MEDICAL PROTOZOLOGY

Protozoa (singular, protozoan), from the Greek ‘protos’ and ‘zoon’ meaning “first animal”, are members of eukaryotic **protists**.

They may be distinguished from other eukaryotic protists by their ability to move at some stage of their life cycle and by their lack of cell wall.

Occurrence of protozoa

Protozoa are found in all moist habitats. They are common in sea, in soil and in fresh water.

These organisms occur generally as a single cell. Colonies of protozoa might also occur in which individual cells are joined by cytoplasmic threads and form aggregates of independent cells.

However, distinct types of protozoa, include a resistant cyst (non-motile) stage to survive adverse environmental conditions, such as desiccation, low nutrient supply, and even anaerobiosis.

Morphology of protozoa

Protozoa are predominantly microscopic, ranging in size from 2 to more than 100μm.

Morphologically, they are within a mass of protoplasm, consisting of a true membrane – bound nucleus and cytoplasm.

The nucleus contains clumped or dispersed chromatin and central nucleolus or karyosome, which are useful structures to distinguish protozoan species from one another based on the shape, size and distribution of these structures.

Reproduction and regeneration of protozoa

As a general rule, protozoa multiply by asexual reproduction. This is not to say that sexual processes are absent in the protozoa. Some parasitic forms may have an asexual phase in one host and a sexual phase in another host.

Importance of protozoa

Protozoa serve as an important link in the food chain and ecological balance of many communities in wetland & aquatic environments.

They are also important in biological sewage treatment, which involves both anaerobic digestion and/or aeration.

In addition, protozoa are important laboratory organisms in research areas, by which their asexual reproduction enables clones to be established with the same genetic make-up.

These are useful in the study of cell cycles and nucleic acid biosynthesis during cell division.

Transmission

In most parasitic protozoa, the developmental stages are often transmitted from one host to another within a cyst. The reproduction process is also related to the formation of the cyst. Asexual reproduction of some ciliates and flagellates is associated with cyst formation, and sexual reproduction of Sporozoa invariably results in a cyst. Pathogenic protozoa can spread from one infected person to another by:

- Faecal – oral transmission of contaminated foods and water.
- Insect bit inoculums or rubbing infected insect faeces on the site of bite.
- Sexual intercourse

Pathogenesis

Protozoan organisms are virtually always acquired from an exogenous source, and as such, they have evolved numerous ways to enter the body of the human host. Factors that are important for pathogenecity include:

- Attachment to the host tissue followed by replication to establish colonization.
- Toxic products released by parasitic protozoa.
- Shifting of antigenic expression to evade the immune response and inactivate host defenses.

Classification of Protozoa

Protozoa of medical importance are classified based on their morphology and locomotive system as described below:

Amoebas - *Entamoeba histolytica* (Amoebiasis)

Flagellates - *Giarda lamblia* (Giardiasis or lambliosis), *Trichomonas vaginalis* (Trichomoniasis), Trypanosoma spp (Tripanosomiasis), Leishmania spp. (Leishmaniasis)

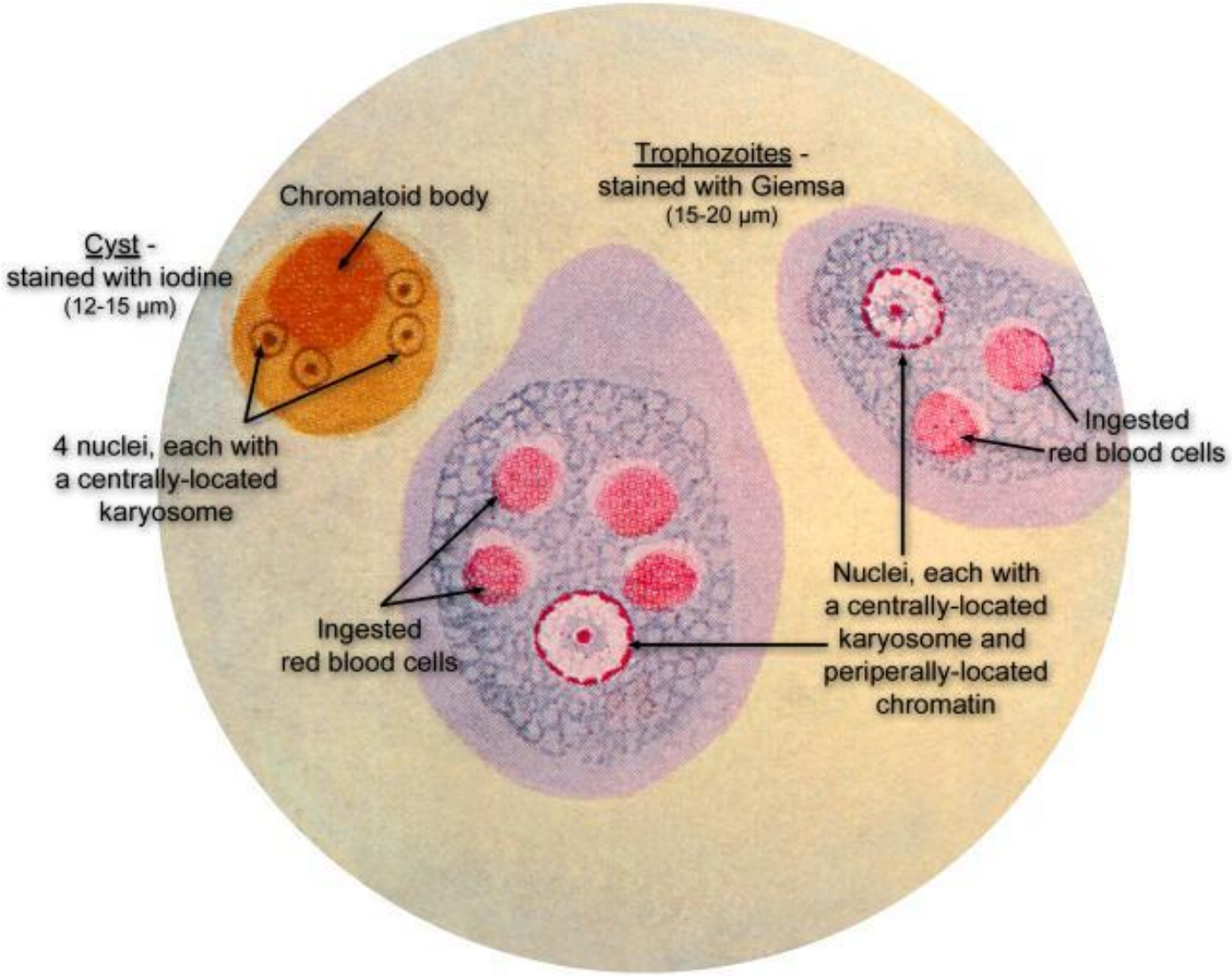
Ciliates - *Balantidium coli* (Balantidiasis)

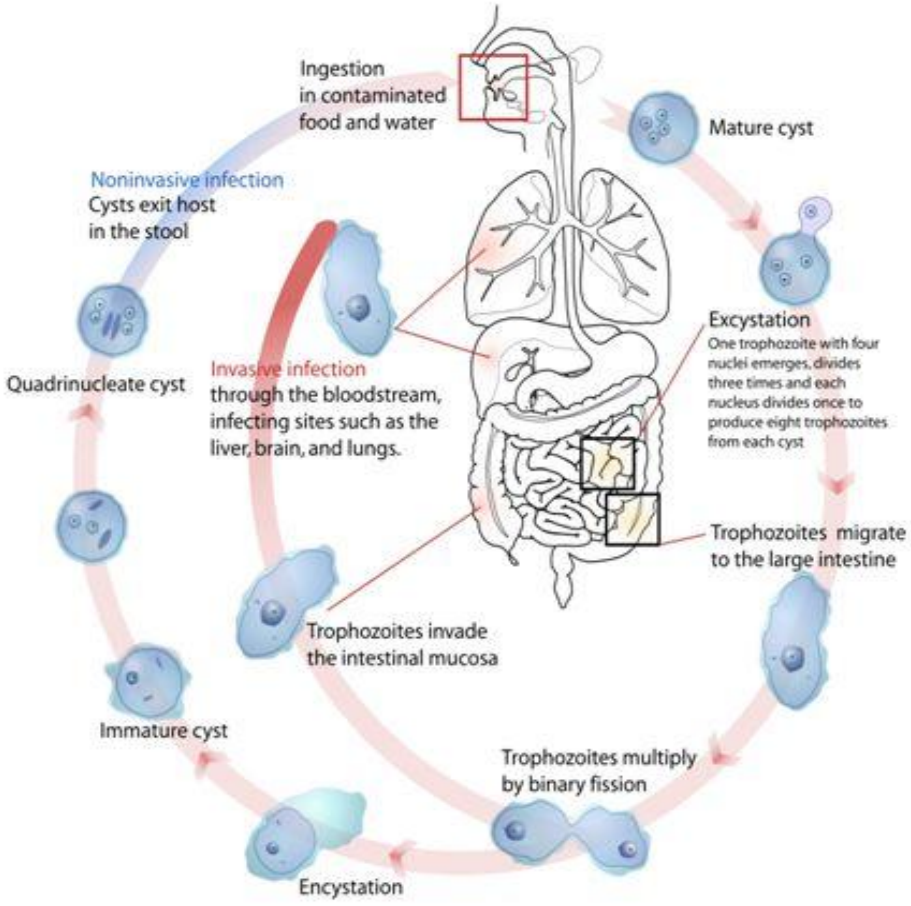
Sporozoa (Coccidian) - *Toxoplasma gondii* (Toxoplasmosis), Plasmodium spp. (Malaria).

1. Parasitic amoeba

Amoebiasis.

Entamoeba histolytica





Wikipedia

Ingestion of cysts

↓

Excystation in small intestine

↓

Division of quadrinucleate cyst into 4 and then 8 trophozoites

↓

Trophozoites move to colonize the colon

↓

Excystation

↓

Excretion of cysts

↓

Ingestion of cysts by the patient

Symptoms:

- Abdominal pain,
- Mild diarrhea, bloody diarrhea,
- Perforation and tissue death. This last complication may cause peritonitis.
- People affected may develop anemia due to loss of blood, weakness

Invasion of the intestinal lining causes amoebic bloody diarrhea or amoebic colitis. If the parasite reaches the bloodstream it can spread through the body, most frequently ending up in the liver where it causes amoebic liver abscesses.

Disease occurs when amoeba comes in contact with the cells lining the intestine. It then secretes the same substances it uses to digest bacteria, which include enzymes that destroy cell membranes and proteins. This process can lead to penetration and digestion of human tissues, resulting first in flack-shaped ulcers in the intestine.

Entamoeba histolitica ingests the destroyed cells by phagocytosis and is often seen with red blood cells inside when viewed in stool samples.

Transmission:

- It is usually transmitted by fecal-oral route, but it can also be transmitted indirectly through contact with dirty hands or objects as well as by anal-oral contact.
- Infection is spread through ingestion of the cyst form of the parasite. This form found in feces. Trophozoites may also be present in stool. These are rarely the source of new infections.
- Contaminated food (vegetables and fruits) and water.
- Soil cultivation.

Diagnosis:

- Microscopy of feces
- Serological tests. Serology becomes positive about 2 weeks after invasion.

Treatment:

Entamoeba histolitica infections occur in both the intestine and in tissue of the intestine and/or liver. As a result, two different classes of drugs are needs to treat the infection, one for each location. Such anti-amoebic drugs are known as amoebicides.

