الجامعة المستنصرية

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فرع علم الحيوان ZOOLOGY

المرحلة الثالثة

الطفيليات الابتدائية

Protozoan Parasites

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Parasitology

Introduction to Parasitology:

Definitions:-

1. ***Parasite:*** is an organism that obtains food and shelter from another organism and derives all benefits from this association
2. ***Parasitology*** :- the science or study of host-parasite relationships.
3. ***Medical parasitology:*** study of parasites which infect humans.
4. ***Vector****:* "carrier" of a parasite from one host to another. Often an insect.
5. ***Host:*** the partner providing food and/or protection. Some parasites require more than one host to complete their life cycle; or may not require a host during some stage(s).

Types of host:

a. ***definitive host****:* the host in which sexual maturity and reproduction takes place. Man is usually a definitive host.
b. ***intermediate host***: **harbors the larval stages of the parasite or an asexual cycle of development takes place. In some cases, larval development is completed in two different intermediate hosts, referred to as first and second intermediate hosts** (malaria).
c. ***Reservoir (carrier) host***: a host that makes the parasite available for the transmission to another host and is usually not affected

by the infection

d. ***Natural host***: a host that is naturally infected with certain species of parasite.
e. ***Accidental host:*** a host that is under normal circumstances not infected with the parasite.

Symbiosis: "living together," a close association between two organisms.

This relationship may be characterized as:-

a. ***mutualism:*** both organisms are benefited (bacteria in bowel)
b. ***commensalism:*** "eating at the same table"; one organism is benefited, the other is unaffected.
c. ***Parasitism*:** one organism is benefited at the expense of another (host).

DIFFERENT KINDS OF PARASITES:

- ***Ectoparasite:*** a parasitic organism that lives on the outer surface of its host, e.g. lice, ticks, mites etc.
- ***Endoparasites:*** parasites that live inside the body of their host, e.g. *Entamoeba histolytica.*
- ***Obligate Parasite*:** This parasite is completely dependent on the host during a segment or all of its life cycle, e.g. Plasmodium spp.
- ***Facultative parasite*:** an organism that exhibits both parasitic and non-parasitic modes of living and hence does not absolutely depend on the parasitic way of life, but is capable of adapting to it

if placed on a host. E.g. Naegleria fowleri

- ***Accidental parasite*:** when a parasite attacks an unnatural host and survives. E.g. *Hymenolepis diminuta* (rat tapeworm).
- ***Erratic parasite*:** is one that wanders in to an organ in which it is not usually found. E.g. *Entamoeba histolytica* in the liver or lung of humans.

Effects of the parasite on the host:

Parasitic damage to host:

1. trauma - damage to tissues, intestine, liver, eye.
2. lytic action - activity of enzymes elaborated by organism.
3. tissue response - localized inflammation, eosinophilia.
4. blood loss - heavy infection with hookworm may cause anemia.
5. secondary infections - weakened host susceptible to bacterial infection, etc.

SOURCES OF PARASITIC INFECTIONS

Parasitic infection may happen by many ways such as:

1. **Damp soil:** It contains worm's eggs or protozoan cysts.
2. **Contaminated water**: It contains protozoan cysts, helminthes egg, worm larvae and else.
3. **Vegetables, Food and Meat** :It is food that contaminated by worms eggs, encysted larvae of worms, or protozoan cysts, it may be the uncooked well meat or unwashed vegetables and fruits.

4. **Animals** :that lives with man such as dogs, cats, rats and else, also Arthropods which transport the parasitic infection mechanically or biologically.

Methods or mode of infection

1. Mouth
2. Skin (Touch, Penetration).
3. of contaminate dust
4. Placental.
5. Vaginal or Anal.
6. Blood.

Types of specimens which can be examined for diagnosis of parasites:

€ **Stool -€ Blood -€ Urine € - Sputum -€ Cerebrospinal fl uid (CSF) € Tissue and aspirates € - Genital specimens..**

Specimen Collection

1. Collect in a clean container - without urine or water (these may be damaging to trophozoites. The entire passage is desirable (this allows for a thorough macroscopic examination).
2. Minimum number of specimens - due to irregular shedding patterns of parasites, a series of three normally passed specimens is preferred.
3. Time frame of collection - Collect on alternate days. Never on same day.

4. Use of faxatives is not permitted - these can mask infection or damage organisms.
5. Exact date and time of collection - important, required information.
6. Proper performance of diagnostic testing is critical - no shortcuts! Follow the procedure, do not modify it for convenience.

Techniques of Stool Examination:

A. Gross examination.
B. Direct Wet Mounts (fresh or formalin preserved specimens).
C. Stained Wet Mounts.
D. Concentration Techniques.
E. Identification by Molecular Methods.
F. In Vitro Cultivation of Parasites - primarily used for blood & tissue protozoa. Can culture intestinal protozoa, but not generally done.
G. Animal inoculation.

Use of Other Specimens:

A. Anal Swabs / Scotch Tape Preparation for *Enterobius vermicularis* - must be collected in the morning prior to bathing or bowel movement.
B. Genital Specimens for *Trichomonas vaginalis* - vaginal, urethral, prostatic exudates are examined via wet mounts, looking for motile organisms.

C. Urine Specimens - *T. vaginalis.*

D. Sputum Specimens.

E. Aspirates and Biopsies.

F. Abscess aspirates - usually for extra-intestinal amoebiasis - wall of abscess is best area to examine.

Procedures for Detecting Blood Parasites:

A. Collection of Blood Samples

B. Examination of Blood Samples

1. Wet Mounts - screening for motile organisms (trypanosomes & filariae)
2. Permanent Stained Smears

a.Stains used -

1) Wright's - alcohol based.
2) Giemsa - water based, preferred stain, all things considered.

b.Thick Blood Films

1) Used in the identification of malaria parasites, trypanosomes, and microfilariae.

c.Thin Blood Films

1) Used in the identification of malaria parasites, trypanosomes, and microfilariae.

Concentration Techniques - Thick Blood smears

The Intestinal Protozoa

Introduction

The Phylum Protozoa is classified into four major subdivisions according to the methods of locomotion and reproduction.

a. ***The amoebae (Superclass Sarcodina, Class Rhizopodea:*** move by means of pseudopodia and reproduce exclusively by asexual binary division.

b. ***The flagellates (Superclass Mastigophora, Class Zoomasitgophorea)*** typically move by long, whiplike flagella and reproduce by binary fission.

c. ***The ciliates (Subphylum Ciliophora, Class Ciliata)*** are propelled by rows of cilia that beat with a synchronized wavelike motion.

d. ***The sporozoans (Subphylum Sporozoa)***lack specialized organelles of motility but have a unique type of life cycle, alternating between sexual and asexual reproductive cycles (alternation of generations).

Number of species - there are about 45,000 protozoan species; around 8000 are parasitic, and around 25 species are important to humans.

General Features

– The single protozoal cell performs all functions.

– Most of the protozoa are completely nonpathogenic¬ but few may cause major diseases such as malaria, leishmaniasis, and sleeping sickness.

– Protozoa exhibit wide range of size (1–150 µm)

Structures

1. Cytoplasm

It has 2 portions:

A. Ectoplasm: Outer homogeneous part that serves as¬ the organ for locomotion and for engulfment of foodby producing pseudopodia.

B. **Endoplasm**: The inner granular portion of cytoplasm that contains **Chromatoid Body** :

Extranuclear chromatin material is called chromatoid body (e.g., as found in Entamoeba histolytica cyst).

- **Karyosome** :It is a DNA containing body, situated peripherally or centrally within the nucleus and found in intestinal amoeba, e.g. E. histolytica, E. coli.
- **Kinetoplast** :Non-nuclear DNA present in addition to nucleus is called kinetoplast. It is seen in trypanosomes. Flagellum orginates near the kinetoplast. Point of origin of agellum is called as basal body
- nucleus is called endoplasm

2. Nucleus

The nucleus is usually single but may be double or multiple; some **species** having as many as hundred nuclei in a single cell. The nucleus contains one or more nucleoli or a central¬ karyosome.

Reproduction

Reproduction can be: **Asexual reproduction**¬

Sexual reproduction.¬ Reproduction usually occurs asexually in protozoans; however, sexual reproduction occurs in cillates and sporozoas.

Life Cycle

– **Single Host**: Protozoa like intestinal agellates and¬ cillates require only 1 host, within which they multiply asexually .

– **Second host**: In some protozoa like Plasmodium,¬ asexual method of reproduction occurs in one host (man) and sexual method of reproduction in another host (mosquito).

1. ***trophozoite*** - the motile vegetative stage; multiplies via binary fission; colonizes host.
2. ***cyst*** - the inactive, non-motile, infective stage; survives the environment due to the presence of a cyst wall.

diagnostic features

a. **size** - helpful in identifying organisms; must have calibrated objectives on the microscope in order to measure accurately.

b. ***type of motility*** - directional or non-directional; sluggish or fast.

c. ***cytoplasmic inclusions*** - chromatoid bars (coalesced RNA); red blood cells; food vacuoles containing bacteria, yeast, etc.

d. ***appearance of cytoplasm*** - smooth & clean or vacuolated.

e. ***nuclear structure*** - important in the identification of organisms and species differentiation.

f. ***endosome*** - also called the "karyosome," this is a mass of chromatin within nucleus. The size, shape, and location of this structure are helpful in identification of organisms.

g. ***chromatin -*** nuclear DNA

h. ***chromatoid*** **body or "bar"** - coalesced RNA within the cytoplasm in the cyst stage. This is not always present, but when it is, its size and shape are helpful in determining species identification.

1. Class: Sarcodina ***- The Amoebae***

A. Life cycle -

a) The definitive host ingests the infective cyst stage from fecal contamination in environment.

b) The cyst passes into the small intestine & excystation occurs with transformation to the trophozoite stage.

c) Trophozoites in the large intestine colonize the host by multiplying asexually via binary fission. They can remain in the lumen or invade the wall of the intestine (pathogenic species only) & multiply, from here they can be transported via the circulation to other organs (liver, lungs, etc.).

d) Cysts and trophozoites are passed in the feces of the infected host.

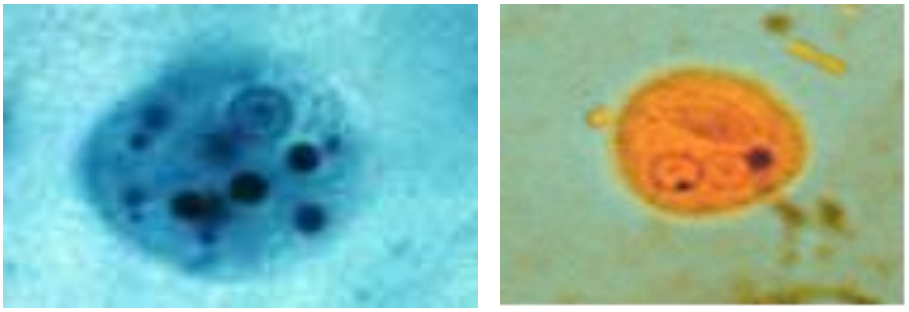
e) ***Infective stage -*** the mature cyst.

f) ***Diagnostic stage -*** the trophozoite or cyst in stool or tissue specimens.

2. ***Genus Entamoeba***

contains the most important of the amoebae causing disease in humans.

Entamoeba histolytica



Entamoeba histolytica trophozoite with ingested red blood cells

Morphological features

Trophozoite

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*.

The cyst ranges between 10 and 30 microns in diameter and contains four nuclei when mature. Cigar-shaped chromatoid bars may be present in some cysts.

a. ***Epidemiology:-*** Occurs worldwide; the highest incidence and prevalence is found in areas with poor sanitation where as many as 80% of a population may be infected. Highest in children >5 years of age; more prevalent in males than in females; common in mental hospitals, prisons, orphanages.

b. ***Pathology and Clinical Manifestations:-*** the 1- The typical manifestation of intestinal amoebiasis is amoebic dysentery The stools are large, foul-smelling, and brownish black, often with bloodstreaked mucus intermingled with feces. The RBCs in stools are clumped and reddish-brown in color,fever vomiting; can become extra-intestinal (liver, lungs, etc.; can be fatal.

c. Small superficial ulcers involving only the mucosa. • Round or oval-shaped with ragged and undermined margin and flask-shaped in cross-section.

Formation of tumor-like masses of granulation tissue (amoeboma).

Mode of transmission: Man acquires infection by swallowing food and water contaminated with cysts. As the cyst wall is resistant to action of gastric juice, the cysts pass through the stomach undamaged and enter the small intestine.

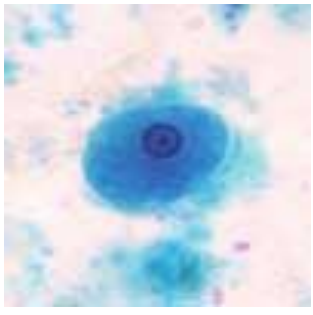
Life Cycle

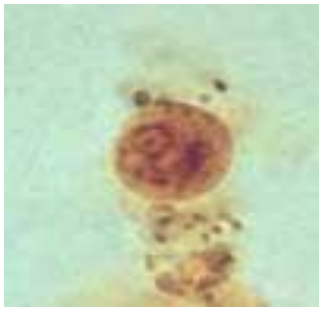
Excystation: When the cyst reaches caecum or lower part of the ileum, due to the alkaline medium, the cyst wall is damaged by trypsin, leading to excystation. The cytoplasm gets detached from the cyst wall and amoeboid movements appear causing a tear in the

cyst wall, through which quadrinucleate amoeba is liberated. This stage is called the metacyst.

Metacystic trophozoites: The nuclei in the metacyst immediately undergo division to form 8 nuclei, each of which gets surrounded by its own cytoplasm to become 8 small amoebulae or metacystic trophozoites. If exystation takes place in the small intestine, the metacystic trophozoites do not colonize there, but are carried to the caecum. The optimal habitat for the metacystic trophozoite is the submucosal tissue of caecum and colon, where they lodge in the glandular crypts and grow by binary fission. Some develop into precystic forms and cysts, which are passed in feces to repeat the cycle. The entire life cycle is, thus completed in one host. In most of the cases, E. histolytica remains as a commensal in the large intestine without causing any ill effects

3. <u>Entamoeba hartmanni</u>





Entamoeba hartmanni rophozoitet

Entamoeba hartmanni cyst (iodine stain)

Formerly called the "**small race"** of *Entamoeba histolytica*.

Technologists must be able to differentiate this organism from *E. histolytica* because *E. hartmanni* is **non-pathogenic**.

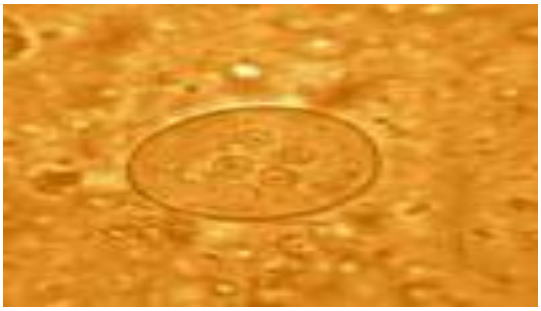
Morphology & Laboratory Identification –

It is much smaller than *E. histolytica*, the trophozoite measuring 4–12 µm and cyst 5–10 µm in size.

Trophozoites: do not ingest red cells and their motility is less vigorous.

The cyst :resembles that of *Endolimax nana*

4. Entamoeba coli



E. coli cyst (iodine)

Entamoeba coli trophozoite

It is worldwide in distribution and a nonpathogenic commensal intestinal amoeba.

Morphology :-

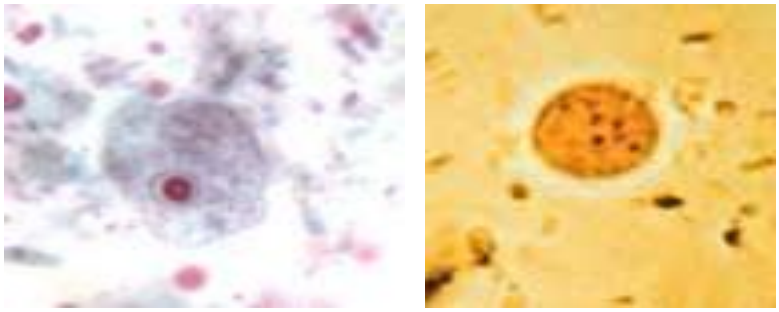
Trophozoites It is larger than *E. histolytica* about 20–50 µm with sluggish motility and contains ingested bacteria but no red cells. The nucleus is clearly visible in unstained fi lms and has a large eccentric karyosome and thick nuclear membrane lined with coarse granules of chromatin.

cysts :Cysts are large, 10–30 µm in size, with a prominent glycogen mass in the early stage. The chromatoid bodies are splinterlike and irregular. The mature cyst has 8 nuclei. The life

cycle is the same as in *E.histolytica* except that it remains a luminal commensal without tissue invasion and is nonpathogenic.

	E. histolytica	***E. coli***	***E. hartmanni***
Trophozoite			
	Size (µm) 12–60	Size (µm) 20–50	Size (µm) 4–12
Motility	Active	Sluggish	Active
Pseudopodia	Finger-shaped, rapidly extruded	Short, blunt slowly extruded	Finger-shaped, rapidly extruded
Cytoplasm	Clearly defi ned into ectoplasmand endoplasm	Diff erentiation not distinct	Clearly defi ned into ectoplasm and endoplasm
Inclusions	RBCs present, no bacteria	Bacteria and other particles, no RBCs	Bacteria and other particles, no RBCs
Nucleus	Not clearly visible in unstained fi lms	Visible in unstained fi lms	Not visible in unstained fi lms
Karyosome	Small, central	Large, eccentric	Small, eccentric
Nuclear Membrance	Delicate, with fine chromatin dots	Thick, with coarse chromatin granules	Coarse chromatin granules
Cyst			
Size (µm)	10–15	10–30	5–10
Nucleiin mature cyst	4	8	4
Glycogen mass	Seen in uninucleate, but not in quadi nucleate stage	Seen up to quadrinucleate stage	Seen in uninucleate, but not in quadi nucleate stage
chromidial	1–4 with crounded ends	Splinter like with angular ends	Many with irregular shape

5. ***Endolimax nana:-***



Endolimax nana trophozoite E. nana cyst (iodine)

This common commensal amoeba is widely distributed. It lives in the human intestine.

