Medical parasitology

Medical parasitology deals with the parasites, which cause human infections and the diseases they produce. It is broadly divided into 2 parts: Protozoology and Helminthology.

Parasites: Parasites are living organisms, which depend on a living host for their nourishment and survival. They multiply or undergo development in the host. The term 'parasite' is usually applied to Protozoa (unicellular organisms) and Helminths (multicellular organisms)

Parasites can also be classified as:

1- Ectoparasite: Ectoparasites inhabit only the body surface of the host without penetrating the tissue. Lice, ticks, and mites are examples of ectoparasites. The term infestation is often employed for patriotization with ectoparasites.

2- Endoparasite: A parasite, which lives within the body of the host and is said to cause an infection is called an endoparasite. Most of the protozoan and helminthic parasites causing human disease are endoparasites.

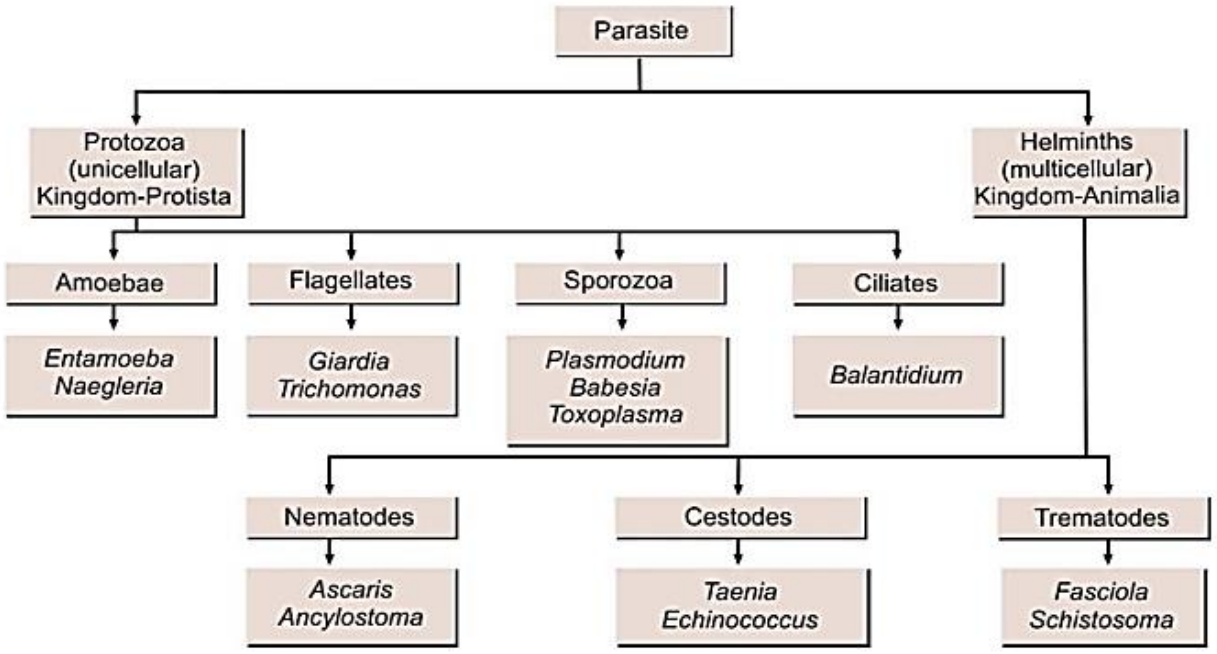
3-Free-living parasite: It refers to non-parasitic stages of active existence, which live independent of the host, e.g., cystic stage of *Naegleria fowleri*

4-Obligate parasite: The parasite, which cannot exist without a host, e.g., Toxoplasma gondii and Plasmodium.

5-Facultative parasite: Organism which may either live as parasitic form or as free-living form.

6-Accidental parasites: Parasites, which infect an unusual host, are known as accidental parasites. *Echinococcus granulosus* infects man accidentally, giving rise to hydatid cysts.

7- Aberrant parasites: Parasites, which infect a host where they cannot develop further, are known as aberrant or wandering parasites, e.g., *Toxocara canis* (dog roundworm) infecting humans.



Host is defined as an organism, which harbors the parasite and provides nourishment and shelter to latter and is relatively larger than the parasite. The host may be of the following types:

1- Definitive host: The host, in which the adult parasite lives and undergoes sexual reproduction is called the definitive host, e.g., mosquito acts as definitive host in malaria. *The definitive host may be a human or any other living being. However, in majority of human parasitic infections, man is the definitive host (e.g., filarial, roundworm, hookworm).

2- Intermediate host: The host, in which the larval stage of the parasite lives or asexual multiplication takes place, is called the intermediate host. In some parasites, 2 different intermediate hosts may be required to complete different larval stages. These are known as first and second intermediate hosts, respectively.

3- Paratenic host: A host, in which larval stage of the parasite remains viable without further development is referred as a paratenic host. Such host transmits the infection to another host.

4-Reservoir host: In an endemic area, a parasitic infection is continuously kept up by the presence of a host, which harbors the parasite and acts as an important source of infection to other susceptible hosts, e.g., dog is the reservoir host of hydatid disease.

5- Accidental host: The host, in which the parasite is not usually found, e.g., man is an accidental host for cystic echinococcosis.

NOTE…. Parasites with man as intermediate or secondary host *Plasmodium spp. Babesia spp. Toxoplasma gondii Echinococcus granulosus ,Echinococcus multilocularis ,Taenia solium Spirometra spp.*

Zoonosis: The word zoonosis was introduced by Rudolf Virchow in 1880 to include the diseases shared in nature by man and animals.

Defined zoonosis as: Those diseases and infections, which are naturally transmitted between vertebrate animals and man". It is of following types:

* **Protozoal zoonoses**, e.g., toxoplasmosis, leishmaniasis, balantidiasis, and cryptosporodiasis

***Helminthic zoonoses**, e.g., hydatid disease, taeniasis

* **Anthropozoonoses**: Infections transmitted to man from lower vertebrate animals, e.g., cystic echinococcosis

* **Zooanthroponoses:** Infections transmitted from man to lower vertebrate animals, e.g., human tuberculosis to cattle.

-Host-parasite Relationships

Host-parasite relationships are of following types:

*Symbiosis

*Commensalism

*Parasitism

-Direct life cycle:

When a parasite requires only single host to complete its development, it is called as direct life cycle, e.g. Entamoeba histolytica requires only a human host to complete its life cycle.

Indirect life cycle:

When a parasite requires 2 or more species of host to complete its development, the life cycle is called as indirect life cycle, e.g. malarial parasite requires both human host and mosquito to complete its life cycle.

Sources of Infection

Contaminated soil and water: *

Soil polluted with embryonated eggs (roundworm, whipworm) may be ingested or infected larvae in soil, may penetrate exposed skin (hookworm). Infective forms of parasites present in water may be ingested (cyst of amoeba and Giardia) Water containing the intermediate host may be swallowed (cyclops containing guinea worm larva Dracunculus Medinensis). Infected larvae in water may enter by penetrating exposed skin, (cercariae of schisotosomes) €Free-living parasites in water may directly enter through vulnerable sites (Naegleria may enter through nasopharynx).

Food: *

Ingestion of contaminated food or vegetables containing infective stage of parasite (amoebic cysts, Toxoplasma oocysts, Echinococcus eggs) Ingestion of raw or undercooked meat harboring infective larvae (measly pork containing cysticercus cellulosae, the larval stage of Taenia solium).

Insect vectors: *

A vector is an agent; usually an arthropod that transmits an infection from man to man or from other animals to man, e.g., female Anopheles is the vector of malarial parasite.

Mutualism - an association in which both partners are metabolically dependent upon each other and one cannot live without the help of the other; . One classic example is the relationship between certain species of flagellated protozoa living in the gut of termites. The protozoa, which depend entirely on a carbohydrate diet, acquire their nutrients from

termites. In return they are capable of synthesizing and secreting cellulases; the cellulose digesting enzymes, which are utilized by termites in their digestion.

Commensalism - an association in which the commensal takes the benefit without causing injury to the host. e.g. Most of the normal floras of the humans' body can be considered as commensals.

Parasitism - an association where one of the partners is harmed and the other lives at the expense of the other. e.g. Worms like Ascaris lumbricoides reside in the gastrointestinal tract of man, and feed on important items of intestinal food causing various illnesses

Laboratory diagnosis

depending on the nature of the parasitic infections, the following specimens are selected for laboratory diagnosis:

a) Blood – in those parasitic infections where the parasite itself in any stage of its development circulates in the blood stream, examination of blood film forms one of the main procedures for specific diagnosis. For example, in malaria the parasites are found inside the red blood cells. In Bancroftian and Malayan filariasis, microfilariae are found in the blood plasma.

b) Stool – examination of the stool forms an important part in the diagnosis of intestinal parasitic infections and also for those helminthic parasites that localize in the biliary tract and discharge their eggs into the intestine. In protozoan infections, either trophozoites or cystic forms may be detected ; the former during the active phase and the latter during the chronic phase. Example, Amoebiasis, Giardiasis, etc. In the case of helmithic infections, the adult worms, their eggs, or larvae are found in the stool.

c) Urine – when the parasite localizes in the urinary tract, examination of the urine will be of help in establishing the parasitological diagnosis. For example in urinary Schistosomiasis, eggs of Schistosoma haematobium are found in the urine.

d) Sputum – examination of the sputum is useful in the following:

• In cases where the habitat of the parasite is in the respiratory tract, as in Paragonimiasis, the eggs of Paragonimus westermani are found.

• In amoebic abscess of lung or in the case of amoebic liver abscess bursting into the lungs, the trophozoites of E. histolytica are detected in the sputum.

e) Biopsy material - varies with different parasitic infections. For example spleen punctures in cases of kala-azar, muscle biopsy in cases of Cysticercosis, Trichinelliasis, and Chagas' disease, Skin snip for Onchocerciasis.

f) Urethral or vaginal discharge – for Trichomonas vaginalis

Indirect evidences – changes indicative of intestinal parasitic infections are:

a. Cytological changes in the blood – eosiniphilia often gives an indication of tissue invasion by helminthes, a reduction in white blood cell count is an indication of kala-azar, and anemia is a feature of hookworm infestation and malaria.

b. Serological tests – are carried out only in laboratories where special

antigens are available.

• **Medical Protozoology** - Deals with the study of medically important protozoa.

PROTOZOA
Sarcodina (Amoebae):
(a) Genus, *Entameba:*
E.g. *Entameba histolytica*
(b) Genus Endolimax
E.g. *Endolimax nana*
(c) Genus *Iodameba*
E.g. *Iodameba butchlii*
Mastigophora (Flagellates):
(a) Genus *Giardia*
E.g. *G. lamblia* (c)

(b) Genus *Trichomonas*
E.g. *T. vaginalis*
(c) Genus *Trypanosoma*
E.g. *T. brucci*
(d) Genus *Leishmania*
E.g. *L. donovani*

Sporozoa

(1) Genus *Plasmodium*
E.g. *P. falciparum*
(2) Genus *Toxoplasma*
E.g. *T. gondi*
(3) Genus *Cryptosporidum*
E.g. *C. parvum*
(4) Genus *Isospora* E.g. *I. beli*

Ciliates

E.g. *Balantidium coli*

CLASSIFICATION OF THE PATHOGENIC PROTOZOA:
PROTOZOA ORGAN OF IMPORTANT HUMAN LOCOMOTION PATHOGENS

1. Rhizopoda Pseudopodia *Entamoeba histolytica*
(Amoeba)

2. Mastigophora Flagella Trypanosomes
(Flagellates) *Leishmania ,Trichomonas ,Giardia*

3. Sporozoa None, exhibit a slight *Plasmodium* spp., Amoeboid movement
4. Ciliates Cilia *Balantidium coli*

Phylum : Sarcomastigophora

Subphylum : Sarcodina

Class : Rhizopoda

Order : Amoebiid

Family : Endamoebidae

Genus : *Entamoeba histolytic*

Amoebas primitive unicellular microorganisms with a relatively simple life cycle which can be divided into two stages:

• Trophozoite – actively motile feeding stage.

• Cyst – quiescent, resistant, infective stage.

Their reproduction is through binary fission, e.g. splitting of the trophozoite or through the development of numerous trophozoites with in the mature multinucleated cyst. Motility is accomplished by extension of pseudopodia (“false foot”)

Morphological features

a) Trophozoites

Viable trophozoites vary in size from about 10-60μm in diameter. Motility is rapid, progressive, and unidirectional, through pseudopods. The nucleus is characterized by evenly arranged chromatin on the nuclear membrane and the presence of a small, compact, centrally located karyosome. The cytoplasm is usually described as finely granular with few ingested bacteria or debris in vacuoles. In the case of dysentery, however, RBCs may be visible in the cytoplasm, and this feature is diagnostic for E.histolytica.

b) Cyst

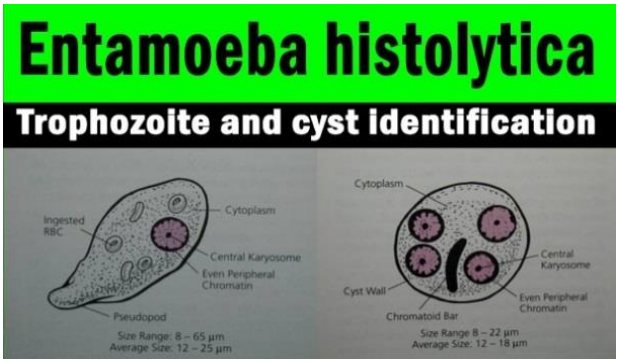
Cysts range in size from 10-20μm. The immature cyst has inclusions namely; glycogen mass and chromatoidal bars. As the cyst matures, the glycogen completely disappears; the chromotiodials may also be absent in the mature cyst

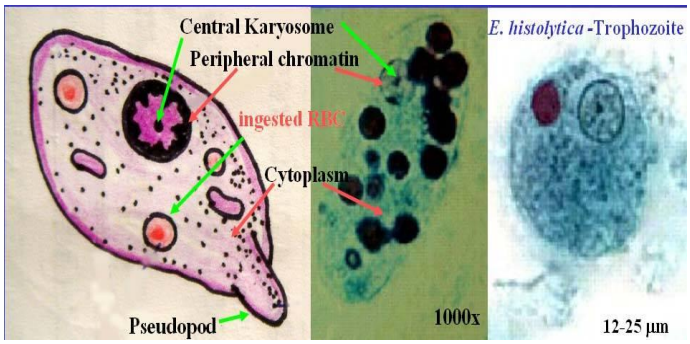
Life cycle

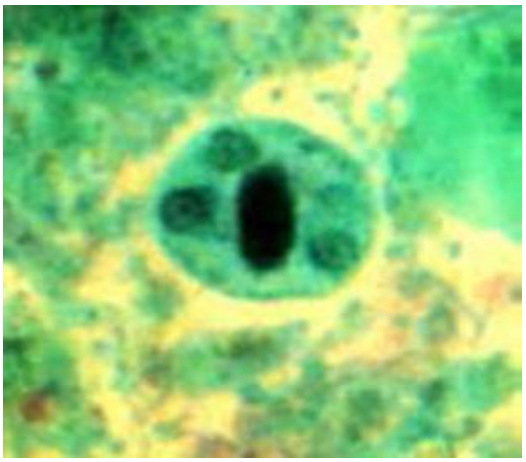
Intestinal infections occur through the ingestion of a mature quadrinucleate infective cyst, contaminated food or drink and also by hand to mouth contact. It is then passed unaltered through the stomach, as the cyst wall is resistant to gastric juice. In terminal ileum (with .alkaline pH), excystation takes place.

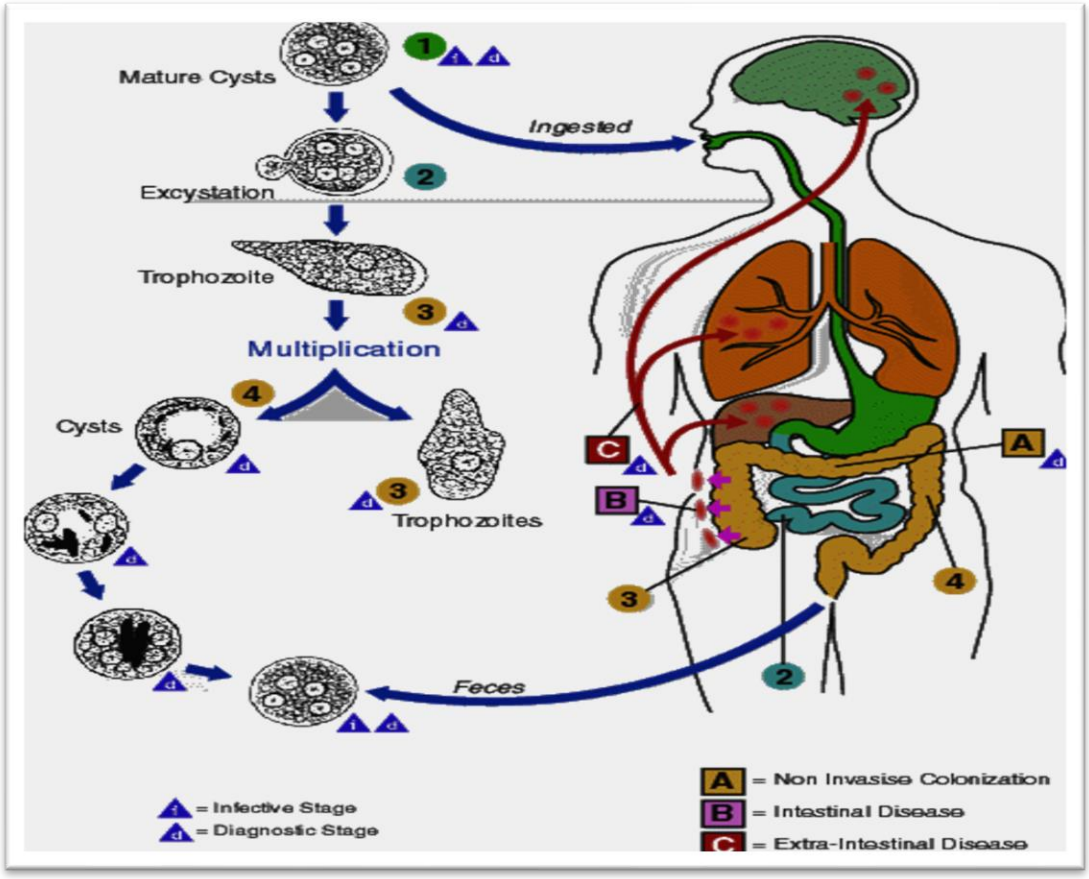
Trophozoites being actively motile invade the tissues and ultimately lodge in the submucous layer of the large bowel. Here they grow and multiply by binary fission. Trophozoites are responsible for producing lesions in amoebiasis. A certain number of trophozoites come from tissues into lumen of bowel and are first transformed into pre-cyst forms. Pre-cysts secret a cyst wall and become a uninucleate cyst. Eventually, mature quadrinucleate cysts

form. These are the infective forms. Both mature and immature cysts may be passed in Faeces. Immature cysts can mature in external environments and become infective.









Clinical features

The outcome of infection may result in a carrier state, intestinal amoebiasis, or exteraintestinal amoebiasis.

1- Diarrhea, flatulence, and cramping are complaints of symptomatic patients..

2- the severe disease is characterized by the passing of numerous bloody stools in a day.

