

Phylum: Ciliphora *Balantidium coli*

Disease: Balantidiasis or Balantidial dysentery:

- *Balantidium coli* : is the only ciliate known to parasitize humans. Ciliates represent a phylum of protozoa characterized by simple or compound ciliary organelles on the surface of their membranes that are used for locomotion.
- Reproduce by transverse binary fission or by conjugation.
- *Balantidium coli* : has 2 contractile vacuoles. Although contractile vacuoles are common to ciliates, they are rare in parasitic protozoa, which suggests that *Balantidium coli* has a unique osmoregulatory capacity.
- *Balantidium coli* : has 2 developmental stages: a trophozoite stage and cyst stage.

Trophozoite stage:

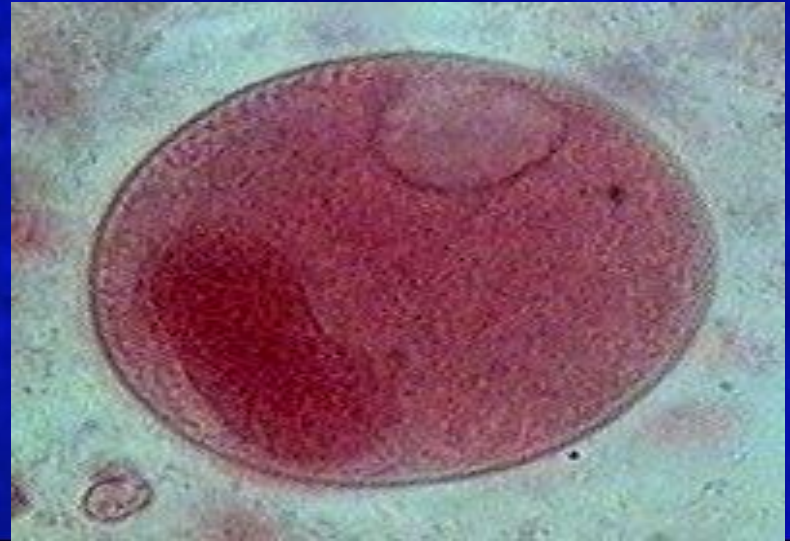
It has 2 nuclei (one macronucleus and other micronucleus).

Cysts:

Are smaller than trophozoites, round and have a tough, heavy cyst wall made of one or two layers.

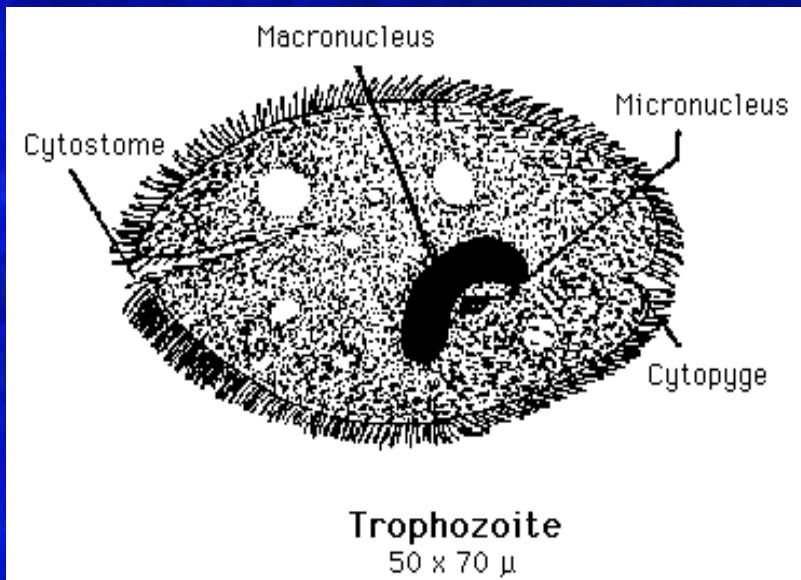
Usually only the macronucleus and sometimes cilia and contractile vacuoles are visible in the cyst.

Living trophozoites and cysts are yellowish or greenish in color.