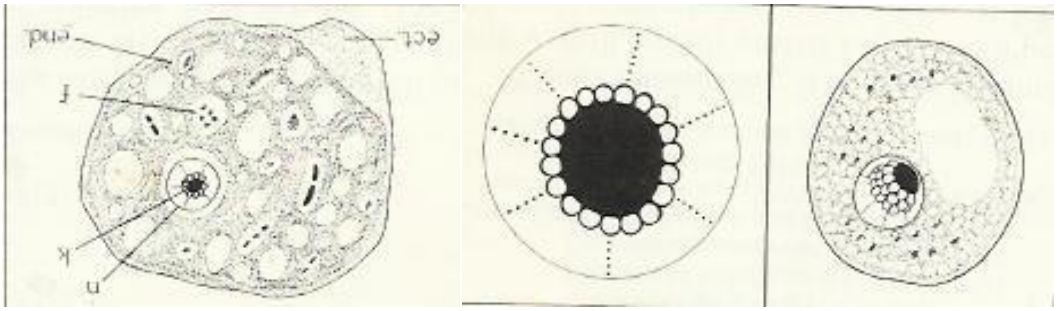
The trophozoite: is small (nana: small), less than 10 µm in size with a sluggish motility. The nucleus has clear karyosome connected to nuclear membrane by one or none coarse strands.

The cyst :is small, oval, and quadrinucleate with glyocgen mass and chromidial bars, which are inconspicuous or absent. .

Pathogenicity: - It is non-pathogenic

Diagnosis: by demonstrate troph. or cyst stage in stool specimen.

6. ***Iodamoeba butschlii:-***



Iodamoeba butschlii trophozoite I. Butschlii cyst

It is cosmopolitan amoeba, but it is less common than *E. coli*, it is non pathogenic commensal living in the lumen of large intestine. This amoeba has trophozoite and cyst stages

Pathogenicity: - none.

Morphology:

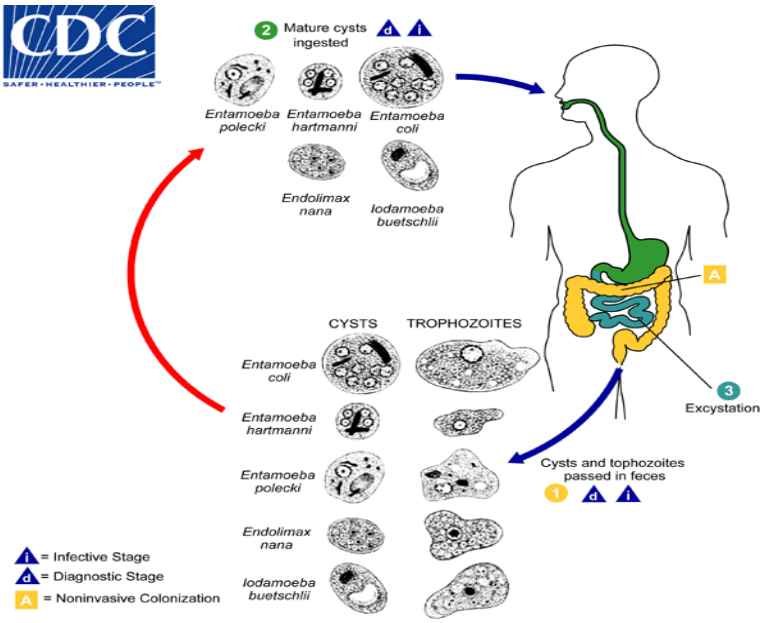
Trophozoite:- small, sluggish, ectoplasm no well differentiated from endoplasm. The nucleus spherical, characterized by having central large karyosome surrounded by achromatic granules. There are 1 or 2 glycogen mass in cytoplasm.

The cyst:- is irregular in shape with single nucleus only, cyst contain well distinct glycogen mass which stain golden brown with iodine solution so this

amoeba called Iodamoeba. The nucleus has large compact eccentric karyosome and around this karyosome there are achromatic granules.

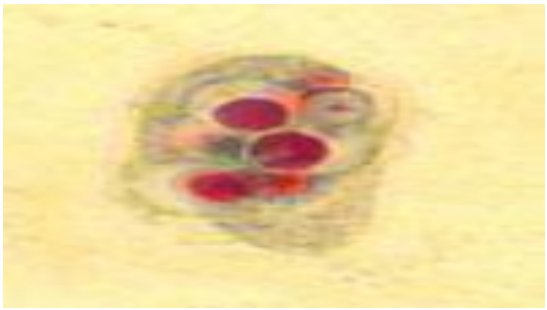
Life Cycles:-

Entamoeba coli, E. hartmanni, E. polecki, Endolimax nana, **and** ***Iodamoeba buetschlii***



Entamoeba coli, *E. hartmanni*, , *Endolimax nana*, and *Iodamoeba buetschlii* are generally considered nonpathogenic and reside in the large intestine of the human host. Both cysts and trophozoites of these species are passed in stool and considered diagnostic. Cysts are typically found in formed stool, whereas trophozoites are typically found in diarrheal stool. Colonization of the nonpathogenic amebae occurs after ingestion of mature cysts in fecally-contaminated food, water, or fomites. Excystation occurs in the small intestine and trophozoites are released, which migrate to the large intestine. The trophozoites multiply by binary fission and produce cysts, and both stages are passed in the feces.

7. ***Entamoeba gingivalis***

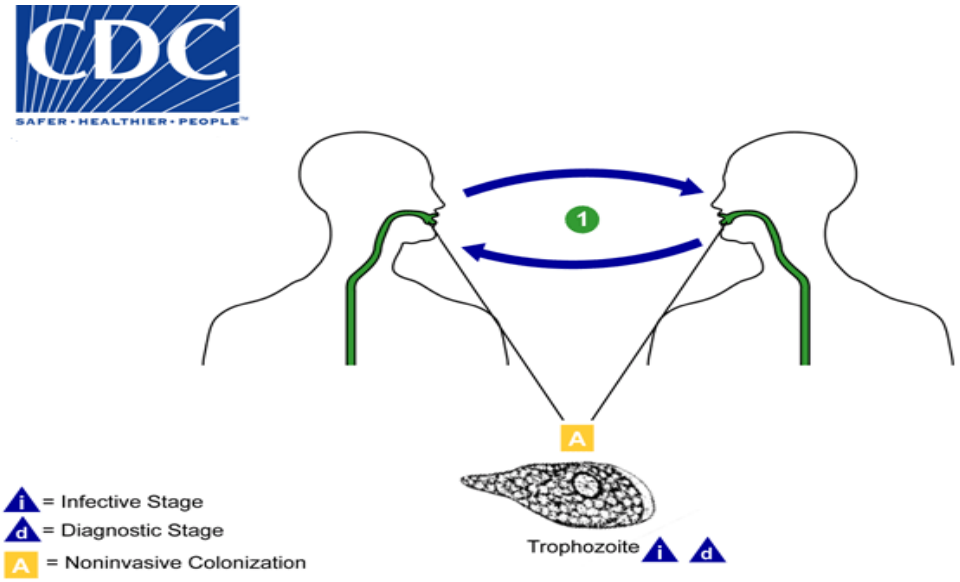


Entamoeba gingivalis trophozoite

Infective site: in the mouth; the organism thrives in diseased gums, but is not considered a causal agent or pathogen. If swallowed, it is destroyed in stomach.

Transmission: contact with fomites (drinking glasses, eating utensils, etc.; kissing**.**

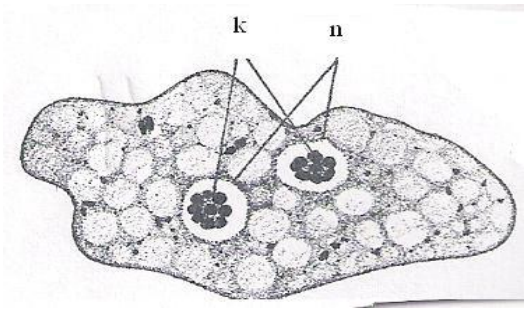
Morphology: The trophozoite is about 10–20 µm, actively motile with multiple pseudopodia. The cytoplasm contains food vacuoles with ingested bacteria, leuocytes, and epithelial cells. Nucleus is round with central karyosome lined by coarse chromatin granules. The amoeba lives in gingival tissues and is abundant in unhygienic mouths. It is a commensal and is not considered to cause any disease.



There is no known cyst stage for *Entamoeba gingivalis*; trophozoites live in the oral cavity of humans, residing in the gingival pockets near the base of the teeth A. They are not considered pathogenic, and feed on bacteria and other debris. Trophozoites are transmitted person-to-person orally by kissing or fomites (such as eating utensils).

8. ***Dientamoeba fragilis:***

D. fragilis was previously considered as an amoeba but has now been reclassifi ed as an amoeboflagellate, based on electron microscopic study and antigenic similarity to Trichomonas



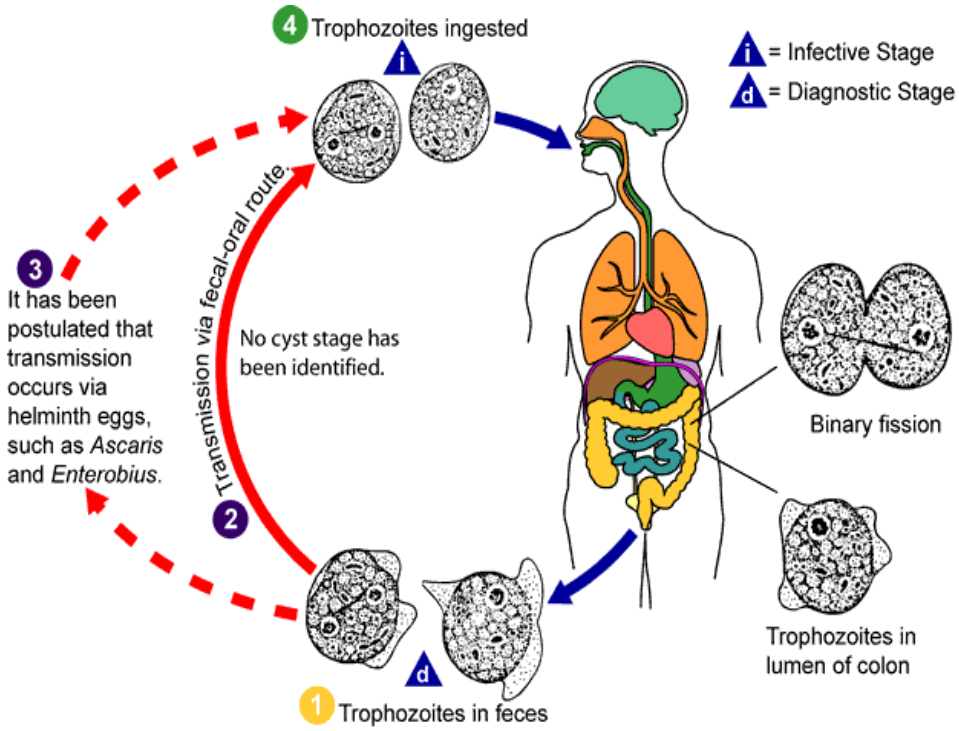
it has only trophozoite stage but no cyst stage. The name

Dientamoeba fragilis is derived from the binucleate nature of trophozoite (Dientamoeba) and the fragmented appearance (fragilis) of its nuclear chromatin. It lives in colonic mucosal crypts, feeding on bacteria. It does not invade tissues, but may rarely ingest RBCs.

Trophozoite :The trophozoite is 7–12 µm in diameter. It is motile with broad hyaline leaflike pseudopodia. They have 1–4 nuclei; the binucleate form being the most common. The nuclear chromatin is present as 3–5 granules in the center, with no peripheral chromatin on the nuclear membrane**.** it is transmitted from persontoperson by the fecaloral route or by the eggs of Enterobius vermicularis and other nematodes, which may serve as a vector**.**

Pathogenicity: Formerly believed to be nonpathogenic, it has now been associated with a variety of symptoms like intermittent diarrhea, abdominal pain, fl atulence, anorexia, nausea, malaise, and fatigue

Diagnosis: by demonstrate trophozoites in formed or diarrhic stool.



Free – Living Pathogenic amoebae:-

Some free-living amoebae (facultative) can cause serious even fatal disease if reach human body. These amoebae has found to have the ability to live in the tissues of mammals.

It can invade human nervous system, usually result in the death of the patient.

Naegleria fowleri (brain – eating amoeba):-

N. fowleri causes the disease **primary amoebic meningi encephalitis** (PAM), a brain infection that leads to destruction of brain tissue.. It is a heat-loving (thermophilic) amoeba that thrives in warm water at low oxygen tension and is commonly found in warm freshwater (e.g. lakes, rivers, and springs) and soil. It is world wide in distribution.

Morphology

N .fowleri occurs in 3 forms:

1. Cyst
2. trophozoite: a-Amoeboid trophozoite form b- Flagellate trophozoite form

Amoeboid form

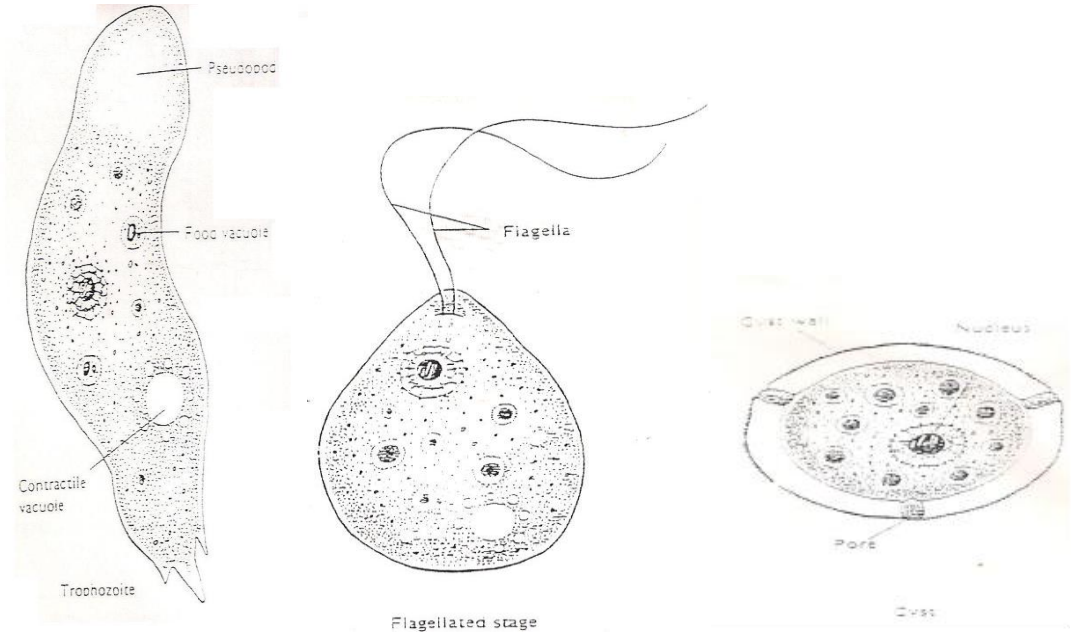
The amoeboid form is about 10–20 µm, showing rounded pseudopodia (lobopodia), a spherical nucleus with big endosome, and pulsating vacuoles. With electron microscopy, vacuole appear to be densely granular in contrast to highly vacuolated body of amoeba and are **called as amoebostomes**. They are used for engulfi ng RBCs and WBCs and vary in number, depending on the species. € Amoeboid form is the feeding, growing, and replicating form of the parasite, seen on the surface of vegetation, mud, and water. € It is the invasive stage of the parasite and the infective form of the parasite.

Flagellate form:

The biff agellate form occurs when trophozoites are transferred to distilled water. This transformation of trophozoites to bifl agellate pear-shaped form occurs within a minute. The flagellate can revert to the amoeboid form, hence *N. fowleri* is classifi ed as amoebofl agellate.

Cyst Stage :

Trophozoites encyst due to unfavorable conditions like food deprivation, dessication, cold temperature, etc. The cyst is 7–10 µm in diameter and has a smooth double wall. € They are the resting or the dormant form and can resist unfavorable conditions, such as drying and chlorine up to 50 ppm.

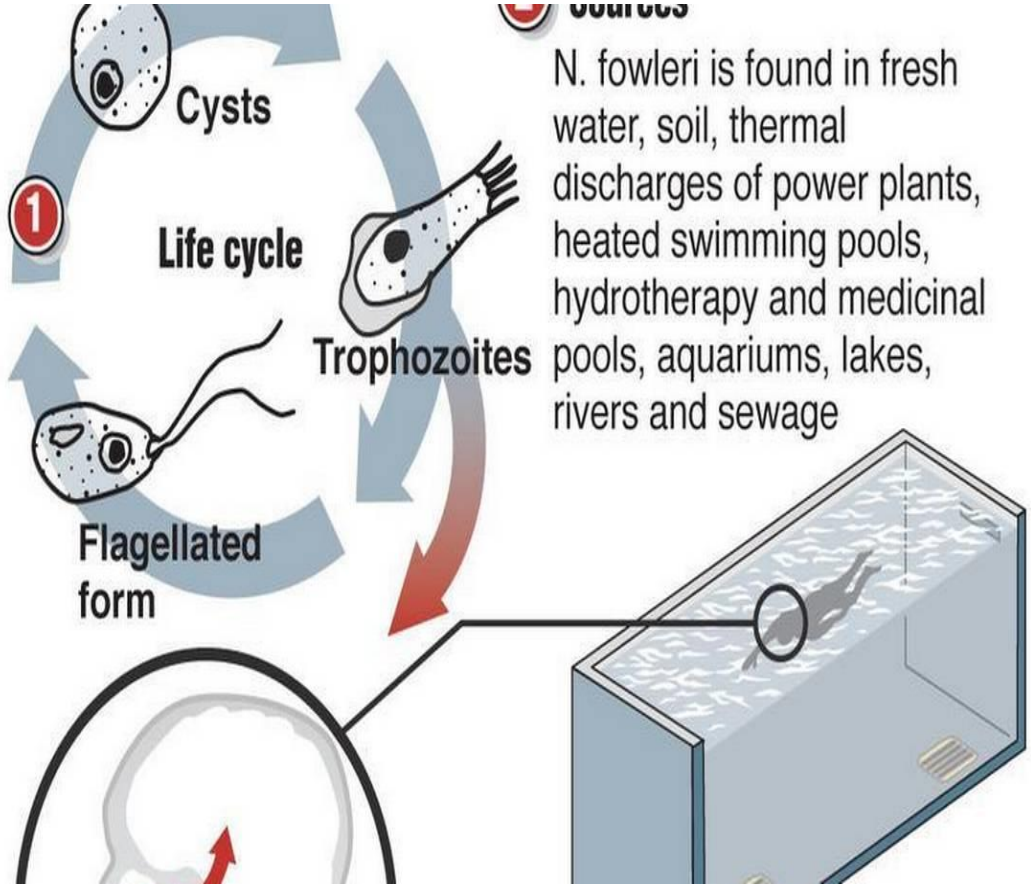


The trophozoites can withstand moderate heat (45°C), but die at chlorine levels of 2 ppm and salinity of 0.7%. € Cysts and fl agellate forms of *N. fowleri* have never been found in tissues of cerebrospinal fl uid (CSF).