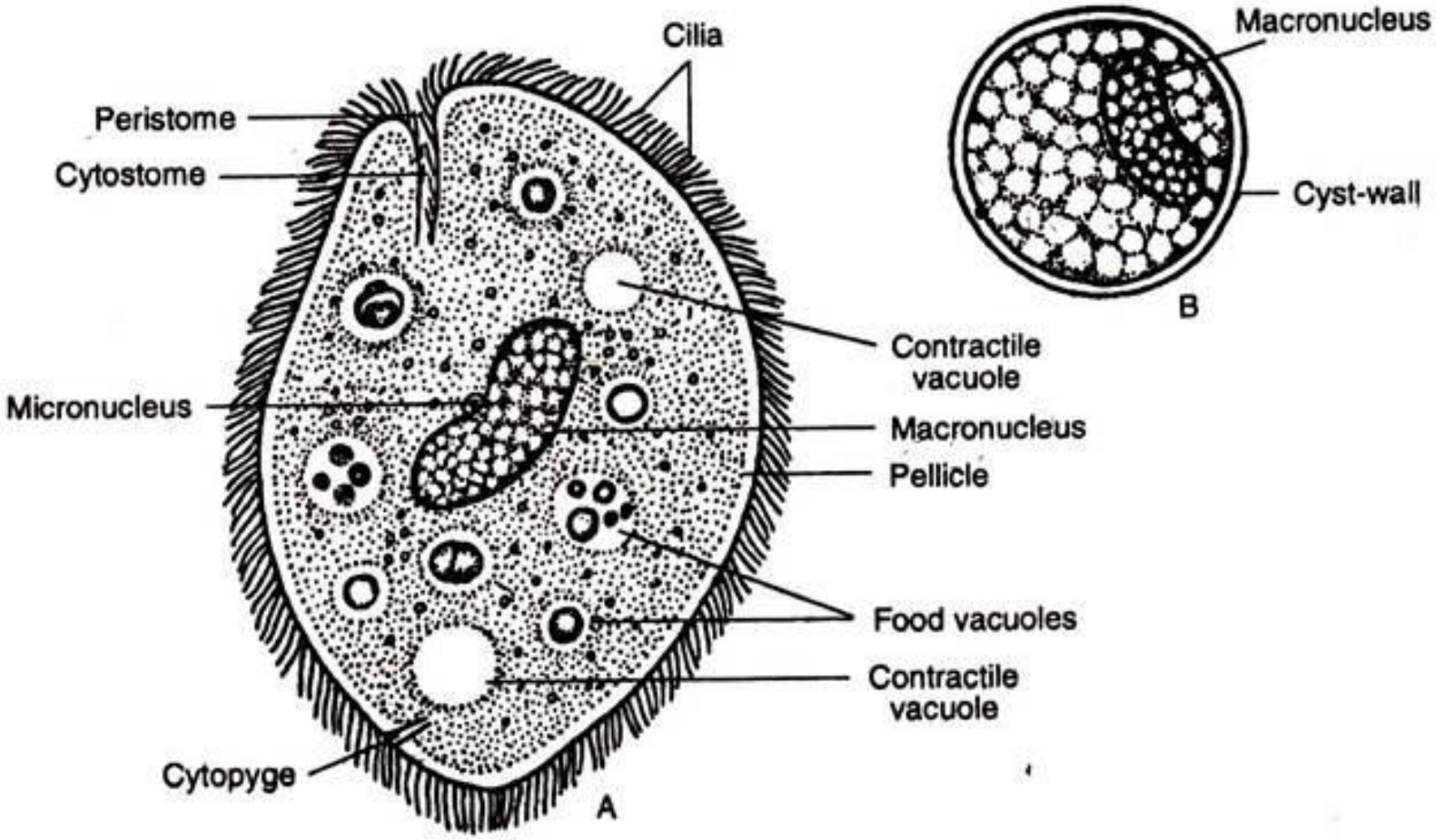
Prevention:

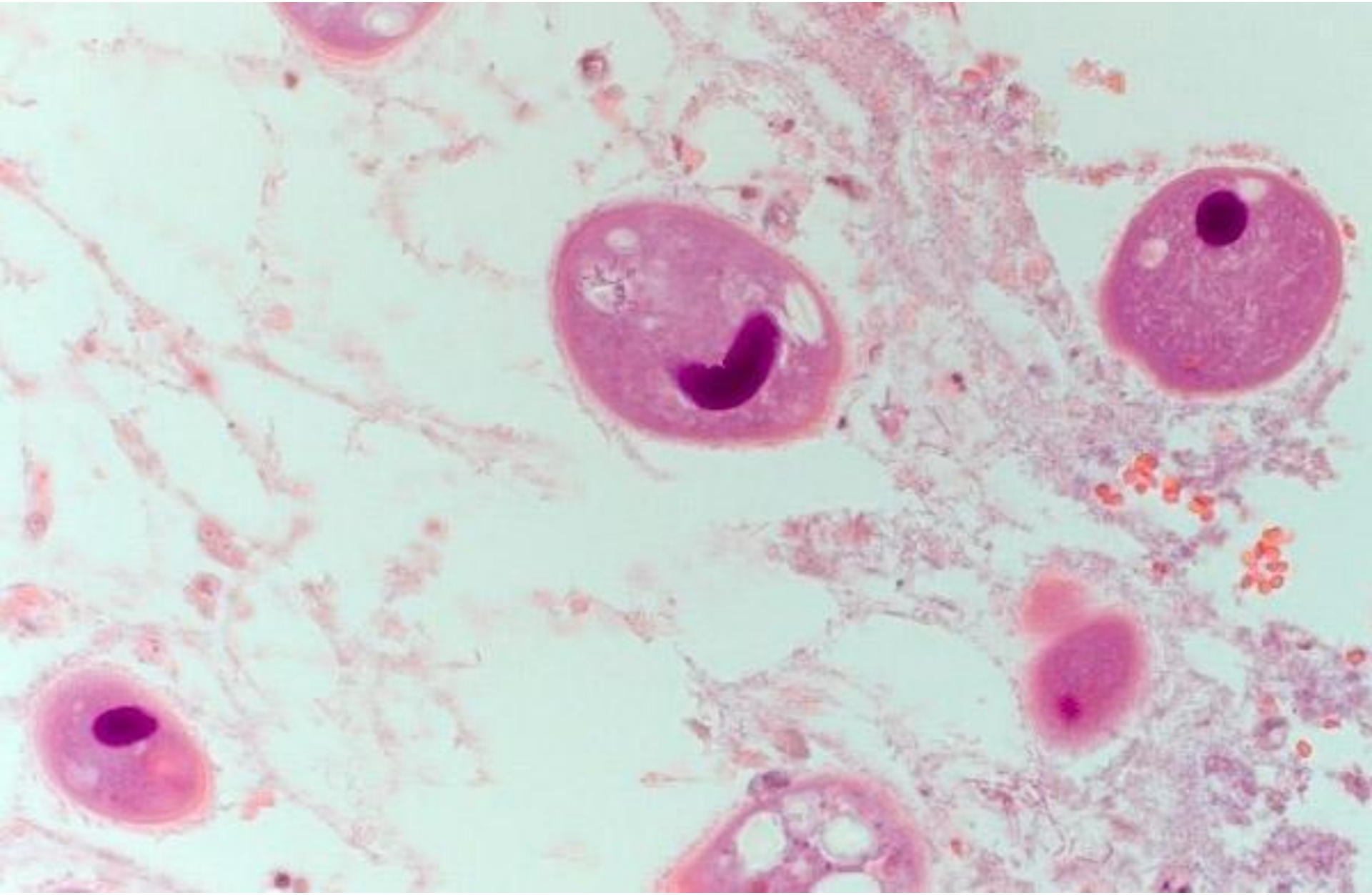
- Wash hands with soap and hot running water
- Clean bathrooms and toilets often
- Avoid sharing towels or face washers
- Clean vegetables and fruits
- Boil water or treat with iodine tablets
- Avoid eating street foods especially in public places where others are sharing sauces in one container.
- Cysts are usually resistant to chlorination; therefore, sedimentation and filtration of water supplies are necessary to reduce the incidence of infection.

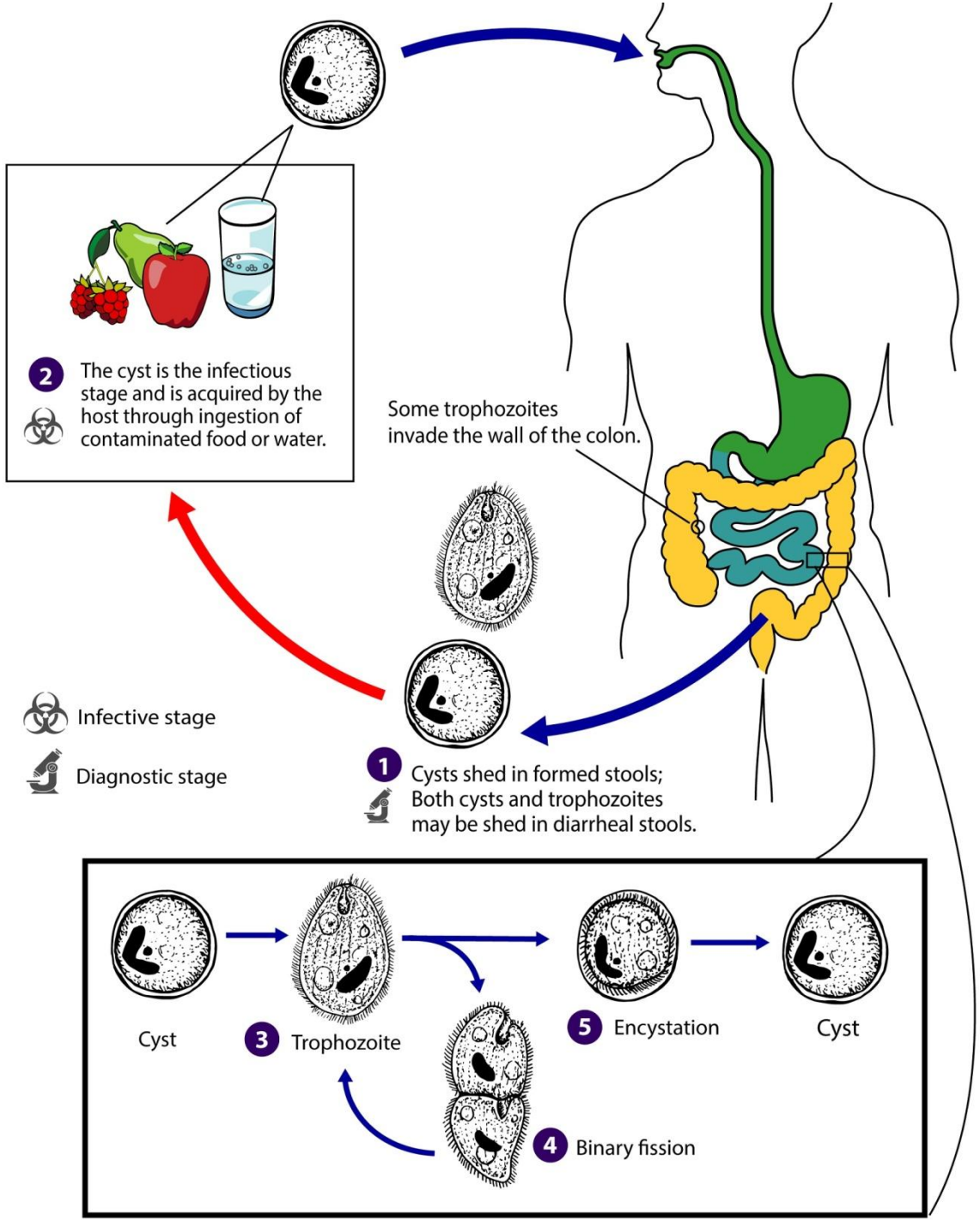
2. Parasitic ciliates

Balantidiasis.

Balantidium coli






2 The cyst is the infectious stage and is acquired by the host through ingestion of contaminated food or water.
Some trophozoites invade the wall of the colon.
Infective stage
Diagnostic stage
1 Cysts shed in formed stools; Both cysts and trophozoites may be shed in diarrheal stools.
Cyst
3 Trophozoite
4 Binary fission
5 Encystation
Cyst

Symptoms:

Can be local due to involvement of the intestinal mucosa, systemic in nature and include either diarrhea or constipation

- Frequent loose muco-purulent, then bloody stools
- Tenesmus
- Pain in the colon, formation of ulcers
- Loss of appetite
- Nausea
- General weakness, sometimes increasing temperature
- Peritonitis

Transmission:

The main source of infection is the pig. But man, releasing ciliates can infect others. Cysts under favorable conditions persist in the feces for several weeks and unable to get into the human digestive tract with contaminated food, water, vegetables and also via contaminated hands. Flies can also carry the cysts.

Diagnosis:

Microscopic examination of stool

Treatment:

Metronidazole

Prevention:

- Is the same as in other intestinal infections: measures that prevent pollution of the environment by feces of humans and pigs. In pig farms the need for good maintenance of pigs, cleaning pig, composting of feces. Special attention should be given to the timely identification and treatment of carriers and patients with this infection.
- Purification of drinking water
- Proper handling of food
- Careful disposal of humans feces
- Monitoring the contacts of balantidiasis patients