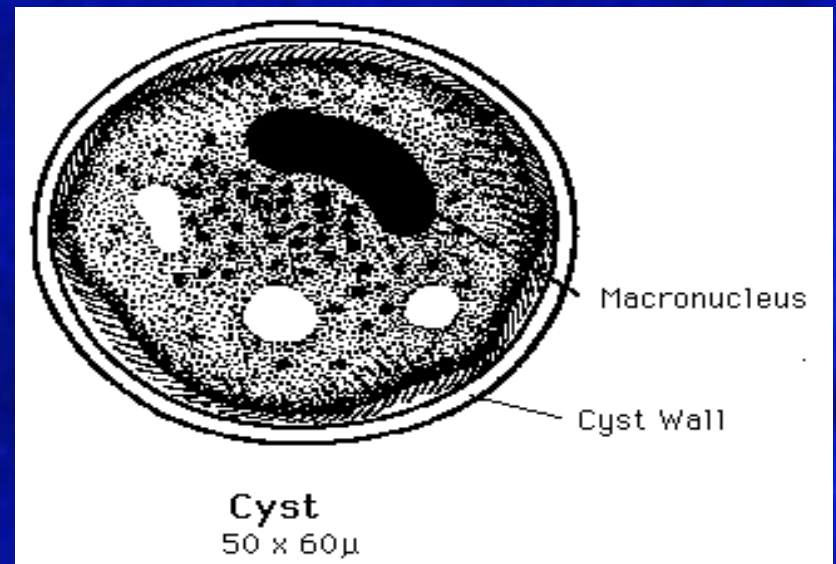


Balantidium coli

Trophozoite stage



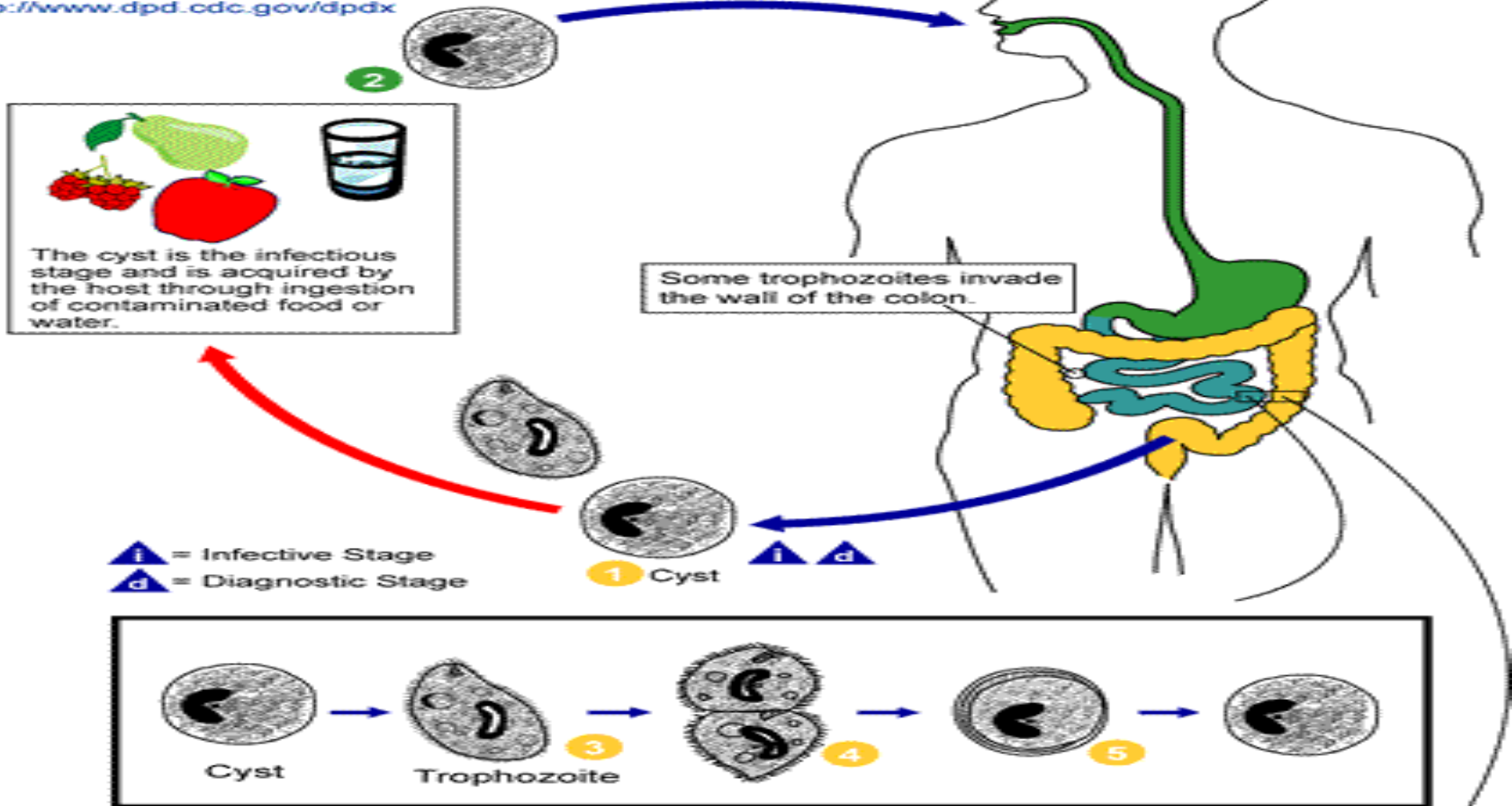
Cyst stage



Life cycle



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Symptoms

Disease

- Symptoms and Pathogenesis of balantidiasis are similar to those seen in amoebiasis including intestinal epithelial erosion, bloody diarrhea, nausea, vomiting and anorexia.
- The bloody diarrhea may persist for long periods of time resulting in acute fluid loss.
- *B. coli* also has ability to penetrate the mucosa resulting in ulceration.
- Extra-intestinal disease has also been reported but rarely.
- **Treatment:** Tetracycline 500 mg 4 times /day for 10 days Or Metronidazole 750 mg 3 times/day.

2-Subphylum: Mastigophora (Flagellates)

- ❑ Mastigophores are the most primitive type of protozoans.
- ❑ They often have many flagella which they are delicate thread-like extension of cytoplasm which arise from blepharoplast, and some are able to form pseudopodia for locomotion.
- ❑ Mastigophora usually reproduce asexually through mitosis, although some varieties can reproduce sexually.
- ❑ They are usually parasitic; that is, they live inside another organism (the host organism) to obtain nutrients and in effect harm the host organism.
- ❑ Some mastigophores are free-living.

Luminal & intestinal flagellates

Giardia lamblia (intestinal flagellate).

Disease: **Giardiasis (lambliasis)**.

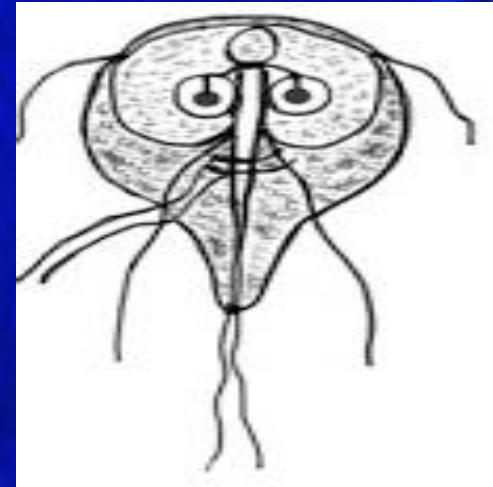
Epidemiology: It has **worldwide** distribution.

- * It is the most common protozoan intestinal disease in world.
- * This parasite more prevalence in children under 10 years old.

Morphology (There are two stages)

Trophozoite:

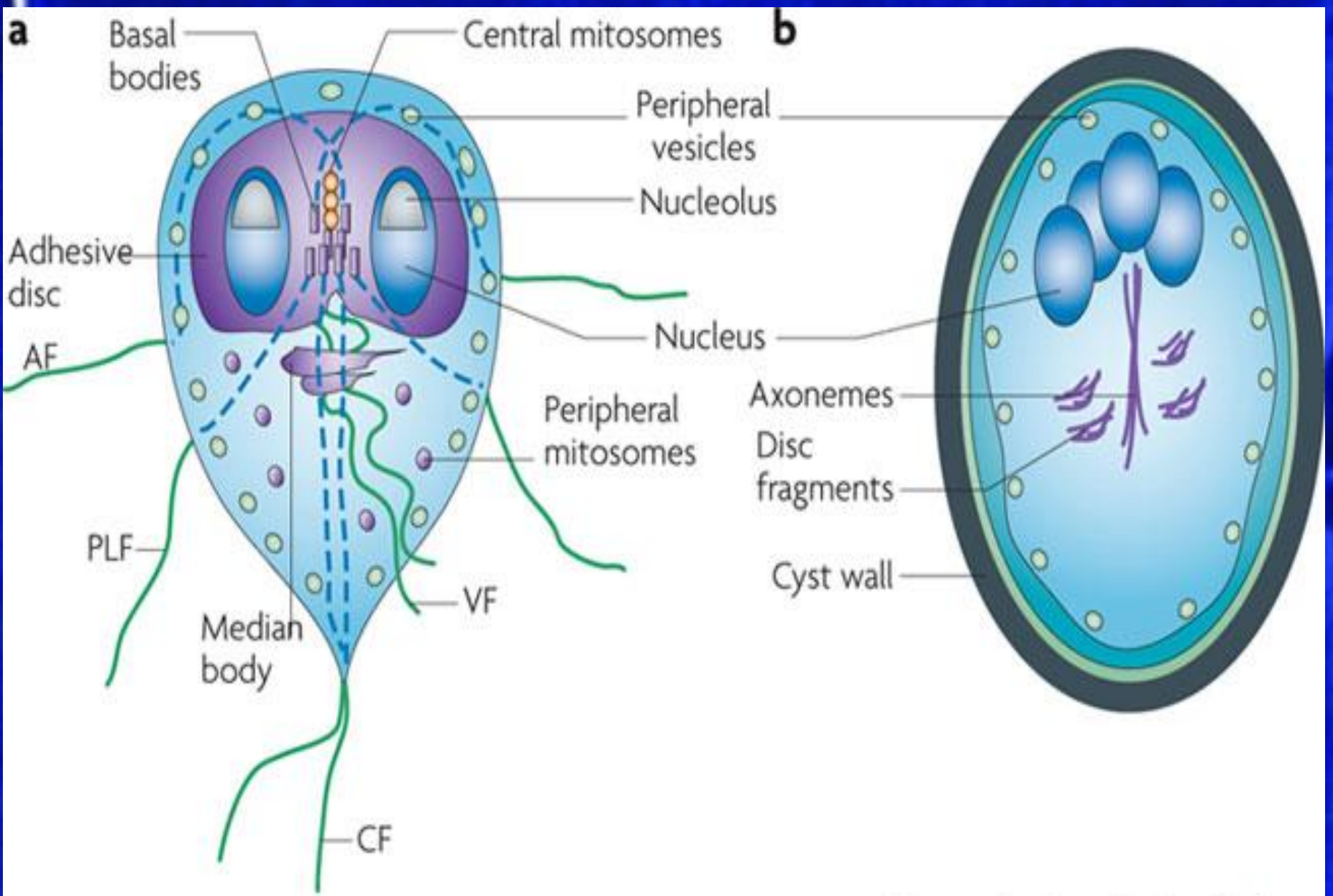
- It is 12-15 μ pear shaped with 8 flagella and, 2 axostyles arranged in a bilateral symmetry.
- There are two anteriorly located large sucking discs.
- It has Mitosome which are small mitochondrial organelle of Giardia an essential for ATP-generating.
- The cytoplasm contains two 2 nuclei with karyosome and 2 Para basal bodies.