3- (fever, leukocytosis, rigors) are present in patients with exteraintestinal amoebiasis.

4- The liver is primarily involved, because trophozoites in the blood are removed from the blood by the portal veins. The right lobe is most commonly involved, thus pain over the liver with hepatomegaly and elevation of the diaphragm is observed.

• Laboratory diagnosis

In intestinal amoebiasis:

 Examination of a fresh dysenteric faecal specimen or rectal scraping for trophozoite stage. (Motile amoebae containing red cells are diagnostic of amoebic dysentery).

 Examination of formed or semi formed faeces for cyst stage. (Cysts indicate infection with either a pathogenic *E.histolytica* or nonpathogenic *E.dispar*) .

Exteraintestinal amoebiasis

• Diagnosed by the use of scanning procedures for liver and other organs.

• Specific serologic tests, together with microscopic examination of the abscess material, can confirm the diagnosis.

Intestinal Protozoa - The Non-Pathogenic Amoebae:

1.Entamoeba hartmanni.

2. Entamoeba. coli.

3. Entamoeba gingivalis.

4.Endolimax nana.

5.Iodamoeba butschlii.

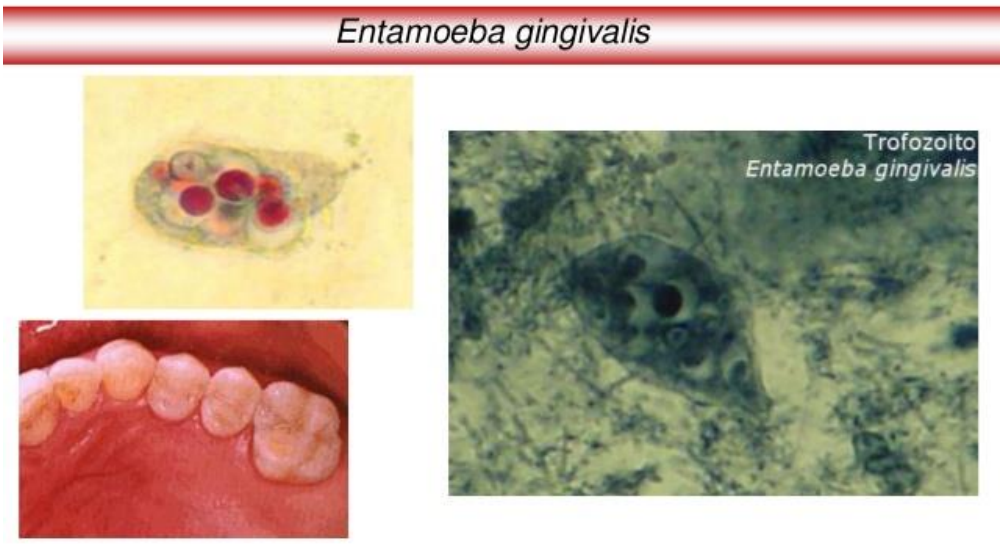
6.D.fragilis(amoebic-flagellates)

These amoebae(except E.gingivalis) are found only in the intestines they do not harm the body. They enter the human body when a person swallows food or water that has been exposed to contaminated stool.

 These amoebae can remain in a person's intestine for weeks, months or years. Studies have shown that these amoebae do not make people sick. Even people who have a weakened immune system are not affected by these amoebas.

Entamoeba gingivalis

Entamoeba gingivalis is an opportunistic Amoebozoa reported by some as an effect of disease; not a cause hence status as a commensal and is the first amoeba in humans to be described. Only the trophozoite stage presents, and encystation probably does not occur. *E.gingivalis* is a commensal, living primarily on exudate from the margins of the gums, and thrives best on unhealthy gums. No specific treatment is indicated. However the presence of *E.giingivalis* suggests a need for better oral hygiene. The infection can be prevented by proper care of the teeth and gums. It is found in the mouth inside the gingival pocket biofilm near the base of the teeth, and in periodontal pockets. Entamoeba gingivalis is found in 95% of people with gum disease and rarely in people with healthy gums. Cyst formation is not present; therefore transmission is direct from one person to another by kissing, or by sharing eating utensils. Only the trophozoites are formed and the size is usually 20 micrometers to 150 micrometers in diameter. *Entamoeba gingivalis* have pseudopodia that allow them to move quickly . Their spheroid nucleus is 2 micrometers to 4 micrometers in diameter and contains a small central endosome. There are numerous food vacuoles, which consists mostly of phagocytized PMN nucleus, blood cells and bacteria.



Phylum : Sarcomastigophora

Subphylum : Sarcodina

Class : Mastigophora

Flagellates

members of this group can inhabit mouth, bloodstream, tissues, gastrointestinal, or urogenital tracts . Mostly uninucleate organisms, that possess, at some time in the life cycle, one to many flagella for locomotion and sensation. (A flagellum is a hair like structure capable of whip like lashing movements that furnish locomotion.) Mastigophora - the flagellates. Inhabit the mouth, bloodstream, gastrointestinal, or urogenital tracts.

Morphological Characteristics

 Flagellum(ae) - organelles of locomotion; an extension of ectoplasm; moves with a whip-like motion.

 Axostyle - a supporting mechanism, a rod-shaped structure; not all

flagellates have these.

 Undulating membrane - a protoplasmic membrane with a flagellar rim extending out like a fin along the outer edge of the body of some flagellates.

 Costa - a thin, firm rod-like structure running along the base of the undulating membrane.

 Cytosome - a rudimentary mouth; also referred to as a gullet.

Identification of a flagellate is based upon:

 Size.

 Shape.

 Motility.

 Number and morphology of nuclei.

 Number and location of flagella

 Location in the body of the host.

Flagellates are classified according to their occurrence in their vertebrate host body

1- intestinal and atrial flagellates which live in the alimentary canal and the urogenital tract.

2-Blood and tissue flagellates which live in the blood, lymph and tissue of the host

Intestinal flagellates

Giardia intestinalis (Giardia lamblia)

Is a flagellate protozoan, that colonizes and reproduces in the small intestine, causing giardiasis. common to human and several other mammalian species such as dogs, cats, bovines .The life cycle includes trophozoite and cyst phases. This organism is now considered to be the only known pathogenic intestinal flagellate It is causing disease

Morphology

 The **trophozoite** is pear shaped, with a broad anterior and much attenuated posterior. It is 10-12µm long and 5-7µm wide, bilaterally symmetrical, and has two nuclei. It is also relatively flattened, with a large sucking disk on the anterior ventral side, which serves as the parasite's method of attachment to the mucosa of the host. The trophozoite also has two median bodies and four pairs of flagella (anterior, caudal, posterior and ventral).

 The *G. intestinalis* **cyst** is egg-shaped, and measures 8-14µm by 7-10µm. After encystation, each organelle duplicates, so each cyst contains four nuclei, four median bodies, eight pairs of flagella--although these organelles are not arraigned in any clear pattern. Upon excystation, each cyst produces two trophozoites called (Giardiasis). The natural habitat to this parasite is duodenum of human intestine.

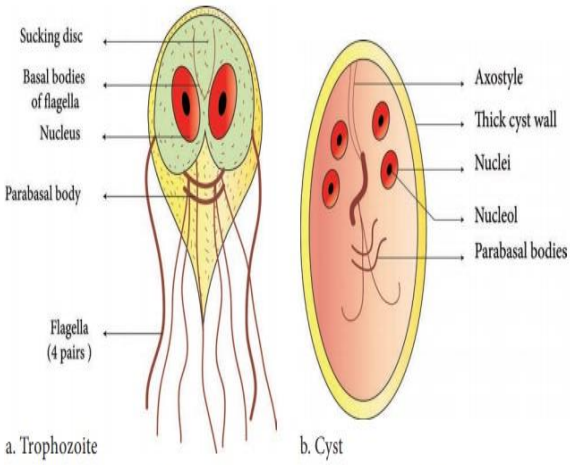
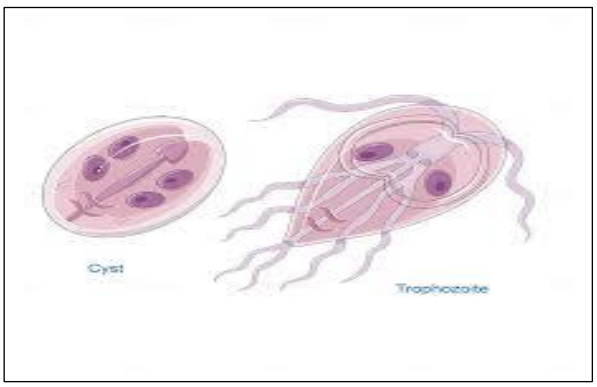
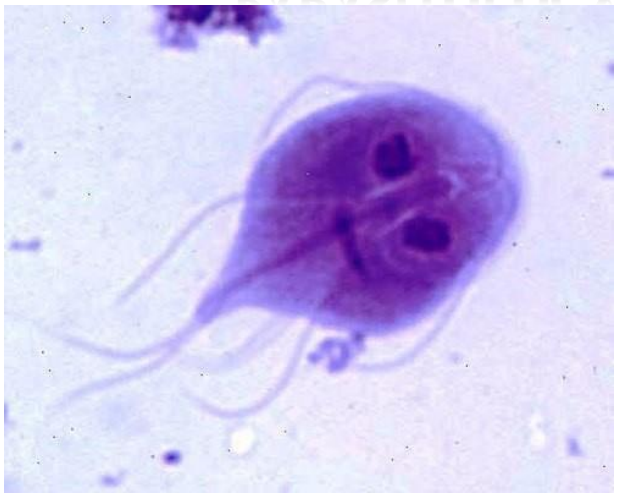


Figure 8.6: Trophozoite and cyst of *Giardia*





Life Cycle

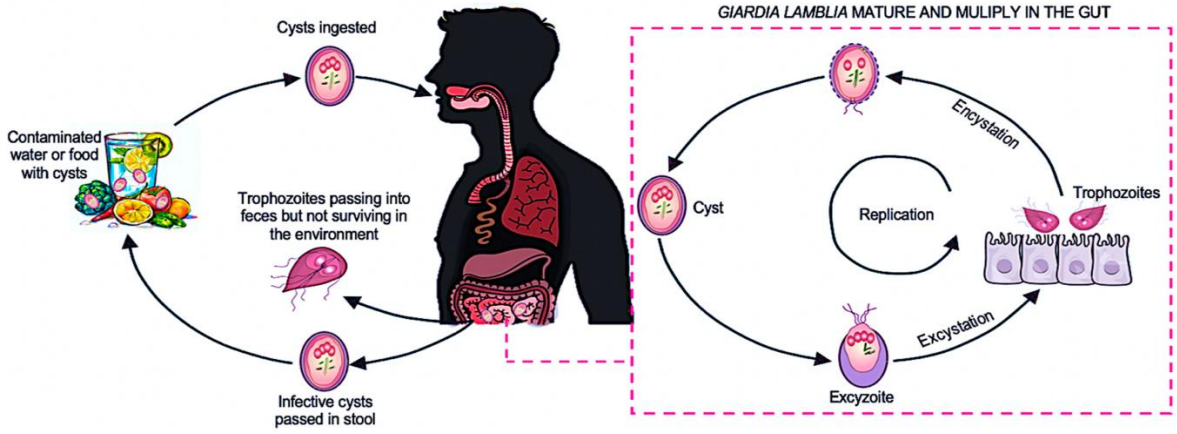
The infective Giardia lamblia cysts enter the stomach , the cyst excyst in the duodenum resulting trophozoites become established and multiply approximately every 8 hours via longitudinal binary fission. The trophozoites feed by attaching their sucking discs to the mucosa of the duodenum . Trophozoites may also infect the common bile duct and the gallbladder . Encystation occurs as the trophozoites migrate into the bowel . The cysts enter the outside environment via the feces and may remain viable for as long as 3 months in water. Trophozoites entering into the outside environment quickly disintegrate.

Notes :

„« Trichrome stain is useful for finding the cysts and trophozoites

„« Trophozoites disintegrate rapidly outside of the body but may be found in fresh , watery stools .

„« Cysts are found in soft and (semi) formed stools .



Pathogenicity

Infection with Giardia intestinalis can range from asymptomatic to severe diarrhea. Trophozoites attach themselves tightly to the free surface of the small intestinal epithelia through the adhesive discs. They cause marked increase of mucous production, diarrhea, dehydration, intestinal pain, flatulence and weight loss. Due to their attachment, the parasite prevent mechanically, the absorption of fats and fat dissolved vitamins. Giardiasis demonstrated by the occurrence of greasy, mucous, watery but not bloody diarrhea. The parasite may enter the bile duct, and the gall bladder which can cause jaundice and colic

Clinical signs

In symptomatic individual, the incubation period lasts 1-3 weeks. Symptoms consist of diarrhea frequently accompanied by abdominal pain, less often there is nausea and vomiting. The acute stage lasts about 3-4 days and in some patients may be prolonged illness with episodes of recurring diarrhea.

Laboratory Diagnosis

1. Stool examination : The stool sample is the specimen of choice for the recovery of Giardia lamblia trophozoites and cysts.

2. Stool antigen detection : available tests use either an immunofluorescent antibody (IFA) assay or enzyme-linked immunosorbent assay (ELISA) against cyst or trophozoite antigens, these examinations are limited to the detection of Giardia lamblia .

3. Stool culture : not useful for diagnosing Giardiasis because the organism cannot be grown from patient samples .

4. Serum antibody detection . 5. String test (entero-test).

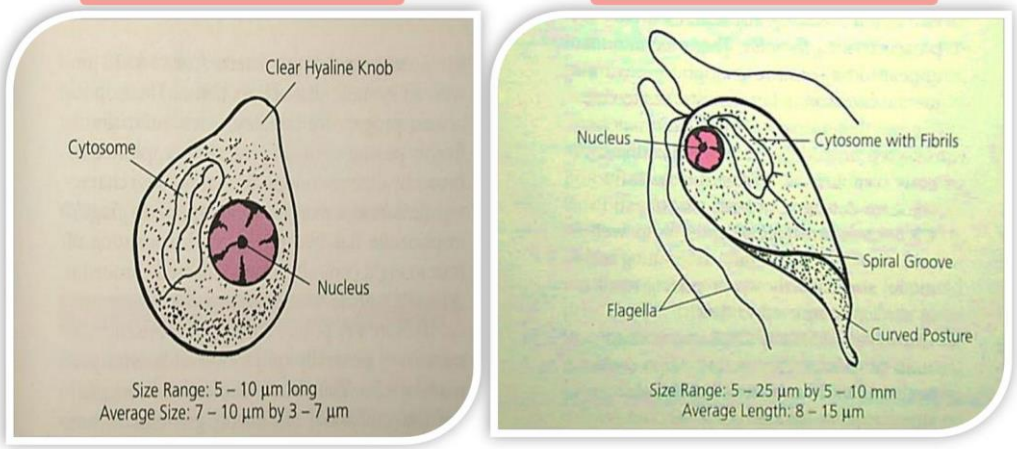
Genus : *Chilomastix mesnili*

Considered to be a non-pathogen, it's resides in cecum and colon . It is cosmopolitan protozoan of the human intestinal tract (especially the large intestine). It has trophozoite and cyst stages and the infection usually acquired from the cysts in contaminated food or drink

The life cycle of this parasite have two stages :

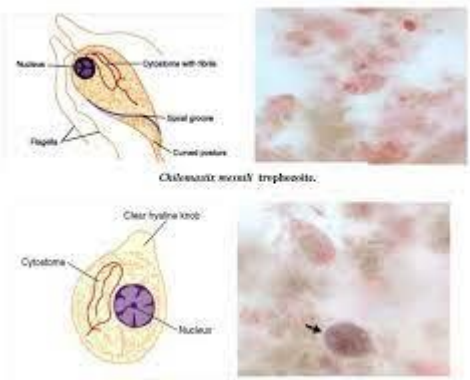
1 – Trophozoite stage :

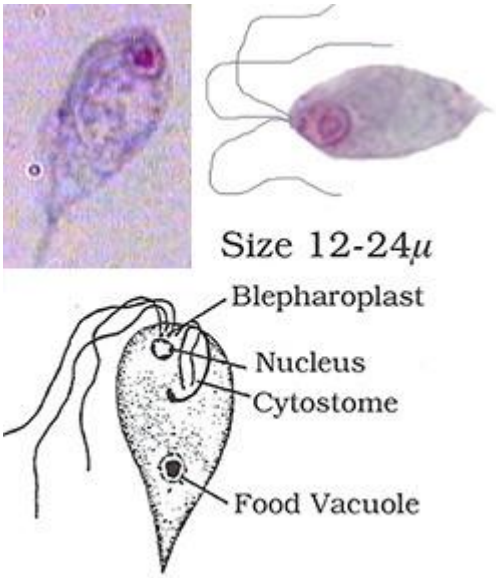
It's pear in shape (the broad anterior end tapers toward the posterior end of the organism) .Stiff , rotary motility in a directional pattern is typically of the Chilomastix mesnili trophozoites . Have the single nucleus , is located in the anterior end with small centric or eccentric karyosome , and no peripheral chromatin . Have four flagella , (three extending from anterior end , one extending posteriorly from Cytosome) .Also Prominent Cytosome (with fibrils) extending 1/3 to 1/2 of the body length and it is located to one side of the nucleus Typical spiral groove , the presence of this spiral groove results in a curved posture at the posterior end . The Cytosome arising from the anterior pole three thin flagella, two of them extends to the left back direction of the body while the third one lies within the cytostome, and two stiffer curved fibrils, one on each side of the cytostome “It is believed that the third flagellum consist an undulating membrane, pushing the food particles to the depth of the cytostome”. The nucleus is situated at the extreme anterior end. The parasite moves forward with a jerky movement in a spiral path. Multiplication is by longitudinal binary fission.



2– Cyst stage :

It's usually lemon shape and possess a clear anterior hyaline knob. Have a large single nucleus with large central karyosome , and no peripheral chromatin .The well-defined Cytosome (with its accompanying fibrils) may be formed to one side of the nucleus. The cyst is lemon-shaped, measures 7-9µ; it has a relatively thick hyaline wall, nucleus, and cytostome. In unformed stools the majority of this organism is motile trophozoite; in formed stool, only cysts are seen. There is no evidence that this organism is pathogenic, but it could be say that this parasite benefits and multiplied in the intestinal infections, but it does not cause the disease.





Laboratory Diagnosis

Stool examination : examination of freshly passed liquid stools from patients infected with Chilomastix mesnili typically reveal only trophozoites , while examination of formed stool samples from such patients usually reveal only cysts . Samples of semi formed consistency may contain both trophozoites and cysts.

Genital, Intestinal, Oral flagella

Trichomonas Spp.

Its includes a group of flagellated protozoa ,It infect humans and animal. flagellates of the digestive tract and genital organs :

A-inhabiting the mouth, intestine, and genital tract are typically lumen parasites ex. *Giardia lamblia* in the duodenum and *Trichomonas vaginalis* in the vagina or *Trichomonas hominis* in intestine.

B-flagellates of the blood and tissues ex.*Leshmania* spp. & *Trypanosoma* spp

Phylum : Sarcomastia

Subphylum : Mastigophora

Class : Zoomastigophora

Order : Diplomonadida

Family : Hexamitidae

Trichomonas Characteristic

These flagellate exist only in trophozoide stage.they have four flagella and one lateral flagellum which is attached to the surface of the parasite to form undulating membrane,its supported by a rod-like structure called costa. The axostyle runs down the middle of the body and ends in the pointed lail-like extremity.A round nucleus is located in the anterior portion.