Life Cycle

Typically, infection occurs when people go swimming or diving in warm freshwater river or ponds and poorlymaintained swimming pools or nasal irrigation using contaminated tap water . The life cycle of N. fowleri is completed in the external environment. The

amoeboid form of trophozoite multiplies by binary fi ssion. Under unfavorable conditions, it forms a cyst and which undergoes excystation in favorable conditions. Flagellate form of trophozoite helps in the spread of *N. fowleri* to new water bodies. Since the amoeboid form is the invasive stage, hence, the flagellate forms revert to amoeboid forms to become infective to man.



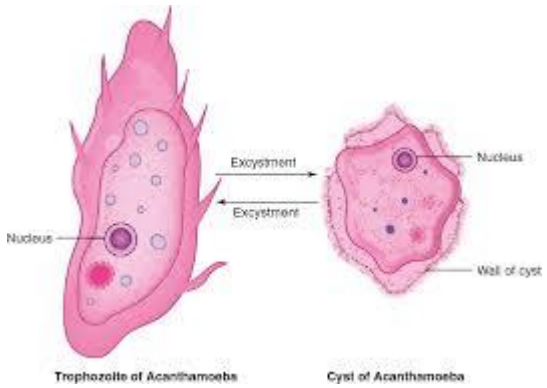
Pathogensis:-

Early in the infection the patient complains of upper respiratory tract symptoms e.g. raning nose, sore throat, fever and headache. Within 2 – 3 days the headache becomes more sever and there may be vomiting, stiff neck, mental confusion, coma as a result of intracranial pressure. Death usually with 10 days of the onset of symptoms.

Diagnosis:-

1. Direct demonstration of motile amoebae in unstained CSF or nasal discharge.
2. Stained smear of CSF.
3. Stained section of brain tissue at autopsy.
4. Culture on non-nutrient agar medium coated with E. coli bacteria.
5. Serological tests.

Acanthamoeba spp.



it is found in **moist soil and in the air and water**. Acanthamoeba exists as active trophozoite form and a resistant cystic form.

The trophozoite

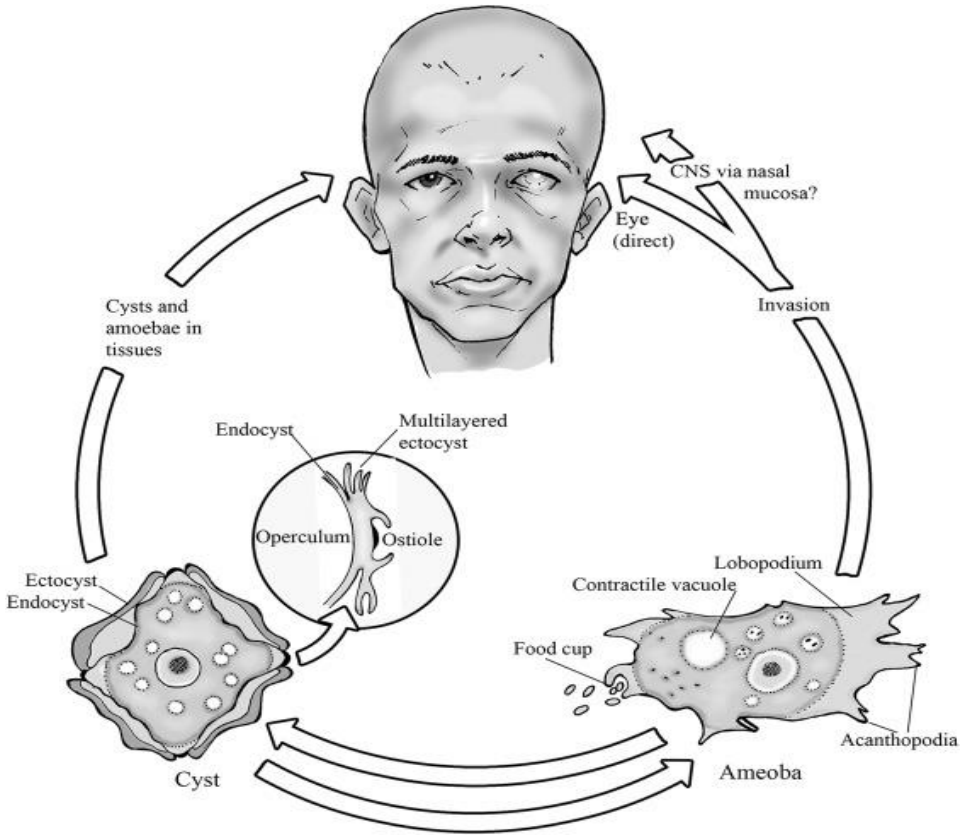
is large, 20–50 µm in size and characterized by spine-like pseudopodia (acanthopodia). It diff ers from Naegleria in not having a fl agellate stage and in forming cysts in tissues.

Cyst

The polygonal double-walled cysts are highly resistant. The cysts are present in all types of environment, all over the world.

Life Cycle:-

Both trophozoites and cysts are infective. Human beings acquire by inhalation of cyst or trophozoite , ingestion of cysts, or through traumatized skin or eyes. After inhalation of aerosol or dust containing trophozoites and cysts, the trophozoites reach the lungs and from there, they invade the central nervous system through the blood stream, producing granulomatous amoebic encephalitis (GAE).



Symptoms: slow onset (10 or more days). Presents as chronic, granulomatous lesions in brain. In eye lesions, the infection resembles a herpes virus infection.

Diagnosis:

1. By finding amoebae in wet mounte (10% KOH) of corneal ulcer scraping or in stained smear.

2. By isolation of amoebae form contact lenses or washing solutions.

Superclass Mastigophora

the flagellates; members of this group can inhabit mouth, bloodstream, tissues, gastrointestinal, or urogenital tracts.

Morphological Characteristics:-

1. Flagellum(ae) - characteristic organelle of locomotion. It is an extension of ectoplasm and resembles a tail; moves with a whip-like motion.
2. Axostyle - a supporting mechanism; a rod-shaped structure; not all Genera exhibit these.
3. Undulating membrane - a protoplasmic membrane with a flagellar rim extending out like a fin along the outer edge of the body of some flagellates. Moves in a wave-like motion.
4. Costa - a thin, firm rod-like structure running along the base of the undulating membrane in some flagellates.
5. Cytosome - a rudimentary mouth; also referred to as a gullet.

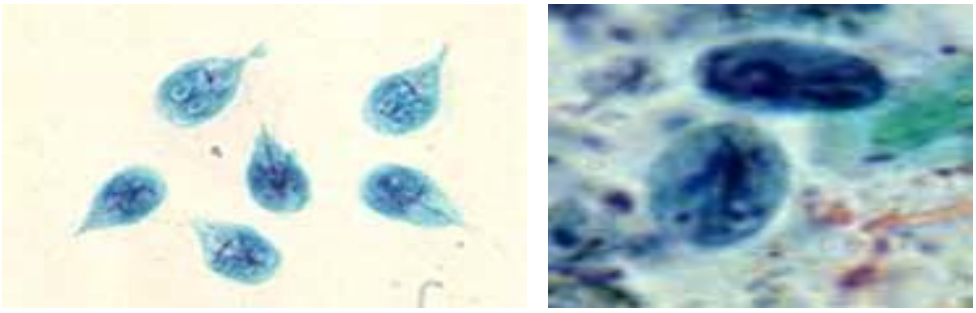
Note: Identification of a flagellate is based upon:-

1. Size
2. Shape
3. Motility
4. Number and morphology of nuclei
5. Number and location of flagellae

6. Location in the body of the host.

Intestinal flagellates:

Giardia lamblia: probably the first described protozoan pathogen of humans.



Giardia lamblia trophozoites G. lamblia cysts

Most common protozoan parasite in the U.S.A.

***Morphology*:** very distinctive. Dorsal-ventrally flattened, and Bi-laterally symmetrical.

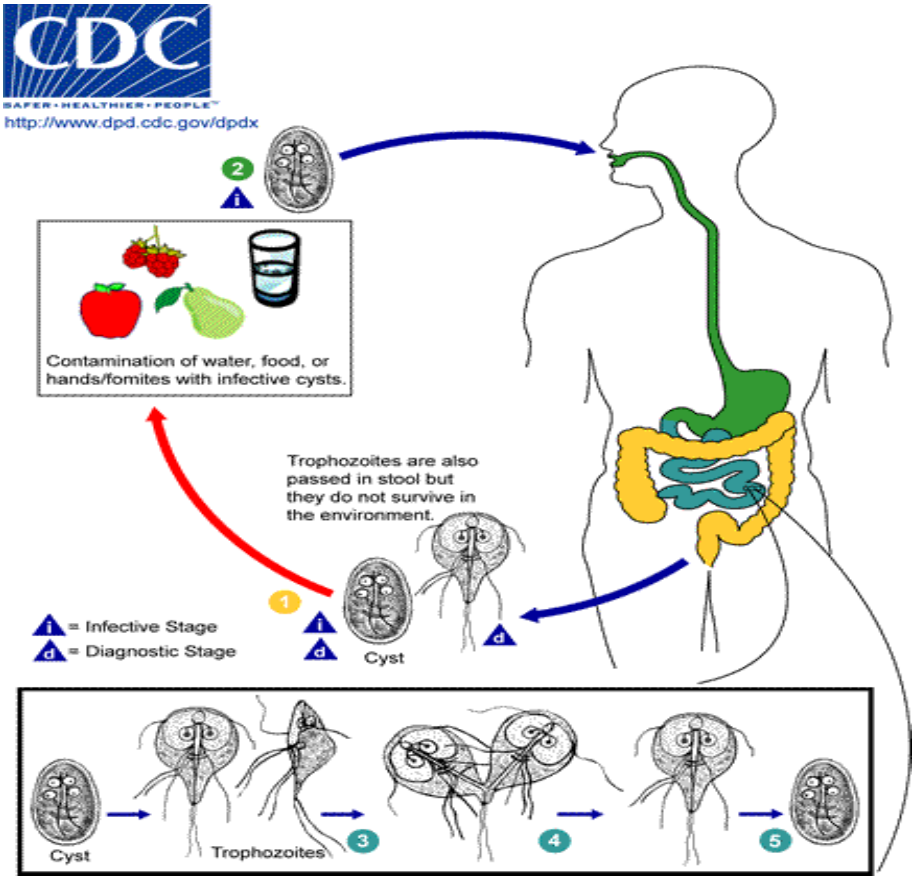
Cyst: Measures 9 x 12 micrometers and contain 2 to 4 nuclei; the karyosome is centrally located, with little or no peripheral chromatin; parabasal bodies are present.

Trophozoite : - Four pairs of flagella - one pair located anterior, two pair located ventral, and one pair located posterior. An axostyle and parabasal bodies are present. motility resembles a “falling leaf” uses “sucking discs” to adhere to intestinal wall; interferes with absorption of nutrients.

Life cycle: man ingests cysts from fecally contaminated environment; the organism excysts in the upper intestine;

trophozoites multiply and attach to the intestinal mucosa, sometimes entering secretory tubes, even the gall bladder. Trophozoites and cysts are passed in the feces.

***intestinalis Lifecycle*:-**



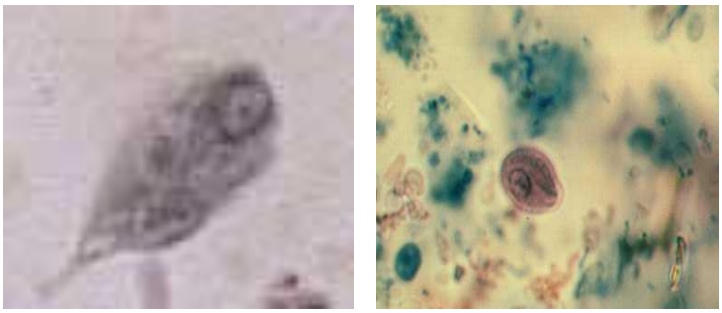
Diagnosis: - identification of cysts or trophozoites in stool specimens or duodenal contents. Irregular shedding pattern results in a "showering" of organisms at times, while being difficult to detect at other times.

Epidemiology: - prevalence 1 to 30%, depending upon the population surveyed; often occurs in epidemics, especially in children's day care centers; can be transmitted in water. Cysts remain viable as long as 3 months when protected from direct sunlight and excess heat; resistant to chlorination.

Pathology and Clinical Manifestations: - symptoms can be severe; diarrhea, foul - smelling, greasy, mucus-laden stools, flatulence, nausea, cramps. Most infections are asymptomatic; chronic cases

experience weight loss, malabsorption of fat, protein, folic acid, and fat-soluble vitamins.

Chilomastix mesnili



Chilomastix mesnili trophozoite C. mesnili cyst

Chilomastix mesnili is cosmopolitan in distribution although found more frequently in warm climates. It is thought to be non-pathogenic although the trophozoite has been associated with diarrhoeic stool. This is the largest flagellate found in man with an incidence of 1-10% being in the large intestine. Found in cecum and colon Transmission by ingestion of mature cysts.

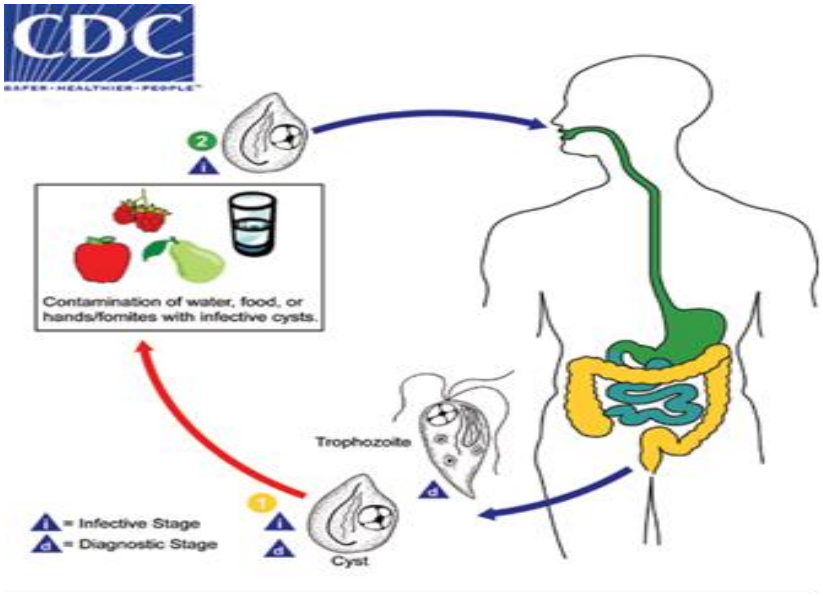
Morphology of the Trophozoite:

The ***trophozoites*** of *C. mesnili* are pear shaped and measure 6-20m m in length. They have 1 large nucleus with a small karyosome and 3 flagella that extend from the nucleus at the anterior end of the parasite. A distinct oral groove or cytosome can be seen near the nucleus with its sides being supported by two filaments. They are known to move in a directional manner.

The cysts: are 6-9m m, they have a large single nucleus with a large

karyosome. They also have a prominent side knob giving it a characteristic lemon shape. The cytosome is evident with a curved shepherds crook fibril. It also has a characteristically coiled filament which when stained is darker in color.

Laboratory Diagnosis: The characteristic lemon shaped cysts can be seen in a formol-ether concentrate.



Life Cycle

The cyst stage is resistant to environmental pressures and is responsible for transmission of *Chilomastix*. Both cysts and trophozoites can be found in the feces (diagnostic stages) . Infection occurs by the ingestion of cysts in contaminated water, food, or by the fecal-oral route (hands or fomites). In the large (and possibly small) intestine, excystation releases trophozoites. *Chilomastix* resides in the cecum and/or colon; it is generally considered a commensal whose contribution to pathogenesis is uncertain. Animals may serve as a reservoir for *Chilomastix*.

Clinical Presentation:

Chilomastix mesnili is considered nonpathogenic. The presence of cysts and/or trophozoites in stool specimens can however be an indicator of fecal contamination of a food or water source, and thus does not rule-out other parasitic infections.

Trichomonas

Trichomonas diff ers from other fl agellates, as they exist only in trophozoite stage. Cystic stage is not seen.

Genus *Trichomonas* has 3 species, which occur in humans

- *T. vaginalis*
- *T. hominis*
- *T. tenax*

a. Characteristics:

1) Undulating membrane - protoplasmic membrane with flagellar rim extending out like a fin along outer edge of body. Moves in a wave-like fashion.
2) Flagella - several in a tuft, provides locomotion
3) Axostyle - functions for support
4) Costa - firm rod-like structure running along base of the undulating membrane.
5) Cytostome - rudimentary mouth

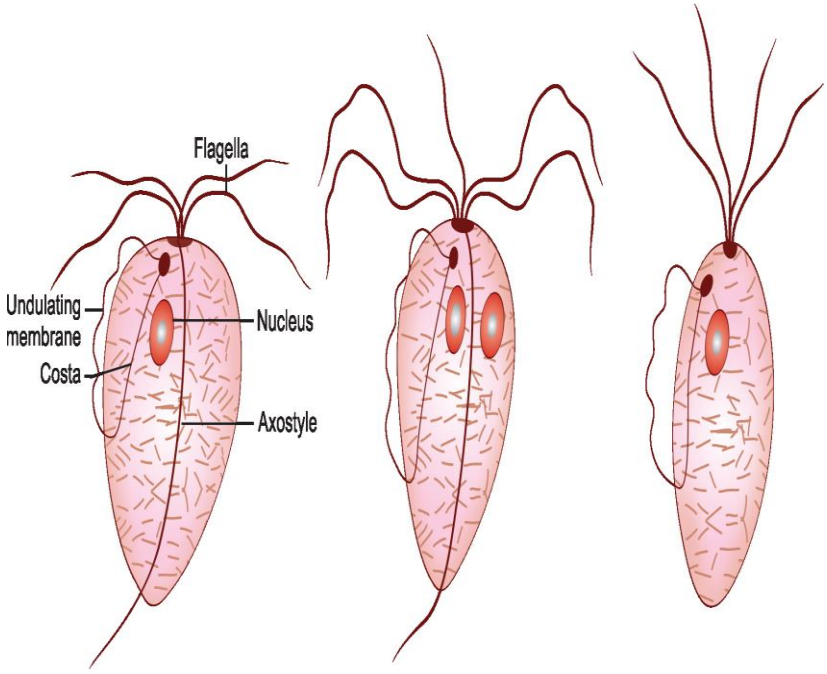


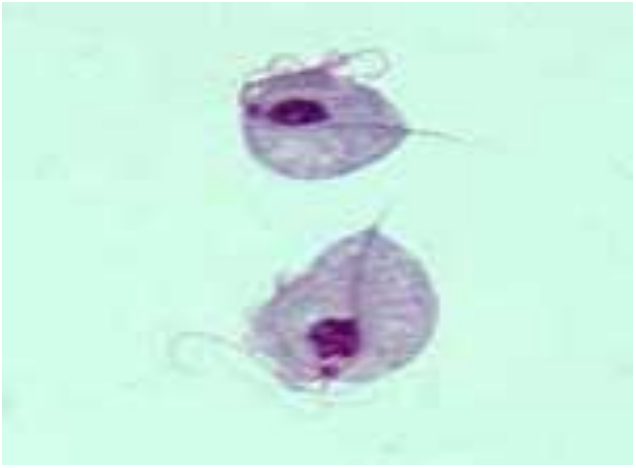
Fig. 4.4: *Trichomonas* species. **A.** *T. vaginalis*; **B.** *T. hominis*; **C.** *T. tenax*

Trichomonas hominis

This flagellate is non-pathogenic and live in the lumen of the large intestine.