Cyst:

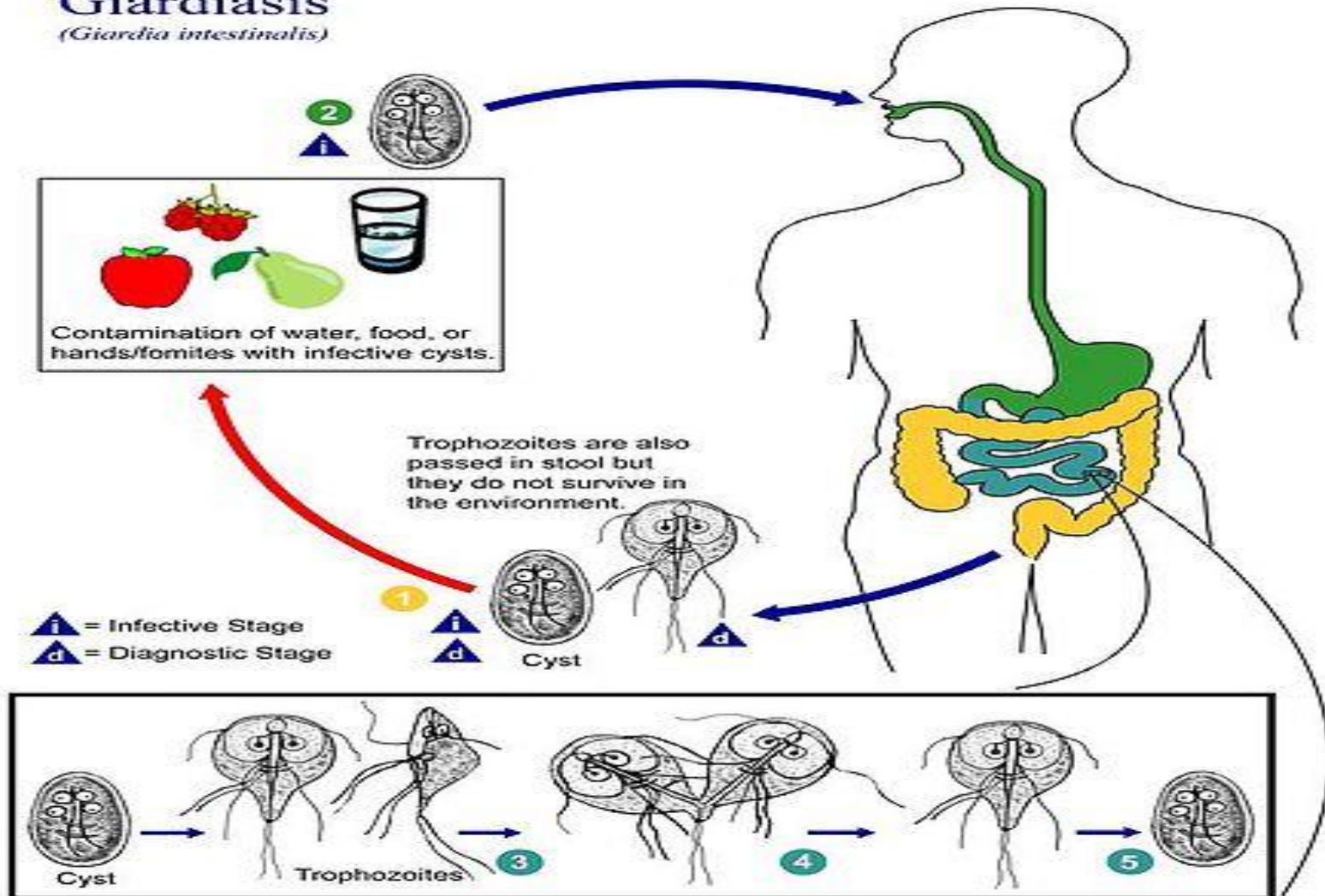
- *Giardia* cysts are 9-12 μ .
- Ovoid in shape with well-defined cyst wall.
- The cytoplasm contains **4 nuclei** and many structures of the trophozoite like **2 axostyles** and **parabasal bodies**.