Trichomonas include three species infect human:

-1-*T. tenax*: living around ,in cavities of carious teeth,its harmless commensal of the human mouth.Transmitted by kissing or salivary droplet

-2-*T. hominis*: Inhibits caecum of man,it does not invade the intestinal mocusa (not pathogenic).occasionally been found in the diarrheic stool. Transmitted by oral- fecal mode.

-3-*T. vaginalis*

Genus : *Trichomonas vaginalis* or (Genital flagellate)

Trichomonas vaginalis has a worldwide distribution It is responsible for one of the most widespread sexually transmitted disease worlwide, trichomoniasis or “trich”. It is closely related to *T. hominis* and it can be distinguished by having a short undulating membrane that extend only about half the distance to the posterior end of the body, with no free flagellum. Most importantly, the two organisms are site specific and live in totally different sites and many unsuccessful attempts have been made to introduce *T. hominis* into vagina. It is an anaerobic, parasitic flagellated protozoan, is the causative agent of trichomoniasis.there is no known cyst form of *T. vaginalis* .It have only trophozoite stage *T. vaginalis* trophozoites reside on the mucosal surface of the vagina in infected women and the most common infection site of *T. vaginalis* in males is the prostate gland region and the epithelium of the urethra .. It is causes persistent urethritis , persistent vaginitis and infant infections (respiratory infections and conjunctivitis)

Morphology

A unicellular trophozoite is oval as well as flagellated, Five flagella arise near the Cytosome, In addition, a conspicuous barb-like Axostyle used for attachment to surfaces and may also cause the tissue damage noted in Trichomoniasis infections. lives in close association with vaginal, urethral and prostatic tissue. While *T. vaginalis* does not have a cyst form, *T. vaginalis* obtains nutrients by transport through the cell membrane and by phagocytosis. The organism is able to maintain energy requirements by the use of a small amount of enzymes to provide energy via glycolysis. multiples by binary fission. There is no cyst in the life cycle, so transmission is via the trophozoite stage.

Trophozoite stage

1-It appear ovoid , round , or pear like in shape .Rapid , jerky molity.

.2-have (4-6) flagella , all of witch originate from the anterior end only one of the flagella extends posteriorly.

.3-The single nucleus is ovoid , nondescript , and not visible in unstained preparations.

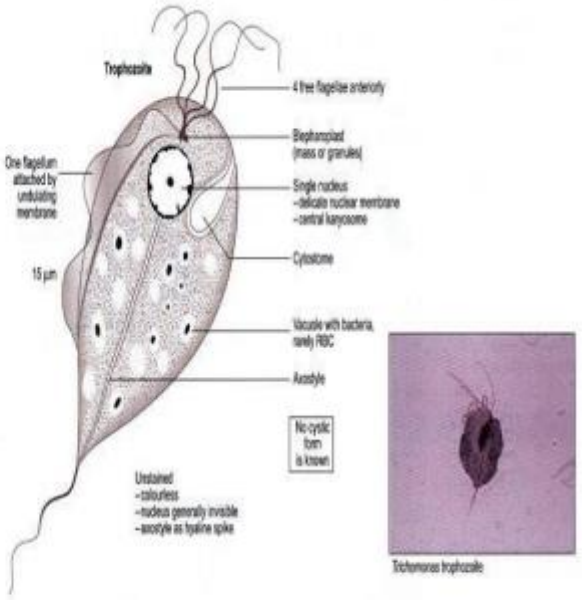
.4-Undulating membrane is short relatively speaking extending only one half of the body length.

.5-Prominent axostyle that often curved around the nucleus and extends posteriorly beyond the body granules may be seen along the axostyle.



Strawberry cervix" due to T. vaginalis

Strawberry cervix" due to *T. vaginalis*



Life cycle

T. vaginalis colonizes the vagina of women and the urethra (sometimes prostate) of men. Infection occurs primarily via sexual contact, although non-venereal infections are possible. The organism does not encyst and divides by binary fission which is favored by low acidity (pH > 5.9; the normal pH is 3.5 to 4.5). There is no non-human reservoir.Trichomonads reproduce by a special form of longitudinal fission, leading to large numbers of trophozoites in a relatively short time.

 Manifestation of infection

The main signs of a Trichomonas infection in women are abdominal pain, itching, and presence of a foul-smelling discharge with abundant leukocytes, while in men the infection is mostly asymptomatic, although it can sometimes lead to urethritis, prostatitis, and epididymitis. Infection with this organism is also associated with severe complications, such as infertility and enhanced predisposition to neoplastic transformation in cervical tissues. asymptomatic-vast majority . symptomatic - vaginitis, prostatitis, urethritis .

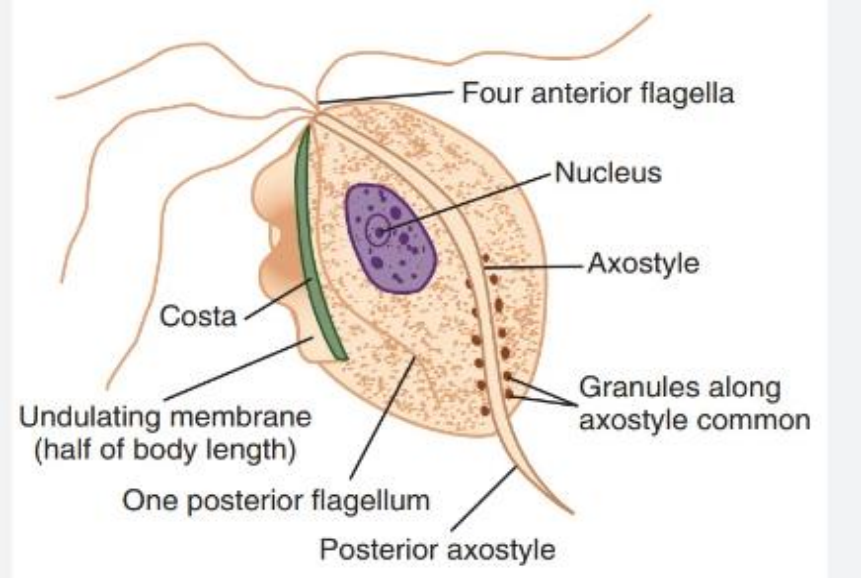
Pathogenesis of Trichomonas infections occurs by:

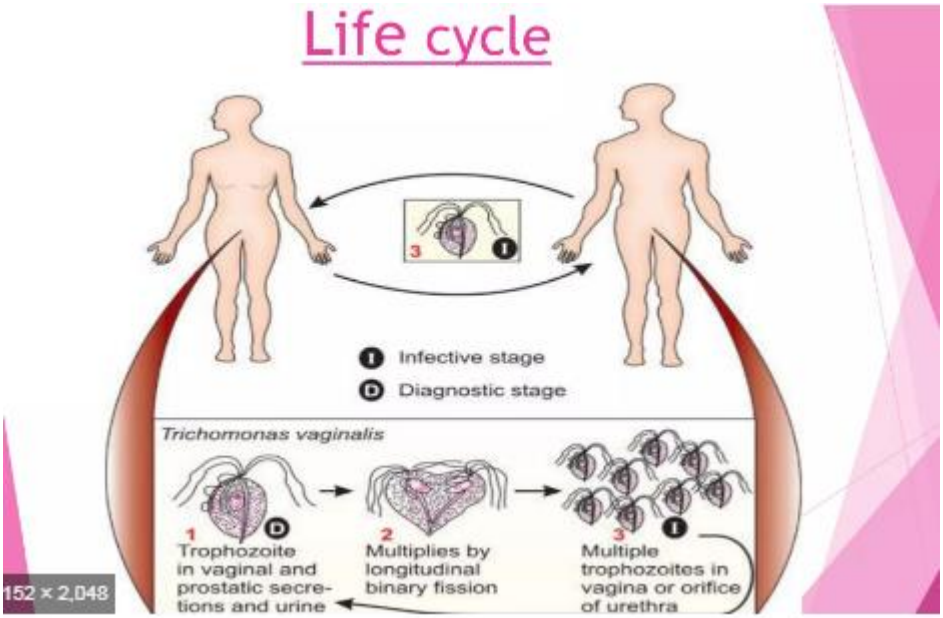
(1)cytopathogenicity against vaginal epithelial cells .

(2)Adhesion of the parasite to the target cell is essential for the maintenance of infection and for cytopathogenicity.

Laboratory Diagnosis

Depends on finding trophozoites in secretions of the genital tract from men or women. In cases where the numbers of organisms are very low, the trophozoites can be cultured to .increase their numbers





Trichomonas tenax / Oral flagellates

Considered to be a non-pathogen and no chemical treatment is normally indicated . There is a known cyst stage of T. tenax .It is located in the tartar between the teeth , tonsillar . crypts , pyorrheal pockets and gingival margin around the gums

Trophozoite stage

1- It's oval to pear in shape.

2-The single ovoid vesicular nucleus is filled with several chromatin granules and is usually located in the center anterior portion of the organism.

3-Has five flagella , all originating anteriorly (4 extend anteriorly, 1extend posteriorly.

4-Undulating ng membrane extending 2/3 of the body length .

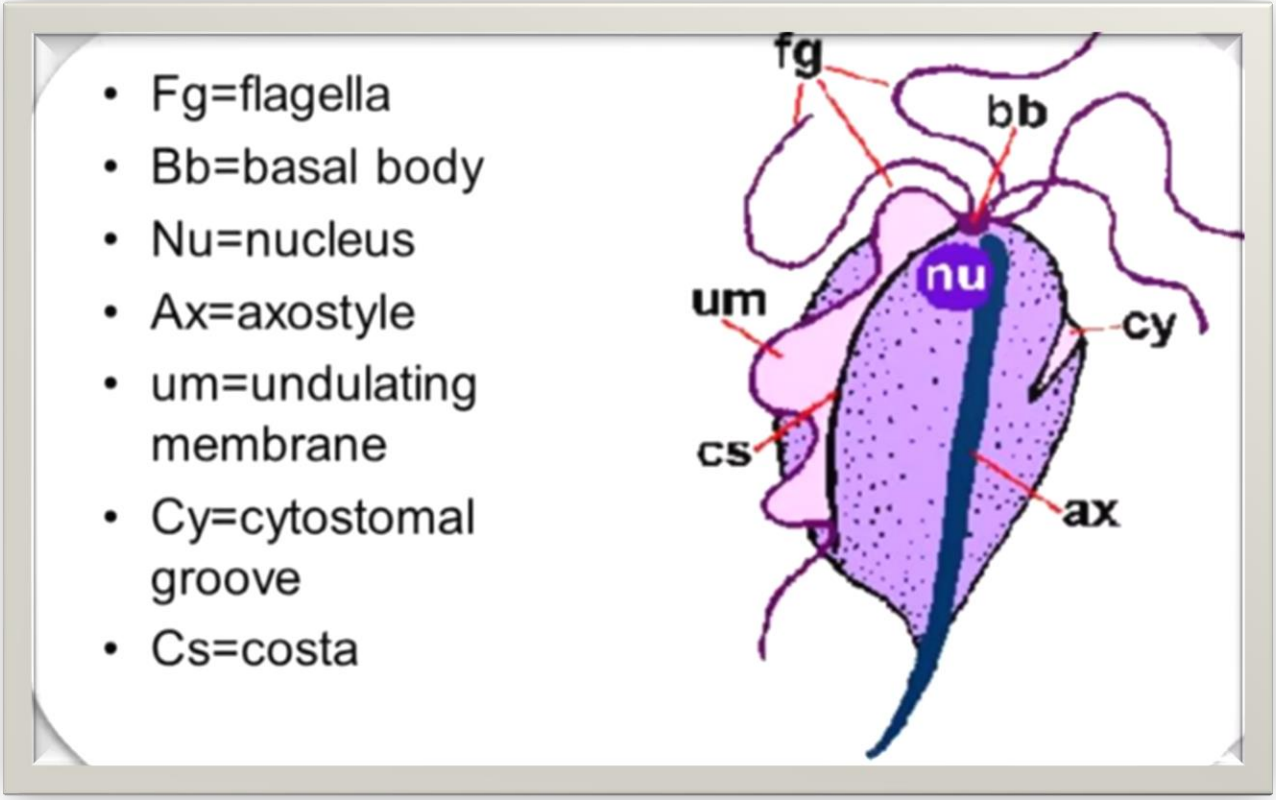
5- Costa typically lie next to the posterior flagellum.

6-A thick axostyle runs along the entire body length curving around the nucleus and extends posteriorly beyond the body of the organism.

7-A small anterior cytosome is located next to the axostyle opposite the undulating membrane.

Laboratory Diagnosis

The specimen of choice for diagnosing ***T. tenax*** trophozoites is mouth scraping . Micro scoping examination of tonsillar crypts and pyorrheal pockets of patients suffering from *T. tenax* infections often yield the typical trophozoites .



Trichomonas hominis

Considered to be a non-pathogen . Treatment , therefore ; is usually not indicated. There is no known cyst form of Trichomonas hominis

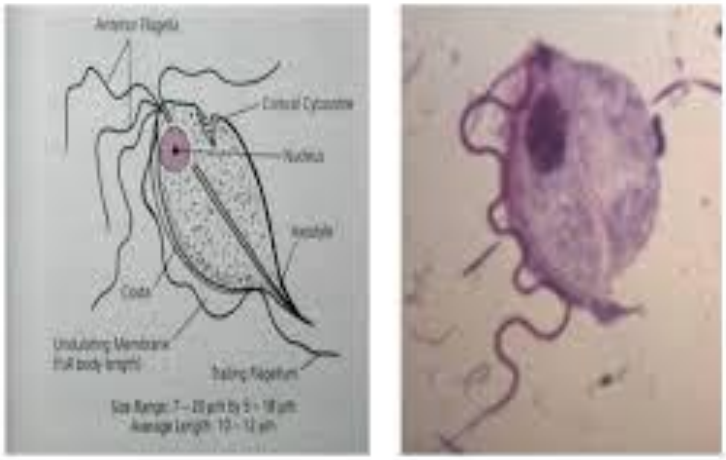
Trophozoite stage:

It's pear in shape .The characteristics nervous , jerky motility is accomplished with the assistance of a full body-length undulating membrane .The single nucleus is located in the anterior region of the organism with small central karyosome is surrounded by a delicate nuclear membrane , and no peripheral chromatin .Trophozoite is supported by an axostyle that extends beyond the posterior end of the body . A conical-shaped cytosome cleft may be seen in the anterior region of the organism lying ventrally opposite the undulating membrane. Has 3 – 5 flagella that originate from anterior end , the single posterior flagellum

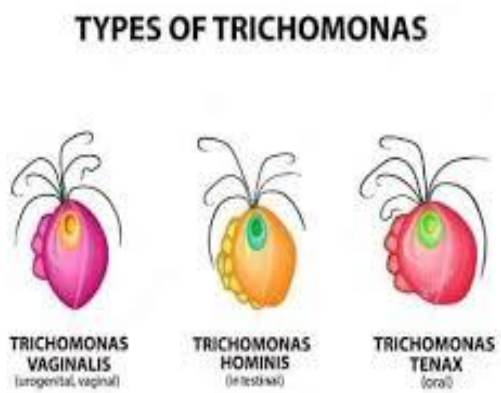
is an extension of the posterior end of the undulating membrane .The Costa connects the undulating membrane to the trophozoite body.

Laboratory Diagnosis

Stool examination : The stool sample is the specimen of choice for the recovery of Trichomonas hominis trophozoites.



Trichomonas hominis Trophozoite



Phylum: Sarcomastigophora

Subphylum: Ciliophora

Class: Cilliata

Order: Euciliata

Kingdom: Animalia
Phylum: Ciliophora
Class : Litostomatea
Order : Vestibuliferida
Family : Balantiididae

Genus: *Balantidium coli*

Balantidium coli is a protozoan parasite responsible for the disease Balantidiasis. *B.coli* is the largest protozoan, *B.coli* is a species of ciliate protozoan. This parasite is the only member of this family known to be pathogenic to humans. Hosts include pigs, rats, primates (including humans), horses, cattle and guinea pigs. Infection is transmitted within or between these species by fecal-oral transmission. Pigs are the most significant reservoir

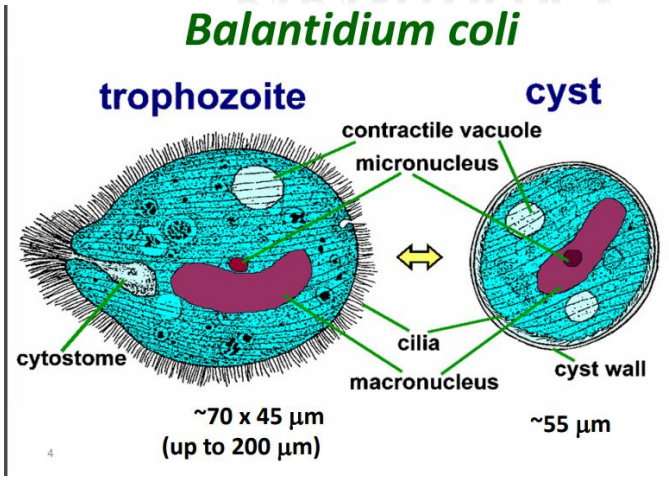
hosts. The protozoa are found worldwide, usually with a prevalence of less than 1%. Infection is rare, but is likely to occur in places where humans live closely with swine and where water sanitation is poor or non-existent.

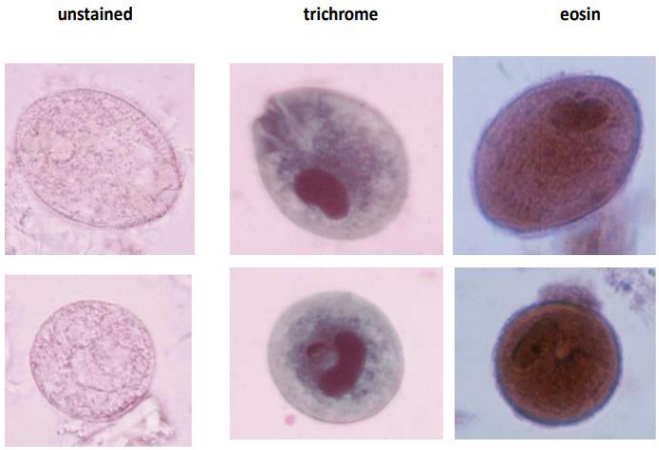
Trophozoite

The trophozoite lives in the large intestine, feeding on cell debris, bacteria, starch grains and other particles. The trophozoite is a large ovoid cell, about 60 to 70 μm in length and 40 to 50 μm in breadth. Very large cells,up to 200 μm are sometimes seen.The anterior end is narrow and the posterior broad. At the anterior end is a groove (peristome), leading to the mouth (cytostome). Posteriorly there is a small anal pore (cytopyge). The cell is covered all over with short delicate cilia. The cell has two nuclei—a large kidney-shaped macronucleus and lying in its concavity a small micronucleus. The cytoplasm has one or two contractile vacuoles and several food vacuoles.

Cyst

The cyst measures approximately 50-75 μm long in the bottom figure and has a thick, refractile cyst wall, within which the organism may be seen. In stained specimens the macronucleus can usually be seen within the cyst wall, but other structures usually are not observed.