Home work. Task 1. *Amoeba and ciliates*

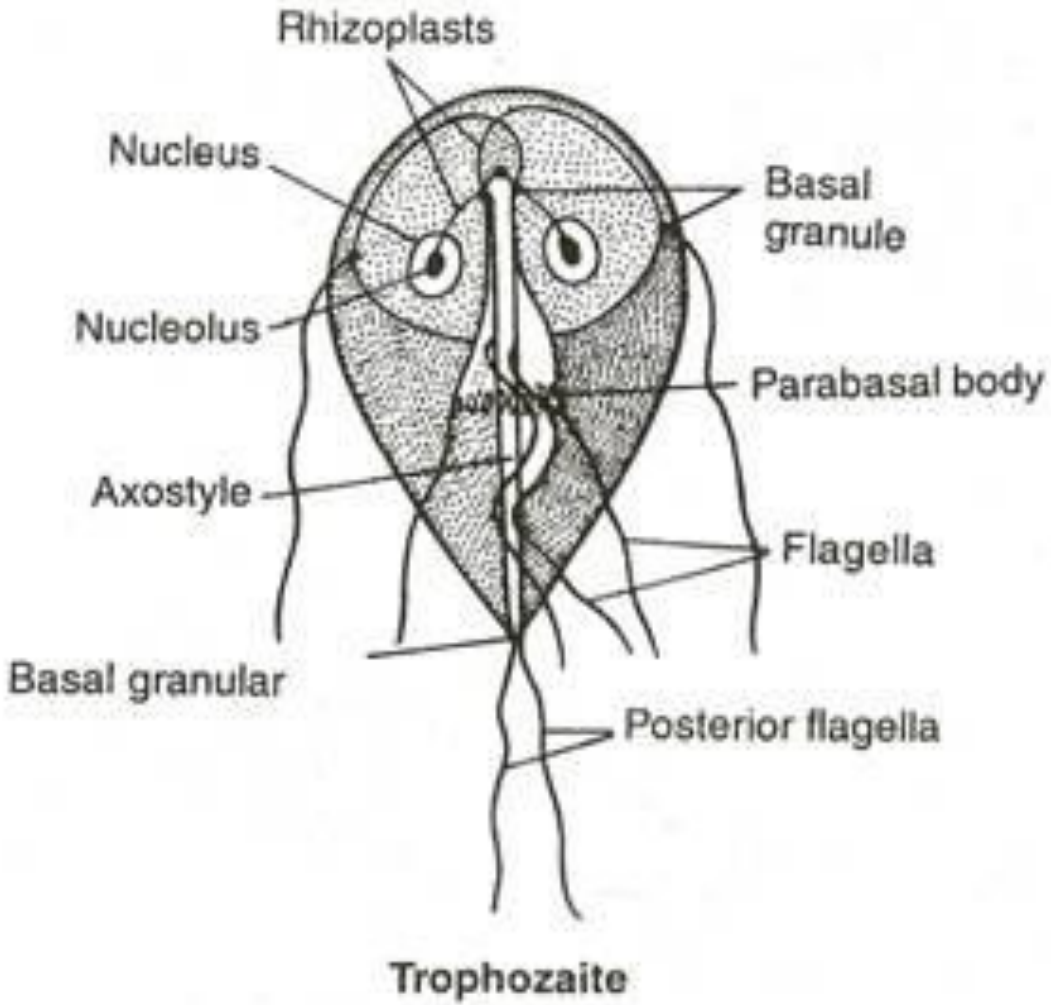
	Amoeba	Ciliates
Name of disease	**Amoebiasis**	**Balantidiasis**
Latin name of parasite	*Entamoeba histolitica*	*Balantidium coli*
Morphology (Forms of parasite)		
Infective form (Trophozoit or cyst)		
Transmission		
Symptoms		
Diagnosis		
Treatment		
Prevention		

3. Parasitic Flagellates:

3.1. Intestinal and vaginal Flagellates

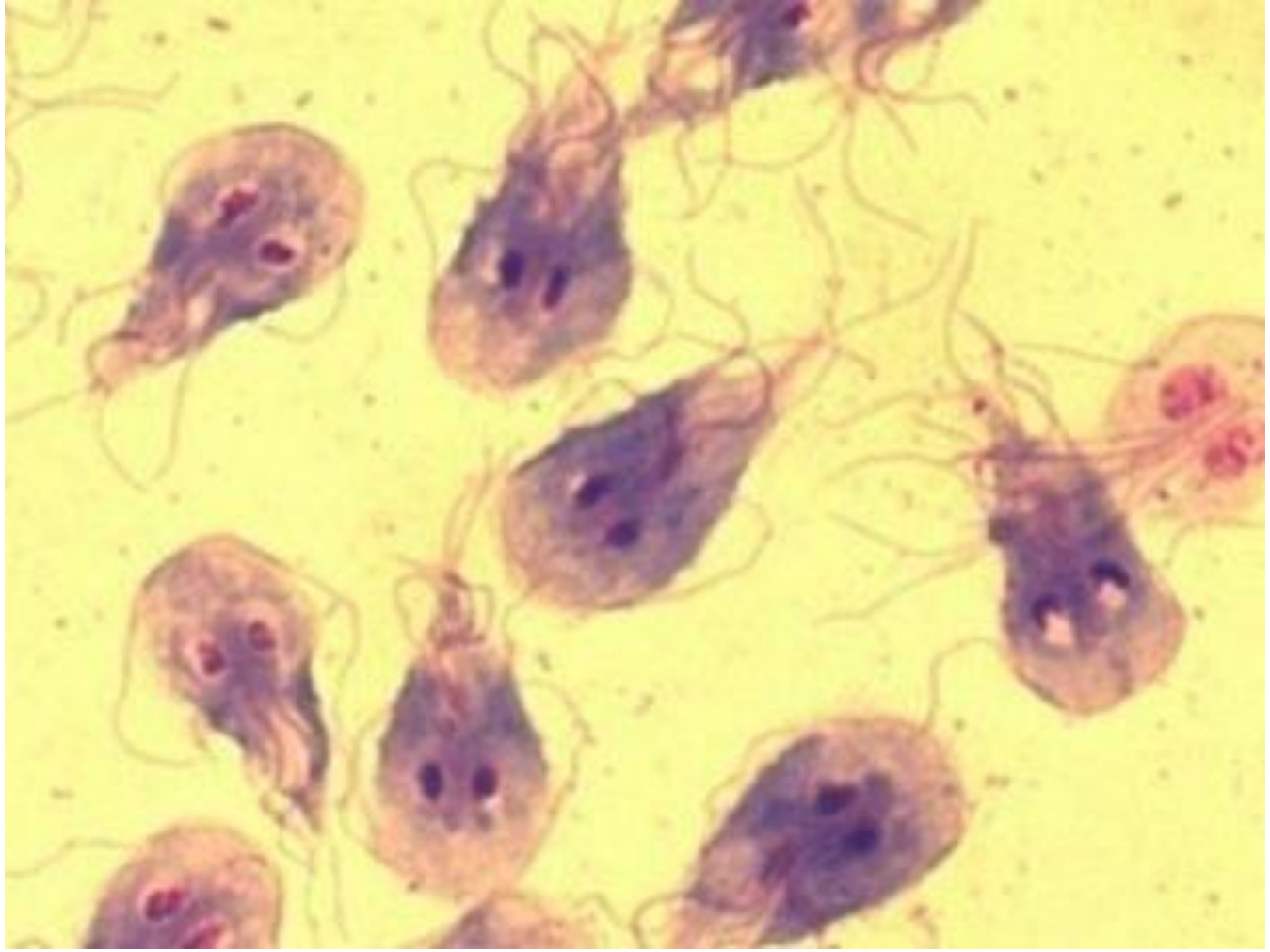
Giardiasis or **lambliosis**

Giardia lamblia



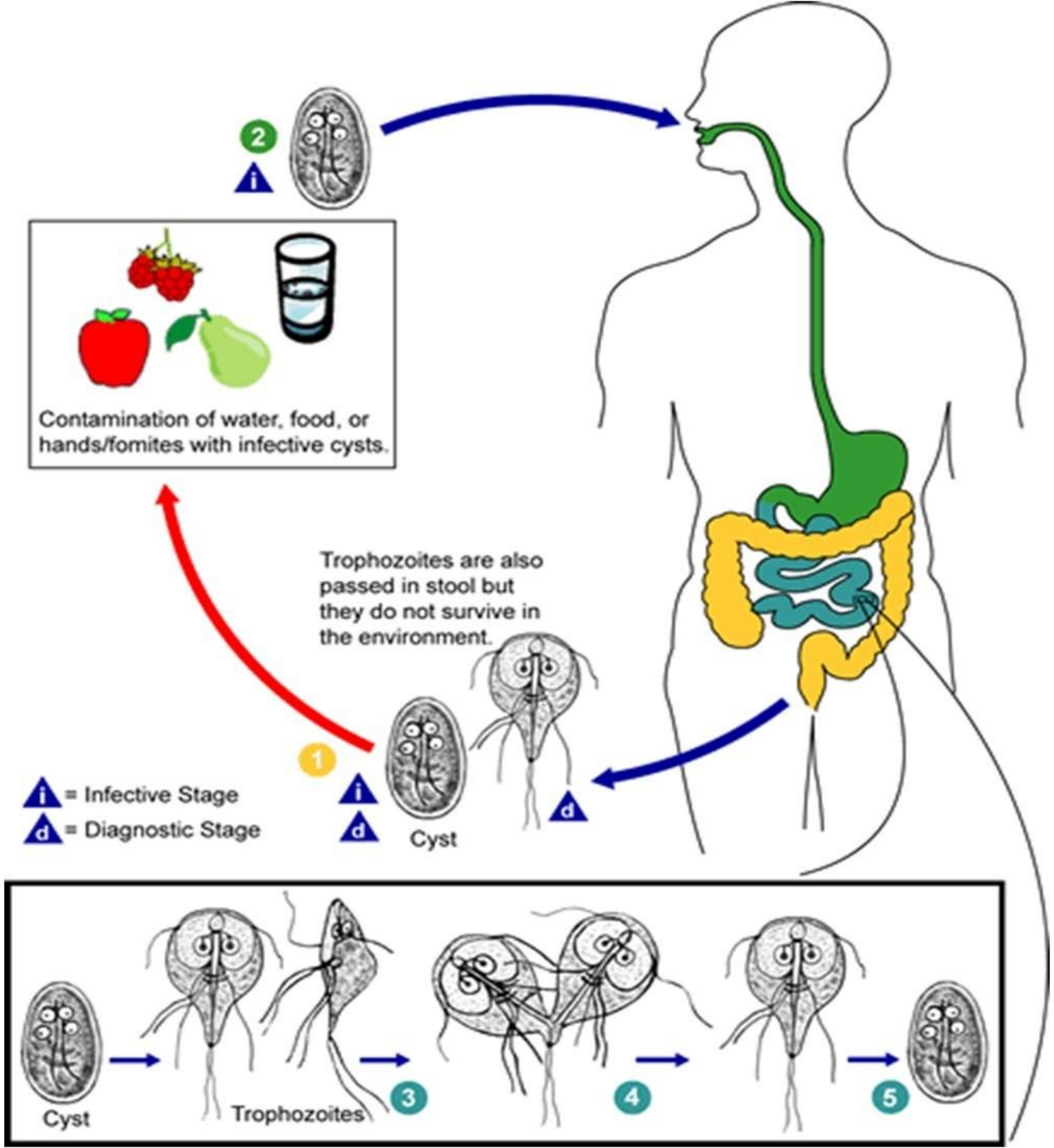
Trophozaite

Cyst



<u>Transmission</u>:

The source of infection is people. Transmission is by ingestion of the infective cyst. Infection occurs by eating infected cysts food, especially not subjected to heat treatment (vegetables, fruits, berries) and water. Also cysts via contaminated hands and household items.


2
i
Contamination of water, food, or hands/fomites with infective cysts.
Trophozoites are also passed in stool but they do not survive in the environment.
1
i
d
Cyst
i = Infective Stage
d = Diagnostic Stage
Cyst
Trophozoites
3
4
5

Symptoms:

The main habitat of giardia in human body are duodenum and initial part of colon. Parasites attached to the villi of the mucous membrane of the small intestine and, apparently, eat food parietal digestion.

Mechanical irritation of interoreceptors Giardia the small intestine can lead to reflex disturbance of its function, which apparently can be cause of a violation of the physiology of digestion.

Diarrhea, smelling gas, headaches, weight loss, fatigue, loss of appetite, fell sick in general, abdominal pain, particularly cramping, bloating, nausea with/or without vomiting, malaise. Fever is unusual.

Diagnosis:

- Antigene testing of the stool for the presence of giardial proteins
- Examination of stool under microscope for cyst or trophzoites
- Examination of fluid from the duodenum
- Biopsy of the small intestine

Treatment:

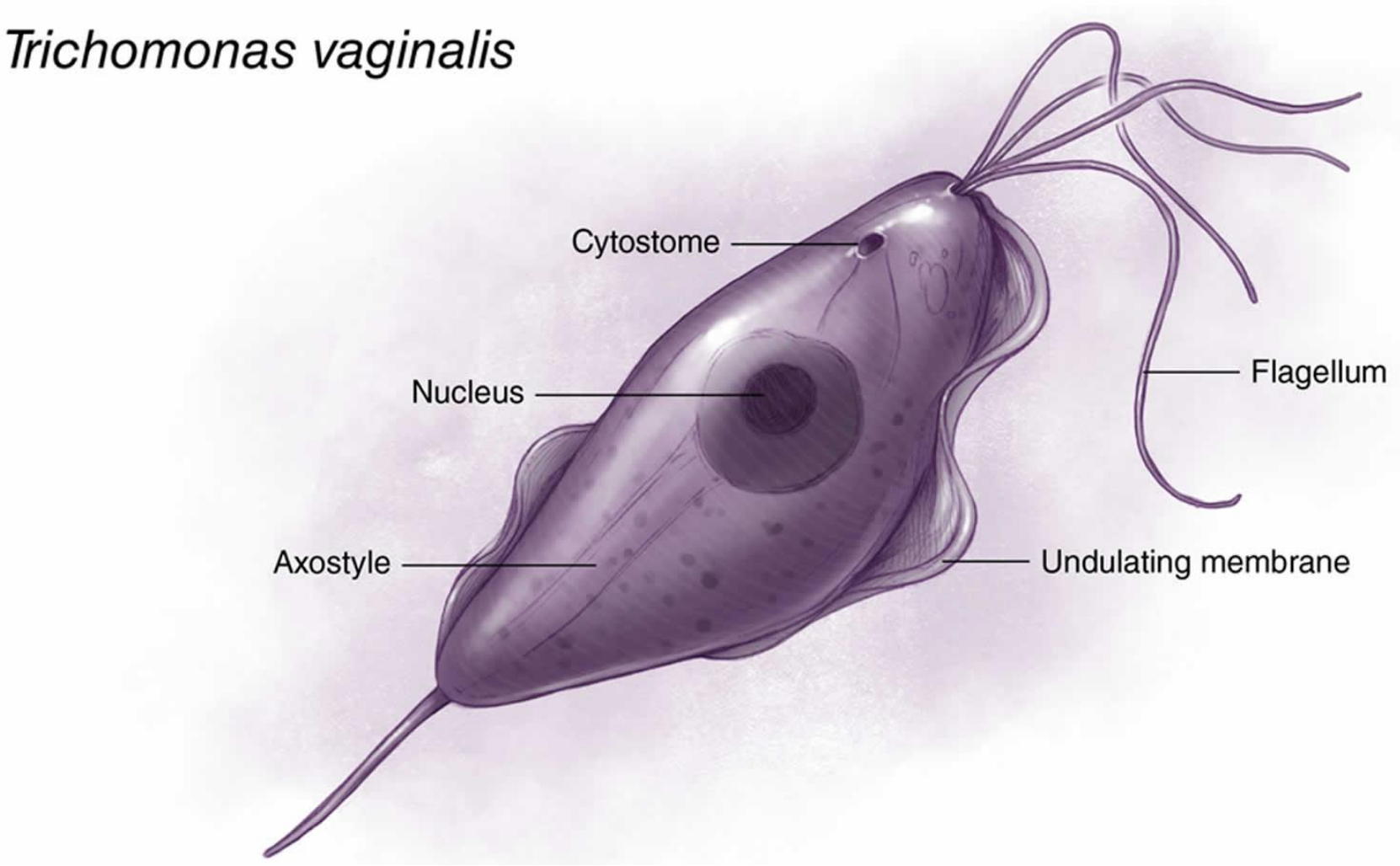
Antiparasitic drugs – metronidazole, tinidazole, paromomycin

Prevention:

- Washing hands
- Don’t swallow water from rivers and lakes
- Drink clean water, don’t drink tap water
- Avoid eating uncooked food. Clean vegetables, fruits and berries before eating.