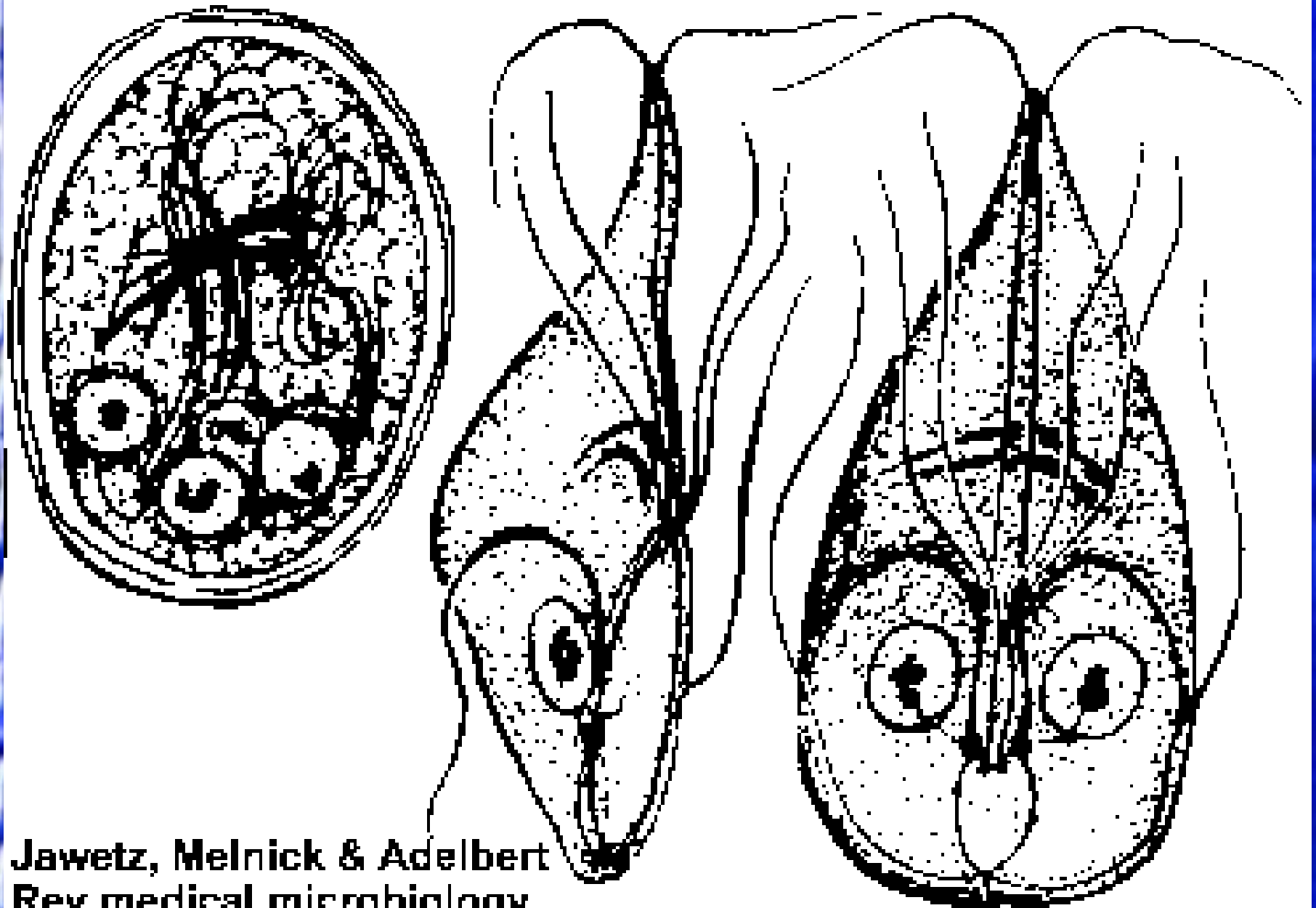
Life cycle of *Giardia lamblia*

Giardiasis (*Giardia intestinalis*)



- **Habitat:** duodenum and upper jejunum.
- **Infective stage:** Mature quadrinucleate cyst.
- **Pathogenic stage:** Active trophozoite.
- * **Diagnostic stage:** Mature cyst and trophozoite in stool sample.
- * **Mode of infection:** Contamination of food and water with quadri-nucleated oval shaped cyst .
- * **Route of entry:** is mouth or orally.





Jawetz, Melnick & Adelbert
Rev medical microbiology

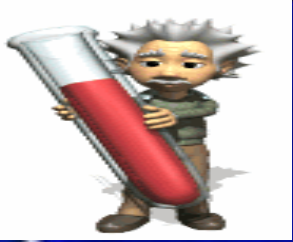
Life cycle of *G. Lamblia*

- **Life cycle:** Infection occurs by ingestion of cysts, usually in contaminated water.
- **Ex-cystation** occurs in duodenum and trophozoites colonize the **duodenum** where they may swim freely or attach to the **sub-mucosal epithelium** via the ventral **sucking disc**.
- The free trophozoites **encysted** in the **colon**.
- The cysts are passed in the stool.
- Man is the primary host and some animals (e.g. pigs and monkeys, mice.....etc) are also infected and serve as reservoirs. Afterward, person-to-person transmission is possible.

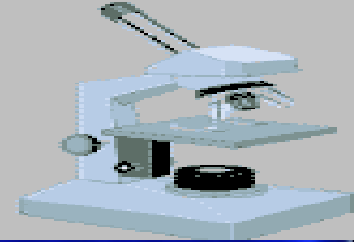
Pathology and Symptoms

- Signs of giardiasis do not begin for at least seven days following infection.
- The severity of the symptoms may vary greatly from mild or no symptoms to severe symptoms.
- The trophozoite not invade the intestine, it feed on its secretion.
- The most common manifestations of giardiasis are diarrhea and abdominal pain.
- Steatorrhea is excessive excretion of fecal fat, stool may have bad smelling when the Giardia interferes with the absorption of fat from the intestine (malabsorption), sometimes gallbladder and bile duct may be infected.

- The malabsorption may cause loss of weight.
- The more chronic stage is associated with vitamins A and B12 Malabsorption.
- Some patients recover from their giardiasis, with or without treatment, but symptoms continue, perhaps because of a condition referred to as post infectious irritable bowel syndrome(IBS).
- The cause of the continuing symptoms is due to bacterial overgrowth of the small intestine



Diagnosis



Identification of cysts in stool sample.

- More than one sample is recommended (at least 3 stool samples with two days between each), since the presence of cysts in the stool can be highly irregular, and cysts may not be present until a week after symptoms appear.
- Trophozoites break up rapidly in the stool.
- **A serological test (ELISA)** may be used to detect Giardia antigens in the stool.
- **Enterotest**: a patient swallows a capsule with a string attached, and when it is passed into the small intestine, trophozoites stick to the string.
- **A duodenal biopsy** may also be useful to detect trophozoite presence

***GIARDIA* Treatment**

Treatment:

Keep the child hydrated. If it is dehydrated:

Using drugs

1-Metronidazole (Flagyle).

2-Tinidazole a new *Giardia* alternative drugs

(but it has a shorter treatment course) used
for **under 3 years children.**

Immune response

- It has been confirmed the presence of cellular and humoral immune response against Giardia infection.
- IgM and IgG Specific antibodies against Giardia.
- IgM is short lives and IgG antibody remains high in titer for months.
- IgA and IgG antibodies very important in the breast milk.
- IgA antibody inhibit Giardia trophozoite adherence on intestine.
- IgA antibody help in clearance of Giardia trophozoite from intestine.

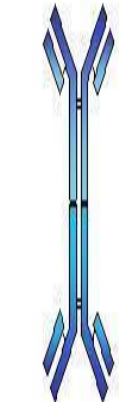
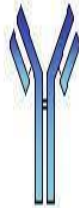
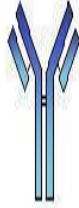
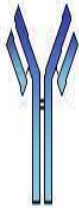
IgM

IgA

IgG

IgE

IgD



Immune cell binds antigen



Activated immune cell makes more antibodies



Antibodies bind antigen



Macrophage eats antigen



Macrophage destroys antigen

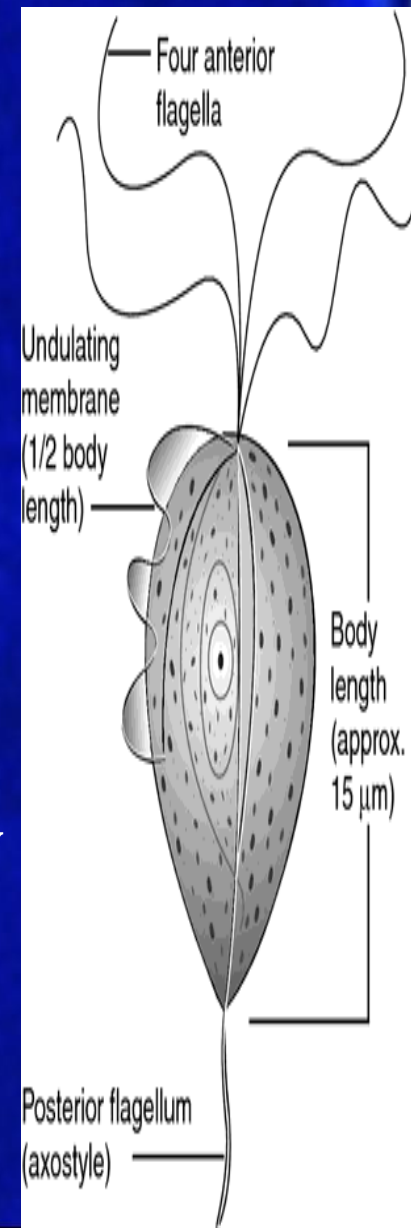
Some immune cells are activated to produce antibodies (such as IgE) against the food toxin.

