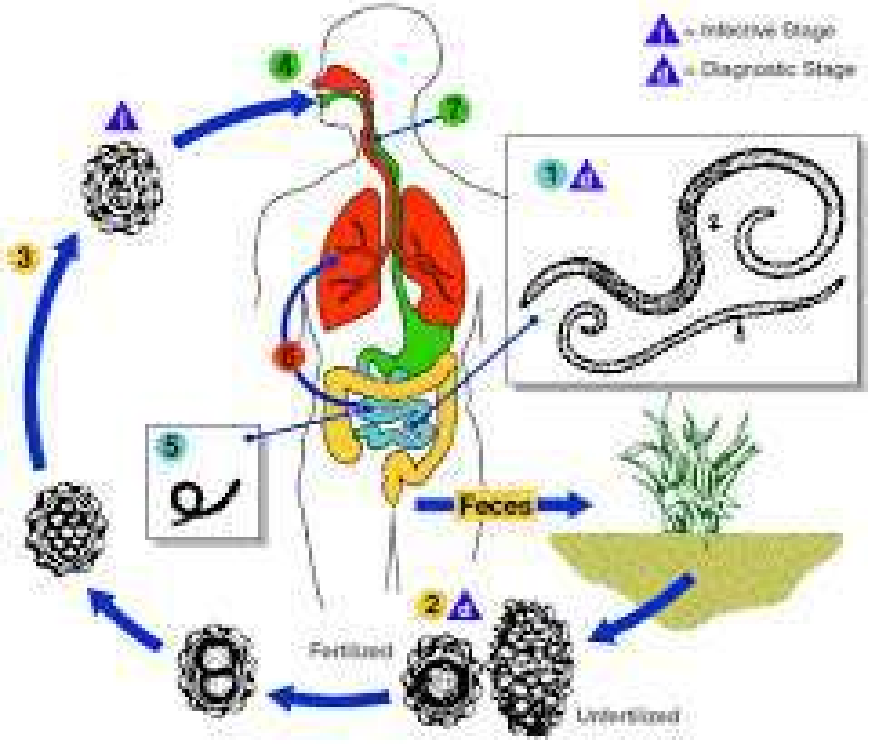
- *Ascaris,* which cause ascariasis, is the largest intestinal roundworm; its distribution is worldwide and most prevalent in tropical and sub-tropical countries. It has a complex life cycle (heart-lung-intestine).
- Adult worms reside is small intestine, mainly in jejunum and upper part of ileum. Adult males are 15-30 cm with strongly curved tail with two spicules but has no copulatory bursa;
- females are 20-35 cm with straight tail. Life span of adults is 1-2 years.

- Female produces 200.00 ova per day from the vulva which is located down the mouth about third of the length of the body.
- Both male female are characterized by smooth fine cuticle and the mouth has three lips covered with small papillae.
- **The fertilized ovum** is oval and has a shell covered with heavy albuminous layer,
- **unfertilized ovum** is longer in shape and has thinner layer. *Ascaris* ova are very resistant to strong chemical substances, desiccation and low temperature.

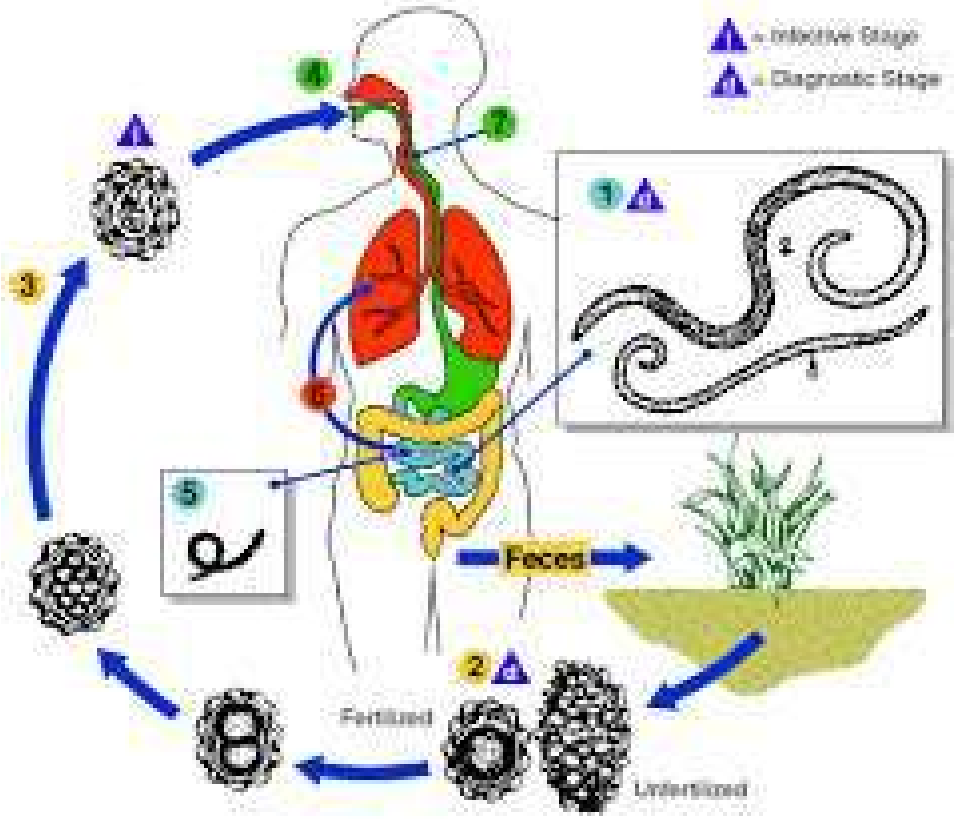
Life cycle:

- **Ova pass with feces to soil and they need abput 10-14 days to be infective rhabditiform L2 inside the ovum. Frequent mode of transmission is by the following:**

1. Vegetable grow in fields irrigate with sewage (human feces)
2. Children playing in mud can transmit ova to mouth through dirty fingers.
3. In heavy soil contamination, the ova become air-borne along with wind- dust and get inhaled.



- Ingested ovum with L2 is hatched of an active motile rhabditiform larva in duodenum, these larvae penetrate intestinal wall to enter the blood circulation then undergo an extraordinary migration through the body (liver + heart + lung), in lung, they molt twice and become L4 and this leads to damage in the alveoli.
- In three weeks, the L4 pass from the respiratory system to be coughed-up and swallowed, and thus, returned to the small intestine where they mature to adult male and female worms who can mate for another life cycle.



Symptoms and pathology:

- Infection with ova may remain asymptomatic but symptoms appear due to larval migration throughout of the body and adults gathering.
- Through pulmonary larval migration phase, pneumonitis may be provoked with low fever, cough, asthma and eosinophilia.
- In intestine phase, symptoms appear when adults pass through pancreas, bile ducts, gallbladder, or liver with abdominal pain and intermittent colic;
- large number of adults usually lead to bowel obstruction.

Diagnosis:

- Most diagnosis is made by identifying the appearance of the worm or eggs in feces due to the large quantity of eggs laid.
- Larvae may be found in gastric or respiratory secretion in pulmonary disease.

- **Treatment:**
- Mebendazole or Albendazole.


Thank you