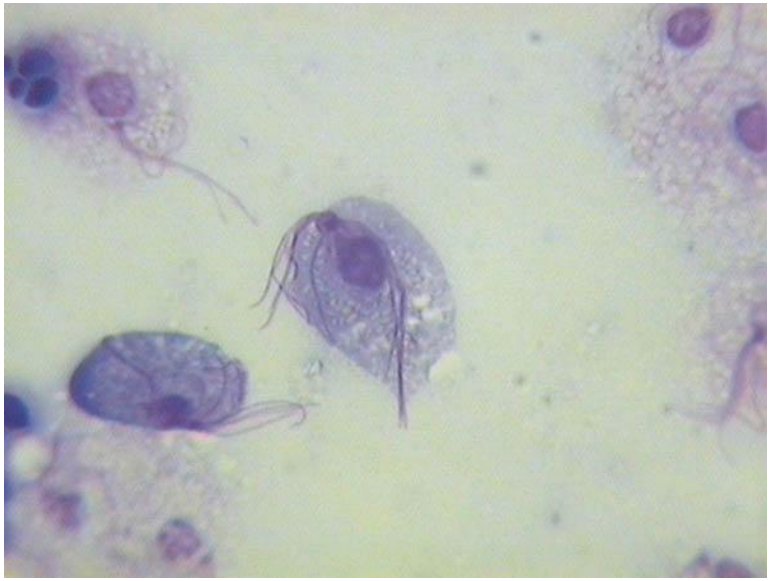
Trichomoniasis.

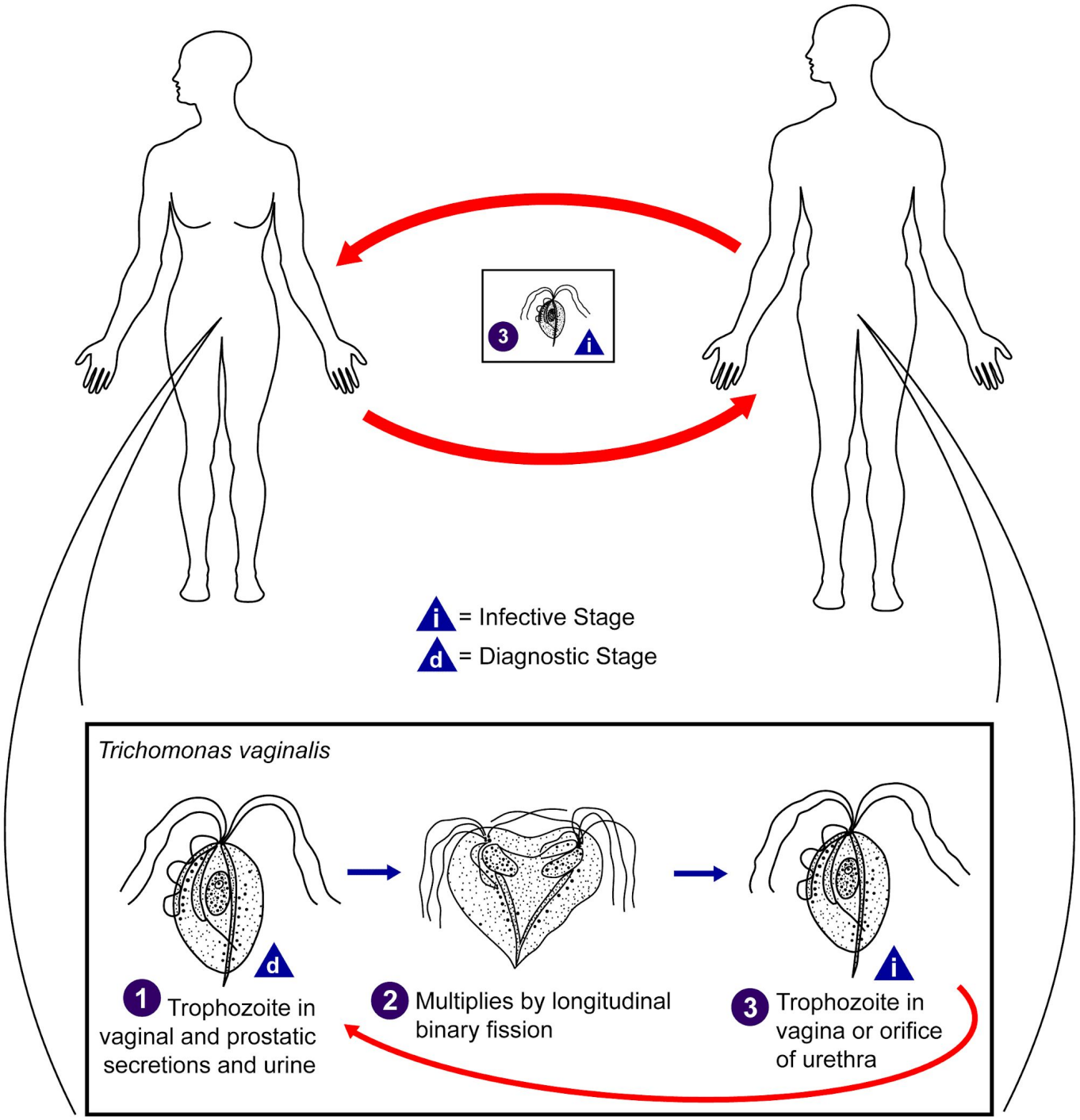
Trichomonas vaginalis



Is a sexually transmitted infection. It caused by Trichomonas vaginalis. It is very common. This tiny parasites travels from person to person via genital contact. Fortunately it is also easily treatable. Often has no symptoms, especially in men. Symptoms can begin anywhere from 3 to 28 days after being infections.

It is a pear-shaped organism with a central nucleus and four anterior flagella; and undulating membrane extends about two-thirds of its length. It exists only as a trophozoite form, and measured 7-23µm long and 5-15µm wide. Transmission is by sexual intercourse.




3
i
i = Infective Stage
d = Diagnostic Stage
Trichomonas vaginalis
d
1 Trophozoite in vaginal and prostatic secretions and urine
2 Multiplies by longitudinal binary fission
i
3 Trophozoite in vagina or orifice of urethra

Symptoms:

- Vaginal discharge, which may be white, gray, yellow or green and usually has unpleasant smell.
- Genital redness or swelling
- Pain during urination or sexual intercourse.
- An urge to urinate frequently
- Discharge from the urethra
- Burning during urination
- An urge to urinate frequently

Diagnosis:

Physical test and laboratory test: cell culture, DNA examing samples of vaginal fluids.

Treatment:

Metronidazole is the drug of choice. If resistant cases occur, re-treatment with higher doses is required.

Prevention:

- Both male and female sex partners must be treated to avoid reinfection
- Good personal hygiene, avoidance of shared toilet articles and clothing.
- Safe sexual practice.

Home work. Task 2.
Amoeba, Ciliates, intestinal and vaginal flagellates

Name of disease	Amoebiasis	Balantidiasis	Giardiasis	Trichomoniasis
Latin name of parasite	*Entamoeba histolitica*	*Balantidium coli*	*Giardia lamblia*	*Tricomonis vaginalis*
Morphology (Forms of parasite)				
Infective form (Trophozoit or cyst)				
Transmission				
Symptoms				
Diagnosis				
Treatment				
Prevention				

3. Parasitic Flagellates:
3.2. Hemoflagellates

Leishmaniasis.

Is a disease caused by protozoan parasites of the genus ***Leishmania*** and spread by the bite of certain types of sandflies.

Definitive host: human

Intermediate host: sandfly

Symptoms:

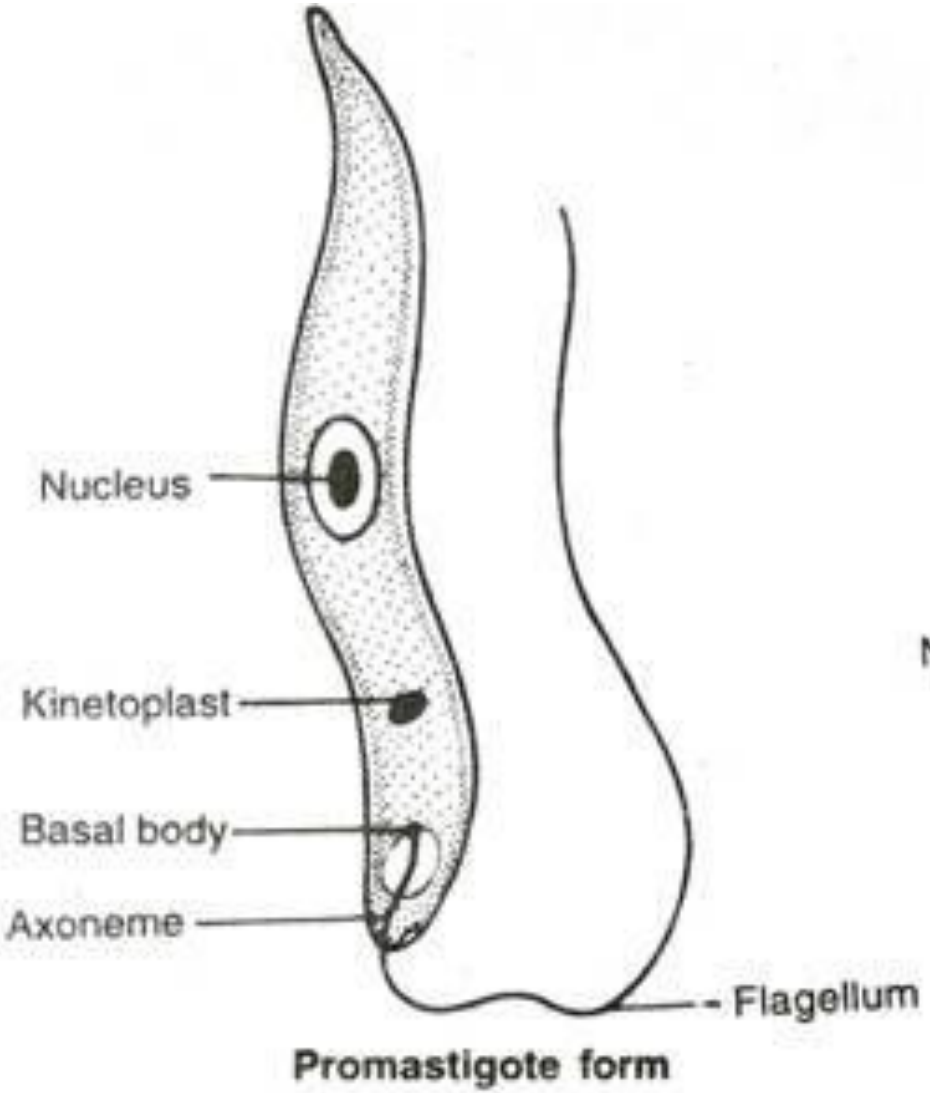
The disease can present in three main ways: **cutaneous, mucocutaneous,** or **visceral**.