It has an undulating membrane, also, it has 3–5 free anterior flagella, and other flagellum borders an undulating membrane, but becomes free beyond the posterior end.Axostyle projects posteriorly, one anterior nucleus, cytostome small and anterior,has no cyst stage. Mode of infection occurred by ingestion of trophozoites stageswhich can resist the gastric juice.This parasite exists in the human intestine, and the scientists consider it nonpathogenic, despite its existence cause intestinal disorder and diarrhea.

b. Trichomonas vaginalis:



Trichomonas vaginalis trophozoites

[Habitat]

- found in the vagina, especially when vaginal secretion is less acidic than normal (pH 5 – 7.5, average 5.5).* It may also live in cervix, uterus or the urethra and urinary bladder.
- In males, it may inhabit the urethra, urinary bladder, prostate and seminal vesicle.

[Morphology]

- The trophozoite is similar to that of T. hominis, but differs in:
- Larger, may reach up to 30 micron in length.
- Undulating membrane is short reaching about half the body length.
- The fourth flagellum has no free end.

[Transmission]

During sexual intercourse, trophozoites stages are transmitted.

[Pathogenesis & Symptoms]

In female:

- The patient feels itching in the external genitals, burning feeling and frequency of urination.
- These symptoms are accompanied with milky yellowish exudates from the vagina.

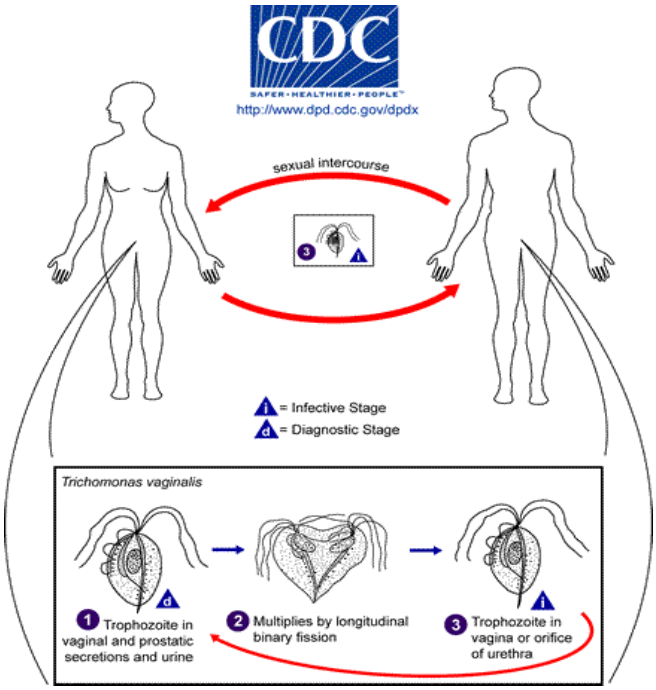
In male:

- Infection is generally asymptomatic, when symptoms occur; they consist of burning feeling during urination, accompanied with a yellowish discharge from the urethra.
- If infection extends to posterior urethra and the prostate, urethritis becomes chronic.

[Diagnosis]

- In females, detecting trophozoites stages in vaginal discharge or vaginal scrapings.
- In males, by finding flagellates in the urethral discharge got by prostatic massage.

Life cycle: - trophozoite lives in the vagina, urethra, epididymis, and prostate; multiplies via longitudinal fission; no cyst stage.

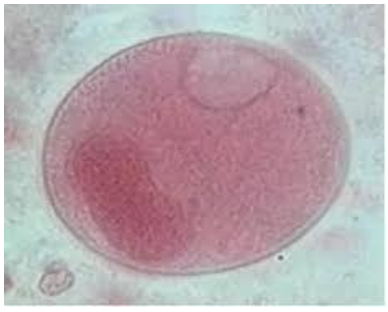
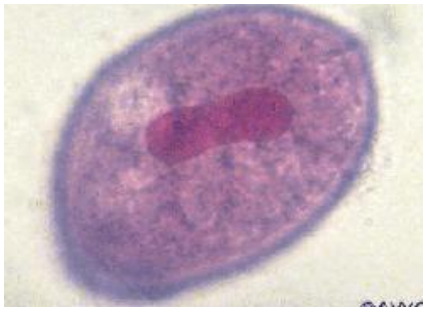


Diagnosis - ID of trophs in body fluids - wet mounts of discharges or on PAP smears.

1. ***Trichomanas tenax*** – was first recovered from the mouth, specifically in tartar from the teeth. There is no known cyst stage. The trophozoite has a pyriform shape and is **smaller and more slender than that of *T.hominis***. Diagnosis is based on the recovery of the organism from the teeth, gums, or tonsillar crypts, and no therapy is indicated.

2. Class Kinetofragminophora:- The Ciliate

1-Balantidium coli

Balantidium coli cyst Balantidium coli trophozoite)

Habitat

B. coli resides in the large intestine of man, pigs, and monkeys.

Morphology:-

Trophozoite stage: reniform large macronucleus Smaller micronucleus in the concavity of macronucleus 30-300 μm in size, 2 contractile vacuoles Cytostome locate at anterior end as funnel-shape

Locomotion with cilia, embedded in pellicle in longitudinal rows

Cystic stage: The cyst is spherical in shape and measures 40–60 μm in diameter. It is surrounded by a thick and transparent doublelayered wall. The cytoplasm is granular. Macronucleus, micro nucleus, and vacuoles are also present in the cyst. The cyst is the infective stage of *B. coli.* It is found in chronic cases and carriers.

Epidemiology:

Rarely found in USA. This is the only ciliate parasite of humans. It is prevalent in tropical areas, or where poor sanitation, hygiene, and crowding occur. Increase numbers of infections are expected in those with close, continuing contact with swine.

Pathology & Symptoms:-

Most infections are asymptomatic.

- Symptomatic disease or **balantidiasis** resembles amoebiasis causing diarrhea or frank dysentery with abdominal colic, tenesmus, nausea, and vomiting.

- *Balantidium* ulcers may be secondarily infected by bacteria.
- Occassionaly, intestinal perforation peritonitis and even death may occur.
- Rarely, there may be involvement of genital and urinary tracts.

Life cycle:

Life Cycle

B. coli passes its life cycle in one host only (monoxenous).

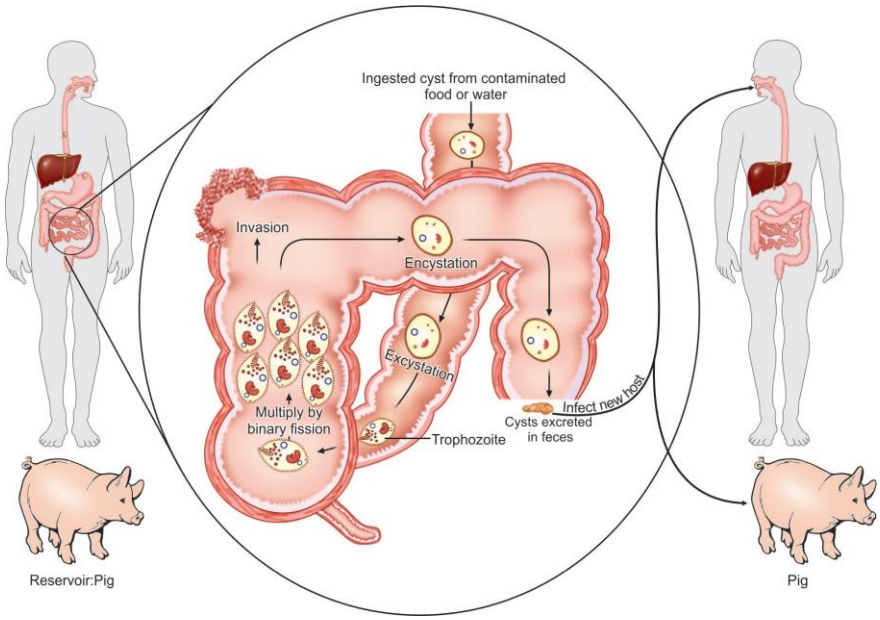
Natural host: Pig.

Accidental host: Man.

Reservoirs: Pig, monkey, and rat.

Infective form: Cyst.

Infection occurs from close association with pigs , Human to human transmission may also occur , Cyst is the infective stage of this parasite . Excystation occurs in small intestine Multiple in large intestine by binary fission Conjugation at the anterior end for a few minutes



Diagnosis:

Diagnosed by observing cysts & trophs in fecal samples. Cysts are easily missed – cysts stain very dark with iodine, so the structures used in identification (buccal cavity & macronucleus) are not always readily visible.

Disease names:

Balantidiasis, balantidial dysentery

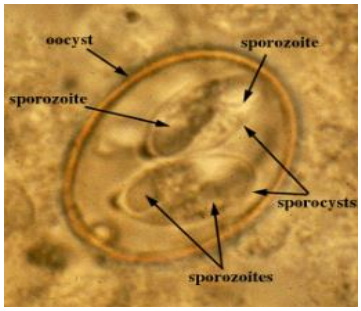
V. Intestinal Coccidia

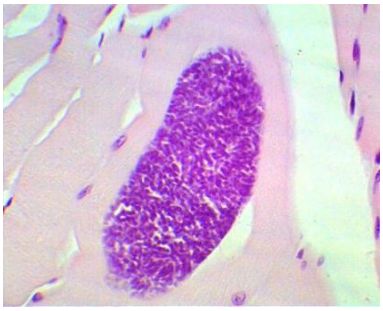
Introduction:

1. A number of species parasitize humans: *Isospora, Sarcocystis, Cryptosporidium, & Toxoplasma*
2. Have complex life cycles - most have 2-host life cycle.
3. ***Schizogony:-*** asexual binary fission.
4. ***Sporogony***: - sexual reproduction
5. Diagnostic stages are often difficult to locate. They are easily overlooked due to their nearly transparent appearance.

Permanently stained smears not helpful. Acid fast and giemsa stains are more often used. Oocysts do not stain with iodine.

1- <u>Sarcocystis:-</u>





species of genus Sarcocystis can infect humans 1-*S. hominis* (transmitted through cattle) Humans are the definitive host 2-€ *S. suihominis* (transmitted through pig)

Sarcocystis species produce cyst in the muscle of the intermediate hosts. These cysts, called Sarcocysts contain numerous merozoites (bradyzoites) .

When sarcocyst is eaten by **the definitive host**,: the **merozoites** are released in the intestine, where they develop into male and female gametes.

After fertilization, the **zygote** develops into an **oocyst** containing 2 sporocysts, each having 4 sporozoites .

These **oocysts** are shed in feces and are ingested by intermediate host.

In the intermediate hosts: the sporozoite invade the bowel wall and reach the vascular endothelial walls, where they undergo schizogony producing merozoites (tachyzoites).

These spread to muscle fibers and develop into sarcocysts.

Cow is the intermediate host for ***S. hominis***. Human infection is acquired by eating raw or undercooked beef. Oocysts are shed in human feces, which contaminate grass and fodder eaten by cows.

pig is the intermediate host In the case of ***S. suihominis***, human infection is obtained through eating contaminated pork. Human infection with *S. hominis* and *S. suihominis* is related to food habits.

Definitive host: - humans. Pig (sui-) and cow (bovi-) are intermediate hosts.

Infective stage: - ingestion of sarcocysts in meat (intestinal); ingestion of oocysts from animal feces (muscle).

Life cycle

Species of the protozoa Sarcocystis have an obligatory 2-host life cycle. The intermediate host is usually an herbivore or omnivore. The definitive host is a carnivore.

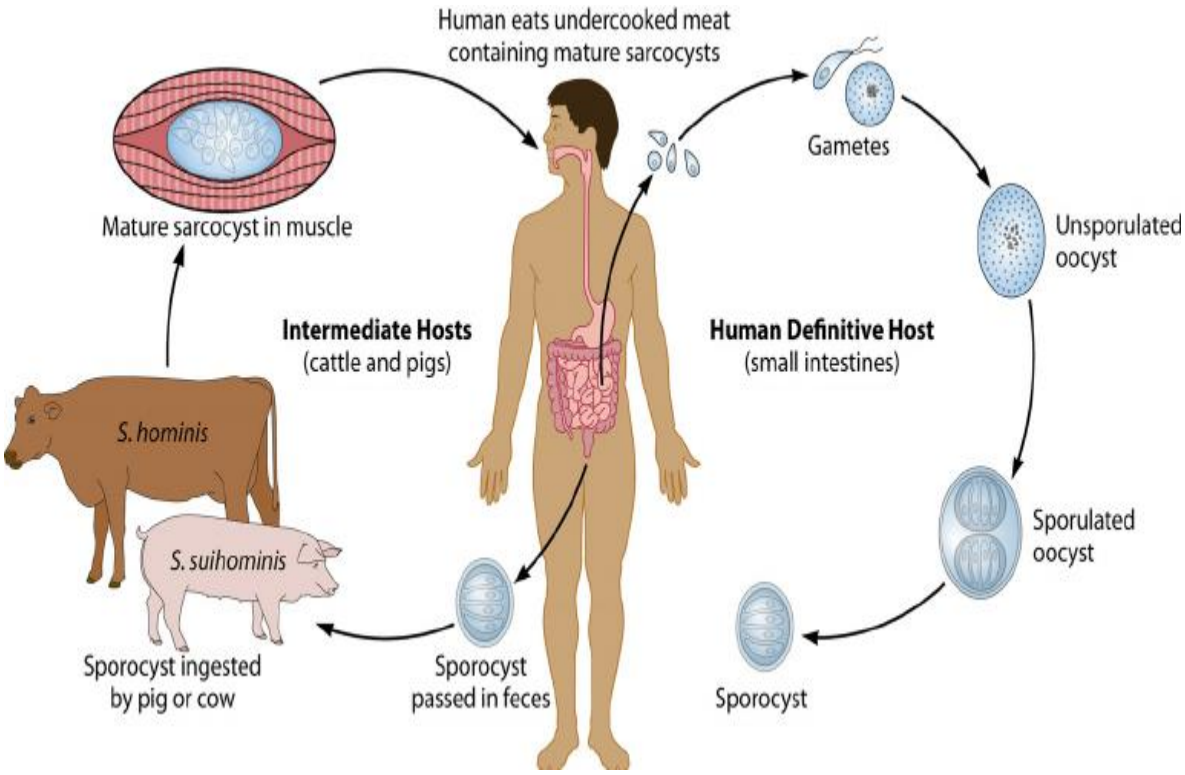
Sexual Reproduction. The cycle begins when adult male and female parasites sexually reproduce in the definitive host's epithelial cells. The newly created oocysts are sporulated in the host. In passing, the thin-walled sporulated oocysts often rupture, releasing infective **sporocysts**. The host then sheds both sporocyts and sporulated oocysts in the feces.

Transmission. In the next stage of the cycle, the intermediate host ingests infective sporocysts fecal-orally by contamination of feed or water.

Asexual reproduction. Sporozoites excyst from the sporocysts and invade the intestinal mucosa to reach endothelial cells. An asexual cycle begins, whereby initial reproduction forms **schizonts**.

Within the schizonts are **merozoites**, which are released and eventually find their way to muscle tissue, where they form **sarcocysts**. The merozoites divide into pairs within the tissues of the cyst, forming **metrocytes**. Metrocytes continue to undergo maturations, until finally forming mature, banana-shaped **bradyzoites**, which are slowly dividing, and also infective to definitive hosts.

When the muscle tissue containing sarcocysts with infective bradyzoites is consumed by the definitive host (a carnivore), the bradyzoites penetrate the mucosa and transform into **macrogametes** and **micrgametocytes** in the cells of the intestinal epithelium. The microgametocytes produce flagellated **microgametes**, which penetrate the macrogametes. The zygote lays down a resistant wall and sporulates endogenously.



Clinical Features

Intestinal sarcocystosis is usually asymptomatic. Patients may have nausea, abdominal pain, and diarrhea.

Muscular sarcocystosis is also usually asymptomatic but may cause muscle pain, weakness, or myositis, depending on the size of the cyst.

Laboratory Diagnosis

Stool Examination Characteristically sporocysts or occasionally oocysts can be demonstrated in feces of human beings. Species identifi cation is not possible with microscopy.

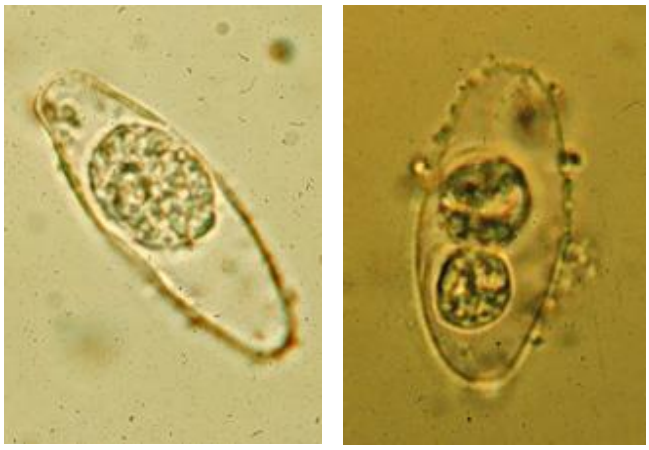
Muscular Sarcocystosis Diagnosis can be made by demonstration of sarcocysts in the skeletal muscle and cardiac muscle by biopsy or during autopsy.