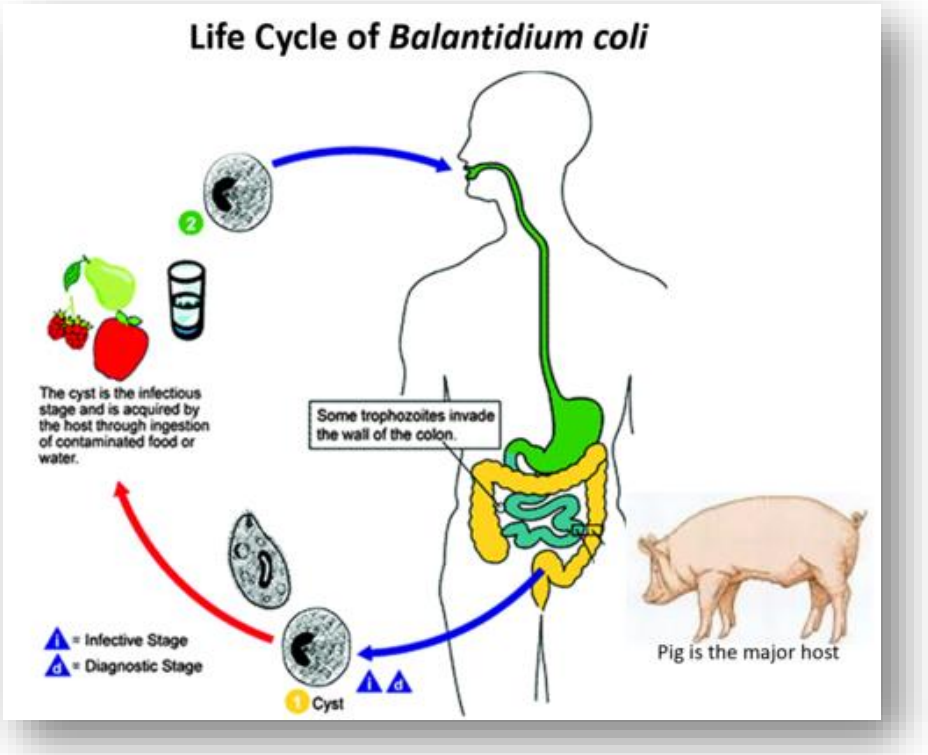


Clinical manifestation

who are infected with *B. coli* remain asymptomatic. Although Balantidium coli usually resides in the lumen of its host, trophozoites can invade the mucosa of the large intestine (cecum and colon) and cause ulcerations. The parasite secretes a substance like Proteolytic enzyme and cytotoxic enzyme, which helps degrade intestinal tissue and facilitates penetration of the mucosa. Other bacteria in the intestine may enter the ulcer leading to secondary infections. Ulcerations of the large intestine can be viewed using sigmoidoscopy. Common symptoms of Balantidiasis include chronic diarrhea, occasional dysentery (diarrhea with passage of blood or mucus), nausea, foul breath, colitis (inflammation of the colon), abdominal pain, weight loss, deep intestinal ulcerations, and possibly perforation of the intestine. After ingestion of an infective *Balantidium coli* cyst, days to weeks may pass before infection occurs.

Life Cycle of *Balantidium coli*

The cyst is the infective stage of ***Balantidium coli*** . Once the cyst is ingested via feces-contaminated food or water, it passes through the host digestive system. The tough cyst wall allows the cyst are resist to acidic environment of the stomach and the basic environment of the small intestine until it reaches the large intestine. There, excystation takes place. Excystation produces a trophozoite from the cyst stage. The motile trophozoite feeding on intestinal bacterial flora and intestinal nutrients.Trophozoites multiply by asexual binary fission or sexual conjugation,The trophozoite may become invasive and penetrate the mucosa of the large intestine,Trophozoites are released with the feces, and encyst to form new cysts. Encystation takes place in the rectum of the host as feces are dehydrated or soon after the feces have been excreted.



Diagnosis

Examination of stool samples, looking for trophozoites and cysts ,Trophozoites are readily identified because of their large size and the fact that B. coli is the only ciliate that parasitizes humans ,The infection may disappear spontaneously or the host may become asymptomatic, with the host remaining as a carrier Several drugs that are taken orally are known to eliminate the infection, Microscopic examination of faeces for trophozoite and cysts is

performed. The trophozoite is very large, varying in length from 50 to 200μm and in width from 40 to 70μm. The surface is covered with cilia.

Blastocystis hominis

is an inhabitant of the human intestinal tract previously regarded as non-pathogenic . Its pathogenicity remains controversial. The organism is found in stool specimen from asymptomatic people as well as from people with persistent diarrhea. *B.hominis* is capable of pseudopodia extension and retraction, and reproduces by binary fission or sporulation. The classic form that is usually seen in the human stool specimen varies tremendously in size, from 6-40μm. There are thin –walled cysts involved in autoinfection, and thick–walled cysts responsible for external transmission via the faecal-oral route. The presence of large numbers of these parasites(five or more per oil immersion microscopic field) in the absence of other intestinal pathogens indicates disease. Treatment with iodoquinol or metronidazole has been successful .

Clinical Features

The infection is frequently asymptomatic (carriers).Types of symptomatic disease:

1) Mild blastocystosis: The most common type; manifests with diarrhea (without blood in stool), abdominal pain, nausea, weight loss, and sometimes fine macular rash.

2) Acute blastocystosis: Very rare; typically manifests with watery diarrhea, spastic abdominal pain, and occasionally fever; may lead to gastrointestinal (GI) bleeding.

Irritable bowel syndrome (IBS) has been associated with blastocystosis in some studies, although a causal relationship has not been proven

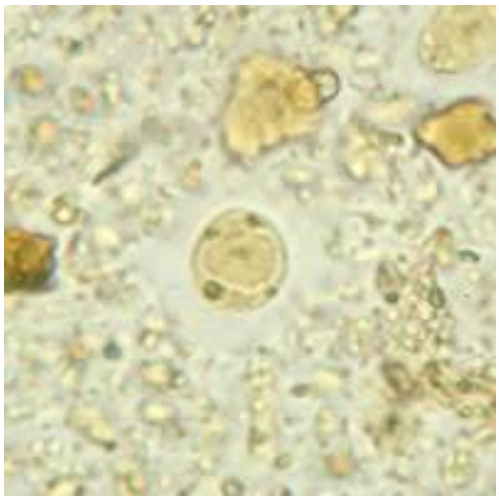
Diagnostic Criteria

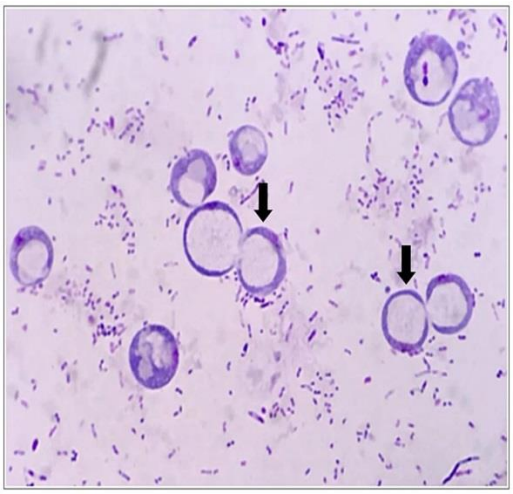
Diagnosis is made based on microscopic identification of any morphologic forms of *B hominis* in stool. Repeat stool sampling is usually recommended, several days apart, as the parasite is excreted in shifted temporal patterns and in varying amounts. Because of the morphologic diversity of the different forms of B hominis, standard direct stool examination

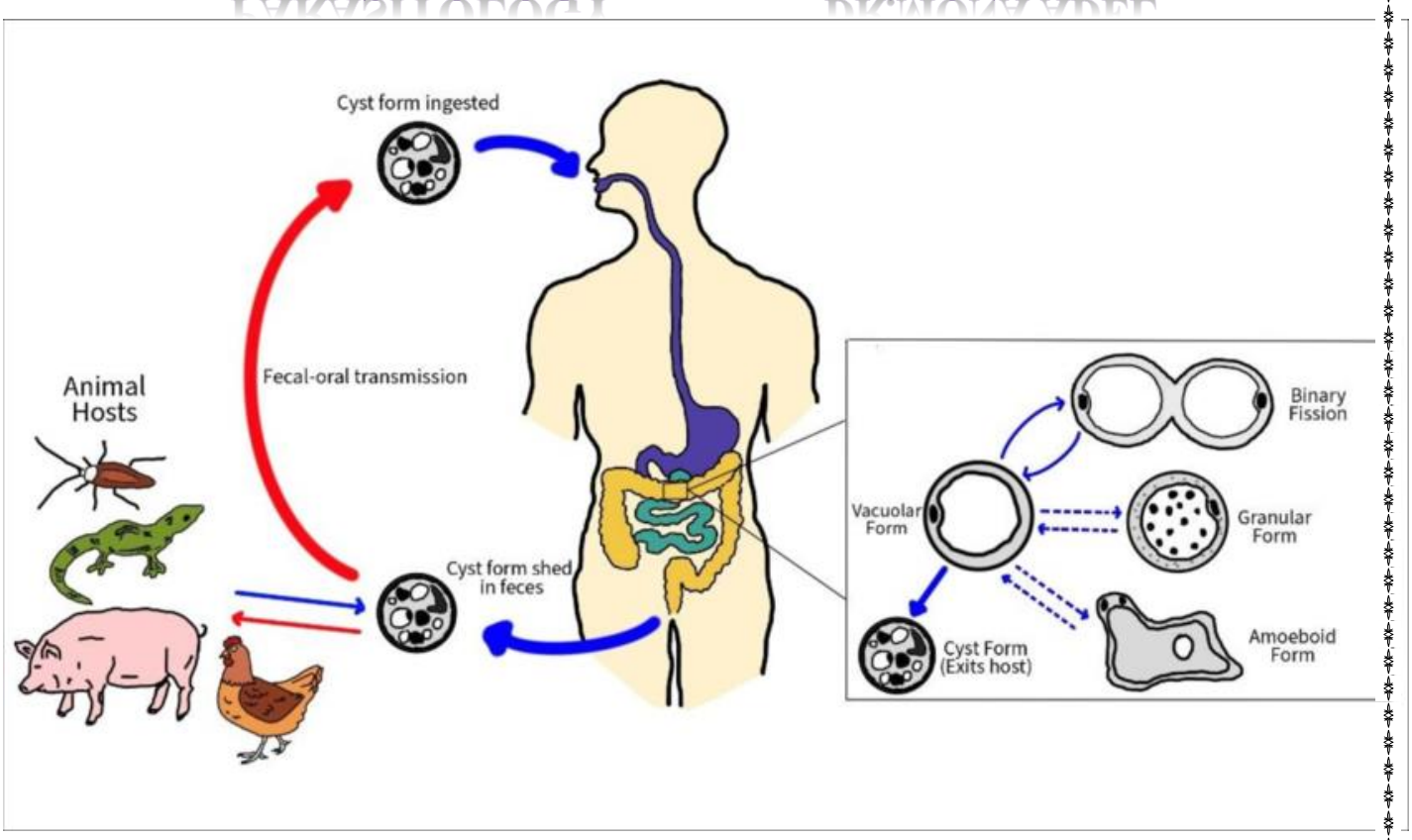
should be accompanied by the analysis of permanent Trichrome-stained smears. Polymerase chain reaction (PCR) assays are now available and have high sensitivity and specificity. However, as B hominis is frequently found in asymptomatic individuals and its pathogenicity is not fully known, the significance of a positive PCR test result is debatable.

 Lacks a cell wall but has a mitochondrion with protozoan morphology. Capable of pseudopodial extension and retraction Asexual reproduction by binary fission or sporulation under strict anaerobic conditions Optimal growth= 37 oC in the presence of bacteria; does not grow on fungal media

Parasite Biology Multiplication of *Blastocystis hominis* is by binary fission Transmitted by fecal-oral route Morphological forms: 1. Vacuolated 2. Ameba-like 3. Granular 4. Multiple fission 5. Cyst 6. A vacuolar







Apicomplexa:

The phylum: Apicomplexa are a large group of protozoa, characterized by:

 The presence of an apical complex (apicoplast) that functions in the invasion of host cell.

 They are unicellular, spore-forming, and exclusively parasites of animals or human.

 Motile by gliding motility. Motile structures such as flagella or pseudopods are absent except in certain gamete stages.

 Is a diverse group including organisms such as coccidia *(Cryptosporidium sp., Isospora belli, Sarcocystis and Toxoplasma gondii) (Plasmodium spp.).*

 Most members have a complex life-cycle, involving both asexual (schizogony or merogony) and sexual (gametogony and sporogony) reproduction.

 Typically, a host is infected via an active invasion by the oocyst containing numerous sporocyst, which divide to produce sporozoites that enter its cells.

 Within the host cells, the sporozoites transform in to trophozoites (schizonts), and each releasing merozoites internally. Eventually, the cells burst, releasing merozoites which infect new cells.

 This may occur several times, until some merozoites transform into male (microgametocyte, each one will develops into several microgametes) and female (macrogametocyte, each one will develops into one macrogamete), which forming gametes that fuse to create new cysts (oocyst).

 There are many variations on this basic pattern, however, and many Apicomplexa have more than one host.

Cryptosporidium parvum **(cryptosporidiosis)**

The first human cases of cryptosporidiosis were reported in 1976.

Initially it was believed to be a rare and exotic infection.

Now recognized as a common human pathogen and a frequent cause of diarrhea in humans.

In immunocompetent individuals this diarrhea is self-limiting and last about two weeks.

The disease is quite serious and potentially life-threatening in immunodeficient patients (especially AIDS) and is characterized by a profuse watery diarrhea.

Mode of Infection with *Cryptosporidium parvum*:

 Ingestion of thick-walled oocysts: In contaminated food or drink (called heteroinfection).

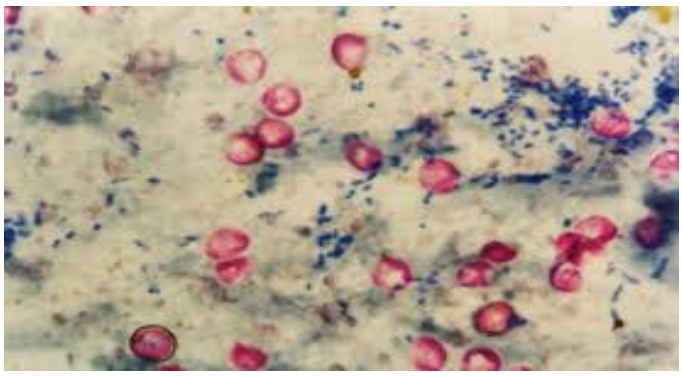
By faeco-oral route (hand to mouth) in already infected patient (called external autoinfection).

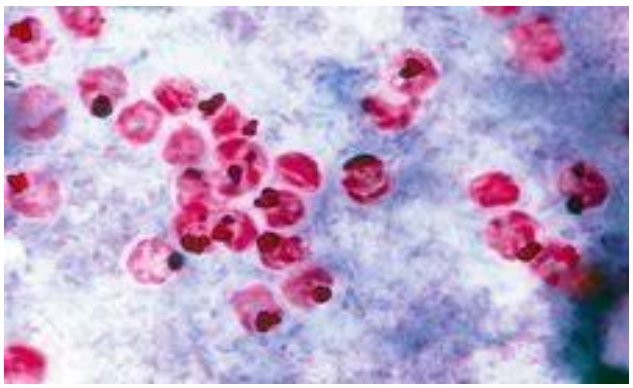
 Thin-walled oocysts in intestinal lumen of already infected patient causes internal autoinfection

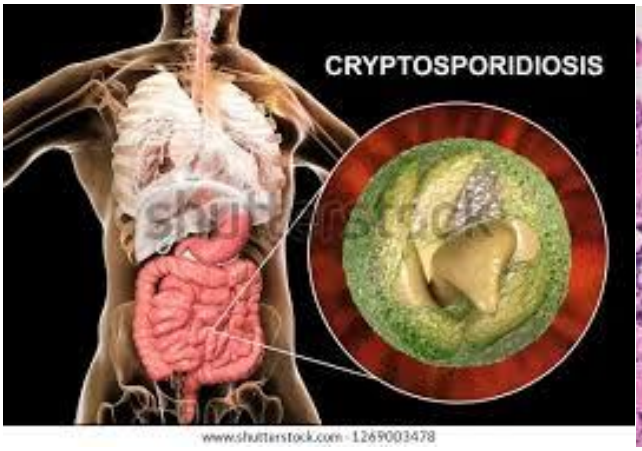
Pathogenesis:

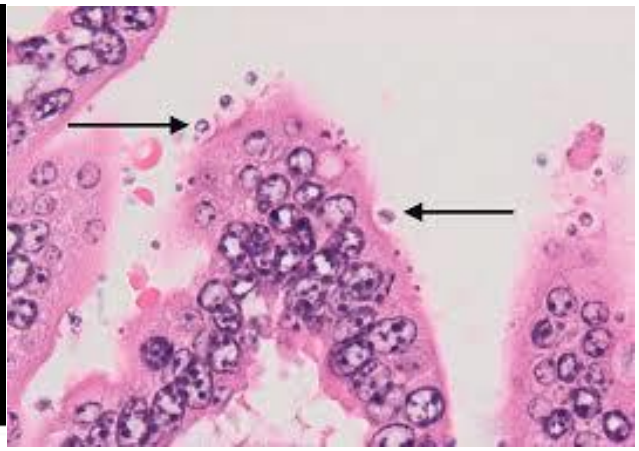
The most common clinical manifestation of cryptosporidiosis is a mild to profuse watery diarrhea. This diarrhea is generally self-limiting and persists from several days up to one month. Recrudescences are common. Abdominal cramps, anorexia, nausea, weight loss and vomiting are additional manifestations which may occur during the acute stage. The disease can be much more severe for persons with AIDS which manifests as a chronic diarrhea

lasting for months or even years. Diarrhea can have osmotic, inflammatory, or secretory components.









Phylum: Apicomplexa

Class: Sporozoea

Subclass: Coccidia

Order: Eucoccidiida

Suborder: Eimerina

Family: Sarcocystidae

Genus: *Sarcocystis* spp.

There are about 130 recognised species in this genus reported from the striated muscles of mammals, birds, reptiles and humans. The parasites derive their name from the intramuscular cyst stage (sarcocyst) present in the intermediate host. • Sarcocystosis is one of the most zonosis parasitic disease of livestock and infects animals. • Most Sarcocystis species, infecting human and domestic animals, are species-specific for their intermediate

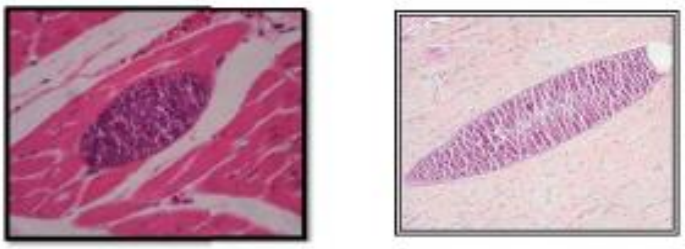
hosts and family-specific for their final hosts. • Sarcocystis infections in the intermediate host are usually asymptomatic. Gastrointestinal disease is occasionally reported in humans. • Sarcocysts are found in striated and heart muscles and may be either microscopic or visible to the naked eye and contain metrocytes initially, and bradyzoites when mature.