1. The **cutaneous form** presents with skin ulcers,

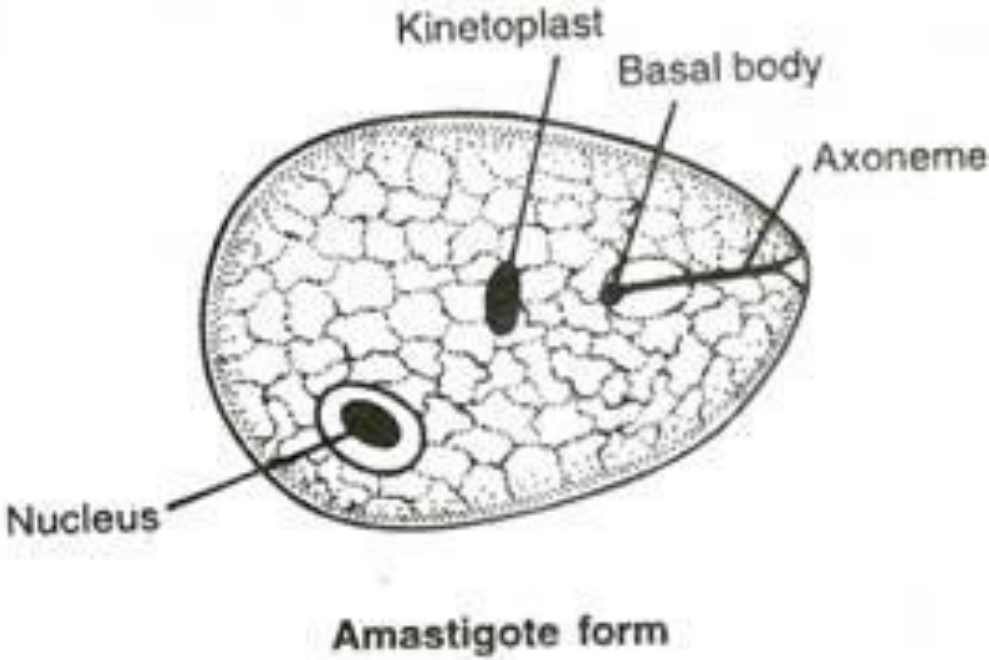
2. while the **mucocutaneous form** presents with ulcers of the skin, mouth, and nose.

3. **Visceral form** starts with skin ulcers and then later presents with fever, low red blood cells, and enlarged liver or spleen.

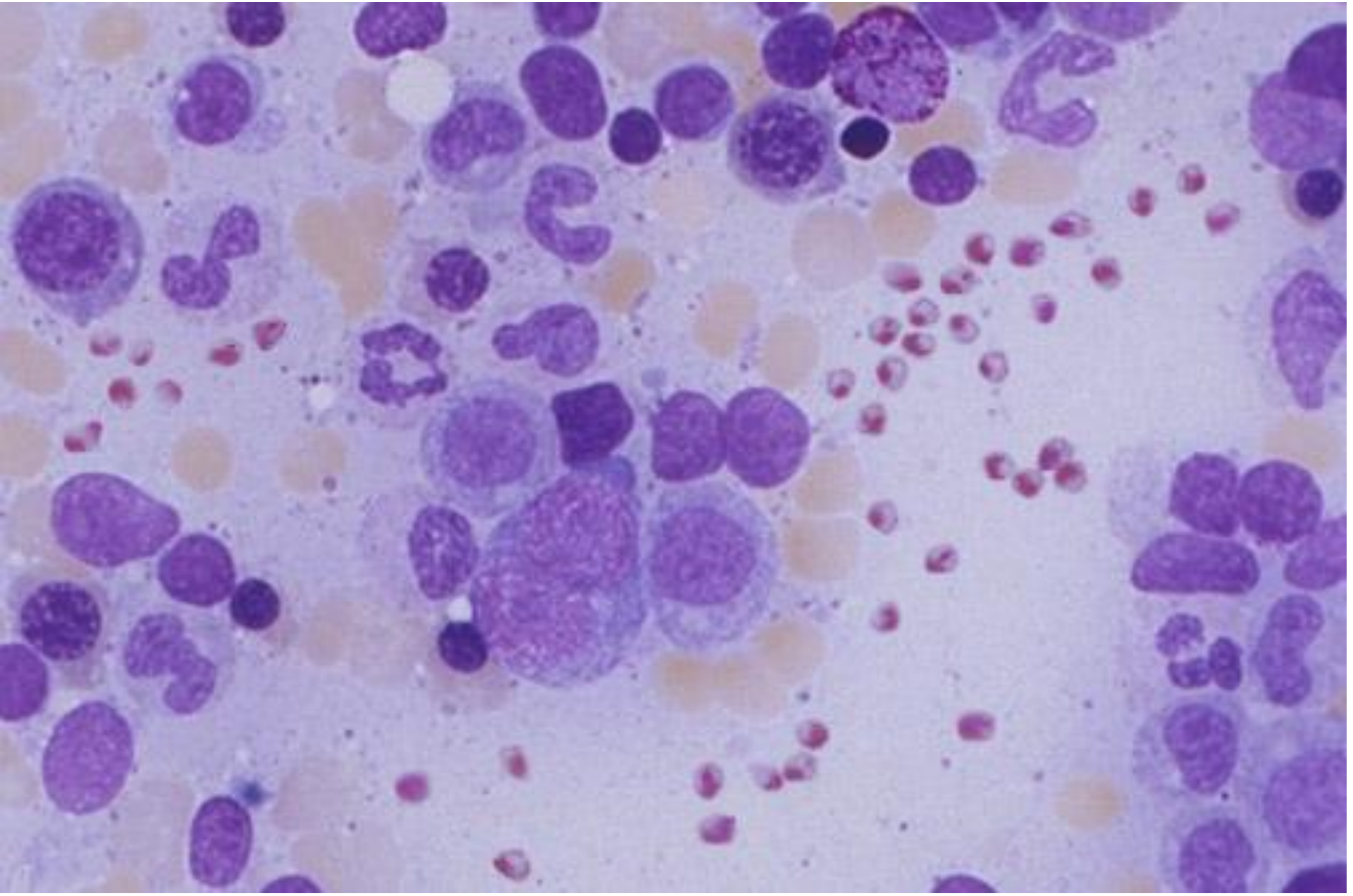
Morphology



Promastigote form



Amastigote form

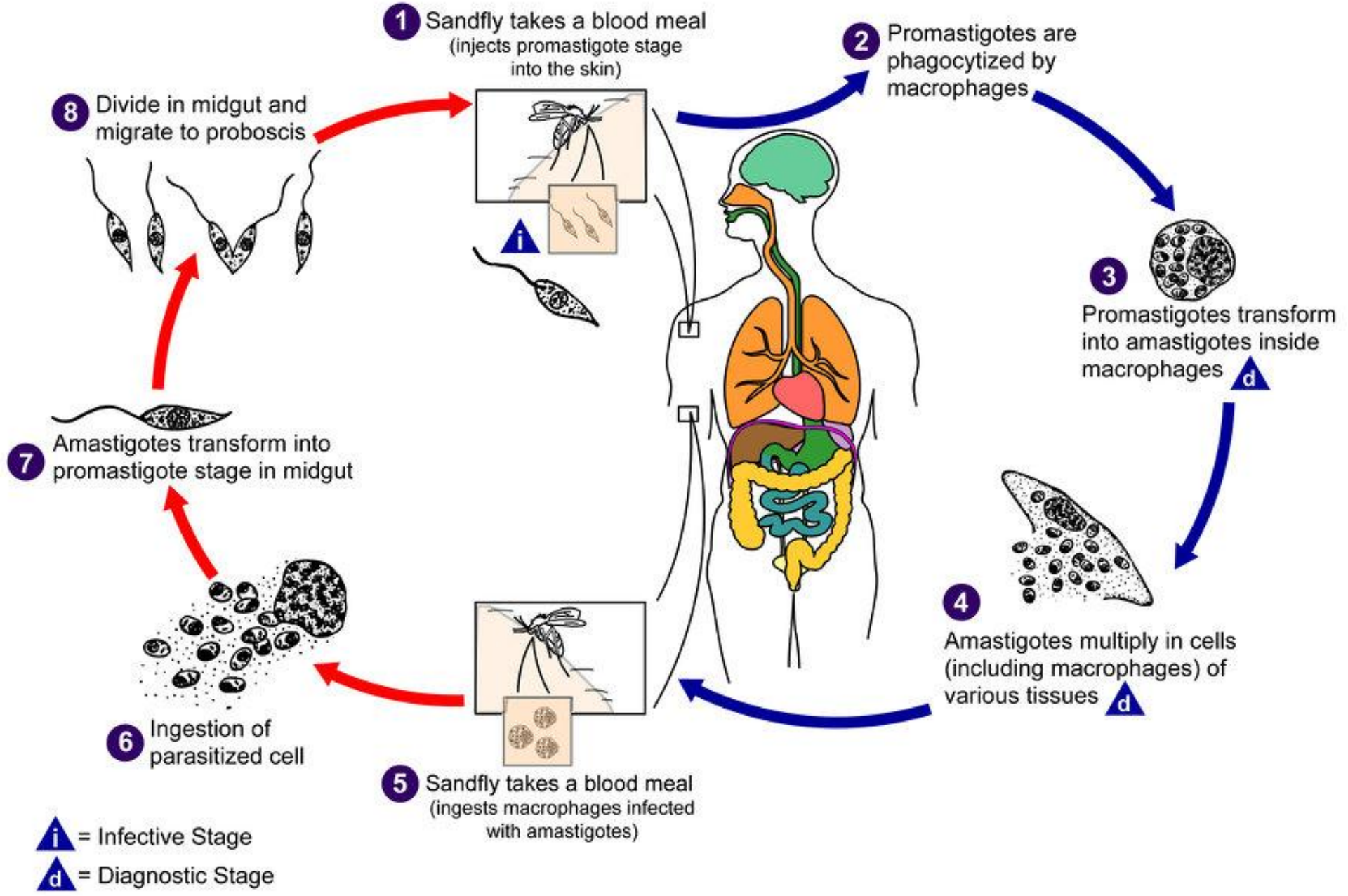


Pathogenesis

In visceral leishmaniasis, the organs of the reticuloendothelial system (liver, spleen and bone marrow) are the most severely affected organs. Reduced bone marrow activity, coupled with cellular distraction in the spleen, results in anaemia, leukopenia and thrombocytopenia. This leads to secondary infections and a tendency to bleed. The spleen and liver become markedly enlarged, and hypersplenism contributes to the development of anaemia and lymphadenopathy also occurs. Increased production of globulin results in hyperglobulinemia, and reversal of the albumin-to-globulin ratio.

Clinical features

Symptoms begin with intermittent fever, weakness, and diarrhea; chills and sweating that may resemble malaria symptoms are also common early in the infection. As organisms proliferate and invade cells of the liver and spleen, marked enlargement of the organs, weight loss, anemia, and emaciation occurs. With persistence of the disease, deeply pigmented, granulomatous lesion of skin, referred to as post-kala-azar dermal leishmaniasis, occurs. Untreated visceral leishmaniasis is nearly always fatal as a result of secondary infection.


1 Sandfly takes a blood meal
(injects promastigote stage into the skin)
i
2 Promastigotes are phagocytized by macrophages
3 Promastigotes transform into amastigotes inside macrophages
d
4 Amastigotes multiply in cells (including macrophages) of various tissues
d
5 Sandfly takes a blood meal
(ingests macrophages infected with amastigotes)
6 Ingestion of parasitized cell
7 Amastigotes transform into promastigote stage in midgut
8 Divide in midgut and migrate to proboscis
i = Infective Stage
d = Diagnostic Stage

Diagnosis:

Seeing the parasites under the microscope.

Visceral disease can be diagnosed by blood tests.

Examination of tissue biopsy, spleen aspiration, bone marrow aspiration orlymph node aspiration in properly stained smear (e.g. Giemsa stain).

The amastigotes appear as intracellular and extra cellular L. donovan (LD) bodies.

Culture of blood, bone marrow, and other tissue often demonstrates the promastigote stage of the organisms.

Serologic testing is also available.

Leishman skin test

Treatment:

Liposomal amphotericin B, miltefosine, paromomycin, fluconazole, pentamidine, allopurinol.

Prevention:

Spraying insecticides to kill sandflies and treating people with the disease.

Trypanasomiasis

This disease caused by **Tripanosoma species**. In humans this include African and South-American types.