Prophylaxis

By avoiding eating raw or undercooked beef or pork

By avoidance of contamination of food and drink with feces of cat, dog, or other carnivorous animals

2-Isospora belli



Isospora belli oocyst (mature) --- Isospora belli oocyst (immature)

Isospora belli is a coccidian parasite which can cause diarrhea in humans. The name belli (from bellium meaning war) was given for its association with war, because several cases of infection with this parasite were seen among troops stationed in Middle East during fi rst world war.

Morphology:

Oocysts of *I. belli* are elongatedovoid and measure 25 µm × 15 µm. Each oocyst is surrounded by a thin smooth 2 layered cyst wall. Immature oocyst seen in the feces of patients contain two sporoblasts. The oocysts mature outside the body. On maturation, the sporoblast convert into sporocysts. Each sporocyst contain 4 crescentshaped sporozoites. The sporulated oocyst containing 8 sporozoites is the infective stage of the parasite.

Definitive host: - humans.

Life Cycle

belli completes its life cycle in one host.

Man gets infection by ingestion of food and water contaminated with sporulated oocyst.

When a sporulated oocyst is swallowed, 8 sporozoites are released from the 2 sporocysts in the small intestine and invade the intestinal epithelial cells.

In the epithelium, the sporozoites transform into trophozoites, which multiply asexually (schizogony) to produce a number of (merozoites). The merozoites invade adjacent epilhelial cells to repeat asexual cycle.

Some of the trophozoites undergo sexual cycle (gameto gony) in the cytoplasm of enterocytes and transform into macrogametocytes and microgametocytes.

After fertilization, a zygote is formed, which secretes a cyst wall and develops into an immature oocyst.

These immature oocysts are excreted with feces and mature in the soil.

Incubation period: 1–4 days

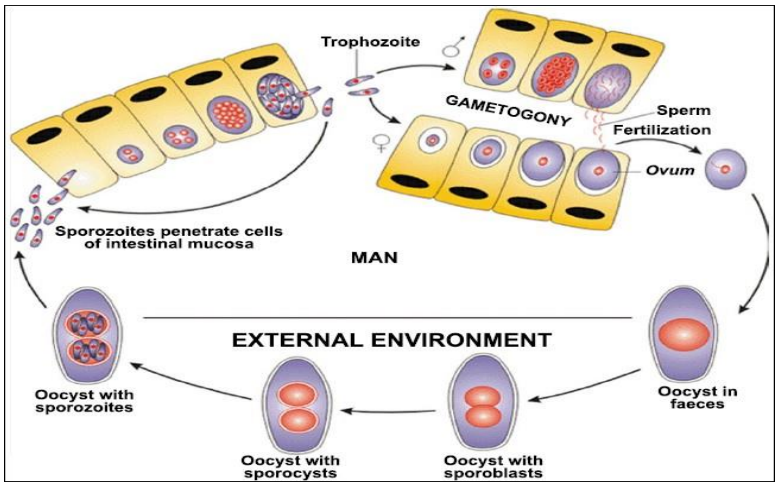
Diagnostic/infective stage:-

a. **Immature oocysts** - contain only one sporocyst, do not stain with iodine; measure 12 x 30 microns.

b. **Mature oocysts** - contain two sporocysts, each of which contain four sporozoites. Similarly, do not stain with iodine.

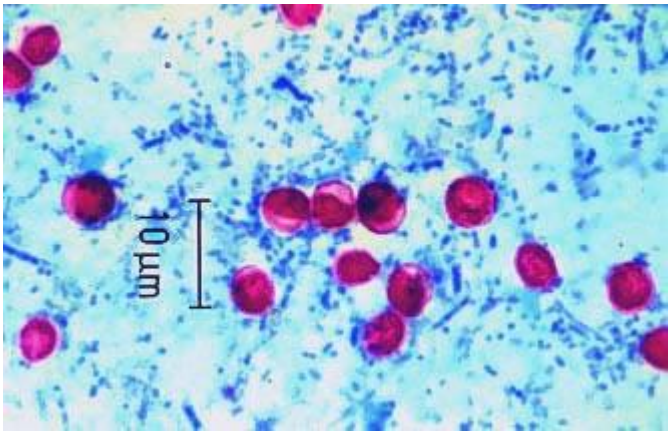
Clinical Features

Infection is usually asymptomatic. Clinical illness includes abdominal discomfort, mild fever, diarrohea, and malabsorption. The diarrohea is usually watery and does not contain blood or pus and is selflimiting. However, protracted diarrohea, lasting for several years can be seen in immunocompromised persons, particularly in the human immunodefi ciency virus (HIV) infected.



Intermediate hosts : none.

3- Cryptosporidium parvum:-



Cryptosporidium parvum oocysts (acid-fast stain)

cryptosporidium has assumed great importance as a frequent cause of intractable diarrohea, in AIDS patients, and immunocompromised subjects. It is worldwide in distribution. Two species of *cryptosporidium, c. hominis and c. parvum* mostly cause human infections.

Habitat

Habitat *c. parvum* inhabits the small intestine. It may also be found in stomach, appendix, colon, rectum, and pulomonary tree.

*C.*parvum inhabits the small intestine. It may also be found in stomach, appendix, colon, rectum, and pulomonary tree.

Morphology

The infective form of the parasite is oocyst. The oocyst is spherical or oval and measures about 5 µm in diameter. Oocysts does not stain with iodine and is acid fast. The wall of the oocysts is thick, but in 20% cases, wall may be thin. These thin walled oocysts are responsible for autoinfection. Both thin walled and thick walled oocyst contain 4 crescentshaped sporozoites . Oocyst can remain viable in the environment for long periods, as it is very hard and resistant to most disinfectants and temperature upto 60°C. It can survive chlrorinated water, but sequential application of ozone and chlorine has been found eff ective in eliminating the cysts.

Life cycle:

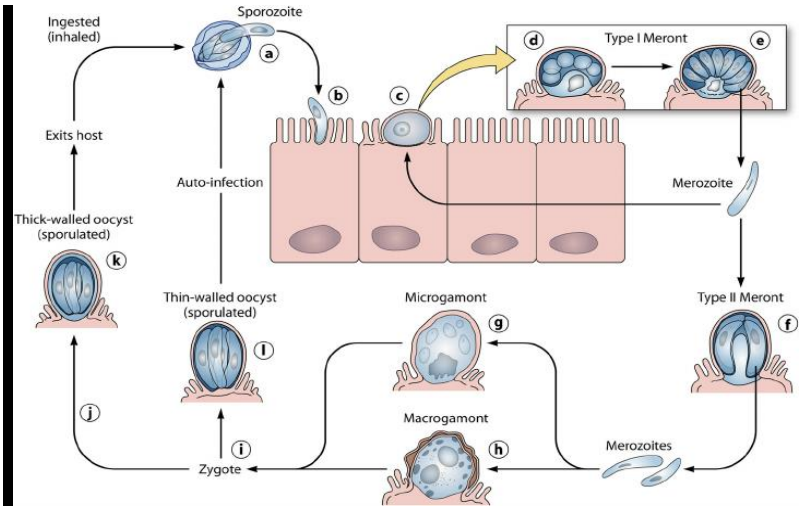
The parasite complete its life cycle, sexual and asexual phases in a single host (monoxenous) .

Suitable host: Man.

Reservoirs: Man, cattle, cat, and dog.

Mode of transmission: Man acquires infection by: € Ingestion of food and water contaminated with feces containing oocysts € Autoinfection. **Infective form**: Sporulated oocysts.

The oocyst contains 4 sporozoites, which are released in the intestine



The sporozoites develop into trophozoites within parasito phorous vacoules in the brush border of the intestine.

The trophozoites undergo asexual multiplication (schizogony) to produce type I meronts.

Eight merozoites are released from each type I meront. These merozoites enter adjacent epithelial cells to repeat schizogony or form type II meronts, which undergo gametogony.

Four merozoites are released from each type II meront. The

merozoites enter host cell to form sexual stages— microgamete and macrogamete.

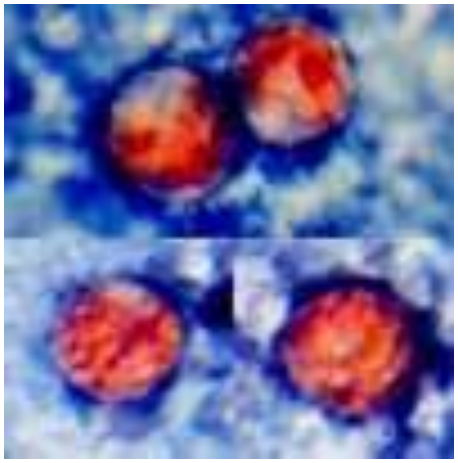
After fertilization, the zygote formed develops into the oocyst. The oocysts undergoes sporogony to form spourlated oocyst, which contain 4 sporozoites. Spourlated oocysts are released into the feces and transmit the infection from one person to another. Some of the oocysts have a thin wall surrounding 4 sporozoites and are called as thin-walled oocysts. These oocysts infect the same host and maintain the cycle of autoinfection.

The oocysts are fully mature on release and are infective immediately without further developmen.

Pathology:- although the condition can be asymptomatic, most infections cause moderate to severe diarrhea of one to two weeks in the immunocompetent patient. In the immunosuppressed patient, the condition is protracted and life threatening. At this time, there is no drug effective against this parasite.

Identification: oocysts are 2 - 5 microns in diameter; do not stain with iodine; and are acid-fast.ElIS

4- Cyclospora cayetanensis



Cyclospora cayetanensis oocysts

(acid-fast stain)

Morphology:

The morphological form found in the feces is an oocyst. The oocyst is a nonrefractile sphere, measuring 8–10 µm in diameter. It contains 2 sporocysts. Each sporocyst contains 2 sporozoites. Hence, each sporulated oocyst contains 4 sporozoites.

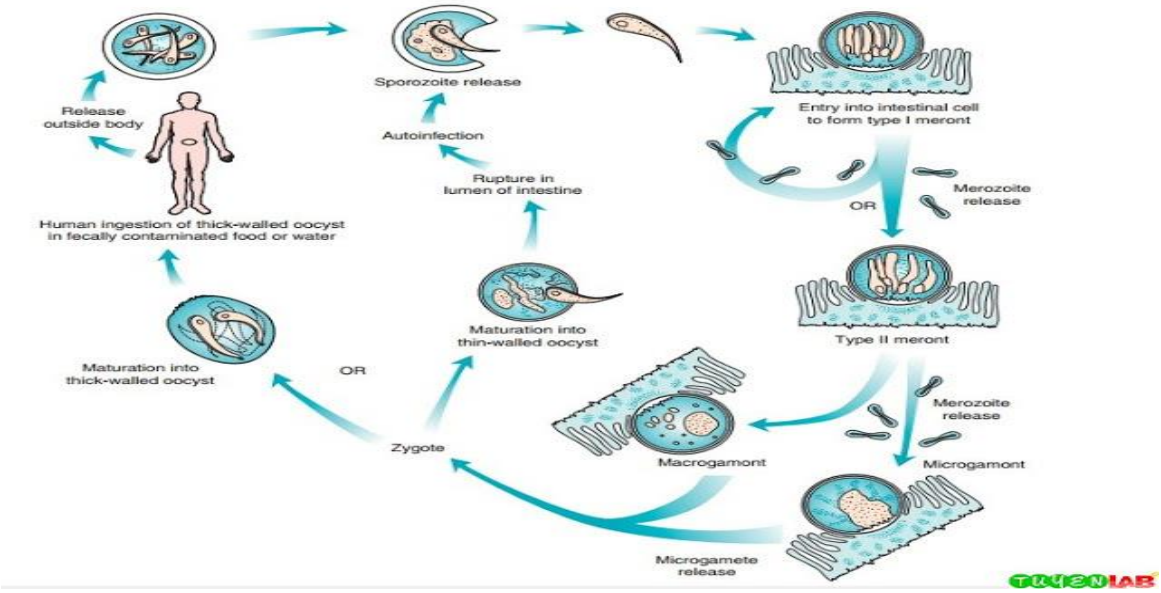
Animal reservoirs:- not known. Other *Cyclospora* species are known to infect a variety of animals, but *C. cayetanensis* is the name designated for the only one known to infect humans (at this time).

Life Cycle

Oocyst shed in feces sporulates outside the host. The sporulated oocysts are infectious to humans. Man acquires infection by ingestion of food and water contaminated with feces containing oocysts. Excystation of the sporocyst releases crescentic sporozoites measuring 9 µm × 1.2 µm.

The sporozoites infect enterocytes in the small intestine.

The sporozoites develop into unsporulated oocysts, which are excreted in feces.

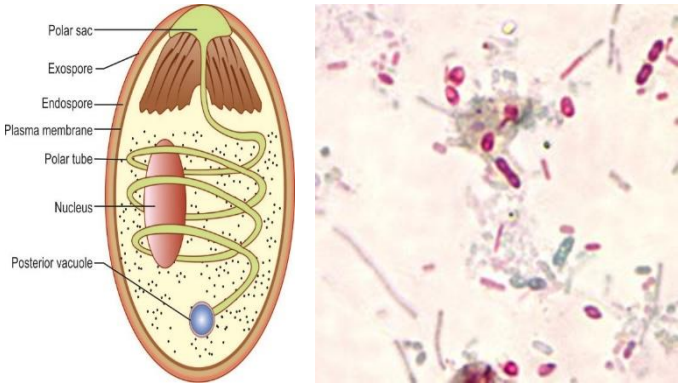


Transmission:- Contaminated food or water , person-to-person.

Pathology: - Infected individuals experience a diarrhea similar to that experienced with *Cryptosporidium* infections. While some cases were less debilitating than others, asymptomatic cases were not thought to have occurred.

Identification:- Oocysts are spherical, 7 - 10 microns in diameter, and present as variably acid-fast. Acid-fast procedures utilizing carbol-basic fuschin proved superior in demonstrating this parasite.

5-Microsporidium:-



Microsporidium spp. spores

General – as of this time, infections are thought to be limited to AIDS patients.

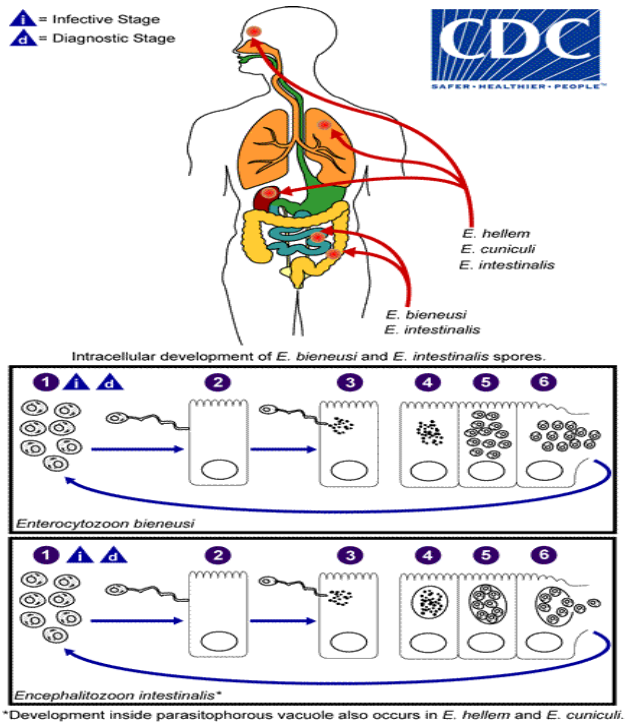
Morphology:-

Microsporidia are unicellular, obligate intracellular parasite. They reproduce in host cells by producing spores¬ (sporogony) Spores are 2–4 µm in size and oval to cylindrical in shape,¬ with a polar lament or tubule (Fig. 8.1). The spores are the infective stage of microsporidia¬ and the only stage of life cycle capable of existing outside the host cell. The polar tubule is an extrusion mechanism for injecting infective spore contents into the host cell. Spores are surround by thick double-layered cyst wall Outer layer (exospore) is proteinaceous and electron-dense Inner layer (endospore) is chitinous and electronlucent Spores are Gram-positive and acid fast.

Life cycle:-

Life Cycle Infection in host is probably by ingestion or inhalation of spores. In the duodenum, the spore with its nuclear material is¬ injected through the polar tubule into the host cell. Inside the cell,

the microsporidia multiply by repeated¬ binary ssion (merogony) and produce large number of spores (sporogony). During sporogony, a thick spore wall is formed that¬ provides environmental protection to the cyst. The spores are then liberated free from the host cell and¬ infect other cells.



Clinical Presentation:-

Human microsporidiosis represents an important and rapidly emerging opportunistic disease, occurring mainly, but not exclusively, in severely immunocompromised patients with AIDS.. The clinical manifestations of microsporidiosis are very diverse, varying according to the causal species with diarrhea being the most common.

Laboratory Diagnosis

Microscopy Diagnosis of microsporidiasis is made by demonstration of the spores in stool, urine, cerebrospinal - uid (CSF), or small intestine biopsy specimen.

Blood & Tissue Protozoa – The Hemoflagellates

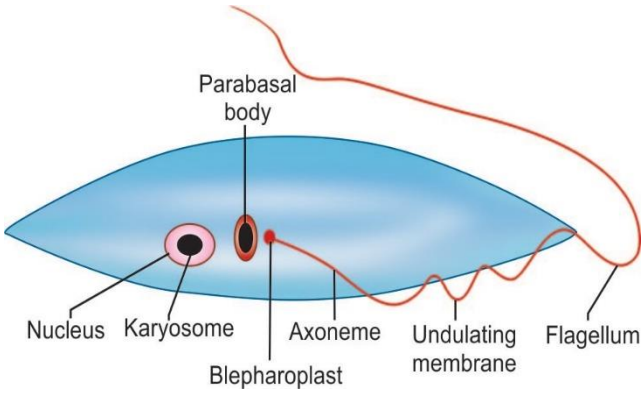
Introduction

1. The family Trypanosomatidae, (includes hemoflagellates) contain only two genera that parasitize humans.
   a. Genus ***Leishmania*** are always intracellular, principally in cells of the reticuloendothelial system.
   b. Genus ***Trypanosoma*** contains members that may be found both in the circulating blood and intracellularly in cardiac muscle. African - blood; American - cardiac muscle.
2. In all probability, the hemoflagellates were originally parasites of insects. They are transmitted by insects, and in them undergo a developmental cycle (the arthropod serves as intermediate host).
   a. "Old World" leishmaniasis - transmitted by the bite of various species of sandflies of the genus ***Phlebotomus*.**
   b. South American leishmaniasis - carried by ***Lutzomyia*** spp. sandflies.
   c. American trypanosomiasis - transmitted by **reduviid bugs**; transmission occurs when infective feces of the bug contaminates the wound made by the insect's bite or an abrasion of the skin.
   d. African trypanosomiasis - transmitted by ***Glossina spp***. tsetse flies.