Life cycle

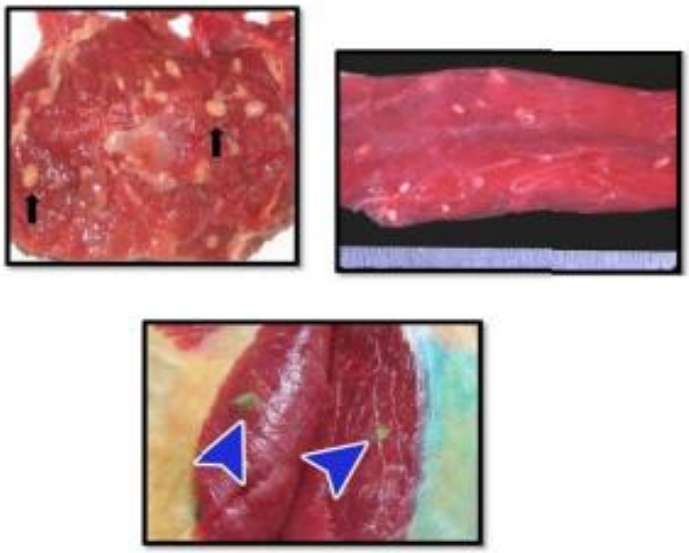
Intermediate host: Herbivores or omnivores (like cattle, pigs, and humans) ingest sporocysts from the definitive host's feces. The parasites undergo asexual reproduction in the intermediate host's tissues, eventually forming sarcocysts (also called "Miescher's tubules") in the muscle.

Definitive host: Carnivores or omnivores (like dogs, cats, or humans) eat the raw or undercooked muscle meat containing sarcocysts. The sarcocysts mature into a form that is infectious to the definitive host, and sexual reproduction occurs in the intestinal lining, releasing sporocysts in the feces.

Parasites in the muscle can be identified by microscopic examination of tissue samples. Eggs and sporocysts in feces can be identified by microscopic examination of feces.



Miesher's tubes



Sarcocystis spp. in Muscles of cattle

Isospora belli

is a parasite that causes an intestinal disease known as Isosporiasis. This protozoan parasite is opportunistic in immune suppressed human hosts. It primarily exists in the epithelial cells of the small intestine, and develops in the cell cytoplasm. The distribution of this coccidian parasite is cosmopolitan, but is mainly found in tropical and subtropical areas of the world such as the Central and South America, India, and Africa. In the U.S., it is usually associated with HIV infection and institutional living.

Geographical Distribution: Widely distributed in the tropical and subtropical countries

Habitat: The epithelial cells/villi of the small intestine.

Morphology:

- Oocyst:

- Size: 20-30µm
- Shape: oval /flask shaped/
- Colour: transparent or occasionally pale yellow.
- Oocyst: immature when released in the faeces and contain granular mass and two sporocysts each with four sporozoites when the oocyst gets mature, smooth thin and colourless with two layers of cyst wall

Diagnosis:

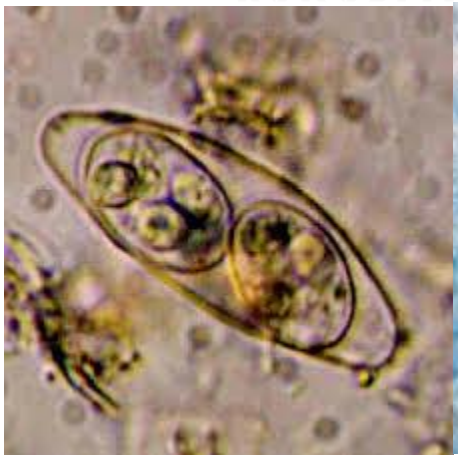
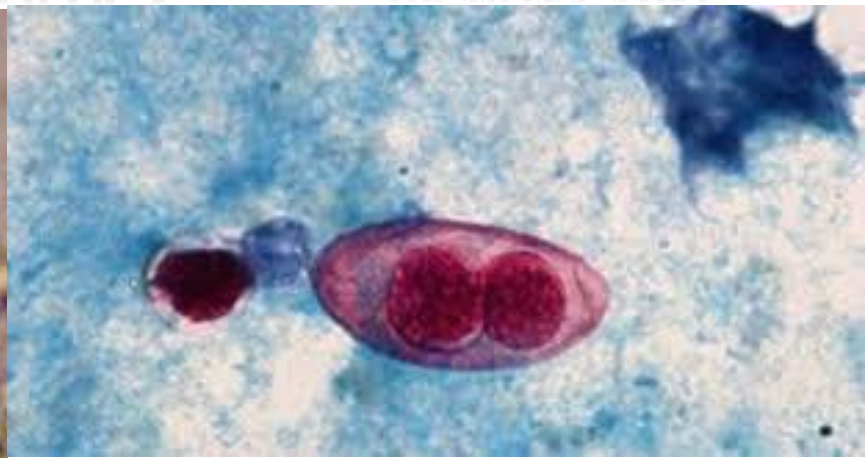
1. *isospora belli* is diagnosed by identification of the oocyst through examining a stool sample under a microscope.

2. The diagnostic stage is the immature oocyst that contains a spherical mass of protoplasm, the oocyst that is diagnosed in the stool sample is unsporulated, and contains only one sporoblast.

3. For stool diagnosis, direct smear, concentration smear, microscopic wet mount, or iodine stains of fecal smears are adequate. But for easy screening, acid-fast stains is recommended. If stool test is negative, and biopsies of the small intestine is performed

Life Cycle

I.belli complete its life cycle in a single host. The infective stage is the mature oocyst containing sporozoites and following ingestion, with contaminated food or drink the parasites excyst and the sporozoites enter epithelial cells of the small intestine where they develop and multiply by schizogony (merogony). Merozoite infect new cells. Some merozoite form male and female gametes. Fertilization→ Zygotes. Zygotes→Oocyst. Oocysts are excreted in the faeces. Feces containing oocyst contaminate water supply, food, etc. The oocysts are immature when passed with the faeces and maturation(sporogonic reproduction) is completed in the external environment. Sporozoites are produced in oocyst by sporogony.

***Cyclospora* spp.**

microscopic protozoan parasites that cause an intestinal illness called cyclosporiasis, with Cyclospora cayetanensis being the species that infects humans. Infection occurs by ingesting contaminated food or water, leading to symptoms like watery diarrhea, abdominal cramps, bloating, nausea, and fatigue. Diagnosis is typically made through a stool sample, and while it can resolve on its own, it may require treatment, especially in those with weakened immune systems.

Infection is caused by consuming food or water contaminated with the parasite's feces. Direct person-to-person transmission is unlikely. Outbreaks are often linked to imported fresh produce like raspberries, basil, cilantro, and snow peas

symptoms

Most common symptom: Watery diarrhea, which can last for weeks or even months and may come and go.

Other symptoms:

Loss of appetite

Weight loss

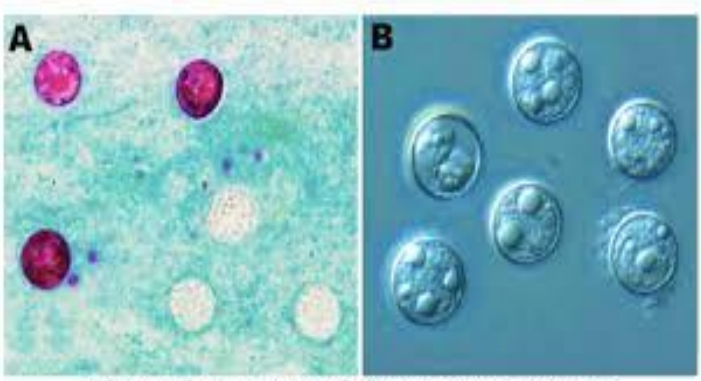
Stomach cramps or pain

Bloating and increased gas

Nausea

Fatigue

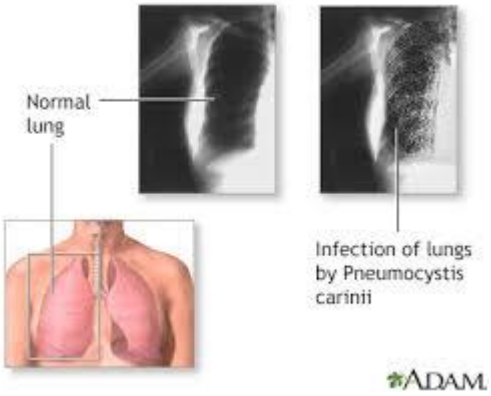
Flu-like symptoms



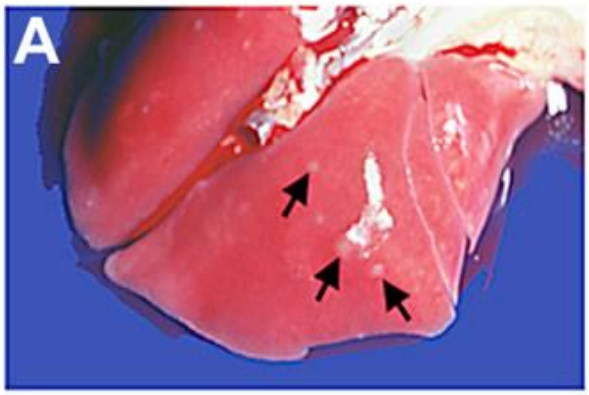
Microscopic appearance of *Cyclospora cayetanensis* oocysts
Source: Emerging Infectious Diseases • www.cdc.gov/eid • Vol. 17, No. 10, October 2011

pneumocystis carinii

Transmission occurs by inhalation, and infection is predominantly in the lungs. The presence of cysts in the alveoli induces an inflammatory response consisting primarily of plasma cells, histiocytes, lymph resulting in a frothy exudate that blocks oxygen exchange. (The presence of plasma cells has led to the name "plasma cell pneumonia.") The organism invade the lung tissue.*P.carinii* is distributed worldwide. It is estimated that 70%



of people have been



Clinical Findings

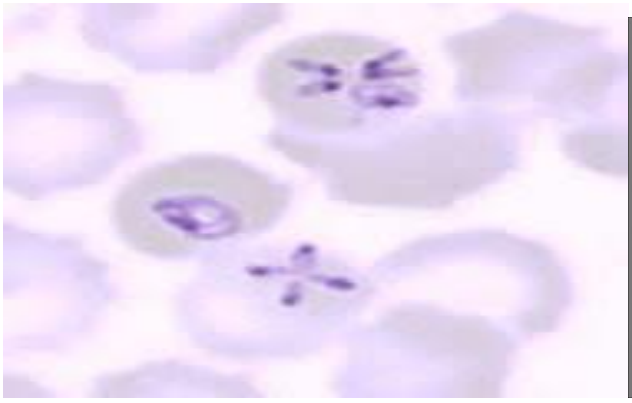
The sudden onset of fever, nonproductive cough, dyspnea, and tachypnea is typical of *Pneumocystis* pneumonia. In infants, the disease usually has a more gradual onset. The mortality rate of untreated Pneumocystis pneumonia approaches 100%.

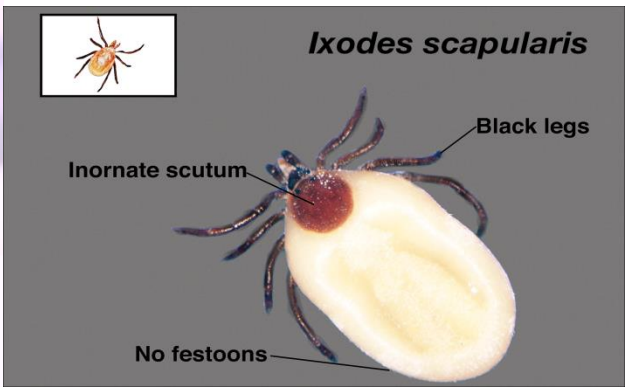
Laboratory Diagnosis

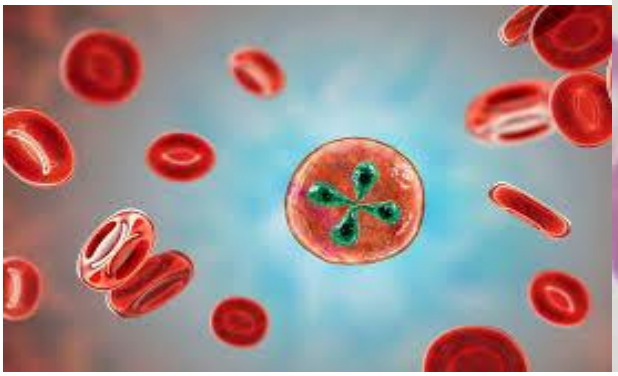
Diagnosis is made by finding the typical cysts by microscopic examination of lung tissue or fluids obtained by bronchoscopy, bronchial lavage, or open lung biopsy. Sputum is usuallyless suitable. The cysts can be visualized with methenamine-silver, Giemsa the cyste appear as a round masses of unstained cytoplasm containing 2-8 purple stained nuclei. Fluorescent-antibody staining is also commonly used for diagnosis. The organism stains poorly withGram stain. There is no serologic test, and the organism has not been grown in culture. PCRbased tests are being developed.

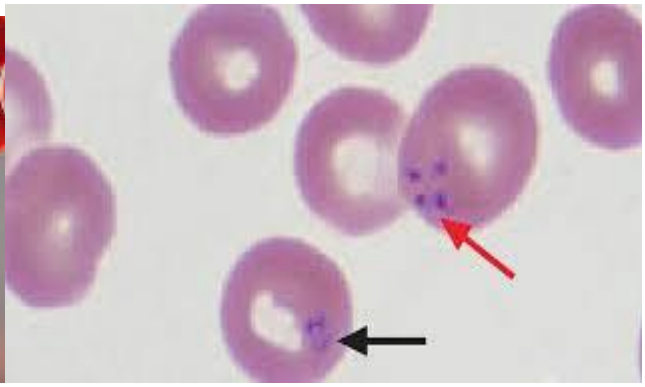
Babesia ssp.

It is transmitted in nature by ixodid, or hard -bodied, ticks.It can be differentiated from malaria by the absence of pigment within infected erythrocytes. The organisms infect the red cells, in which they appear some what poleomorphic ring like structures. Most resembles ring stage of *Plasmodia*. The small parasites appearing much like P.falciparium can be differentiated from malaria parasite by the absence of pigment in the infected erythrocytes. Human infection is diagnosed by identifying the intra-erythrocytic parasite in Giemsa – stained blood films. It can also be diagnosed by serologic test with the indirect immunoflorescent antibody test; which is the most useful in diagnosis.









-Final host: domestic animals, including cattle, sheep, goats, horses, swine, dogs, and cats, as well as numerous wild animals and man.

Intermediate host: Ixodes tick.

-Site of infection: inside the R.B. Cs, reproduce by binary fission inside the R.B. Cs, after transmission.

Diagnostic Technique: Blood examination.

Toxoplasma gondii

Like most of the Apicomplexa, *Toxoplasma* is an obligate intracellular parasite. Its life cycle includes two phases called the intestinal-enteric and Extraintestinal phases.

• The intestinal phase occurs in cats only (wild as well as domesticated cats) and produces "oocysts."

• The extraintestinal phase occurs in all infected animals produces "tachyzoites" and, eventually, "bradyzoites" or “tissue cysts.“

• The disease toxoplasmosis can be transmitted by ingestion of oocysts (in cat feces) or bradyzoites (in raw or undercooked meat).

Transmission

1. Accidental ingestion of oocysts passed in cat feces through contaminated soil or handling of cat litter

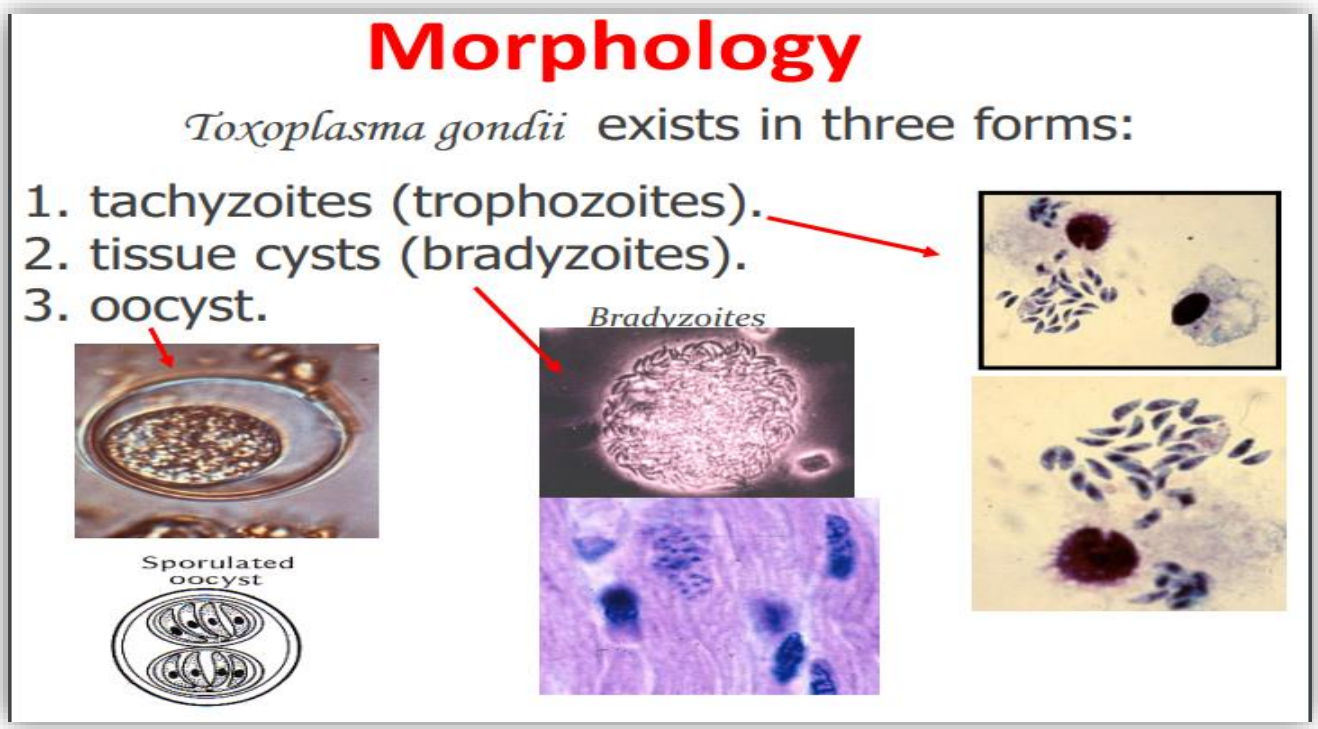
2. Ingestion of tissue cysts with raw or undercooked meat (lamb, pork, beef), drinking unpasterized milk, contaminated water, or unwashed fruits or vegetables

3. Transplacental transmission. Tachyzoites multiply within the placenta and spread to the fetus.

Two different kinds of host are needed in the sexual and asexual generations present in the life cycle of T.gondii.

• Sexual development or Gametogony occurs in the epithelial cells of the small intestine of cats(intraintestinal phase).

• The binary fission and endodyogeny (process of internal budding in which two daughter cells are formed within the body of the mother cell that dies when the progeny are released) of the asexual development takes place in various nucleated cells outside the intestine of many spp. Of mammals and birds.



Clinical Features

Most human infections are asymptomatic. Clinical toxoplasmosis may be congenital or acquired.