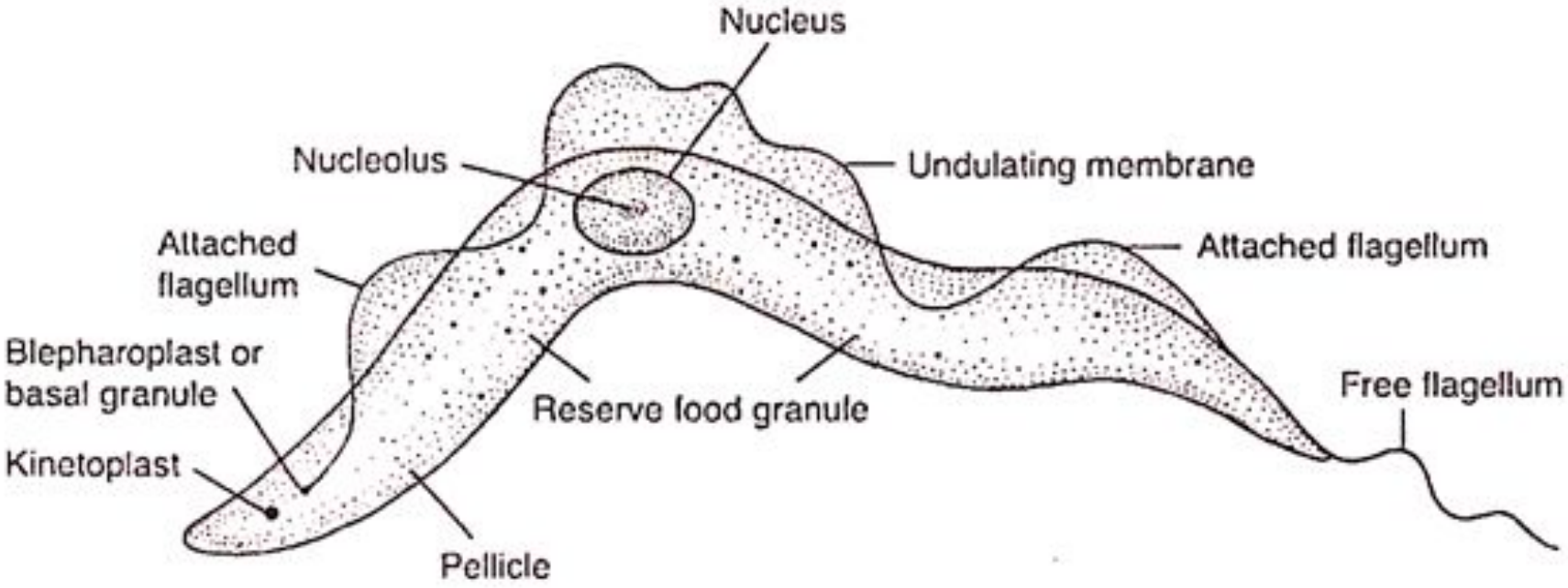
Trypanosoma brucei complex – African trypanosomiasis (sleeping sickness)

Trypanosoma cruzi – American trypanosomiasis (Chagas' disease)

These species may have amastigote, promastigote, epimastigote, and trypomastigote stages in their life cycle. In human trypanosomes of the **African form**, however, the amastigote and promastigote stages of development are absent.

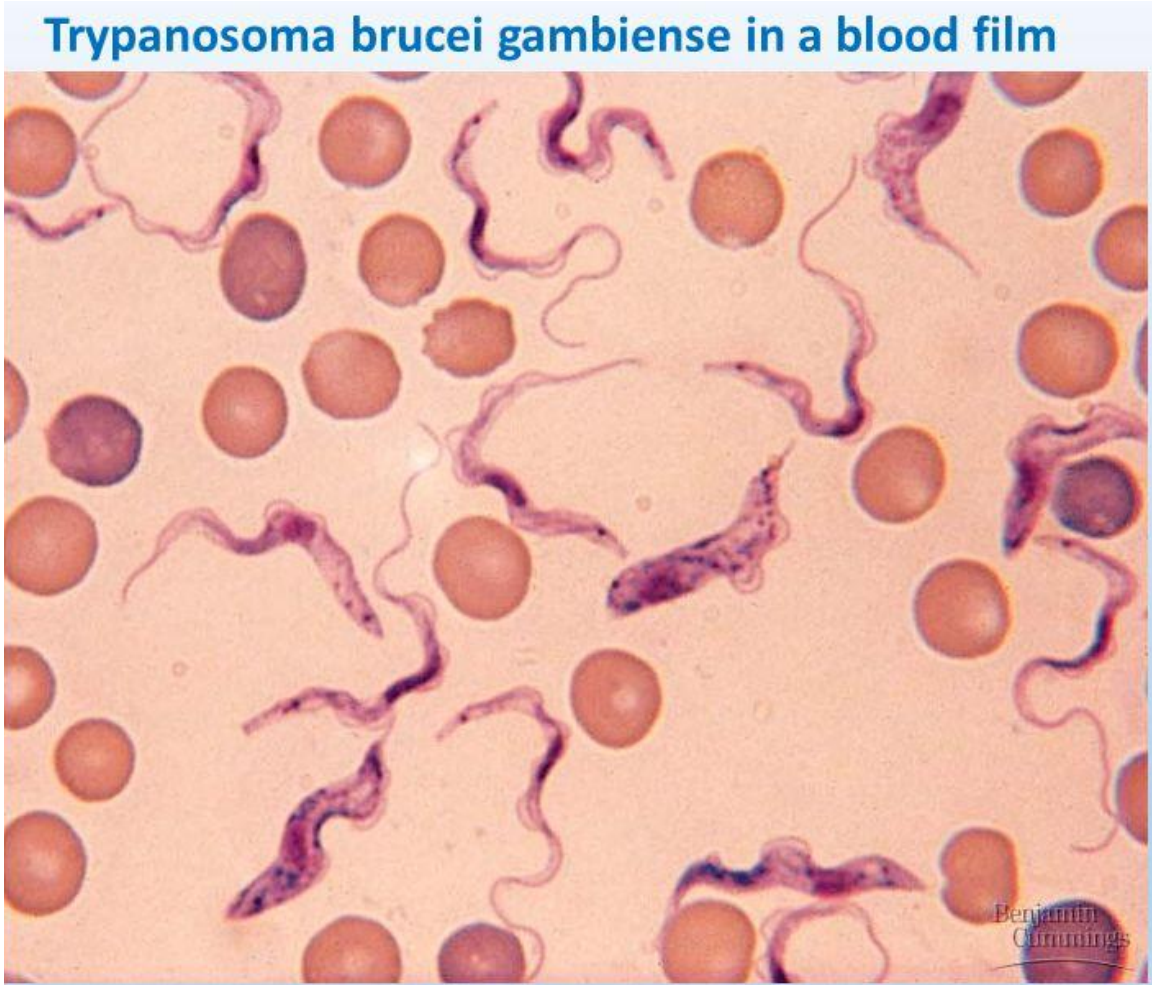
Morphology

Typical trypanosome structure is an elongated spindle-shaped body that more or less tapers at both ends, a centrally situated nucleus, a kinetoplast posterior to nucleus, an undulating membrane arising from the kinetoplast and proceeding forward along the margin of the cell membrane and a single free flagellum at the anterior end.



African type of Trypanosomiasis.

It caused by ***Trypanosoma brucei***. Common name: sleeping sickness. Definitive host: man, intermediate host: tsetse fly. Infective stage: metacyclic trypomastigote.

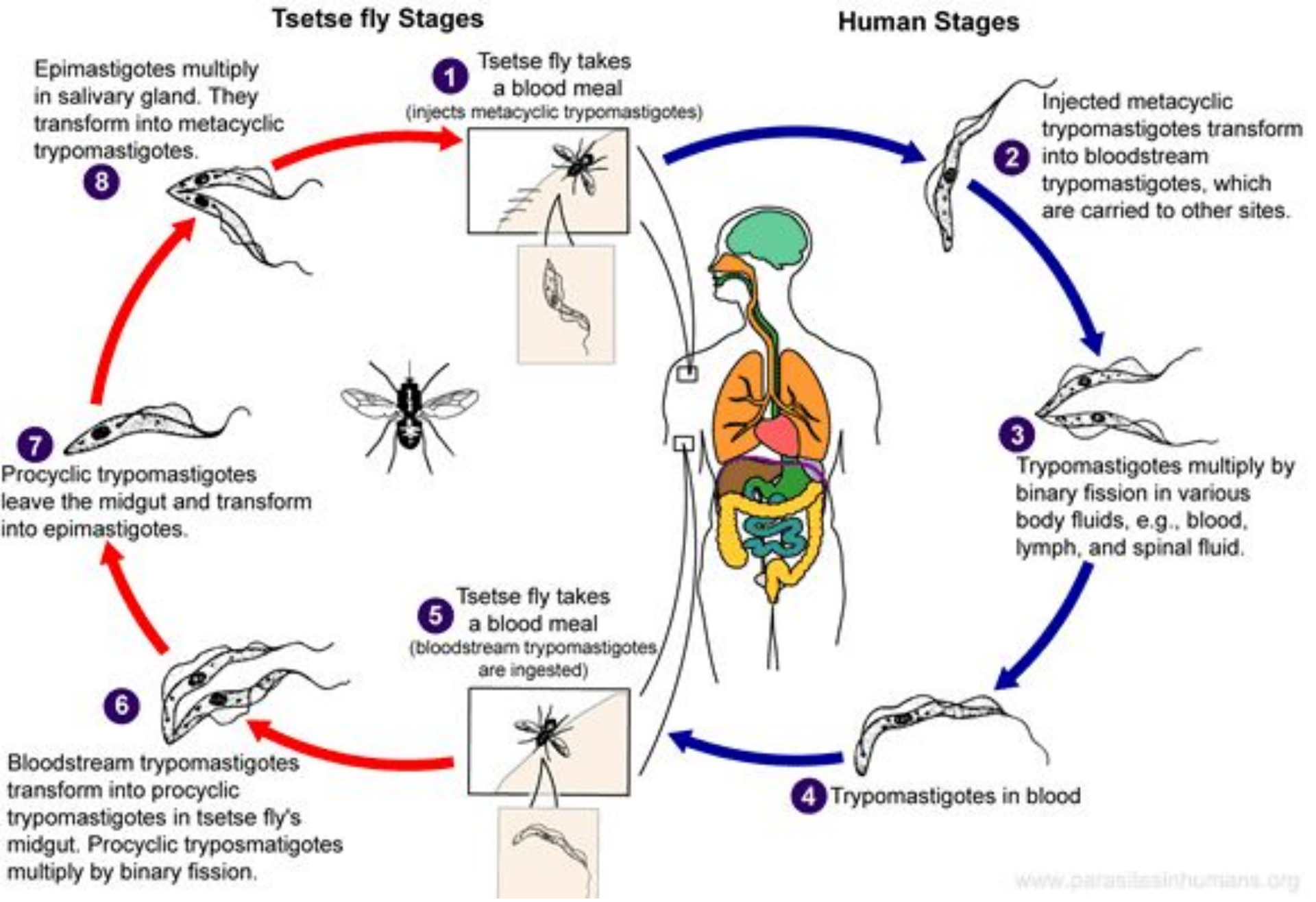


Symptoms

Transmission **by tsetse fly**.

The tsetse fly bite erupts into a red chancre sore and within a few weeks, the person can experience fever, swollen lymph glands, blood in the urine, aching muscles and joints, headaches and irritability. In first phase, the patient has only intermittent bouts of fever with lymphadenopathy together with other non-specific signs and symptoms.

The second and third stage is marked by involvement of the central nervous system with extensive neurological effects like changes in personality, alteration of the biological clock, confusion, slurred speech, seizures and difficulty in walking and talking. Also general toxic symptoms, anemia, bone pain. These problems can develop over many years and if not treated, the person dies.


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)
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.
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.

Amastigotes

Tripomastigotes

Epimastigotes

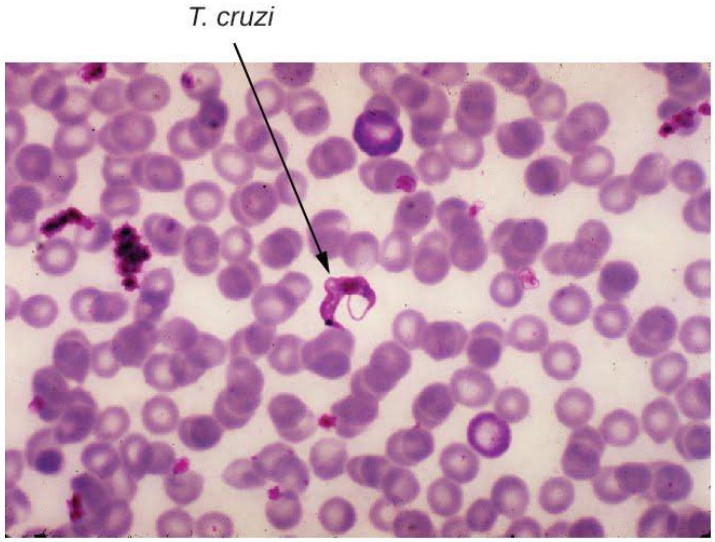
South-American type of Trypanosomiasis.

Common name: Chaga's disease.

It caused by ***Trypanosoma cruzi***.