Morphological forms of hemoflagellates

General Characteristics

1. **Nucleus**: is round or oval and is situated in the central part of the body.
2. **Kinetoplast**: consists of a deeply staining parabasal body and adjacent dotlike **blepharoplast**. The parabasal body and blepharoplast are connected by one or more thin fibrils (Fig 5.1).
3. **Flagellum**: is a thin, hairlike structure, which originates from the blepharoplast. The portion of the flagellum, which is inside the body of the parasite and extends from

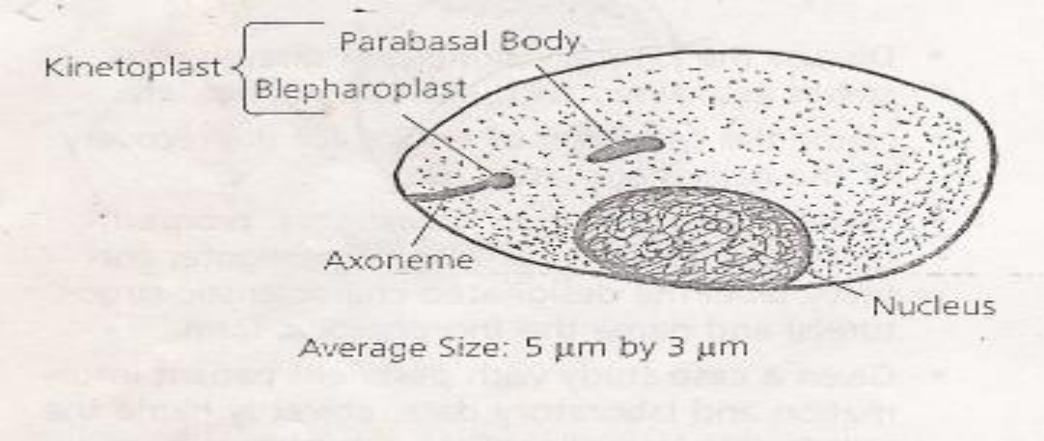


Hemofl agellates exist in two or more of four morphological stages. These forms were formerly called the **leishmanial, leptomonad, crithidial, and trypanosomal stages**. But as these names are also given to diff erent genera within the family, they were changed to **amastigote, promastigote, epimastigote and trypomastigote**.

1- Amastigotes:

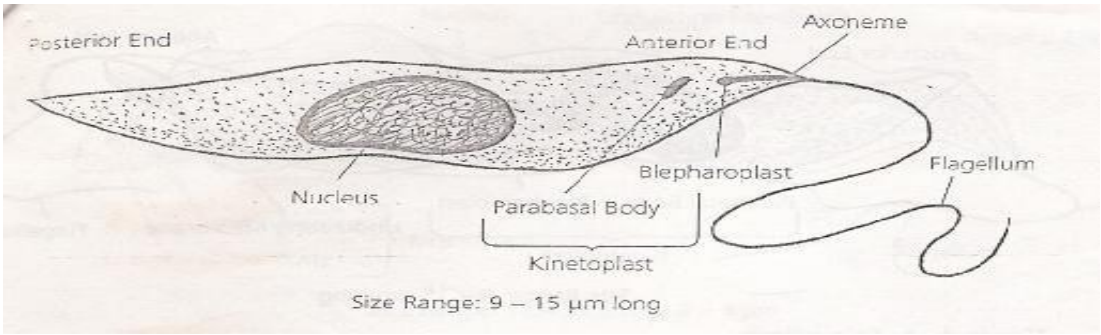
Rounded or ovoid, without any external fl agellum. The nucleus, kinetoplast, and axial fi laments can be seen. The axoneme extends

upto the anterior end of the cell



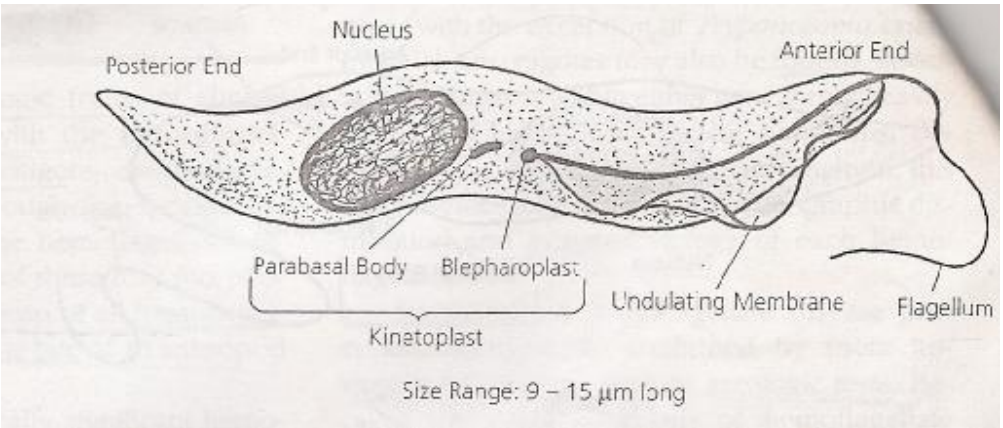
2– Promastigotes :-

Lanceolate in shape. Kinetoplast is anterior to the nucleus (antenuclear kineloplast) near the anterior end of the cell, from which fl agellum emerges. There is no undulating membrane



3– Epimastigotes :-

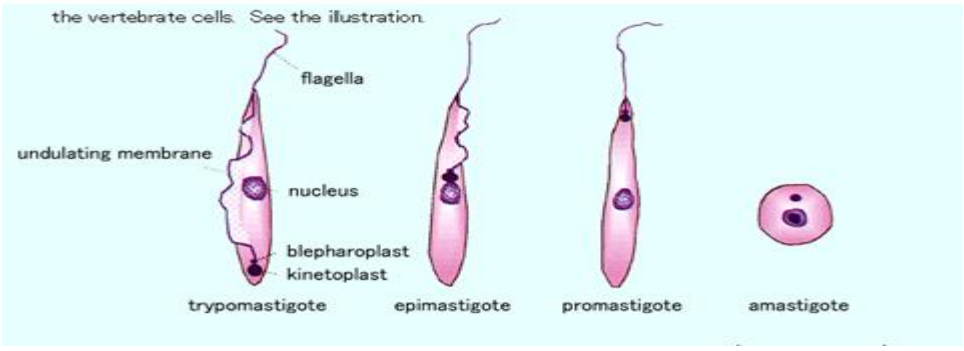
Elongated, with the kinetoplast placed more posteriorly, though close to and in front of the nucleus. The fl agellum runs alongside the body as a short undulating membrane, before emerging from the anterior end



4– Trypomastigotes :-

This stage is elongated, spindleshaped with a central nucleus. The kinetoplast is posterior to the nucleus (postnuclear kinetoplast) and situated at the posterior end of the body. The flagellum runs alongside the entire length of the cell to form a long undulating membrane before emerging as a free fl agellum from the anterior end

Different stages of Haemoflagellates



***1- Trypanosoma*:-**

In the vector, the trypanosomes follow one or two modes of development and are accordingly classifi ed into 2 groups

1. **Salivaria (anterior station):** In salivaria, the trypanosomes

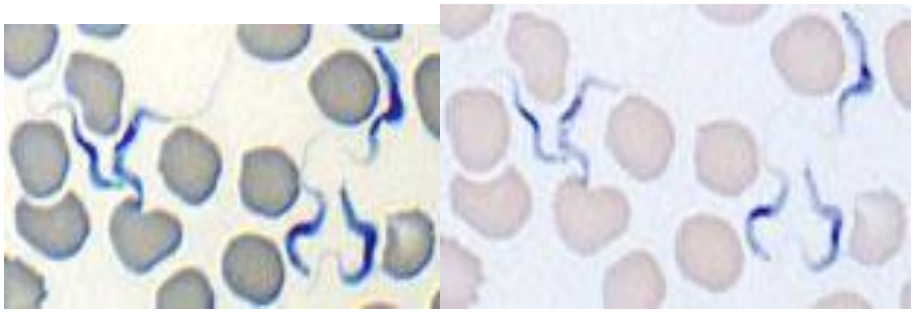
migrate to mouth parts of the vectors, so that infection is transmitted by their bite (inoculative transmission). Examples are ***T. gambiense and T. rhodesiense*** **causing African trypanosomiasis,** which are transmitted by the bite of tsetse fl ies.

2. **Stercoraria (posterior station)**: In stercoraria, the trypanosomes migrate to the hindgut and arepassed in feces (stercorian transmission), e.g. *T. cruzi* causing **Chagas' disease**, which is acquired by rubbing the feces of the vector bug into the wound caused by its bite

1-African trypanosomiasis (sleeping sickness)

2- South American trypanosomiasis (Chagas' disease)

A-Trypanosoma Brucei Gambiense (**West African Trypanosomiasis**)



Trypanosoma b. rhodesiense Trypanosoma b. gambiense

Habitat

Trypanosomes live in man and other vertebrate host. They are essentially a parasite of connective tissue, where they multiply rapidly and then invade regional lymph nodes, blood, and fi nally may involve central nervous system.

Life Cycle

T. brucei gambiense passes its life cycle in 2 hosts.

Vertebrate host: Man, game animals, and other domestic animals.

Invertebrate host: Tsetse fly. Both male and female tsetse fly of Glossina species (G. palpalis) are capable of transmitting the disease to humans.

Infective form: Metacyclic trypomastigote forms are infective to humans.

Mode of transmission

By bite of tsetse fly Congenital transmission has also been recorded.

Reservoirs: Man is the only reservoir host, although pigs and others domestic animals can act as chronic asymptomatic carriers of the parasite.

Development in Man and Other Vertebrate Hosts

Metacyclic stage (infective form) of trypomastigotes are inoculated into a man (definitive host) through skin when an infected tsetse fly takes a blood meal. The parasite transforms into slender forms that multiply asexually for 1–2 days before entering the peripheral blood and lymphatic circulation. These become ‘stumpy’ via intermediate forms and enter the blood stream.

In chronic infection, the parasite invades the central nervous system.

Trypomastigotes (short plumpy form) are ingested by tsetse fly (male or female) during blood meal.

Development in Tsetse Fly

In the midgut of the fly, short **stumpy trypomastigotes** develop into long, slender forms and multiply

After 2–3 weeks, they migrate to the salivary glands, where they develop into **epimastigotes**, which multiply and fill the cavity of the gland and eventually transform into the infective metacyclic trypomastigotes

Development of the infective stage within the tsetse fly requires 25–50 days (extrinsic incubation period).

Thereafter, the fly remains infective throughout its life of about 6 months.

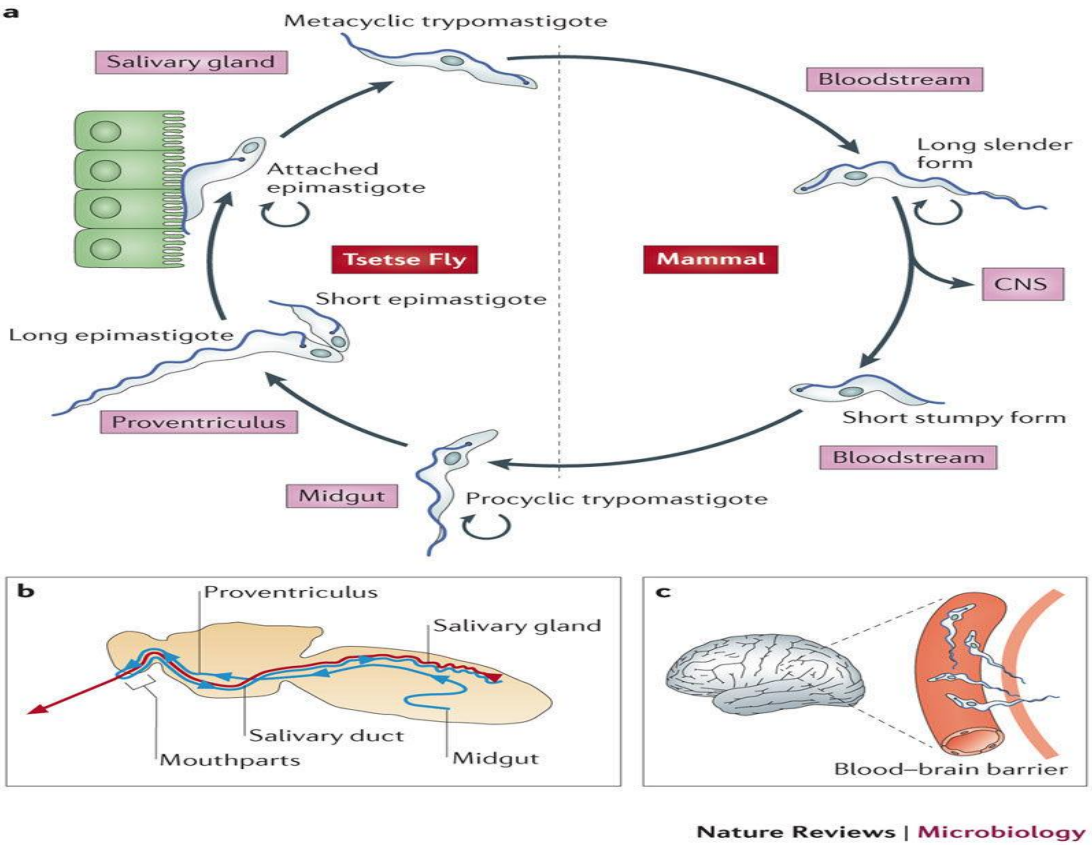
Pathogenecity and Clinical Features

stage I disease. In this stage, there is hepatosplenomegaly and lymphadenopathy, particularly in the posterior cervical region (Winterbottom's sign)**,** Myocarditis,Hematological manifestations seen in stage I include anemia, moderate leucocytosis, and thrombocytopenia**.,**

Stage II disease

involves invasion of central nervous system. With the invasion of central nervous system, which occurs after several months, the 'sleeping sickness' starts. This is marked by increasing headache, mental dullness, apathy, and day time sleepiness

a. ***Diagnosis:*** examine wet mounts of aspirates from chancre (insect bite sight), or blood (buffy coat) for presence of trypanosomes).



2-Trypanosoma Brucei Rhodesiense (East African Trypanosomiasis)

The principal vector is *G. morisitans, G. palpalis*,

Pathogenesis and Clinical Feature

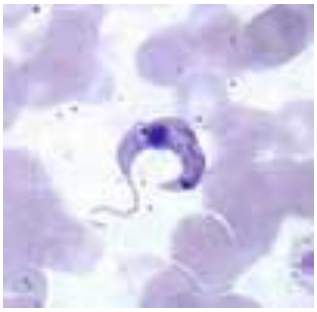
T. brucei rhodesiense causes East African sleeping sickness East African trypanosomiasis is more acute than the Gambian form and appears after an incubation period of 4 weeks.

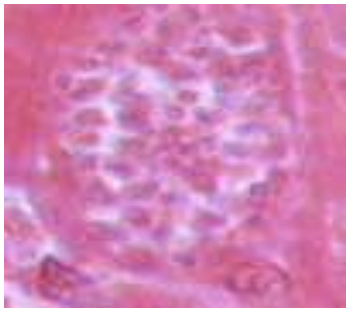
It may end fatally within an year of onset, before the involvement of central nervous system develops.

Pathological features are similar in both diseases with some variations-Edema, myocarditis, and weakness are more prominent in East African sickness. Lymphadenitis is less prominent.

Characteristics	West African	East African
Organism	*T. brucei gambiense*	*T. brucei rhodesiense*
Distribution	West and Central Africa	East and Central Africa
Vector	Tsetse fly (*Glossina palpalis* group)	Tsetse fly (*Glossina morsilans* group)
Reservoir	Mainly humans	Wild and domestic animals
Virulence	Less	More
Course of disease	Chronic (late central nervous system invasions); months to years	Acute (early central nervous system invasion); less than 9 months
Parasitemia	Low	High and appears early
Lymphadenopathy	Early, prominent	Less common
Isolastion in rodents	No	Yes
Mortality	Low	High

B. Trypanosoma cruzi:-





Trypanosoma cruzi trypomastigote Trypanosoma cruzi amastigotes

1. ***Disease: -*** *T. cruzi* is the causative organism of Chagas's disease or South American trypanosomiasis.
2. ***Transmission***: - ***Metacyclic trypomastigote*** *in* ***reduviid bug*** feces is introduced through the skin following the bug's bite. Ingestion

of the bug is also a mode of infection.

3. Habitat

In humans, *T. cruzi* exists in both amastigote and trypomastigote forms. Amastigotes are the intracellular parasites. They are found in muscular tissue, nervous tissue, and reticuloendothelial system. € Trypomastigotes are found in the peripheral blood.

Morphology: the trypomastigote has a characteristic "U" or "C" shape with an undulating membrane and anteriorly extending flagellum; the amastigote is oval to suboval, averages 3 to 5 microns in diameter and contains a nucleus and rod-shaped kinetoplast.

Life Cycle

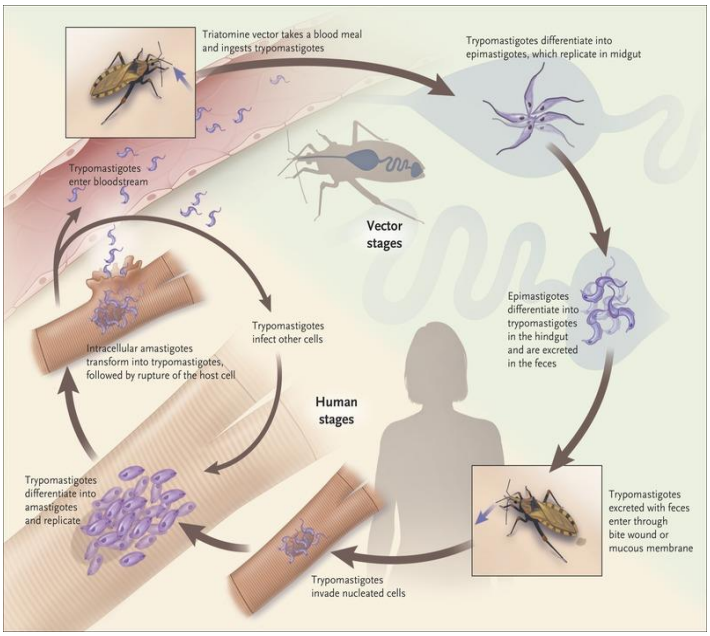
T. cruzi passes its life cycle in 2 hosts.