Congenital Toxoplasmosis

Congenital toxoplasmosis results when infection is transmitted Transplacental from mother to fetus. The risk of foetal infection rises with the progress of gestation, from 25 per cent when the mother acquires primary infection in the first trimester, to 65 per cent in the third trimester. Conversely the severity of fetal damage is highest when infection is transmitted in early pregnancy. Mothers with chronic or latent Toxoplasma infection acquired earlier do not ordinarily infect their babies, but in some women with latent or chronic infection may be to babies.

Acquired Toxoplasmosis

Infection acquired postnatally is mostly asymptomatic.The most common manifestation of acute acquired toxoplasmosis is lymphadenopathy, Fever, headache, myalgia and splenomegaly. The illness may resemble mild ‘flu’ or infectious mononucleosis lymphadenopathy may persist. In some there may be a typhuslike,with pneumonitis, myocarditis and meningoencephalitis, which may be fatal. Another type of toxoplasmosis is ocular. Approximately 35 per cent of cases of chorioretinitis . Toxoplasmosis primarily involving the central nervous system is usually fatal and often found in AIDS, severely in the immunodeficient, brain involvement is common

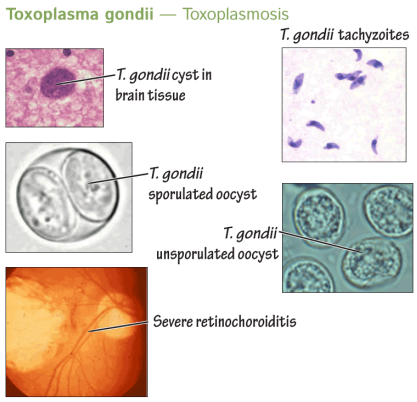
Diagnosis

Laboratory diagnosis may be made by microscopic demonstration of the parasite,by its isolation or by serological tests. Giemsa stained impression smears of lymphnodes, bone marrow, spleen or brain may occasionally show the trophozoites, which can be readily identified by their morphology. The most common method of laboratory diagnosis is by serology. Several serological tests are available. Indirect immunofluorescence, indirect haemagglutination, complement fixation ,ELISA and PCR. The standard test used now is ELISA, separately for IgM and IgG antibodies.The presence of IgM antibody in the absence of IgG denotes current infection,IgM antibody with high titre IgG suggests infection in the recent past; Negative IgM with positive IgG indicates past infection.This is subject to individual variation.In some cases IgM antibody may persist up to 18 months. Serial ELISA provides better information than a sinlge test.

Prevention:

• Cook meat thoroughly to kill the cysts.

• Avoid proximity to cats.



Class: Mastigophora
Tissue and blood flagellate (Haemoflagellates forms)

Phylum : Sarcomastia

Subphylum : Mastigophora

Class : Zoomastigophora

Order : Kinetoplastida

Family : Trypanosomatidae

Genus : *Leishmania* **Spieces:** (*L. donovani, L. tropica , L. braziliensis*)

Tissue and Blood flagellates

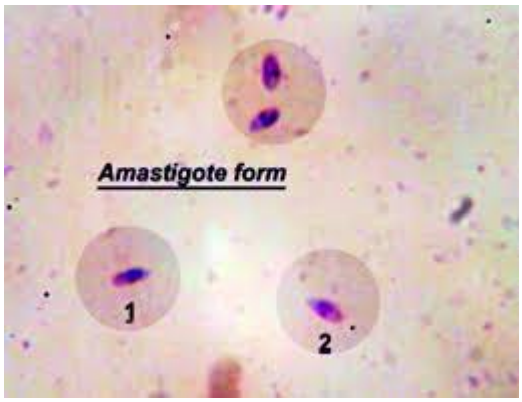
Blood and tissue flagellates of major clinical significance include members of genera Leishmania (*L. donovani, L. tropica and L. braziliensis*) and Trypanosoma *(Trypanosoma brucei gambiense, T. brucei rhodesiense and T. cruzi*). These two genera belong to the family Trypanosomatidae of the order Kinetoplastida. The members of this family are characterized of being parasitic and possess a single locomotory flagellum and a DNA-containing organelle called kinetoplast, which is located close to the flagellar basal body. Hemoflagellates may be present in any of four different stages (amastigotes, promastigotes, epimastigotes, and trypomastigotes) which are named according to their flagellation (Figure 2). The amastigote (Leishman-Donovan body) stage does not have a flagellum and is an obligate intracellular

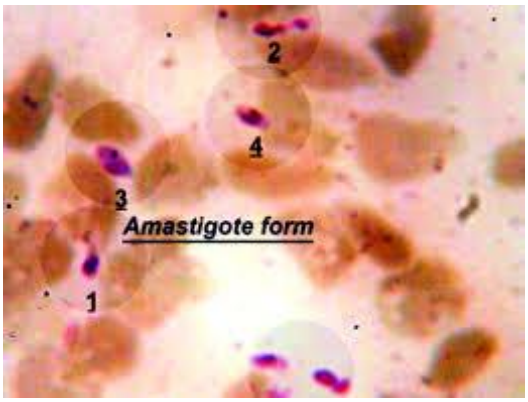
arasite. The promastigote (leptomonas) stage has a free flagellum but no undulating nembrane. The epimastigote (crithidia) has a free flagellum with undulating membrane. The rypomastigote (trypanosome) has a free flagellum and an undulating membrane that extends long with the length of the organism.

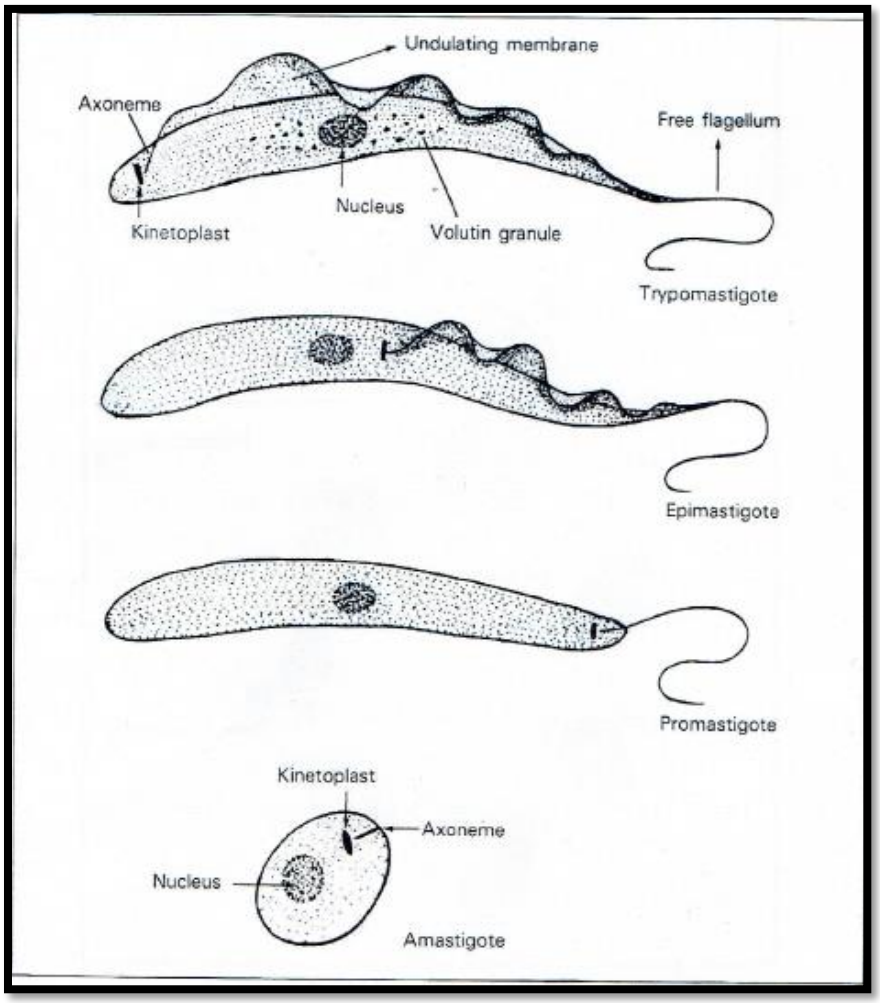
Hemoflagellates have 4 developmental forms:

) Round intracellular stage called amastigote

he amastigote is spherical or subspherical, it lives and reproduces by longitudinal binary ission in macrophage of skin, mucosa, lymph node and RES.In preparation stained with iemsa´s or Wright's stain the cytoplasm is pale blue and the large nucleus is red stain. In their ytoplasm in the median line of the cell, there is a deep red rod like structure called kinetoplast; delicate filament called axoneme extends from near the kinetoplast to the cell membrane.







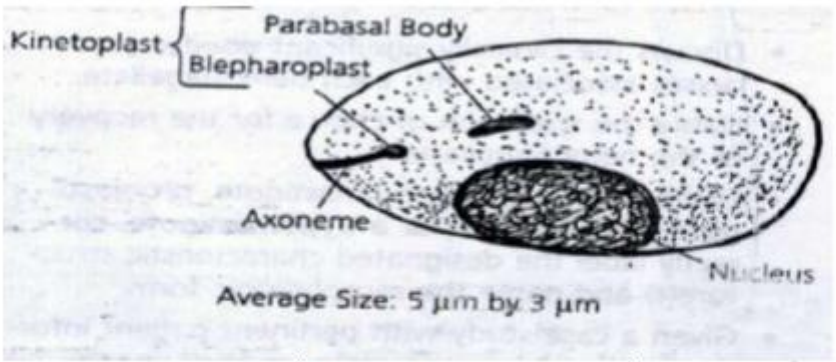


Figure 1: Amastigote

2) Flagellated extracellular stage called promastigote (leptomonas) (Fig. 2):It is is the basic of the hemoflagelate.It is pyriform without an undulating membrane with a kinetoplast at the anterior end. A free flagellum near the anterior end of the cell. There is no undulating membrane.

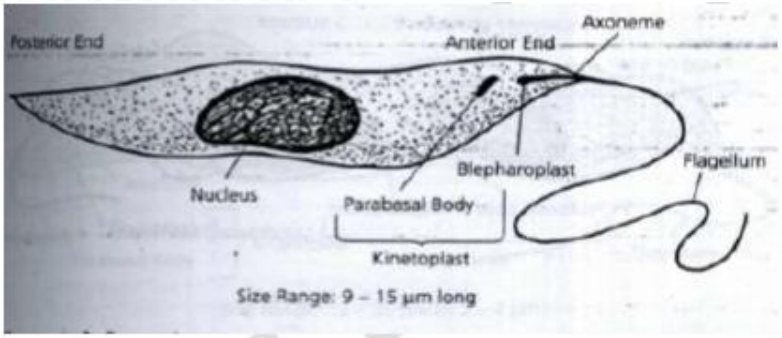


Figure 2: Promastigote

) **Epimastigote (crithidia) (Fig. 3):** Elongated extracellular stage with kinetoplast placed nore posteriorly close to and in front of the nucleus. The flagellum runs alongside the body as hort undulating membrane before emerging from the anterior end .

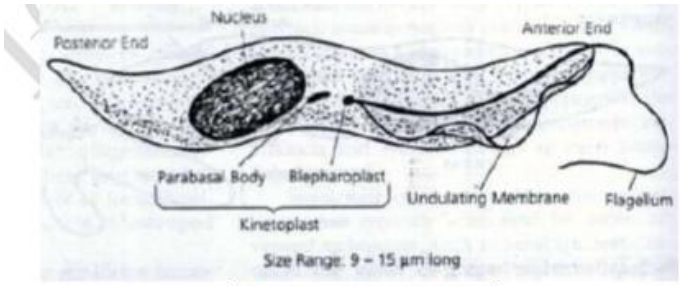
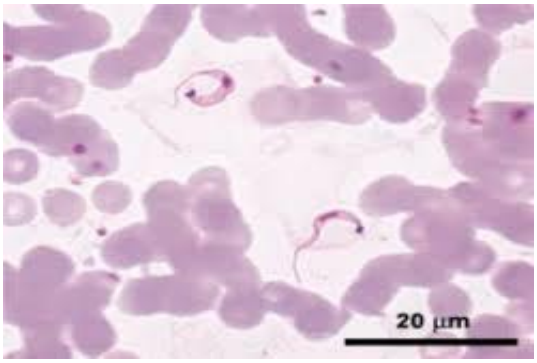


Figure 3 Epimastigote



4) Trypomastigote (trypanosoma) (Fig. 4): The cell is elongated; spindle shaped with a central nucleus and the kinetoplast posterior to the nucleus situated at the posterior end of the body .There is a long undulating membrane and a free flagellum.

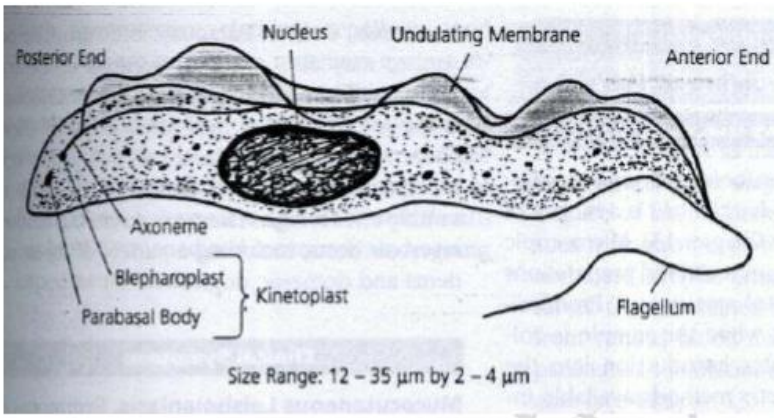
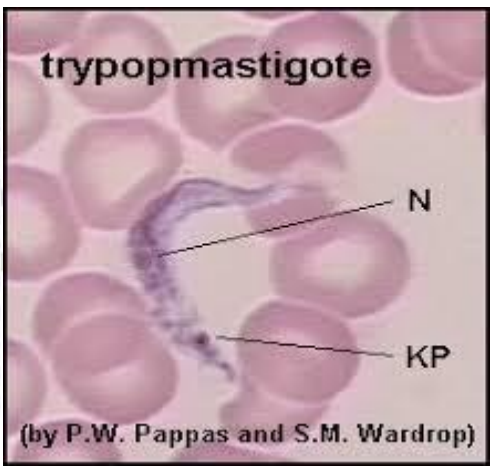


Figure 4 Trypomastigote.



Human leishmaniasis is not a single disease, but a group of diseases. These diseases are caused by infection with protozoan parasites from the Leishmania genus. Most species of Leishmania are zoonotic (affecting animals as well as humans). Many species of Leishmania are pathogenic for humans:

- ***Leishmania donovani*** causes visceral leishmaniasis (Kala-azar, black disease, dumdum fever)
- ***Leishmania tropica*** causes cutaneous leishmaniasis (oriental sore, Delhi ulcer, Aleppo, Delhi or Baghdad boil).
- ***Leishmania braziliensis*** are etiologic agents of mucocutaneous leishmaniasis (espundia, Uta).
- The geographical distribution of leishmaniasis is limited by the distribution of the sandfly, its susceptibility to cold climates, its tendency to take blood from humans or animals only and its capacity to support the internal development of specific species of Leishmania. Both visceral and cutaneous leishmaniasis are found in Iraq, especially in central and southern parts.

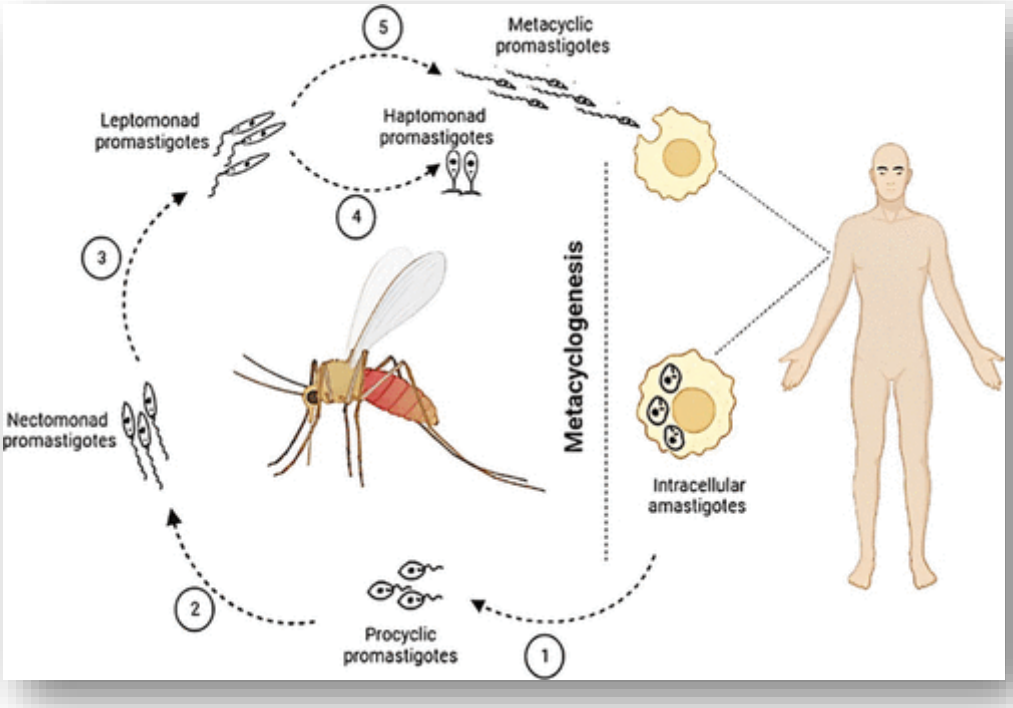
Morphology

The amastigote forms (Leishman-Donovan body) are small, round or oval bodies and measure 3 -5 µm by 1 - 3 µm . The large nucleus and small kinetoplast in the amastigote can be seen when stained with Giemsa or Wrights' stains and sometimes the short intracytoplasmic portion of the flagellum can also be seen. The promastigote stage (leptomonad) is a motile, slender organism (10-15 µm) with a single anterior flagellum and multiplies by longitudinal fission in the gut of the insect.

life cycle

The life cycle of ***Leishmania*** parasites has two distinct forms, amastigote and promastigote . The parasites are transmitted by the bite of several species of blood-sucking sandflies (*Phlebotomus*) which carry the promastigote in the anterior gut and pharynx. During feeding, infected female sandflies inject the flagellated stage (promastigote) into the skin of the vertebrate host. When the promastigotes are engulfed by the macrophages, they transform into intracellular stage (amastigote). The amastigote multiplies by binary fission within the parasitophorous vacuole of the macrophage until the infected cell ruptures. The librated amastigotes infect other cells and the infection spreads by movement of infected cells in the vascular system. The sandfly takes the amastigotes during the blood meal which

transform back into flagellate promastigotes and multiply in the gut until the anterior gut and pharynx are packed. The infected sandfly injects the promastigotes into a new host when it feeds. Dogs and rodents are common reservoir hosts.



life cycle of ***Leishmania*** **spp.**

Leishmania donovani

Obligate ,intracellular parasite of reticuloendothelial cells ,predominat in liver , spleen, bone marrow and lymph node of man and other vertebrate host were occurs in amastigote form. Morphologically ,found in two forms:

.1.Amastigote:

- Size: 5 by 3µm,Shape: oval to round.
- Nucleus: One, eccentric.
- Kinetoplast: Present,Consisting of dot-like blepharoplast, with small axoneme and prabasal body.
- Flagellum absent

.2.promastigote:

- Size: 9-15µm.
- Shape: long and slender.
- Nucleus: one, central.
- Kinetoplast: Anterior end of the organism, no undulating membrane.
- Flagellum: Single, anterior free flagellum.