Luminal or atrial flagellates

Parasites of openings and body cavities such as the gut, mouth and urogenital organisms
UGT.

Genus : *Trichomonas*. General characters.

- All *Trichomonas*; they have 4 free anterior flagellates.
- The fifth one attach to the surface of the body forming undulating membrane.
- Axostyle extended along the length of body, and then emerge from posterior.
- Direct life cycle & reproduce by longitudinal binary division.
- All Spps have no cyst stage.
- All Spps the trophozoite is the infective, diagnostic and pathogenic stage.



Trichomonas: There are three species of genus *Trichomonas*

1-Trichomonas tenax

- an organism found in the oral cavity of human.
- Living in the mouth and in the tartar around the teeth and the pockets.
- Maybe found in tonsillar follicles causing tonsillitis.
- It is 5-10 μm in length. Undulating membrane to two third of the body
- Moving vigorously by flagellum through saliva, feeding on food debris.
- The tissue destruction has been done by toxins produced by this and other pathogens at the site of infection.
- it remains absent from commensal gingival biofilm said appearing healthy.
- Its presence in necrotizing ulcerative gingivitis.

2-Trichomonas hominis.

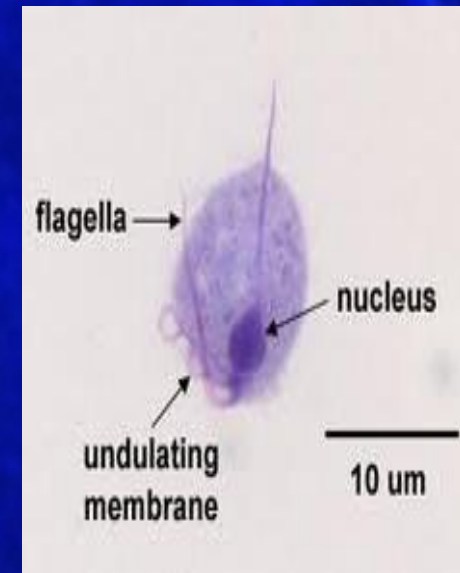
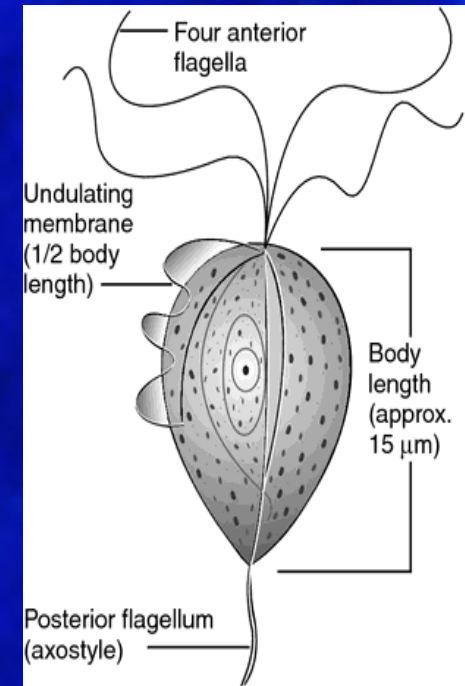
- Lives in the intestine.
- It is cosmopolitan in distribution.
- It is thought to be **non-pathogenic** although it has been associated with **diarrheic stools**.
- If ingested in a protected media like milk can be pass the stomach and small intestine.

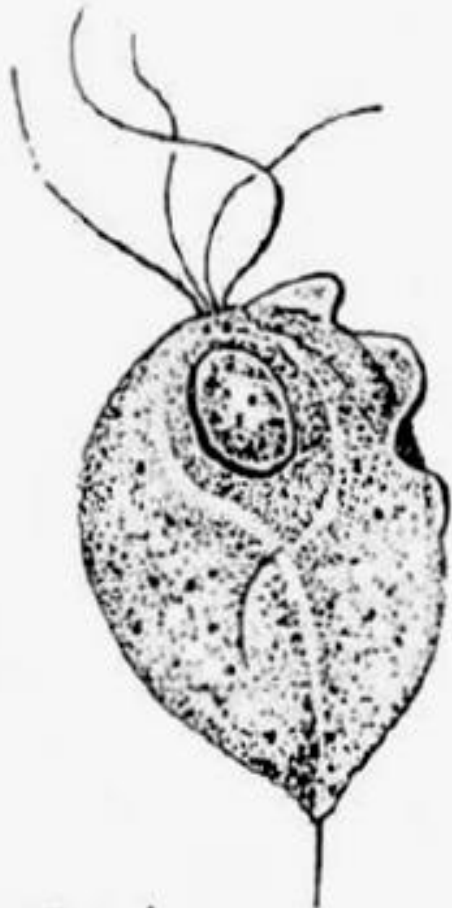
3-Trichomonas vaginalis

- ✿ **Trichomoniasis disease vaginitis in female and proctitis in male**
- ✿ **It is sexually transmitted disease**
- ✿ **Epidemiology: It has a world-wide.**
- ✿ **Trichomonas vaginalis associated with low birth weight and preterm delivery.**
- ✿ **Mode of infection is sexually.**
- ✿ **Habitat: Male ♂ and Female ♀ urogenital systems**

Morphology: (Trophozoite)
pear shaped with a single nucleus and 4 anterior flagella and a lateral flagellum attached by an undulating membrane, which extended to the 1/2 of the body