Definitive host: Man

Intermediate host (vector): Reduviid bug or triatomine bugs.

Reservoir host: Armadillo, cat, dog, and pigs.

Infective form: Metacyclic trypomastigotes forms are the infective forms found in feces of reduviid bugs.



1. ***<u>Pathogenesis:</u>*** ***acute phase*** - shortly after infection (1-4 month's duration; ***chronic phase*** - decades long. Edema of eyelids (Romana's sign). Fever, headache, malaise, myalgia, megacolon, megaesophagus. Cardiac manifestations.
2. ***<u>Reservoir hosts:</u>*** many animals- dogs, opossums, cat, , etc.

Infective **metacyclic trypomastigotes** are deposited on human skin when the reduviid bug takes a blood meal. Trypomastigotes enter the body when the feces are either rubbed into the **bite wound or the eye**. Trypomastigotes invade cells, where they reproduce asexually **as amastigotes**. The cell dies and amastigotes are released. Some will infect other cells and continue the amastigote reproductive cycle, while others will circulate in the blood, where they develop into nondividing trypomastigotes. **The vector ingests** the circulating parasites during a blood meal. The t**rypomastigotes** travel to the bug's gut, where they transform into

epimastigotes and undergo asexual reproduction by binary fission. From here, the parasites travel to the hindgut, where they develop into metacyclic trypomastigotes and migrate to the rectum, ready to be excreted

Prevention:

Most effective approach.

Insecticides

Improving housing conditions

Testing of blood donors

No vaccine

Genus Leishmania:-

- All members of the genus Leishmania are obligate intracellular parasites that pass their life cycle in 2the **mammalian host** and the **insect vector**, female sandfl y.
- Vector : sandfly (phlebotomus).
- Reservoir : digs and rodents .

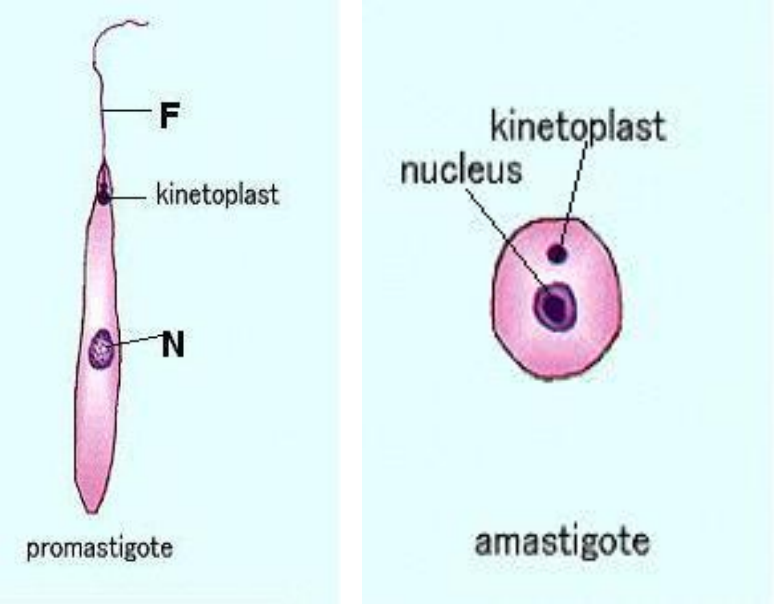
1. **Visceral leishmaniasis**: The species L. donovani complex infecting internal organs (liver, spleen, and bone marrow) of human is the causative parasite.
2. **Cutaneous leishmaniasis**: The species *L. tropica complex, L. aethiopica, L. major and L. mexicana* complex are the causative parasite.
3. **Mucocutaneous leishmaniasis** : It is caused by the L.

braziliensis complex .

Morphology

The parasite exists in 2 forms.

Amastigote form: in humans and other mammals. **Promastigote form**: in the sandfl y and in artifi cial culture.



1- *Leishmania Donovani* (Old World Leishmaniasis)

L. donovani causes visceral leishmaniasis or Kala-azar. It also causes the condition, Post Kala-azar Dermal Leishmaniasis (PKDL)

Habitat

The amastigote (LD body) of L. donovani is found in the reticuloendothelial system. They are found mostly within the macrophages in the spleen, liver, bone marrow and less often in other locations

Mode of transmission:

Humans acquire by bite of an infected female sandfl y. € It can

also be transmitted vertically from mother to fetus, by blood transfusion, and accidental inoculation in the laboratory.

Incubation period: Usually 2–6 months, occasionally it may be as short as 10 days or as long as 2 years.

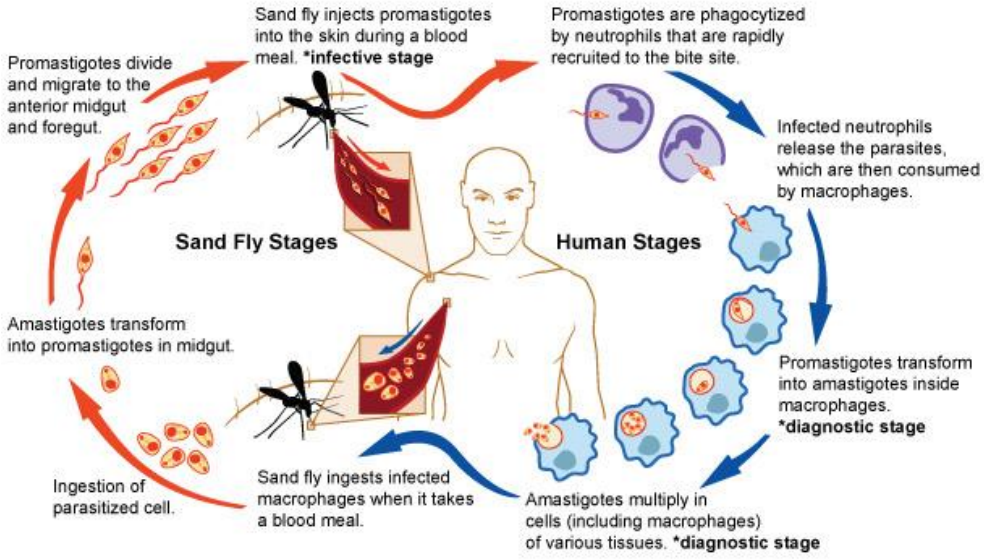
Life Cycle

L. donovani completes its life cycle in 2 hosts (Fig. 5.10).

Defi nitive host: Man, dog, and other mammals.

Vector: Female sandfl y (Phlebotomus species).

Infective form: Promastigote form present in midgut of female sandfl y.



Pathogenicity

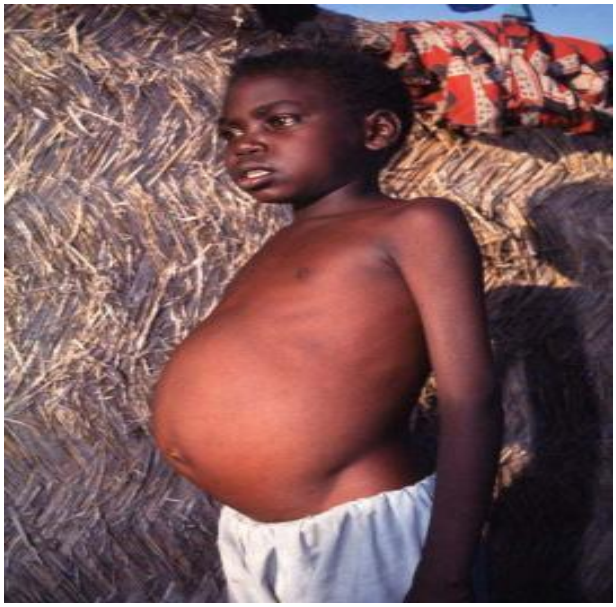
L. donovani causes visceral leishmaniasis or kala-azar.

Kala-azar is a reticuloendotheliosis resulting from the invasion of reticuloendothelial system by L. donovani.

The parasitized macrophages disseminate the infection to all parts

of the body.

In the spleen, liver, and bone marrow particularly, the amastigotes multiply enormously in the fixed macrophages to produce a 'blockade' of the reticuloendothelial system. This leads to a marked proliferation and destruction of reticuloendothelial tissue in these organs.



1- Leishmania tropica

- It includes 3 species:

Leishmania tropica - Leishmania major. Leishmania aethiopica

Disease

All these species **cause old world cutaneous leishmaniasis. The disease is also known as oriental sore, Delhi boil, Bagdad boil,**

a. ***Site of infection*** are essentially the parasite of skin.

b. ***Moist ulcers*** associated wit*h* ***L. tropica major*** - Incubation

period of ***several weeks*** to months is followed by rapid development of weeping ulcers that heal within 6 months.

c. ***Dry ulcers***:- associated with ***L. tropica minor*** - Incubation period may ***last for several years*** before appearance of slowly developing ulcer that is covered with a scaly crust that may take years to heal. Ulcers resemble those produced by the New World form.

A. Life Cycle

The life cycle of *L. tropica* is similar to that of L. donovani except Vectors: The vectors of *L. tropica* complex are Phlebotomus sandflies. The following species of sandflies act as vector.

a. ***Diagnosis:*** - identification of intracellular amastigotes in macrophages from active lesions.-Clinical diagnosis:
   - history of origin specifying the endemic area of residence at the time of development of symptoms
   - morphology of the lesions .immunological and molecular tests
   - **Pathology:** Papule and ulcer are the main pathological lesions. They heal over months to years, leaving scars.

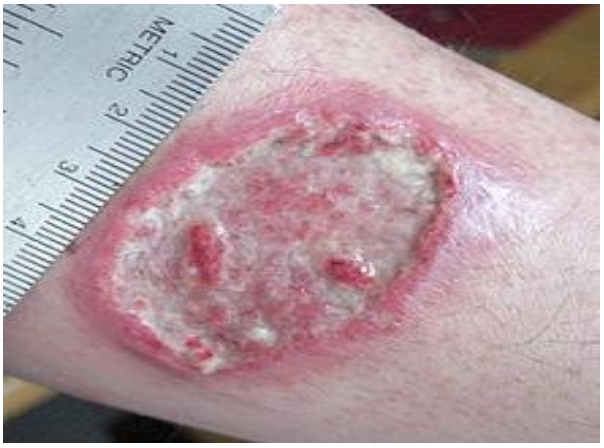
Prophylaxis:

Control of sandfly population by insecticides and sanitation measures.

Personal protection by use of protective clothing and use of insect repellants. Elimination of mammalian reservoir.

-Differences between dry and wet cutaneous leishmaniasis :

Subspecies	***L. tropica***	***L. major***
Distribution	Urban	Rural
Incubationperiod	2~8 mths	1~4 wks
Papule	Brown, 1~3 mm	Red, 5~10 mm
Rupture	3~6 wks	1~3 wks
Lesions	Dry,	Wet,
Site	Face	Lower limbs
Healing	1~2 yrs	3~6 mths



2-Leishmania braziliensis:-

New World Leishmaniasis *L. braziliensis* complex and *L. mexicana*

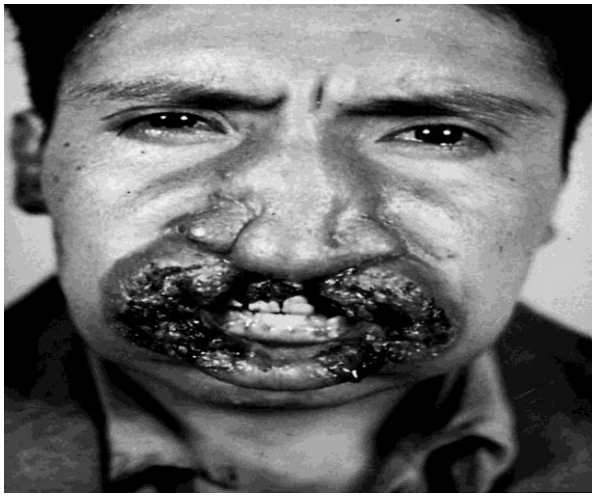
1. ***Disease:*** Mucocutaneous leishmaniasis, espundia.
2. **Site of infection** (oral-nose mucocutaneous tissue)- erosion of oral, nasal structures. Organism spread via bloodstream to other areas, especially the mucous membranes and cartilaginous areas of the nasal and oral structures. Erosion and disfigurement of these areas was called ", **Espundia, Uta, chiclero ulcer)**

Clinical Manifestations:-

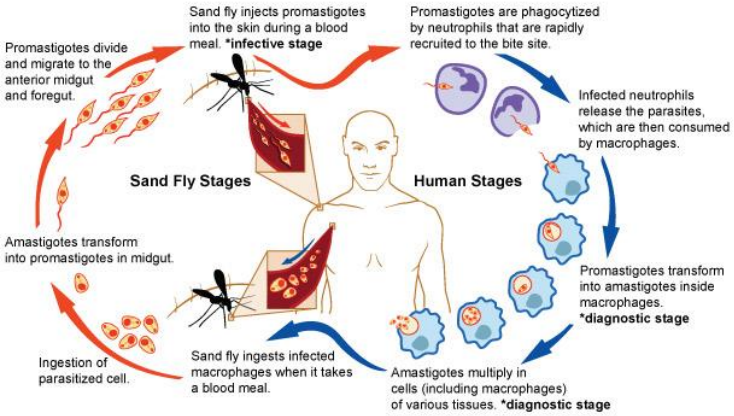
- inital symptoms are similar to that of cutanous leishmaniasis
- single or multpile lesions and ulcers develop at the mucosal regions (nose, mouth, throat cavities) and in the adjacent tissue
- extensive disfiguring of the nasal septum, lips, and palate (does not include the bones)
- extensive disfiguring of the nasal septum, lips, and palate (does not include the bones)

Life cycle

The infection is transmitted to man from animals by bite of sandfly vectors of genus Lutzomiya.



Life Cycle: :(for all)



Leishmaniasis is transmitted by the bite of infected female phlebotomine sandflies. The **sandflies inject the** infective stage (i.e., promastigotes) from their proboscis during blood meals. Promastigotes that reach the puncture wound are phagocytized by macrophages and other types of mononuclear phagocytic cells. Promastigotes transform in these cells into the tissue stage of the parasite (i.e., amastigotes), which multiply by simple division and proceed to infect other mononuclear phagocytic cells. Parasite, host, and other factors affect whether the infection becomes symptomatic and whether cutaneous or visceral leishmaniasis results. Sandflies become infected by ingesting infected cells during blood meals. **In sandflies**, amastigotes transform into promastigotes, develop in the gut in the midgut for organisms in the and migrate to the proboscis.

1. Blood and tissue the Coccidia

a. Have complex life cycles - most have 2-host life cycle.

b. Schizogony - asexual binary fission.

c. Sporogony -sexual reproduction

2. 1-Toxoplasma gondii

Toxoplasma is now recognized as the most common protozoan parasite globally, with the widest range of hosts spread over 200 species of birds, reptiles, and mammals, including humans.

- Cats (both domestic and wild) are the only ***definitive hosts*** and can also be the intermediate hosts
- The disease that *Toxoplasma gondii* caused ***(toxoplasmosis)***

Morphology

T. gondii occurs in 3 forms (Fig. 7.1):

- Trophozoite
- Tissue cyst
- Oocyst.

1) The trophozoite and tissue cyst represent stages in asexual multiplication (**schizogony**), while the the oocyst is formed by sexual reproduction (**gametogony or sporogony**).
2) All 3 forms occur in domestic cats and other felines, which are the defi nitive hosts and support both schizogony and gametogony.
3) Only the asexual forms, trophozoites and tissue cysts are present in other animals, including humans and birds, which are the intermediate hosts.
4) All the 3 forms are infectious to man.

Trophozoites (Tachyzoites)

The trophozoite is crescent shaped, with one end pointed and the other end rounded.

- It measures 3–7 μm in length. The nucleus is ovoid and is situated at the blunt end of the parasite.
- Electron microscopy reveals an **apical complex** at the pointed end

It can invade any nucleated cell and replicate within cytoplasmic vacuoles by a process called **endogony** (**internal budding**), wherein 2 daughter trophozites are formed, each surrounded by a membrane, while still within the parent cell. When the host cell becomes distended with the parasite, it disintegrates, releasing the trophozoites that infect other cells.

During acute infection, the proliferating trophozoites within host cell may appear rounded and enclosed by the host cell membrane. This is, called **pseudocyst** or

colony and can be diff erentiated from tissue cysts by staining reactions.

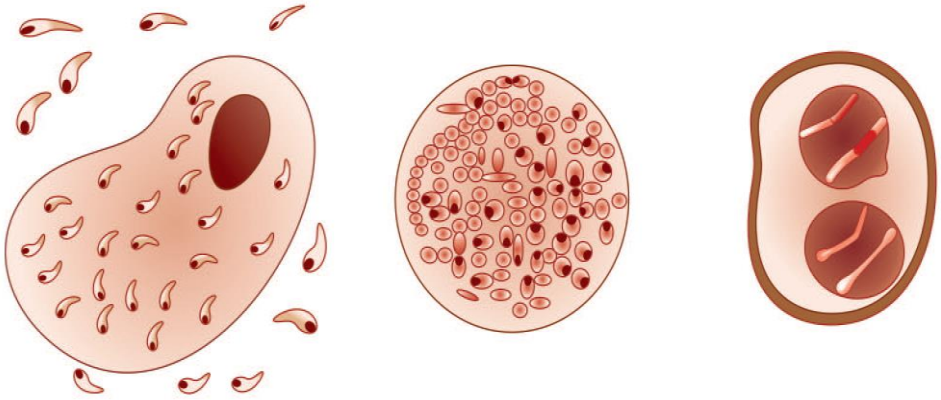
The rapidly proliferating trophozoites in acute infection are called **tachyzoites**

The trophozoites are susceptible to drying, freezethawing, and gastric digestion.

Tissue cyst

Tissue cysts are the resting form of the parasite.

They are found during chronic stage of the infection and can be found in the brain (most common site), skeletal muscles, and various other organs.



Toxoplasma gondii. **A.** Smear from peritoneal fl uid of infected mouse, showing crescentic tachyzoites—extracellular trophozoites and intracellular form within macrophage; **B.** Thickwalled tissue cyst containing rounded formsbradyzoites;

C. Oocyst containing 2 sporocysts with sporozoites inside

The slowly multiplying parasites within the cyst are called **bradyzoites**.

The cyst is round or oval, 10–20 μm in size and contains numerous bradyzoites. Cysts remain viable in tissue for several years.

In immunologically normal hosts, the cysts remain silent, but in the immunodefi cient subjects, they may get reactivated, leading to clinical disease.