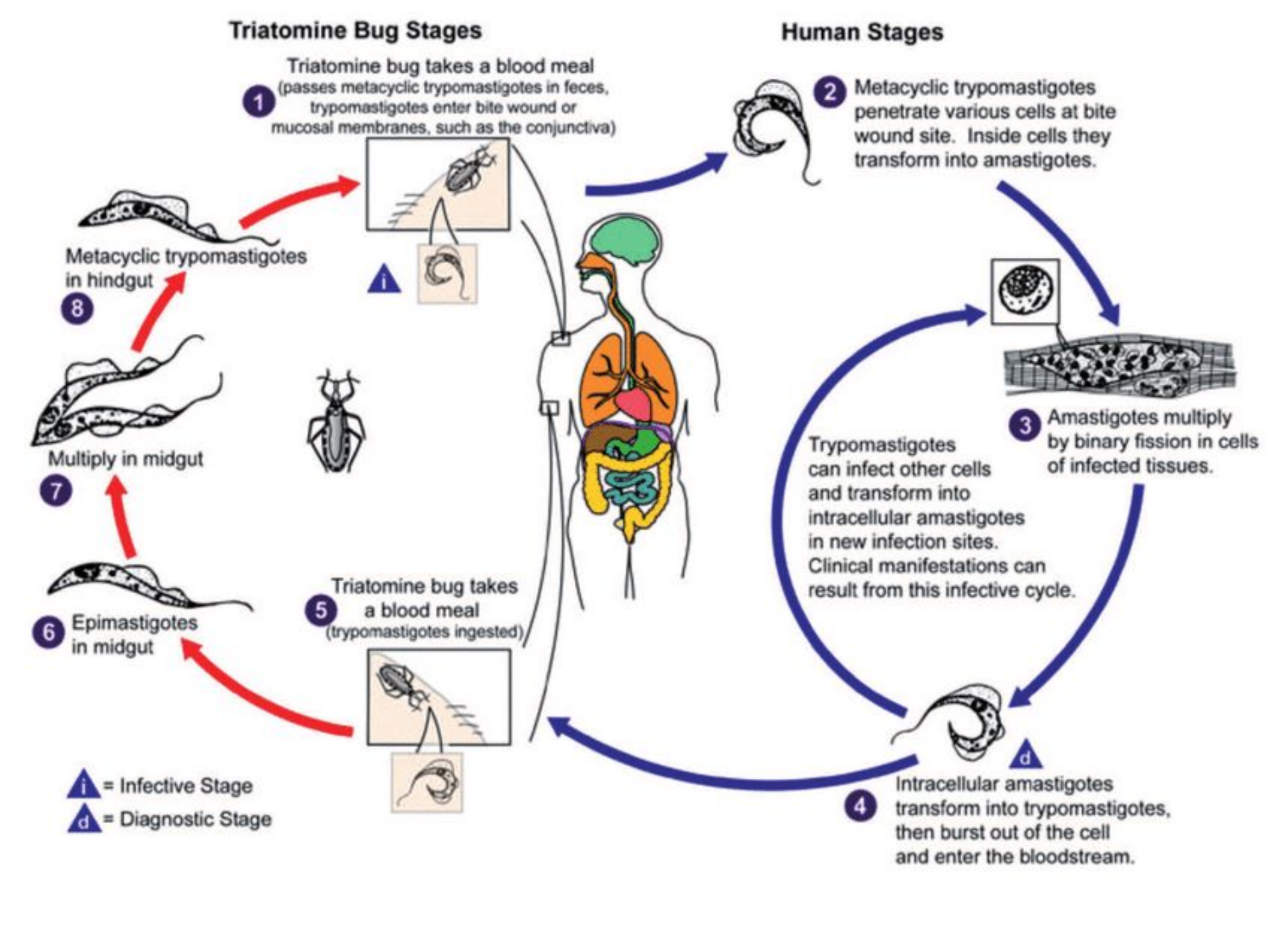
Definitive host: man,

Intermediate host: cone bug.



(a)

(b)


Triatomine Bug Stages
Human Stages
Triatomine bug takes a blood meal
(passes metacyclic trypomastigotes in feces, trypomastigotes enter bite wound or mucosal membranes, such as the conjunctiva)
1
2
Metacyclic trypomastigotes penetrate various cells at bite wound site. Inside cells they transform into amastigotes.
Metacyclic trypomastigotes in hindgut
8
i
3
Amastigotes multiply by binary fission in cells of infected tissues.
Trypomastigotes can infect other cells and transform into intracellular amastigotes in new infection sites. Clinical manifestations can result from this infective cycle.
Multiply in midgut
7
Triatomine bug takes a blood meal
(trypomastigotes ingested)
5
6
Epimastigotes in midgut
d
4
Intracellular amastigotes transform into trypomastigotes, then burst out of the cell and enter the bloodstream.
i = Infective Stage
d = Diagnostic Stage

Symptoms of an acute form:

- Blood and reticulo-endothelial cells predominantly involved
- Fever
- Oedema (lymph blockage)
- Lymphadenopathy
- Enlargement of liver and spleen
- Sometimes encephalitis

Symptoms of chronic form:

- General toxic symptoms and focal signs depending on localization (toxic depression of bone marrow, anemia, hepatomegaly, splenomegaly, lymphadenopathy)
- Predominantly cardiac and CNS manifestations (myocarditis, tachycardia, heart block, encephalitis, general or focal CNS signs and symptoms)
- May be asymptomatic.

Diagnosis:

Often missed in the first phase of the disease due to non-specific nature of symptoms. Examine thin or thick stained preparations for trypomastigotes. Wet preparations should also be examined to look for motile organisms that leave the blood stream and become difficult to find. Biopsy of lymph nodes, liver, spleen, or bone marrow may demonstrate organisms in amastigote stage. Xenodiagnosis - which consists of allowing an uninfected, laboratory-raised reduviid bug to feed on the patient and, after several weeks, examining the intestinal contents of the bug for the organism.

Treatment:

Pentamidine and suramin are used for treatment in the first stage.
Melarsoprol, nifurtimox and eflornithine are used in the second stage.

Prevention:

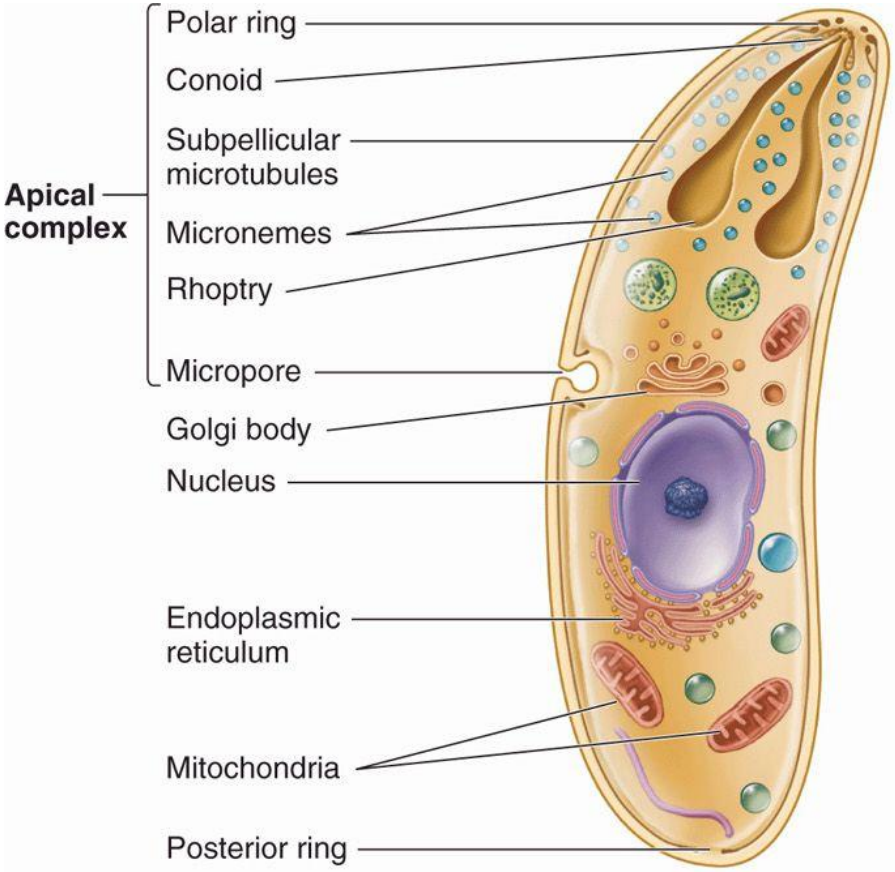
Spraying insecticides, repellents, using mosquito nets.
Treating infected person and exclusion of donors by screening blood.
Development of vaccine.

Home work. Task 3. *Hemoflagellates*

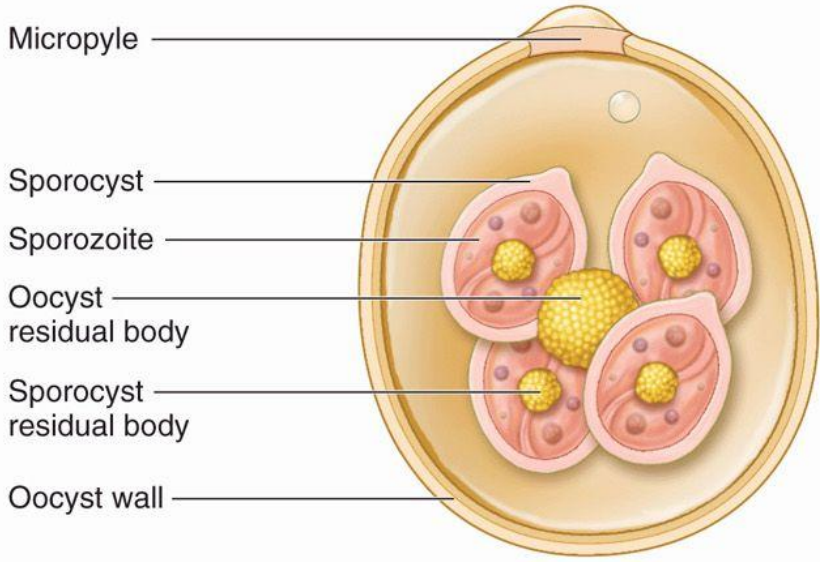
Name of disease	Leishmaniasis	Trypanosomiasis	
Latin name of parasite	*Leishmania donovani*	*Trypanosoma brucei*	*Trypanosoma cruzi*
Morphology (Forms of parasite)			
Infective form			
Transmission			
Symptoms			
Diagnosis			
Treatment			
Prevention			

4. Sporozoa

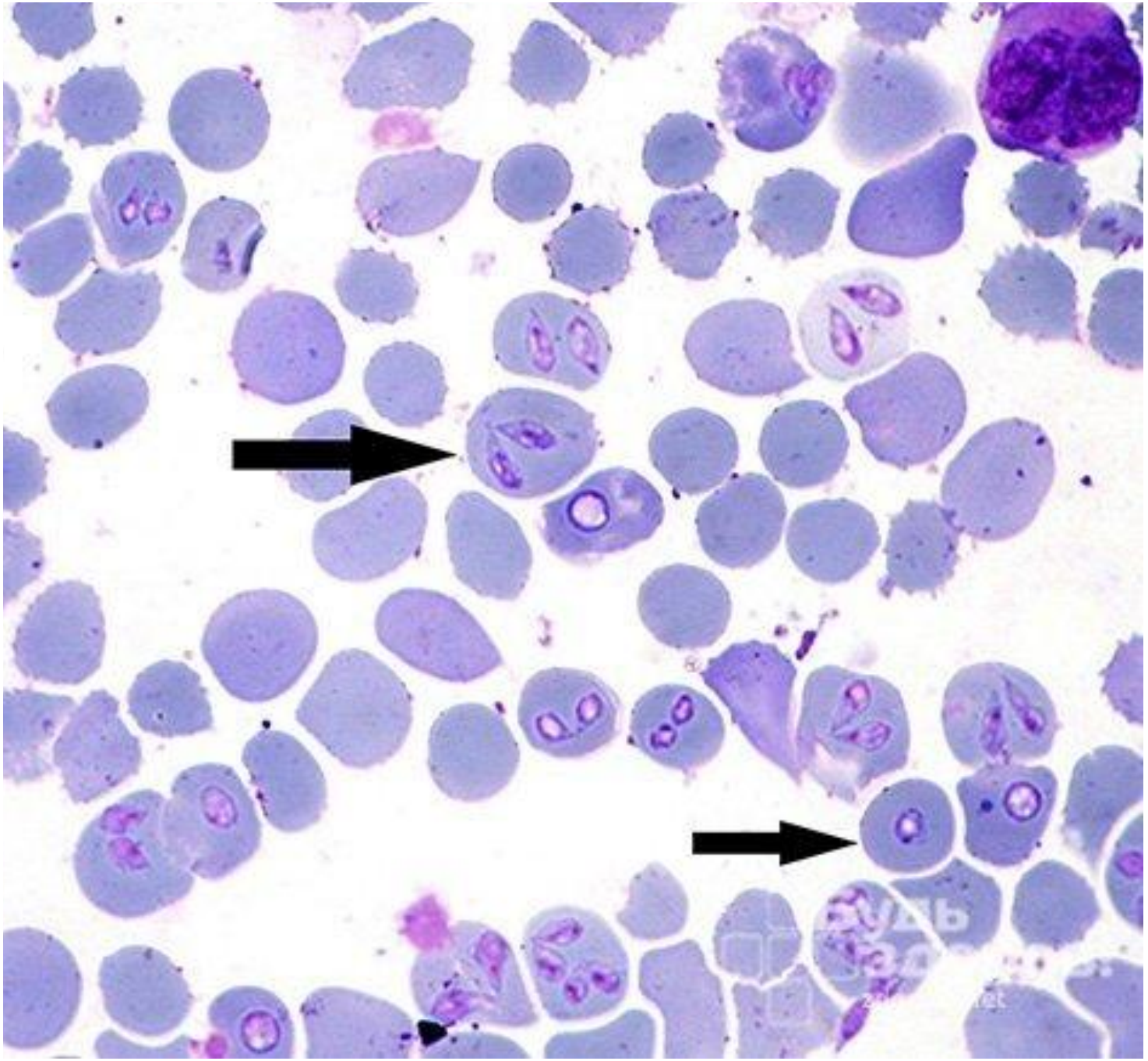
Toxoplasmosis.
Toxoplasma gondii



(a) Merozoite



(b) Oocyst



Transmission:

The **definitive host** is the domestic cat and other felines.
Humans and other mammals are **intermediate hosts**.