2 axostyles arranged asymmetrically.
The organism does not encysted

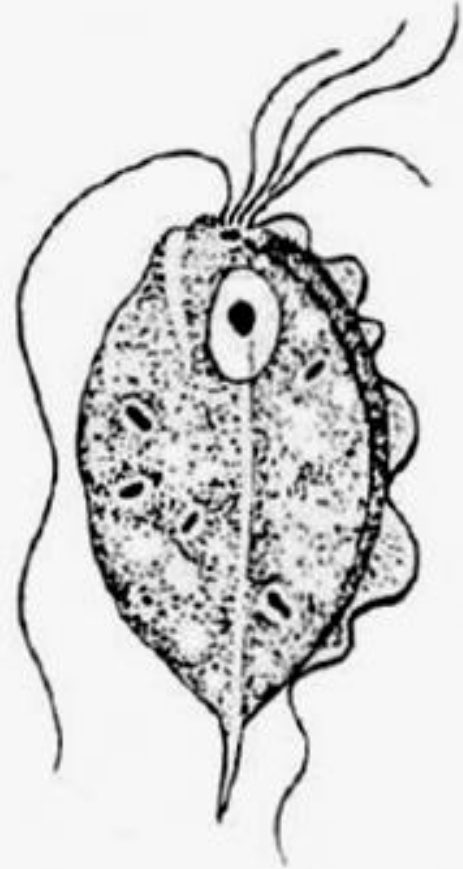




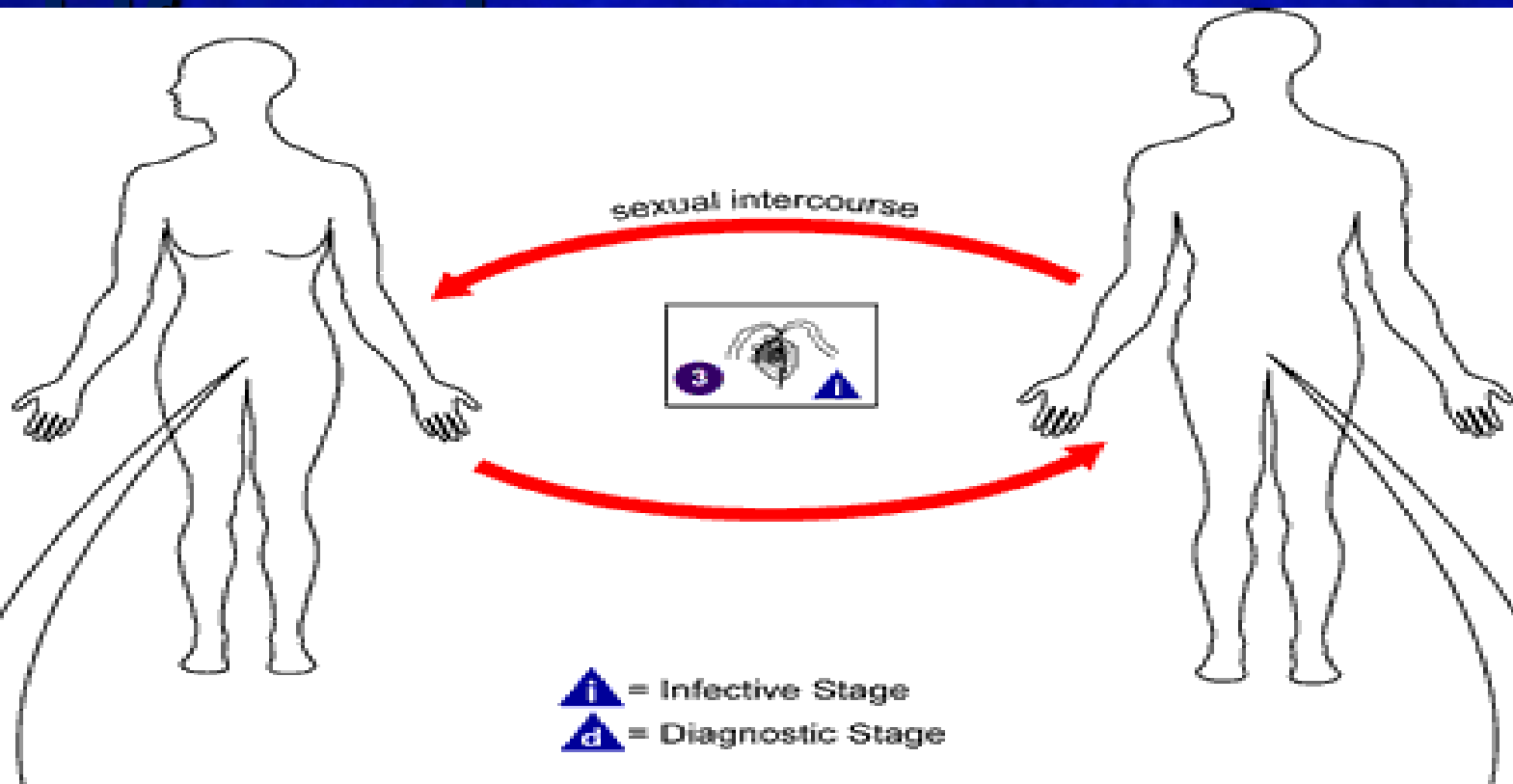
Trichomonas vaginalis



Trichomonas tenax



Trichomonas hominis



Trichomonas vaginalis



LIFE CYCLE OF *T. VAGINALIS*

- Trichomonas vaginalis resides in the female lower genital tract and the male urethra and prostate.
- Where it replicates by longitudinal binary fission.
- The parasite does not appear to have a cyst form, and does not survive well in the external environment.
- Trichomonas vaginalis is transmitted among humans, its only known host, primarily by sexual intercourse.

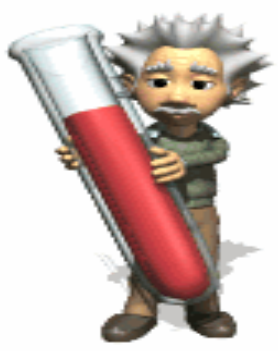
Trichomonas vaginalis

Pathogenesis:

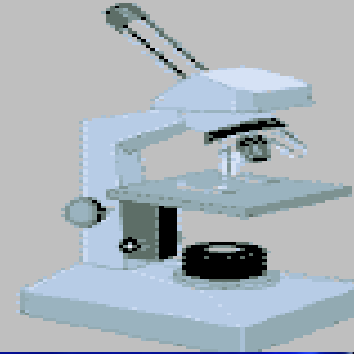
- ❖ The main mechanism of parasite pathogenicity is cell to cell adherence & hemolysis and secreting soluble proteinases.
- ❖ Trophozoite infect the squamous epith. Cells.
- ❖ It is asymptomatic particularly in male.
- ❖ In females causing severe irritation & Inflammation of the vaginal mucosa (vaginitis) or inflammation uterine cervix (cervicitis).
- ❖ In males causing inflammation of urethra and prostate (urithritis and prostatitis).

Clinical symptoms :

- ❖ majority of infected males without symptoms and $\frac{1}{3}$ of infected females without symptoms.
- ❖ both in male and female suffering from genital discharge and pain in urination.



Diagnosis and treatment



■ Diagnosis

- By GUE or males and females UG swab and smear stain with Giemsa stain.

■ Treatment

- 1-Metronidazole(Flagyle) tablets for men and vaginal suppositories for females.
- 2-Tinidazole alternative drug. it has a shorter treatment course.

BLOOD AND TISSUES FLAGELLATES (Haemoflagellates)

- The major clinical significance include members of 2 genera
- 1- Genus: *Leishmania* (*L. donovani*, *L. tropica* and *L. major*.)
- 2- Genus: *Trypanosoma* (*T. brucei* and *T. cruzi*);

- Several species of *Leishmania* are pathogenic for man:
- *L. donovani* causes visceral leishmaniasis (**Kala-azar, black disease, dum dum fever, black fever**)
- *L. tropica* (*L. t. major*, *L. t. minor* cause cutaneous leishmaniasis (**oriental sore, Delhi ulcer, Aleppo boile, Delhi or Baghdad boil**).
- **Epidemiology:** Leishmaniasis is prevalent worldwide, ranging from south east Asia, Mediterranean, north and central Africa, and south and central America.