-It causes a disease called Kala-azar or Dum-Dum fever or visceral Leishmaniasis or black fever.

- Intermediate host :The sand fly
- Definitive host: skin to liver, spleen, bone marrow of mammals
- Infection stage: metacyclic promastigotes

Laboratory Diagnosis

.1.Amastigotes in aspirates of spleen bone marrow, enlarged lymph nodes, and in peripheral blood monocytes.

.2.Promastigotes in culture media

.3.Testing serum for leishmanial antibodies.

.4.Formal get test; is a non-specific screening test for marked increases in IgG

Leishmania tropica

It causes a disease called Dry or urban cutaneous Leishmaniasis or oriental sore or Baghdad boil or Old world cutaneous Leishmaniasis or tropica sore. Morphological and life cycle resemble to Leishmania donovani has two form amastigote in man &

promastigote in sand fly

 Habitat: -Amastigotes: In the endothelial cells of cutaneous tissues, lymph nodes, ulcers.

 Promastigotes: In the gut of sandfly

 Definitive host: Man Intermediate host: Female sandfly

 Reservoir host: Dog, cats, mice, etc.

 Mode of Transmission

1. Inoculation by infected sandfly

2. Direct contact with the ulcer

3. Autoinfection

Leishmania braziliensis

Habitat:

Amastigote:- In the reticulo-endothelial cells of muco-cutaneous tissues of nose, mouth, lips, larynx. Promastigote:- In the gut of sand fly

Morphology: Has amastigote and promastigote stages.

Pathology:-Mucocutaneous leishmaniasis (espundia). Chronic ulceration of mucus membrane of the mouth nose, throat, etc. with destruction of bone and cartilage

Reservoir hosts ;are rodents and some domestic animals

Final hosts : human Infection stage: Promastigote

Mode Of Transmission:

1) By vector the disease is transmitted.

2) By contamination of the bite wound or contact when the insect is crushed during the act of feeding. After the infective blood meal, the sand fly become infective in 6-9 days.

3) Man to man is frequent.

4) Other modes of transmission by blood transfusion, contact inoculation and sexual intercourse.

.Taxonomic Classification of ***Trypanosoma*** spp

- Kingdom: Protozoa
- Phylum: Sarcomastigophora
- Subphylum : Mastigophora
- Class: Zoomastigophora
- Order: Kinetplastida
- Family: Trypanosomatidae
- Genus: ***Trypanosoma***
- Disease : typansomiasis

-1-West African Trypanosomiasis: " West African Sleeping Sickness" caused by *T. brucei gambiense.*

-2-East African Trypanosomiasis: " East African Sleeping Sickness" caused by *T. brucei rhodesiense.* **Chronic** form: caused by *T. brucei gambiense.* While **Acute** Form is caused by *T. brucei rhodesiense.*

•African Sleeping Sickness is the 3 rd important parasitic disease globally after Malaria & Schistosomiasis,West African Sleeping Sickness is in regions along riverside while East African Sleeping Sickness is in Forest regions (Savannas)

-3-American trypanosomiasis (Chagas' disease) is caused by *Trypanosoma cruzi*

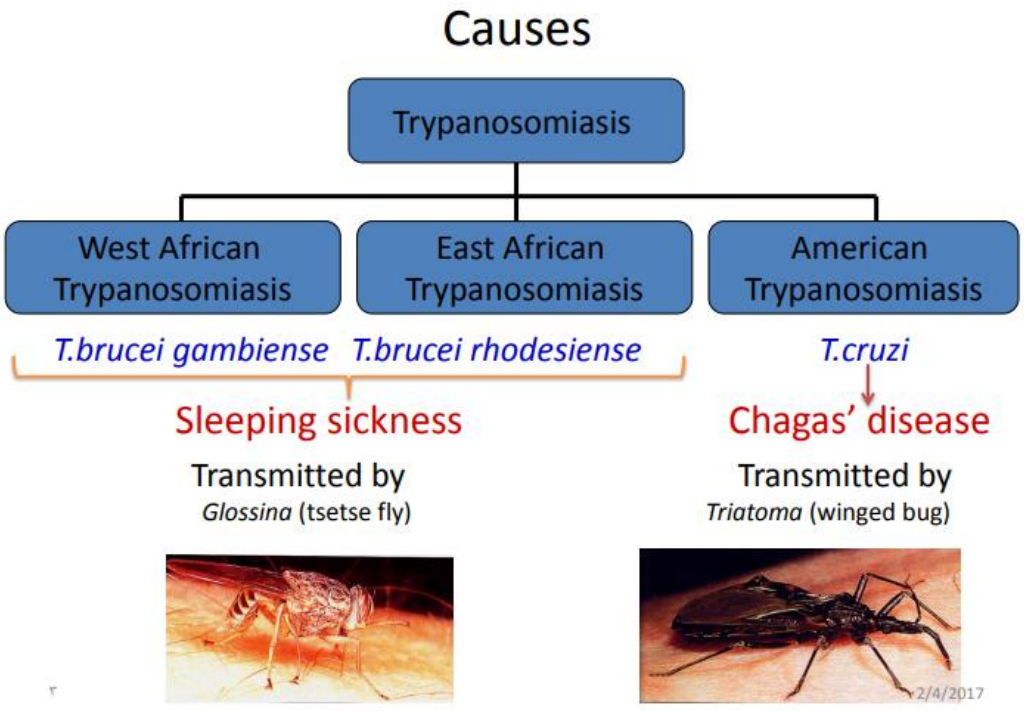
Important features

These species may have four stages in their life cycle:

Amastigote, Promastigote, Epimastigote, Trypomastigote

 In human trypanosomes of the African form, however, the amastigote and promastigote stages of development are absent.

 The typical structure of trypanosome is an elongated spindle-shaped body with 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.



***T. brucei* complex**

Exist into 2 inter forms:

Trypomastigote in Blood/ Lymph / tissue space of various organs & C.N.S is terminal & fatal

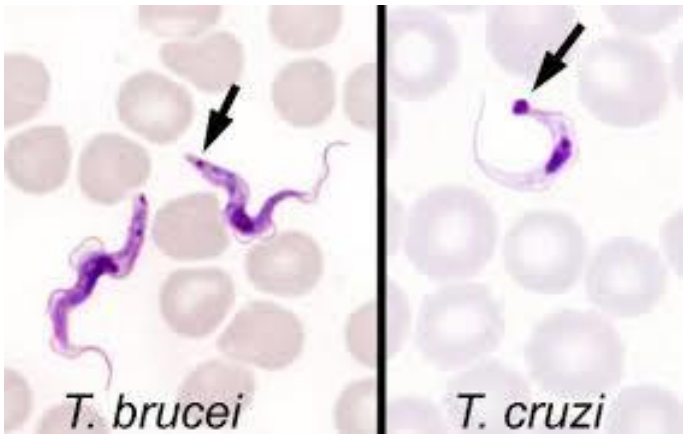
Epimastigote in salivary gland of vector & Culture media.

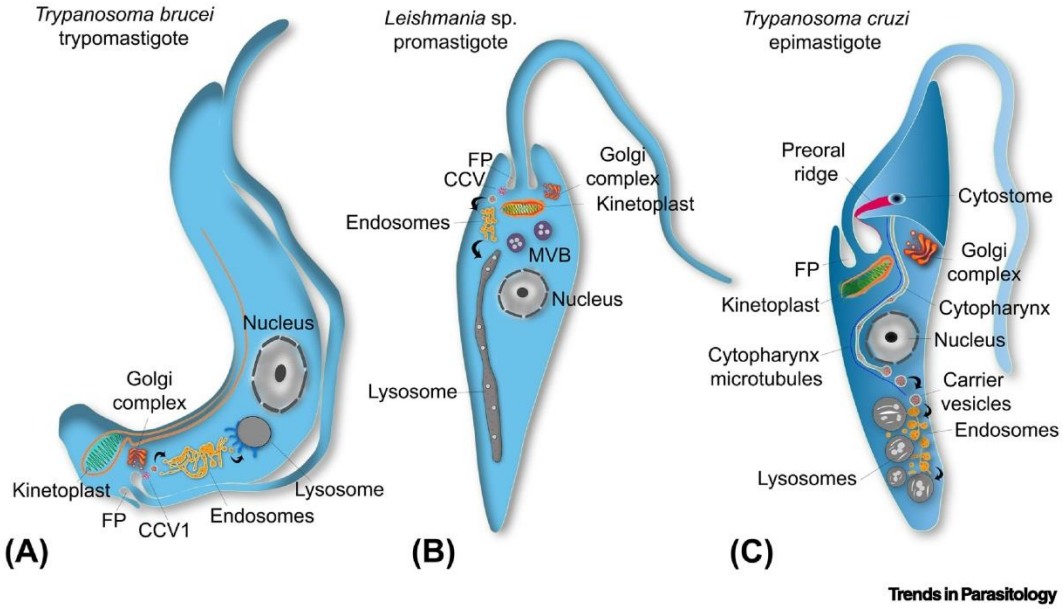
Trypomastigote (Polymorphic Trypanosomes,,,,Spindle shaped – Central nucleus – free flagellum – undulating membrane….. 3 forms

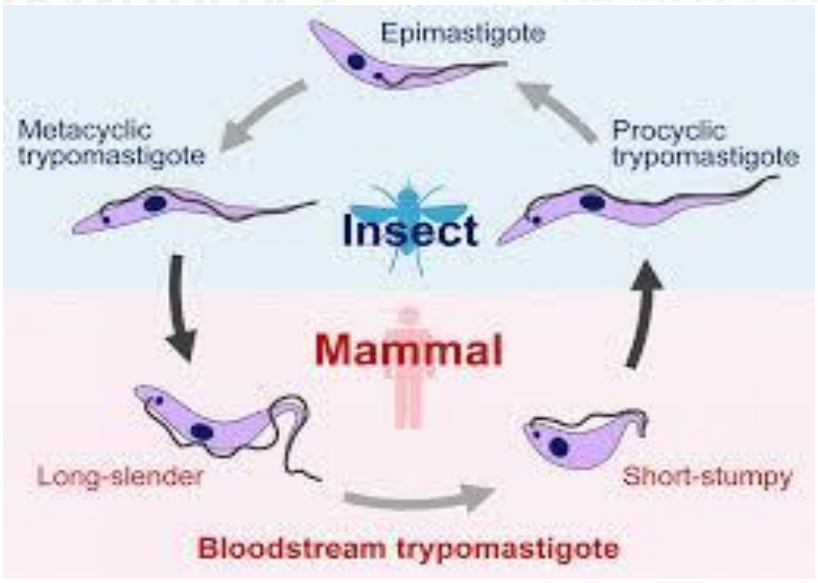
1- long Slender Form (30µ): active motile with free flagellum.

2- Short stumpy Form (15µ): sluggish without free flagellum.

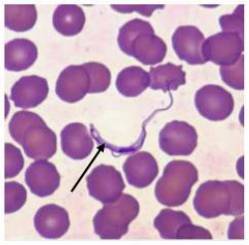
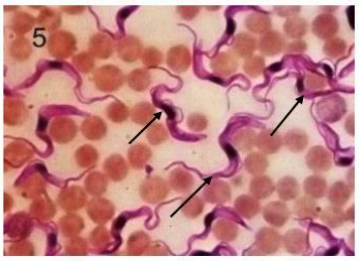
3- Intermediate Form (20µ): with a short free flagellum.







Trypanosoma brucei causing Sleeping Sickness

West Africa	East Africa
T.brucei gambiense	*T.brucei rhodesiense*
	
Less plentiful	More plentiful
Cannot live in lab animals	Can live in lab animals
	Nucleus is shifted posteriorly 
Reservoir host: goats, cattle & pigs	Reservoir host: wild game animals
Transmitted by: *G.palpalis*	Transmitted by: *G.morsitans*

G.palpalis

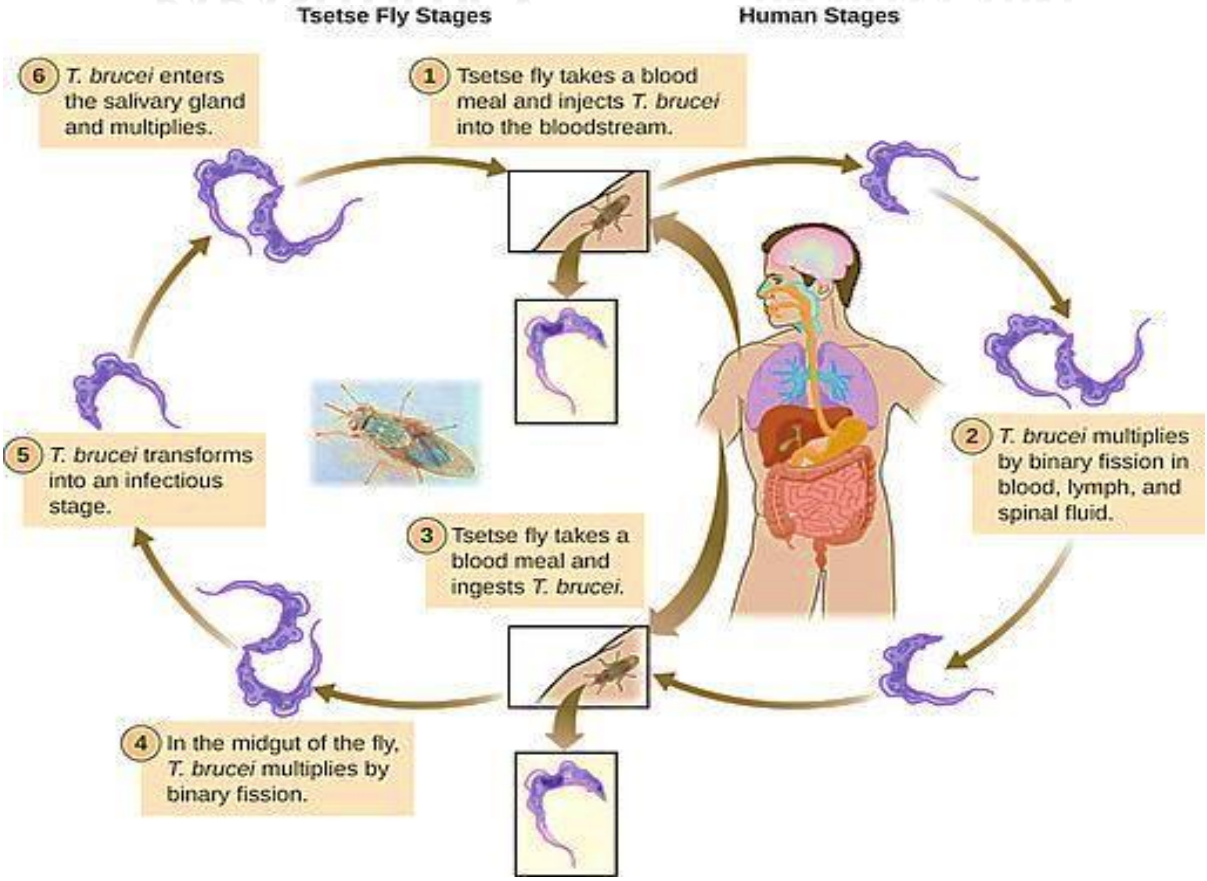
In West Africa

G.morsitans

In East Africa

Life cycle of *Trypanosoma brucei*

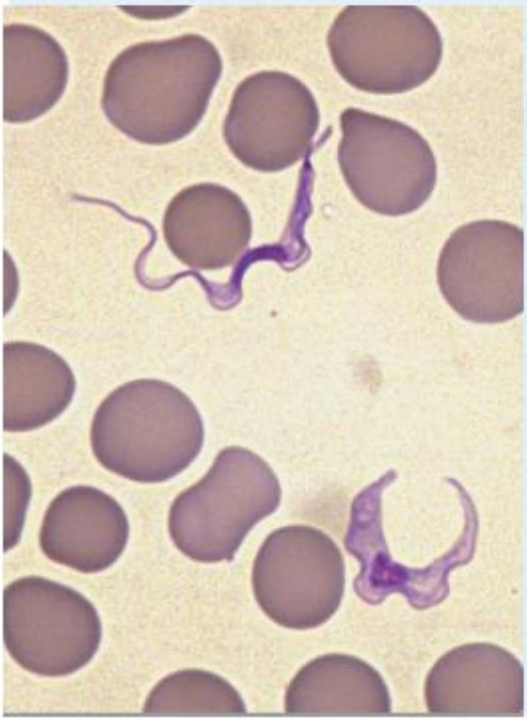
During a blood meal on the mammalian host, an infected tsetse fly (genus ***Glossina***) injects **metacyclic trypomastigotes (infective stage)** into skin tissue. The parasites enter the lymphatic system and pass into the bloodstream (anterior station development). Inside the host, they transform into bloodstream trypomastigotes , are carried to other sites throughout the body, reach other body fluids (e.g., lymph, spinal fluid), and continue the replication by binary fission . The entire life cycle of African trypanosomes is represented by extracellular stages. The tsetse fly becomes infected with bloodstream trypomastigotes when taking a blood meal on an infected mammalian host ,). In the fly's midgut, the parasites transform into procyclic trypomastigotes, multiply by binary fission , leave the midgut, and transform into epimastigotes . The epimastigotes reach the fly's salivary glands and continue multiplication by binary fission . The cycle in the fly takes approximately 3 weeks. Humans are the main reservoir for *Trypanosoma brucei gambiense*, but this species can also be found in animals. Wild game animals are the main reservoir of *T. b. rhodesiense.*

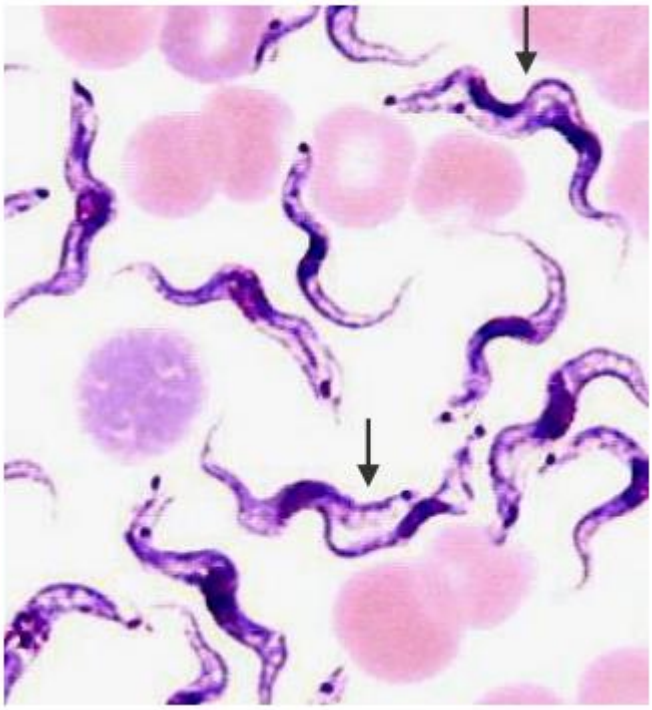


Laboratory

- Examination of thin and thick films
- In aspiration from lymph nodes and concentrated spinal fluid.

Polymorphic Trypanosomes in blood film





Prevention & control

• Control of breeding sites of tsetse flies and use of insecticides.