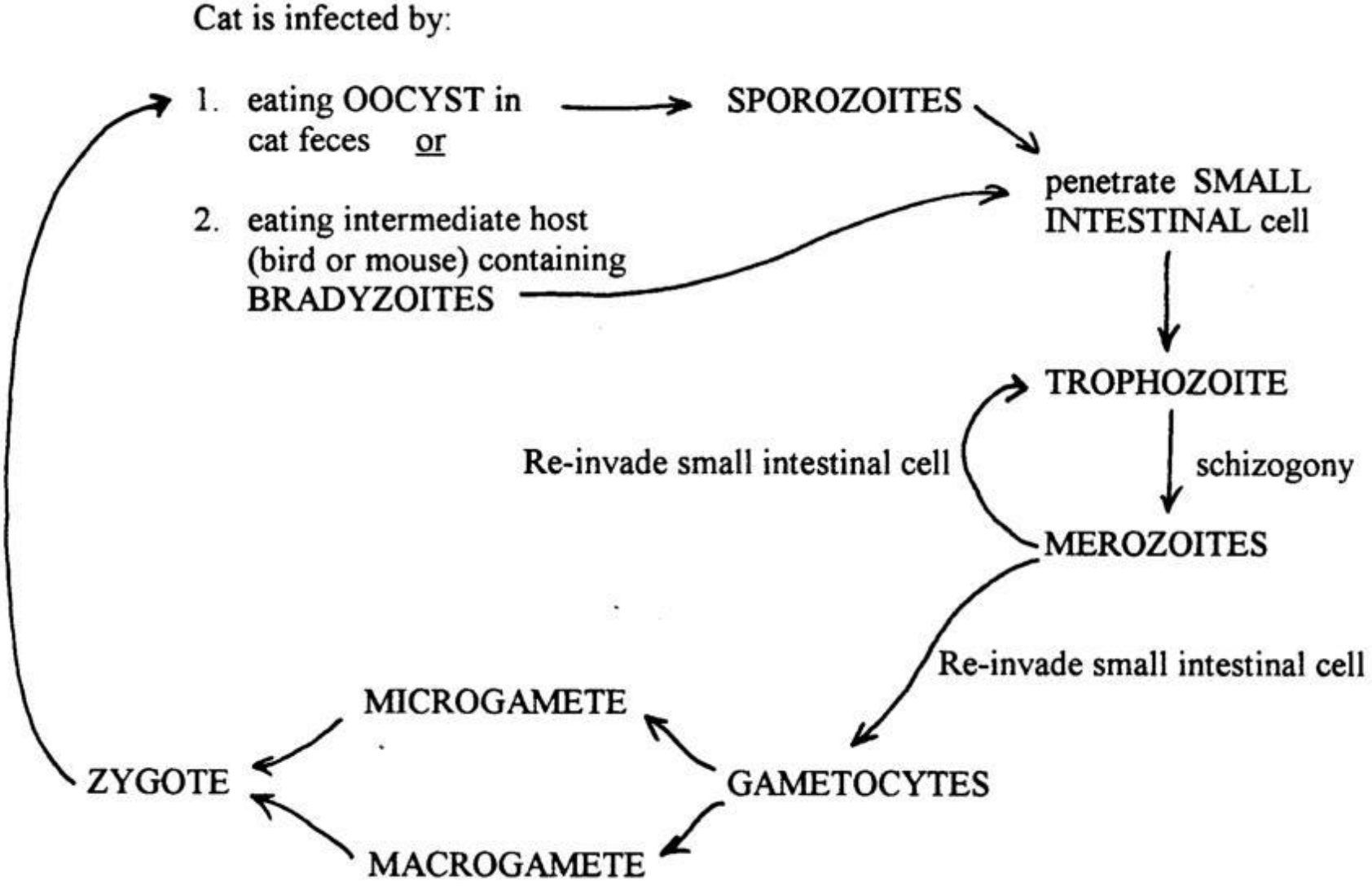
Toxoplasma gondii is usually acquired by ingestion and transplacental transmission from an infected mother to the fetus can occur.
Human–to–human transmission, other than transplacental transmission, does not occur.

Localisation:

After infection of the intestinal epithelium, the organisms spread to other organs, especially the brain, lungs, liver, and eyes.
Most primary infections in immunocompetent adults are asymptomatic.

Life Cycle of *Toxoplasma gondii*

Cycle in the CAT definitive host:



Symptoms:

Infection has 3 stages.

- **Acute toxoplasmosis**. It is often asymptomatic. However, symptoms may manifest and are often influenza-like: swollen lymph nodes, headaches, fever, fatigue or muscle aches and pains that last for a months or more. People with weakened immune system are likely to experience headache, confusion, poor coordination, seizures, lung problems that may resemble tuberculosis or pneumonia. Acute encephalopathy, chorioretinitis, lymphadenopathy, myocarditis, hepatosplenomegaly. It is harmful for pregnant woman and cause fetus death.
- **Latent toxoplasmosis**. This stage associated with numerous disease burdens, neural alterations, and subtle gender-dependent behavioral changes in immunocompetent humans.
- **Cutaneous toxoplasmosis.** Roseola and erythema multiforme eruptions, prurigo-like nodules, urticarial, maculopapular lesions. Newborns may have punctate macules, ecchymoses, or "blueberry muffin" lesions.

Diagnosis:

Biological, serological, histological or molecular methods.

May be detected in blood, amniotic fluid or cerebrospinal fluid by PCR. Used tests to measure IgG antibody and the modified direct agglutination test. In contrast to IgG IgM antibodies can be used to detect acute form.

Treatment:

Acute form – clindamycin, spiramycin, latent form – clindamycin, atovaguone, spiramycin, pyrimethamine, sufradiazine.

Prevention:

- Personal hygiene
- Be careful with cats, control cat's health
- Control populations of rodents (may be used for transmission)

Malaria.
Plasmodium species

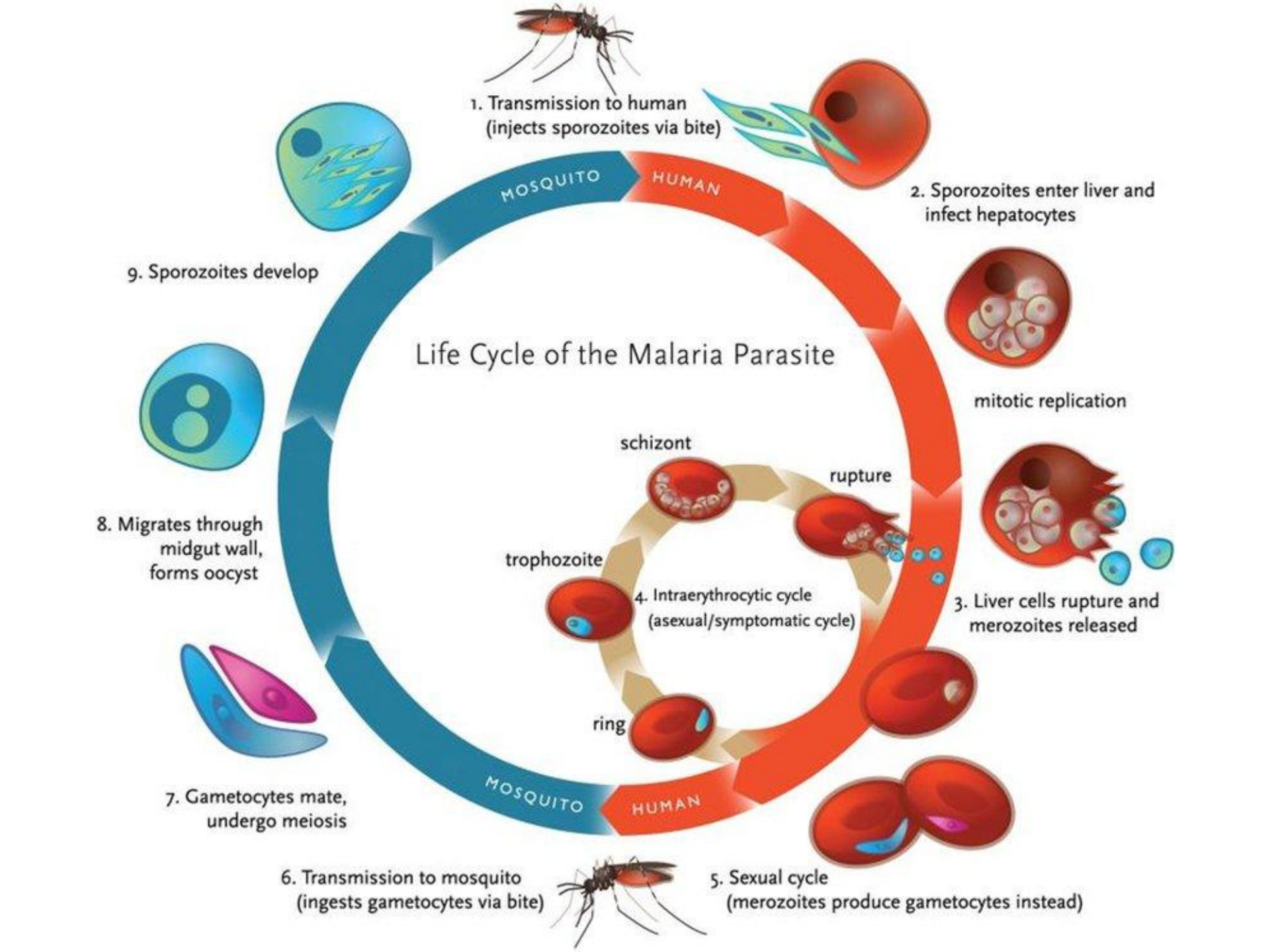
Is a mosquito-borne infectious disease of humans and other animals caused by parasitic protozoans belonging to the **Plasmodium species**. There are four species normally infecting humans, namely, ***Plasmodium falciparum, Plasmodium vivax, Plasmodium ovale, and Plasmodium malariae.***

Definitive host: mosquito *Anopheles*

Intermediate host: human

Distribution:

Tropical and subtropical regions around the equator. Sub-Saharan Africa, Asia, Latin America.


1. Transmission to human
(injects sporozoites via bite)
2. Sporozoites enter liver and
infect hepatocytes
mitotic replication
3. Liver cells rupture and
merozoites released
4. Intraerythrocytic cycle
(asexual/symptomatic cycle)
schizont
rupture
trophozoite
ring
5. Sexual cycle
(merozoites produce gametocytes instead)
6. Transmission to mosquito
(ingests gametocytes via bite)
7. Gametocytes mate,
undergo meiosis
8. Migrates through
midgut wall,
forms oocyst
9. Sporozoites develop
MOSQUITO
HUMAN
MOSQUITO
HUMAN
Life Cycle of the Malaria Parasite

Symptoms:

Fever, fatigue, vomiting, headaches, anemia, hemoglobin in the urine. In severe cases it can cause yellow skin, seizures, coma, death.

Symptoms usually begin 10-15 days after being bitten. If not properly treated, people have this disease some months or years.

The disease is most commonly transmitted by an infected female Anopheles mosquito. The mosquito bite introduces the parasites from mosquito's saliva into a person's blood. The parasites travel to the liver where they mature and reproduce.

Diagnosis:

- Microscopic examination of blood
- Antigen-based rapid diagnostic tests (serology)
- Detection DNA of parasites in blood samples (PCR)
- Microscopy of the urine (hemoglobin in the urine)

Treatment:

Because chloroquine – resistant stains of *P.falciparum* are present in many parts of the world, infection of *P.falciparum* may be treated with other agents including mefloquine, quinine, guanidine, pyrimethamine – sulfadoxine, and doxycycline. If the laboratory reports a mixed infection involving *P.falciparum* and *P.vivax*, the treatment must eradicate not only *P.falciparum* from the erythrocytes but also the liver stages of *P.vivax* to avoid relapses provided that the person no longer lives in a malaria endemic area.

Prevention:

Mosquito control measures:

- Using mosquito nets
- Insecticides, insect repellents

Home work. Task 4. *Sporozoa*

Name of disease	Toxoplasmosis	Malaria (Plasmodium sp.)
Latin name of parasite	*Toxoplasma gondii*	*Plasmodium falciparum* *Plasmodium ovale* *Plasmodium vivax* *Plasmodium malaria*
Morphology		
Definitive host		
Intermediate host		
Infective form		
Transmission		
Symptoms		
Diagnosis		
Treatment		
Prevention		

Table about *Amoebae, Ciliates* and *Flagellates*

	Amoebae	Ciliates	Intestinal flagellate	Genital flagellate	Hemoflagellates		
Name of disease	Amoebiasis	Balantidiasis	Giardiasis	Trichomoniasis	Leishmaniasis	Trypanosomiasis	
Latin name of parasite	*Entamoeba histolitica*	*Balantidium coli*	*Giardia lamblia*	*Tricomonis vaginalis*	*Leishmania donovani*	*Trypanosoma brucei*	*Trypanosoma cruzi*
Morphology (Forms of parasite)							
Definitive host							
Intermediate host							
Infective form							
Transmission							
Symptoms							
Diagnosis							
Treatment							
Prevention							