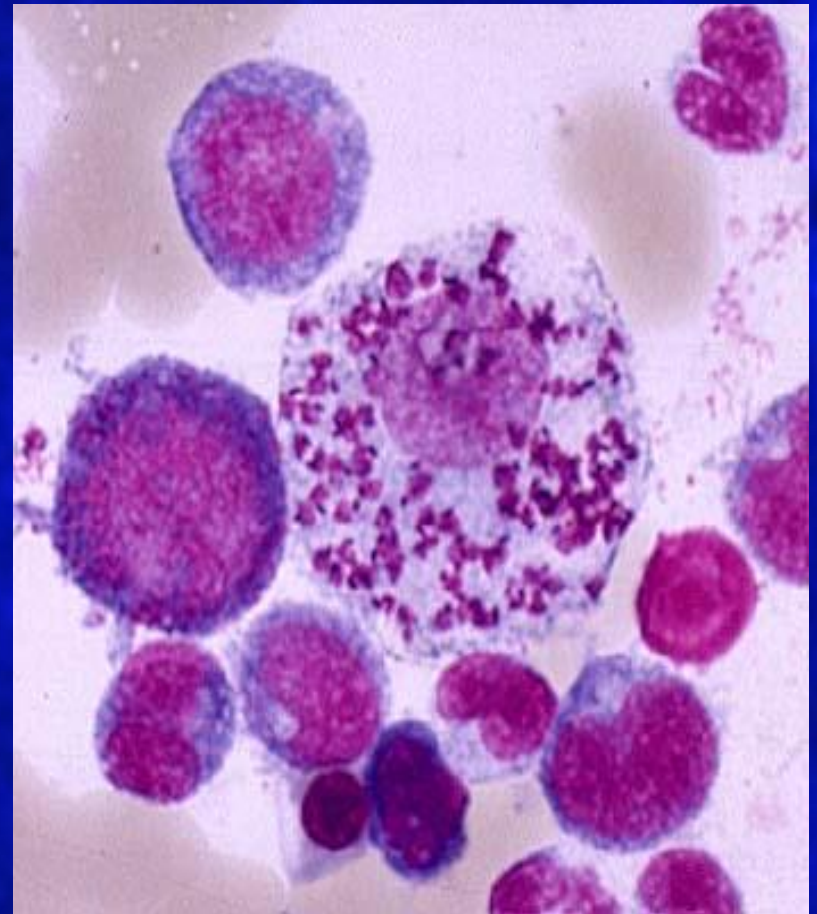
- Most *Leishmania* vector are females sandflies of the genus *Phlebotomus*
- Their primary hosts are vertebrates and Human.
- All species habitat is **obligatory intracellular** (Mostly macrophages).
- **reservoir hosts:** fox, jackal, rodents and wolves .
- All human *Leishmania* are **zoonotic** pathogenic protozoa.
- All human species have **indirect life cycle**.
- **Mode of infection:** by vector sand fly **bit of skin**.
- **Route of infection**→ exposed skin places.
- All human *Leishmania* species are seriously and medically **pathogenic**.

1-Amastigote:

is a stage that does not have a visible external flagella. The term. It is the form the parasite lives in the human macrophages skin.



Amastigote



2-Promastigote.

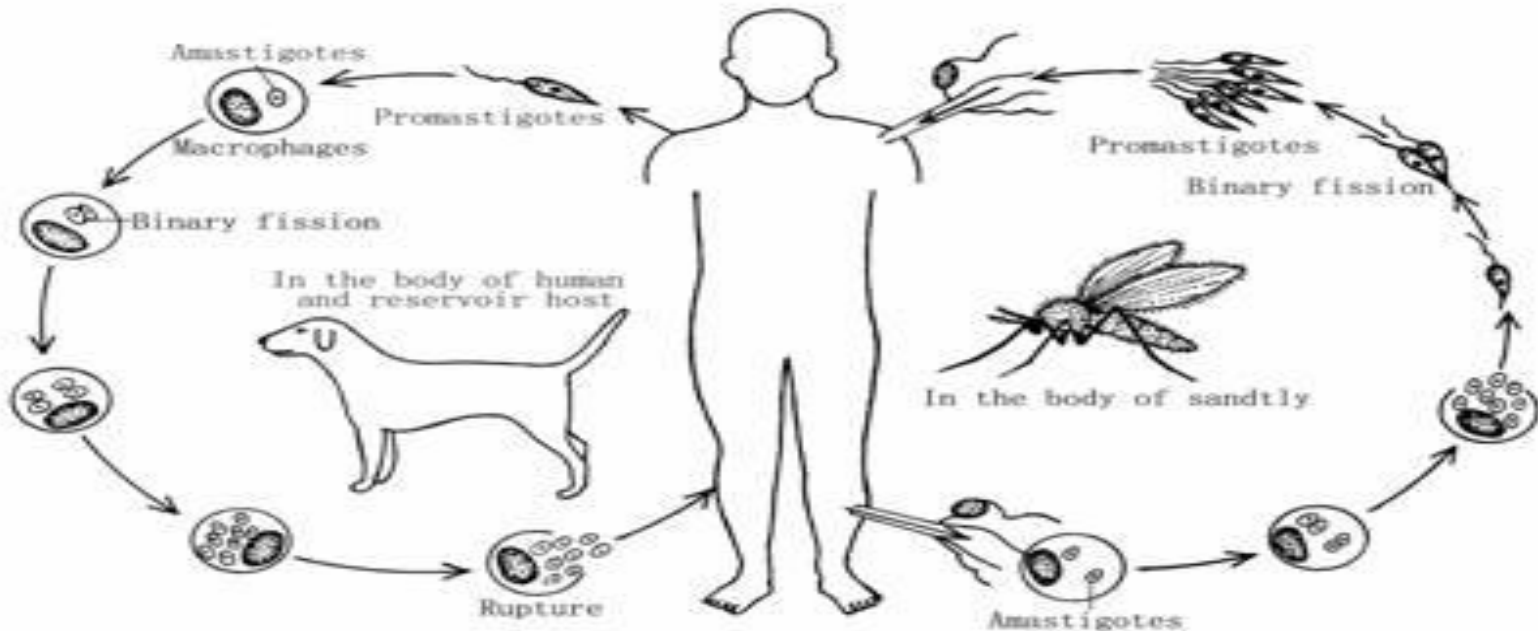
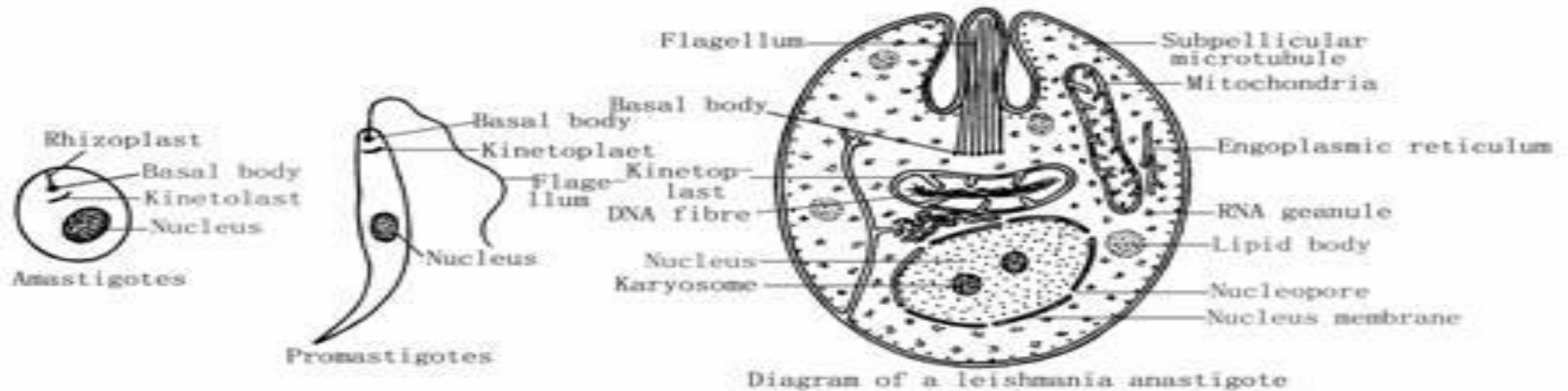
is a stage that does have a visible external flagella. it is the form the parasite lives in the vector sand fly gut.