• Treatment of human cases to reduce transmission to flies.

• Avoiding insect bite by wearing protective clothing & use of screen, bed netting and insect repellants.

- Reduction of contact with ***Glossina*** (vector) through control measures instituted against them - traps - spraying w/ insecticide - skin repellents.

- Reduction of human infection by early diagnosis and prompt treatment

- Chemoprophylaxis in endemic areas [Pentamidine at 4-6 months intervals].

 No vaccine

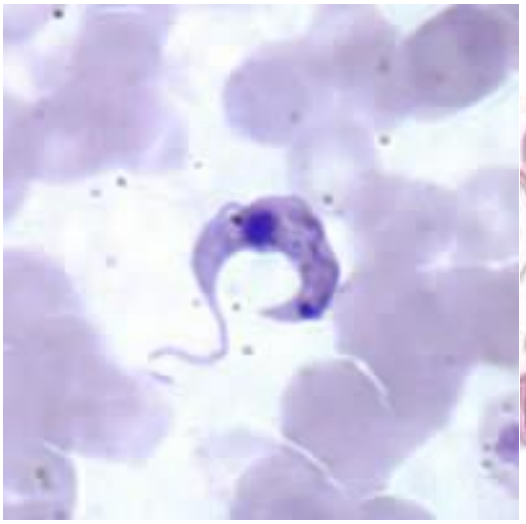
Trypanosoma cruzi ….causing Chagas' disease

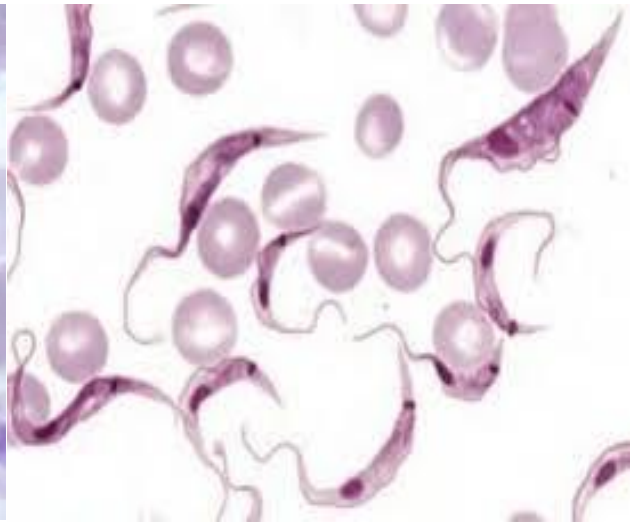
Morphology

Trypomastigote (Monomorphic) Slender shaped (20µ) – Central nucleus – C or U-shaped – Free flagellum 1/3 bodyLarge bulging peripheral kinetoplast

Amastigote:Obligatory intracellular – mainly in cardiac &Skeletal muscles – Brain meninges – Nerve ganglia – cells of GIT …. etc

Epimastigote (Vector only) Spindle shape– Kinetoplast anterior to central nucleus– Undulating membrane is short – terminal free flagellum





Triatoma dimidiata

Rhodnius prolixus

Panstrongylus megistus

Trypanosoma cruzi (American trypanosomiasis / Chaga's disease) **Chaga's disease** is caused by the protozoan **hemoflagellate**, ***Trypanosoma cruzi***. This disease is scattered irregularly in Central and South America, stretching from parts of Mexico to Argentina. It is estimated that 16-18 million people are infected by the parasite and 100 million persons are at risk of infection. About 50,000 people die each year from Chaga's disease. The

morphology depends mainly on the location of the parasite in the body of the host and it occurs in three different forms. The trypanosomal (trypomastigote) form, found in mammalian blood, is 15 to 20 μm long and morphologically similar to African trypanosomes. The crithidial (epimastigote) form is found in the insect intestine. The leishmanial (amastigote) form, found mainly in myocardium and brain and it is round or oval in shape, measures 2-4 μm and lacks a prominent flagellum.

Diagnosis

In endemic areas, cardiac dilation, megacolon and megaesophagus in individuals indicate current or former infection. Although this may make the diagnosis easier, the definitive diagnosis requires the demonstration of trypanosomes by microscopy or biological tests. Antibodies are often detectable by complement fixation or immunofluorescence and provide presumptive diagnosis.

Prevention & control

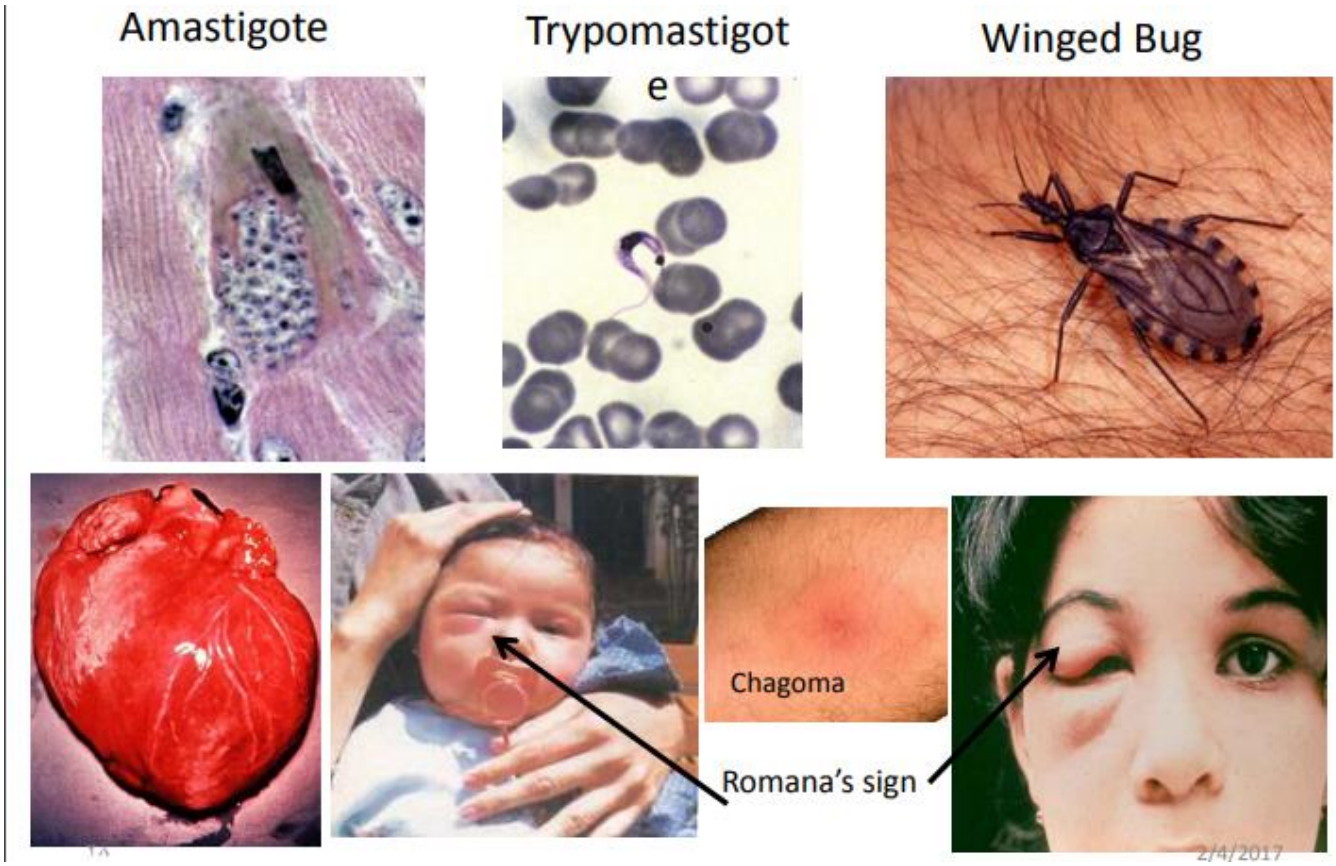
 Control and elimination of domestic and peridomestic vector bugs would help prevent the transmission of disease in endemic areas.

 Triatomine bugs are highly susceptible to chlorinated hydrocarbon insecticides, which form the major weapon for their control.

 Health education

 Sterilization of transfusion blood

The drug of choice in treatment of Chaga's disease is Nifurtimox (Nitrofurfurylidine derivative). It is important to mention that this drug should not be used during pregnancy, control measures are limited to those that reduce contact between the vectors and humans. Attempts to develop a vaccine have not been very successful.



Class: Coccidia (Sporozoa)

Genus: *Plasmodium* species

Malaria

Malaria is one of the most prevalent and weaken diseases afflicting humans. The disease has been reported in more than 90 countries, inhabited by 2.4 billion people. The causative agent is related to class Sporozoa, genus Plasmodium. Although more than 50 species of Plasmodium infect a wide variety of animals, only four species commonly cause malaria in humans. Recently, a fifth species, *Plasmodium knowlesi*, commonly a parasite of Old World monkeys, has been reported to infect humans in Southeast Asia.Regardless of the species responsible for the infection, the certain aspects of the disease such as the life cycle of the infective organism, the chemotherapy and epidemiology are similar, except some medically significant dissimilarities. The four species that cause the disease in the human are:

1. Plasmodium vivax. 2. Plasmodium falciparum. 3. Plasmodium malariae. 4. Plasmodium ovale.

- **Infection stage: sporoziote**
- **Final host: human**
- **Vector host: anopheles mosquitoes**

#General Characteristics

1. Intracellular obligate parasites.
2. Man is intermediate host.
3. Female Anopheles mosquitoes are the definitive hosts.
4. Those species which infect human being are *P.vivax, P.falciparum, P.malariae and P.ovale*
5. Has no animal reservoir host except *P.malariae* in which monkeys are the reservoir hosts
6. Infective stage to man from the insect vector is sporozoites and to the insect vector from man is gametocytes.

***Plasmodium* species**

Four Plasmodium species are responsible for human malaria and these are (*P. falciparum, P. vivax, P. ovale and P. malariae*) . There are an estimated 200 million global cases of malaria leading to mortality of more than one million people per year. *P. falciparum* (malignant tertian malaria) and *P. malariae* (quartan malaria) are the most common species of malarial parasite and are found in Asia and Africa. *P. vivax* (benign tertian malaria) predominates in Latin America, India and Pakistan, whereas, *P. ovale* (ovale tertian malaria) is almost exclusively found in Africa. Plasmodium falciparum is the most dangerous malarial parasite and the infected red blood cells develop surface knobs which cause them to stick to endothelial cells, this causes blockages and brain damage, often resulting in death, which can occur within a few days of infection.

Habitat:

The parasite enters the blood and carried to the parenchyma cells of liver, where they multiply enormously. This is called the pre-erythrocytic or tissue phase. By rupture of the infected cells they enter the RBCs, the erythrocytic phase (Schizogony) and reach all the organs of the blood via the circulating blood, producing parasitaemia.

Diagnosis:

By making (Blood films):

1. Thick blood films: are frequently necessary to detect the parasites. This type allows rapid examination of a large volume of blood in a small area on the slide. Staining of thick blood films is carried out according to (Giemsa technique). These films provide concentration of the parasites.

2. Thin blood films: it is also essential that thin films be prepared because the malarial species can be more readily identified on these, especially less experienced examiners. It is stain by Giemsa stain or Right stain

Plasmodium falciparum malaria is an acute life-threatening disease and rapid diagnosis is required. Diagnosis is based on:

1. Clinical picture: fever, chills, travel history and fever pattern. In Plasmodium falciparum, daily fever and rarely every 2 day fever while in other species, most of the time intermittent fever every 2 or 3 days.

2. Examination of blood: [thin, thick smear, buffy coat and antigen capture (ELISA and PCR).

The examination of Giemsa stained blood smears for characteristics of species depends mainly on:

- The shape and size of trophozoite, schizont and gametocyte.

- Percentage of red blood cells with parasites (very rarley more than 1% parasitemia in *P. vivax, P. ovale or P. malariae*).

 The presence of metabolic debris in red blood cells around parasites (called Schuffner's dots in *P. vivax* infection).

 Size of red blood cells which contain parasites (*P. ovale and P. vivax* infect younger red blood cells.

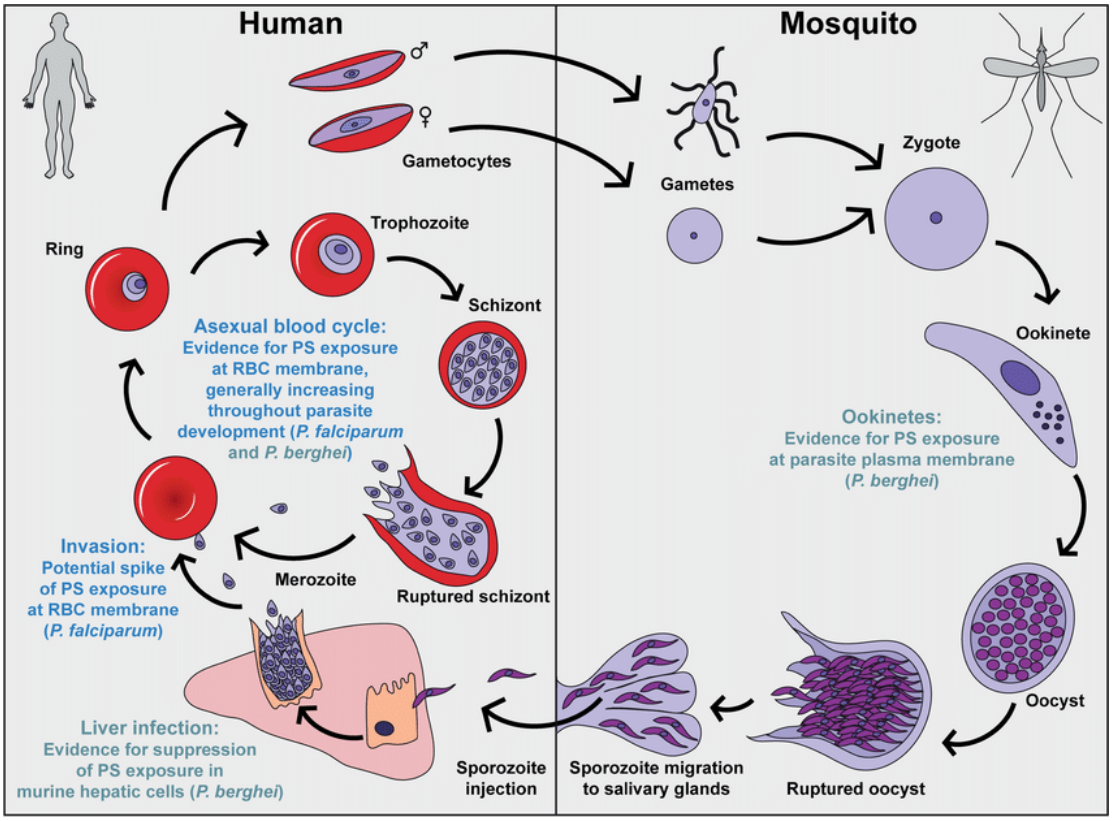
Causative Agents of Human Malaria

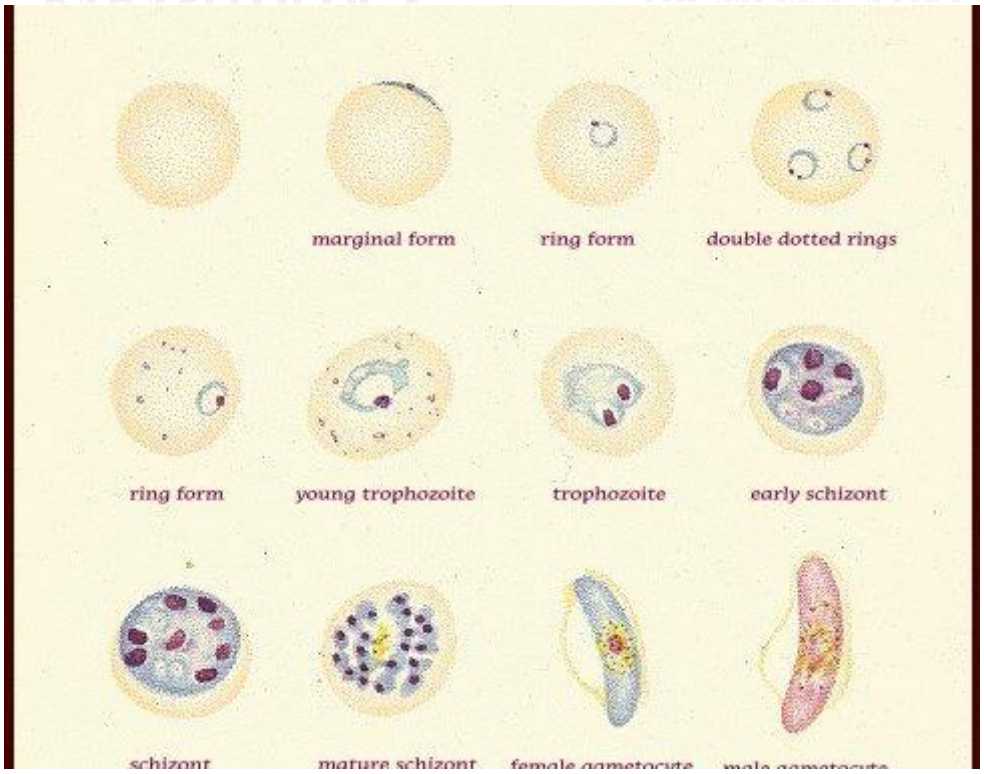
**Plamodium vivax*: Benign Tertian Malaria

**Plasmodium falciparum*: Malignant Tertian Malaria

**Plasmodium malariae*: Benign Quartan Malaria

**Plasmodium ovale*: Benign Tertian Malaria





Species / Stage	Falciparum	Vivax	Malariae	Oval
Ring Stage				
Trophozoite				
Schizont				
Gametocyte				

Causes of anemia in malaria of anemia in malaria

 Destruction of large number of RBCs by complement mediated and

autoimmune hemolysis.

 Suppression of erythropoiesis in the bone marrow

 Increased clearance of both parasitized and non-parasitized RBCs by the spleen.

 Failure of the host to recycle the iron bound in hemozoin pigment

 Antimalarial therapy in glucose-6-phosphate dehydrogenase (G6PD) deficient patients.

Clinical Features

Benign Malaria

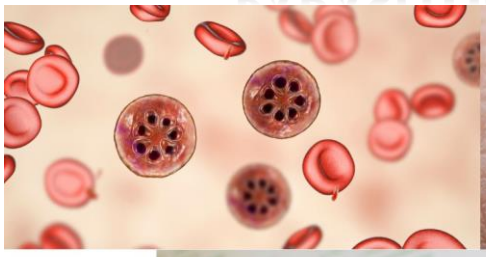
The typical picture of malaria consists of periodic bouts of fever with rigor, followed by anemia and splenomegaly. Severe headache, nausea, and vomiting are common.

 The febrile paroxysm comprises of 3 successive stages: cold stage, hot stage, and sweating stage.

 In the cold stage, that lasts for 15–60 minutes, the patient experiences intense cold and uncontrollable shivering.

 This is followed by the hot stage, lasting for 2–6 hours, when the patient feels intensely hot. The temperature mounts to 41°C or higher.

 After wards comes the sweating stage, when the patient is drenched in profuse sweat. The temperature drop




Malaria : - +


Malaria


Anopheles
Vector for malaria
Photo by James Gathany, CDC