Promastigote



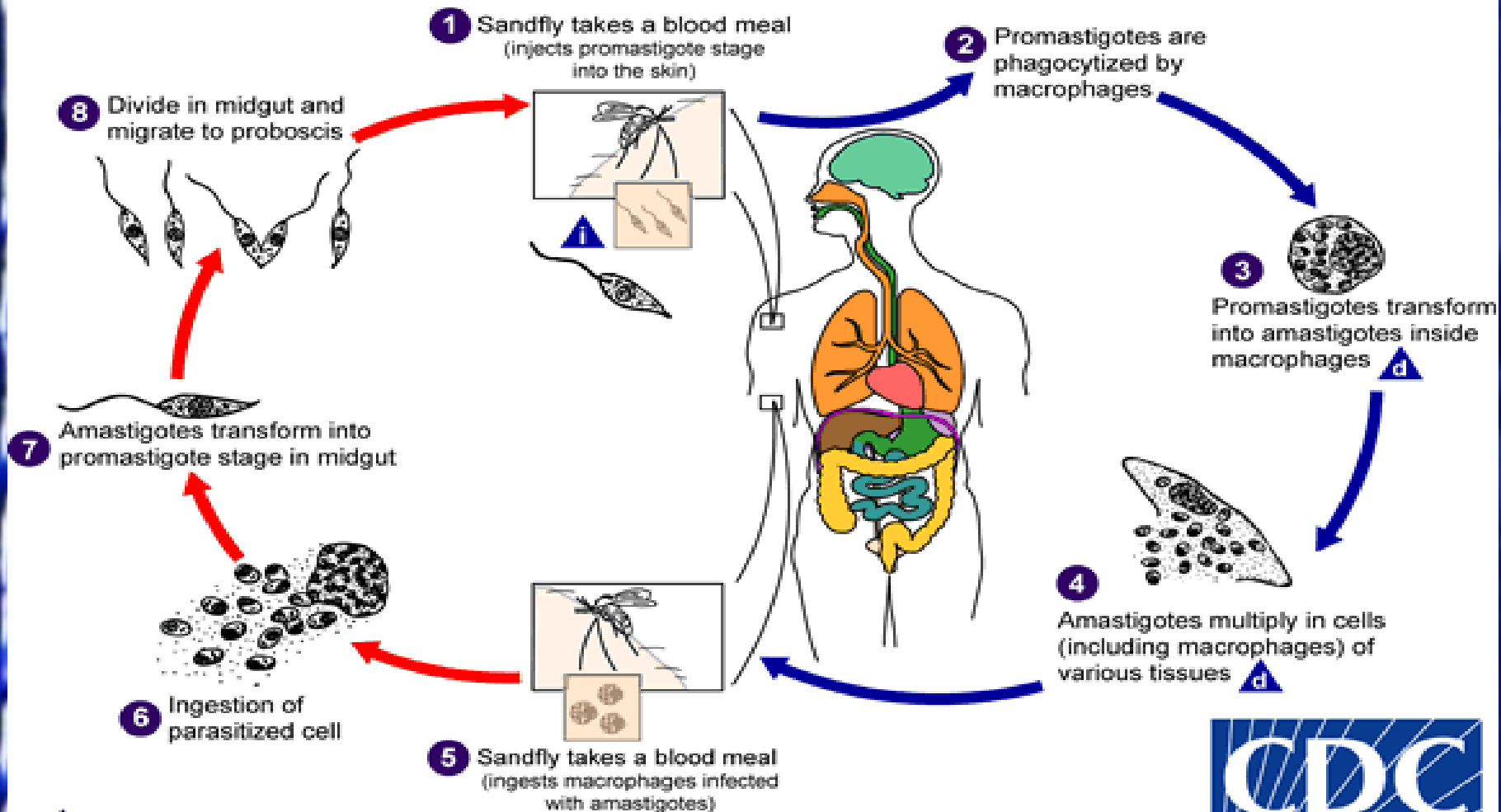
Morphology



Life cycle

Sandfly Stages

Human Stages



i = Infective Stage

d = Diagnostic Stage



Symptoms

- Visceral leishmaniasis caused by *leishmania donovani* (kala-azar, dumdum fever):
- They are localized and multiply in the mononuclear phagocytic cells of spleen, liver, lymph nodes, bone marrow, intestinal mucosa and other organs.
- Cutaneous leishmaniasis (*L. tropica*) cause Oriental sore, Delhi ulcer, Baghdad boil)
- They multiply locally, producing of a papule, 1-2 weeks (or as long as 1-2 months) after the bite, which gradually grows to form a relatively painless ulcer.
- The center of the ulcer encrust while satellite papules develop at the periphery.
- The ulcer heals in 2-10 months even if untreated but leaves a disfiguring scar.
- The disease may disseminate in the case of a depressed immune function.

Pathology & Diagnosis

- **Pathology:** Pathogenesis of leishmaniasis is due to **immune reaction to the organism**, particularly the **cell mediated immunity**.
- **Laboratory examination reveals a marked leukopenia with relative monocytosis and lymphocytosis, anemia and thrombocytopenia.**
- **IgM and IgG levels are extremely elevated due to both specific antibodies and polyclonal activation.**



Diagnosis:

- ❖ Diagnosis is based on the **history of exposure to sand fly**, symptoms and **isolation of the organisms from the lesion aspirate or biopsy**, by direct **examination or culture**.
- ❖ Skin test (**delayed hypersensitivity: Montenegro test**), **detection of anti-leishmanial antibodies** by immunofluorescence are indicative of exposure
- ❖ In **visceral leishmaniasis** a physical exam may show signs of an enlarged spleen, liver (**hepatosplenomegaly**), and **lymph nodes**.
- ❖ The patient may have been **bitten by sand flies**, or was in an area known for **leishmaniasis**

Treatment and Control:

- **Sodium stibogluconate (Pentostam)** is the drug of choice.
- **Pentamidine isethionate** is used as an alternative.
- Control measure involves the **vector control** and avoidance. **Immunization** has not been effective.

Types of macrophages in different tissues.

<u>Name of cell</u>	<u>Location</u>
▪ <u>Dust cells/</u>	<u>Alveolar macrophages lungs</u>
▪ <u>Adipose tissue macrophages</u>	<u>Adipose tissue</u>
▪ <u>Histiocytes</u>	<u>Connective tissue</u>
▪ <u>Kupffer cells</u>	<u>Liver</u>
▪ <u>Microglia</u>	<u>Neural tissue</u>
▪ <u>Epithelioid cells</u>	<u>Granulomas</u>
▪ <u>Osteoclasts</u>	<u>Bone</u>
▪ <u>Hofbauer cell</u>	<u>Placenta</u>
▪ <u>Sinusoidal lining cells</u>	<u>Spleen</u>
▪ <u>Giant cells</u>	<u>Connective tissue</u>
▪ <u>Peritoneal macrophages</u>	<u>Peritoneal cavity</u>

**ALL THESE CELLS ARE CALLED
MACROPHAGES WHICH ORIGINATED
FROM MONOCYTE AND THEIR FUNCTIONS
ARE**

- 1- DEFENCE MECHANISM.**
- 2- PHAGOCYTES OR ENGULF AND DIGEST**
- 3- ELIMINATE FOREIGN BODIES AND PATHOGENS AND
CELLULAR DEBRIS.**

Macrophage populations

Less-flexible programming—determined during ontogeny

Specific transcription factors and epigenetic modifications direct lineage commitment