It is relatively resistant and when the raw or undercooked meat containing the cysts is eaten, infection occurs.

The cyst wall is disrupted by peptic or tryptic digestion and the released parasites initiate infection by invading intestinal epithelial cells.

They reach various tissues and organs through blood and lymphatic dissemination.

Oocyst

Oocysts develop only in defi nitive hosts – in the intestine of cats and other felines but not in humans.

It is oval in shape and measures 10–12 μm in diameter. Each cyst is surrounded by a thick resistant wall.

The oocysts is formed by sexual reproduction (gametogony).

Cats shed millions of oocysts per day in feces for about 2 weeks during the primary infection. The freshly passed oocyst is not infectious. When the infective oocyst is ingested, it releases sporozoites in the intestine, which initiates infection.

Life Cycle

T. gondii completes its life cycle in 2 hosts (Fig. 7.4).

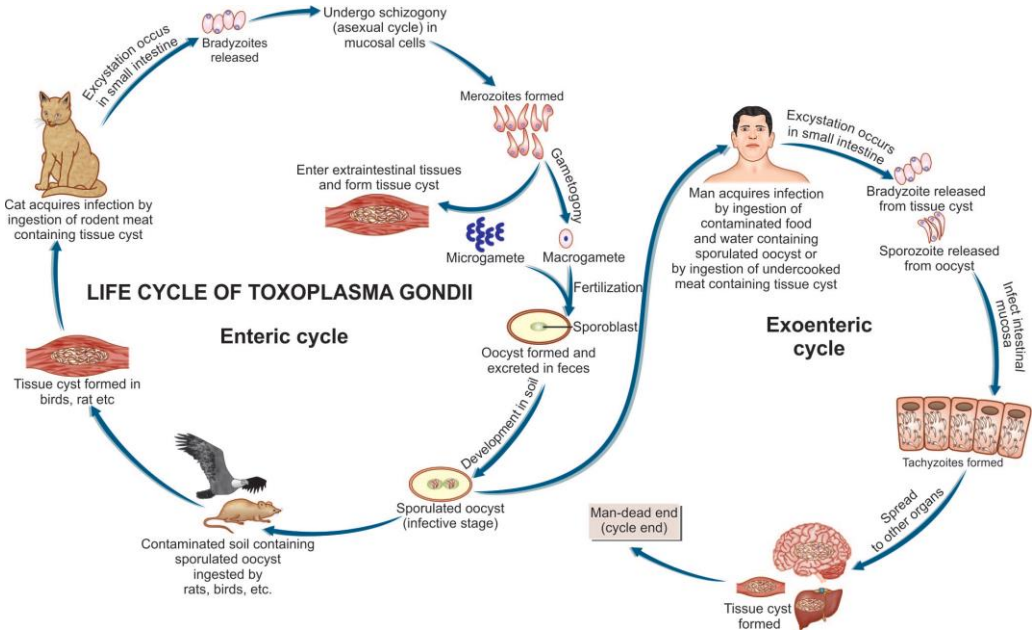
Defi nitive host: Cats and other felines, in which both sexual and asexual cycle takes place.

Intermediate hosts: Man and other mammals, in which only the asexual cycle takes place.

T. gondii has 2 types of life cycles:

Enteric cycle

Exoentric cycle.



Enteric cycle

Enteric cycle occurs in cat and other definitive hosts

Both sexual reproduction (gametogony) and asexual reproduction (schizogony) occur within the mucoscal epithelial cells of the small intestine of the cat.

Cat acquires infection by ingestion of tissue cysts in the meat of rats and other animals or by ingestion of oocysts passed in its feces.

The bradyzoites are released in the small intestine and they undergo asexual multiplication (schizogony) leading to formation of merozoites.

Some merozoites enter extraintestinal tissues resulting in the formation of tissue cysts in other organs of the body.

Other merozoites transform into male and female gametocytes and sexual cycle (gametogony) begins, with the formation of **microgamete** and **macrogamete**.

A macrogamete is fertilized by motile microgamete resulting in the formation of an oocyst, which passes through maturation stages (**sporulation**) in the soil after being excreted from host through feces.

A mature oocyst containing 8 sporozoites is the infective form which may be ingested by rats or other mammals to repeat the cycle

Exoenteric cycle

Exoenteric cycle occurs in humans, mice, rats, sheep, cattle, pigs and birds , which are the intermediate hosts. Humans acquire infection after:

- Eating uncooked or undercooked infected meat,particularly lamb and pork containing tissue cysts
- Ingestion of mature oocysts through food, water, or fingers contaminated with cat feces directly or
- indirectly
- Intrauterine infection from mother to fetus (**congenital toxoplasmosis**)
- Blood transfusion or transplantation from infected donors.

Sporozoites from the oocysts and bradyzoites from the tissue cysts enter into the intestinal **mucosa** and multiply **asexually** and **tachyzoites** are formed (**endodyogeny**).

Tachyzoites continue to multiply and spread locally by lymphatic system and blood.

Some tachyzoites also spread to distant extraintestinal organs like brain, eye, liver, spleen, lung, and skeletal muscles and form **tissue cysts**. The slowly multiplying forms inside the tissue cysts are known as **bradyzoites**, which remain viable for years.

The dormant bradyzoites inside the cyst may be reactived in immune suppression causing renewed infection in the host

Note: Human infection is a dead end for the parasite

Pathogenicity and Clinical Features

The outcome of *Toxoplasma* infection depends on the immune status of the infected person.

Active progression of infection is more likely in immunocompromised individuals. Toxoplasmosis has

acquired great importance as one of the major fatal complications in acquired immunodeficiency syndrome (AIDS).

Most human infections are asymptomatic.

Clinical toxoplamosis may be congenital or acquired.

Congenital toxoplasmosis

Congenital toxoplasmosis results when *T. gondii* is transmitted transplacentally from mother to fetus.

This occurs when the mother gets primary toxoplasma infection, whether clinical or asymptomatic, during the pregnancy.

The risk of fetal infection rises with progress of gestation; from 25%, when the mother acquires primary infection in first trimester

to 65% 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, the tissue cyst may be reactivated during pregnancy and liberate trophozoites, which may infect the fetus *in utero*

The manifestations of congenital toxoplasmosis include chorioretinitis, cerebral calcifications, convulsions, strabismus, deafness, blindness, mental retardation, microcephaly, and hydrocephalus.

How do people get toxoplasmosis?

- Eating undercooked, contaminated meat (especially pork, lamb, and venison).
- Accidental ingestion of undercooked, contaminated meat after handling it and not washing hands thoroughly (Toxoplasma cannot be absorbed through intact skin).
- Eating food that was contaminated by knives, utensils, cutting boards and other foods that have had contact with raw, contaminated meat.
- Drinking water contaminated with Toxoplasma gondii.
- Mother-to-child (congenital) transmission.
- Receiving an infected organ transplant or infected blood via transfusion, though this is rare.
- Organ transplantation or blood transfusion from an infected

person.

Prevention:

- ***Wear gloves when you garden or handle soil***.
- Don't eat raw or undercooked meat.
- Wash kitchen utensils thoroughly.
- Wash all fruits and vegetables.
- Don't drink unpasteurized milk.
- Cover children's sandboxes.

Diagnosis:

- Microscopic Examination
  - Smears and Sections
  - Specimens
  - Blood, Sputum, CSF, bone marrow
  - Tissue Biopsy
- Animal Inoculation
- Serological tests – IHA, IFA, ELISA (IgM/IgG)
- PCR & DNA probes

Subphylum Sporozoa - Malaria

Introduction

There are four species normally infecting humans

P. vivax Benign tertian/ vivax malaria

P. falciparum Malignant tertian/ falciparum malaria, black water

fever

P. malariae Quartan malaria

P. ovale Tertian/ Ovale malaria

P. knowlesi Often cause severe malaria

Host:

- **The mosquitoes**: play as essential hosts that has the sexual stages, **Man**: act as intermediate host being harbor the asexual stages of parasites.

- **Exoerythrocytic cycle** :In the human body, there are two cycles, one in the liver which called the Exoerythrocytic cycle or Pre-erythrocytic cycle.
- **Erythrocytic cycle**: take place in the red blood cells, so called the Erythrocytic cycle.

Geographical distribution

P. vivax is abundant in tropical and temperate countries, while *P. falciparum* is

more common in tropical zones, *P. ovale* and *P. malaria* are present in temperature zones, but are less common in tropical climates.

1. **Primary tissue phase**
    - Sporozoite gradually changes in liver cell to trophozoite form.
    - This trophozoite becomes amoeboid in shape and consumes liver cell to grow and mature.
    - After maturation, each trophozoite divides extremely and changes to a preerythrocytic schizont

- During that division, the nucleus of trophozoite divides into a large number of
- nuclei, followed by division of cytoplasm to form thousands of merozoites inside the liver cell.
- The time needed for the pre-erythrocytic schizont (incubation period) to form and the number of merozoites in, it differ according to species of *Plasmodium*.
- The incubation period for *P. vivax* is eight days, *P. malaria* 12 days, *P. ovale* nine days and finally five days for *P. falciparum*.

2. Secondary tissue phase

- At the same time, some of the pre-erythrocytic merozoites invade the red blood cells thus initiating the erythrocytic schizogony.
- In *P. falciparum*, once pre-erythrocytic merozoites are liberated from the liver
- cells, they invade only the red blood cells and never reinvade the liver, so the relapses are absent in *P. falciparum*.
- The reinvasion of the liver cells in the three benign malaria species is responsible for relapses after apparent cure.

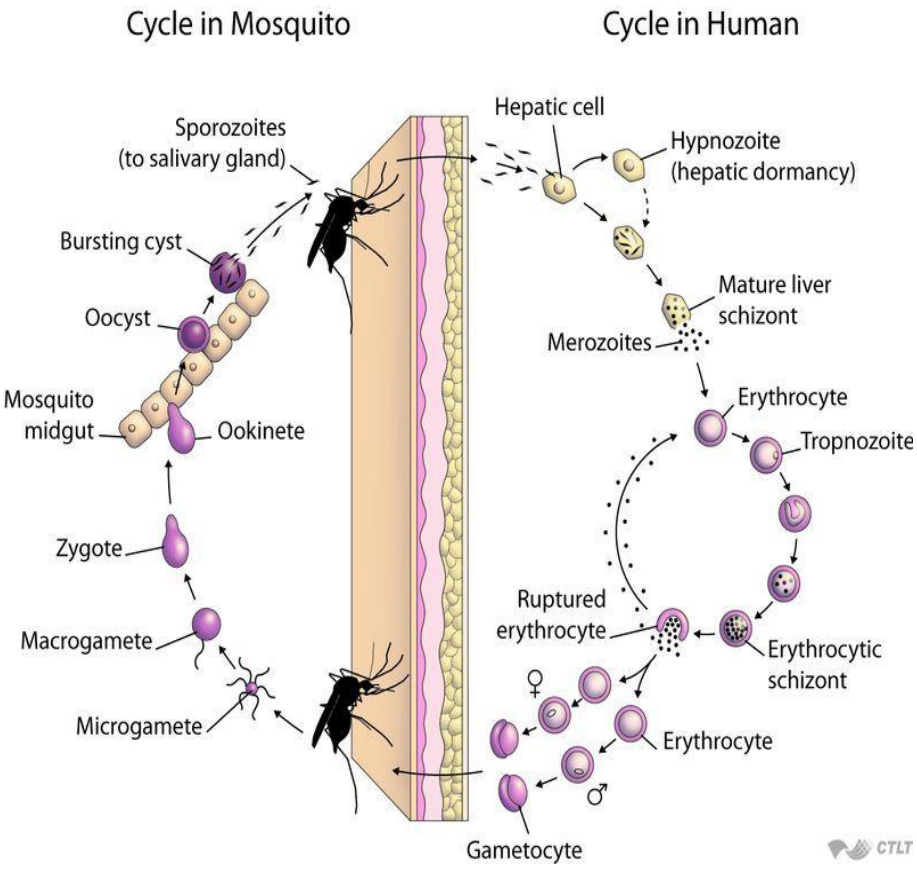
3. Erythrocytic Schizogony

- Merozoites discharged from pre-erythrocytic schizont enter the red cells by invagination of its cell membrane.

- The merozoite is transformed into:
  1) Ring stage or young trophozoite
  2) Amoeboid stage or old trophozoite, which digests hemoglobin to form the
  3) malarial pigments namely **Schuffner's dots in *P. vivax*** and ***P. ovale*, Ziemann's**
  4) **dots in *P. malaria*** and **Maurer's dots in *P. falciparum***.
- The old trophozoite undergoes schizogony to form schizont stage containingmerozoites. The liberated merozoites invade new red blood cells, repeating the cycle. Rupture of erythrocytic schizonts release haematin pigments and parasite toxins, which responsible for fever and sweating.
- When the schisonts rupture, fever and other symptoms take place, and this reoccurs every third day, and thus the fever is **called tertian fever**, and the disease is **called benign tertian malaria**.
- In *P. falciparum*, the **fever is irregular**, it may be tertian, quatrain or even continuous.
- After completing few schizogony cycles, some merozoites develop into male microgametocytes and female macrogametocytes.
- All erythrocytic stages of *Plasmodium* are found in peripheral blood, except trophozoites and **schizonts of *P. falciparum*, which are trapped in blood vessels of internal organ.**

4. The Sexual cycle (Sporogony)

- This occurs inside the insect which is an anopheline mosquito. When it sucks a blood meal of a patient containing all stages and the gametocytes, all stages of malaria are digested in the insect mid-gut except the gametocytes.
- The male microgametocyte nucleus divides into 4-8 fragments, to form finally the microgametes.
- The female macrogametocyte throws two polar bodies to reduce its chromatin before the fertilization occurs.
- After fertilization by fusion of the macro and micro gametes, the zygote is formed.
- The zygote throws out pseudopodia to form a motile stage called Ookinete.
- Ookinete penetrates the mucous membrane of gut, to settle beneath it, which is called oocyst.
- Its nucleus divides many times to produce fine spindle shaped called sporozoites.
- The oocyst ruptures and sporozoites are free to migrate to the salivary glands, they pass with saliva into human body while the mosquito is taking a blood meal.



Pathogenesis]

Three organs show gross pathological lesions namely the liver, the spleen andbone marrow. Liver shows necrosis and enlargement, spleen may enlarge andtender, bone marrow becomes vascular, chocolate brown due to deposition ofpigment.

- In P. *falciparum*, the disease is characterized by its common malignant features,due to:

1. Agglomeration of parasitized and non-parasitized red blood cells inside capillaries of the internal organs, this causes plugging of the human capillaries, and causes thrombosis and sometimes hemorrhages

2. In chronic malignant malaria, rapid intravascular haemolysis occurs, leads to passage of dark urine due to presence of haemosiderin, which called **"black water fever".**

[Diagnosis]

- Clinical symptoms and signs such as fever, sweating, enlarged and tender spleen and liver, and Jaundice may occur.
- Direct methods such as thin and thick blood film.
- Serological test.

[Prevention and control]

- Treatment of patients.
- control the *Anopheles* mosquitoes, adults and larvae

1. ***Transmission*: -** naturally acquired infections are via the bite of infected female *Anopheles* mosquitoes. Malaria is also transmitted via blood transfusion, sharing of contaminated needles among IV drug abusers, and congenital transmission also has been documented.
2. ***Immunity: -*** incomplete immunity follows infection. Some persons get reinfected over and over.

a. *Sickle cell trait:* - the malaria parasite is not successful at utilizing the "S" haemoglobin. This trait does not confer immunity to infection, but does offer resistance to infection.

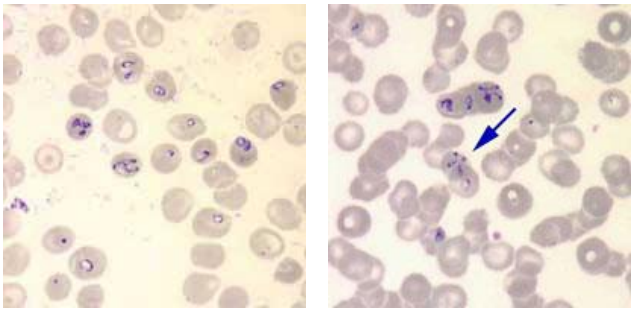
b. ***Duffy factor*** :- represents the "portal of entry" antigen for

P. vivax. Persons without the factor are immune to this species (but not the others).

Pathology/Pathogenesis:

1. ***Prodromal stage :–*** a time period which can include premonitory symptoms such as headache, myalgia, anorexia & nausea prior to the paroxysm.
2. ***Paroxysm: -*** period of chills & fever followed by profuse sweating.

A. ***Babesia:-***



Babesia sp. rings in red blood cells

Babesia sp. "maltese cross" (arrow)

Babesia are intraerythrocytc sporozoan parasites that morphologically resemble *plasmodium* and cause tickborne malaria like illness in domestic and wild animals. It causes opportunistic infection in humans.

Habitat

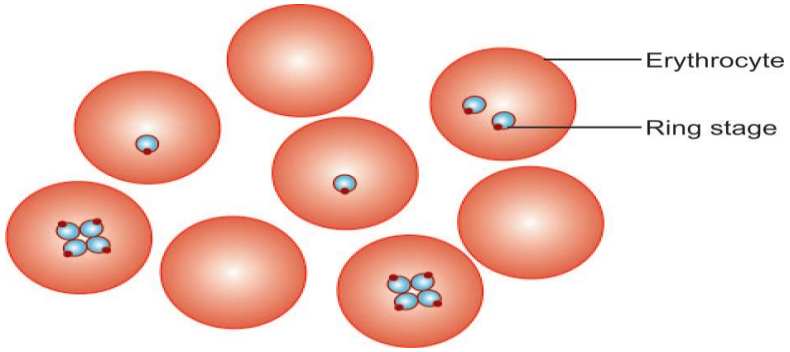
The parasite is present in erythrocytes and resembles the ring stage of *P. falciparum.*

Morphology:

Morphology

Trophozoites are pleomorphic 2–5 μm in diameter found inside the red cells. The shape may be pyriform, amoeboid, or spindle-like, usually in pairs and are often mistaken as ring form of *Plasmodium*. Merozoites may be spherical or oval or pyrifom bodies, found in pairs.

Disease name: - **babesiosis, piroplasmosis. "Piro" from "pyro"- Latin for "fire".** Because the disease is fever (fire) inducing. The "piroplasms" are a group of fever-inducing organisms or infections. Malaria is a piroplasm also.



Life Cycle

Defi nitive host: *Ixodid* ticks.

Intermediate host: Man or other mammals.

Infective form: Sporozoites are the infective form for humans.

Mode of Transmission: Infection in vertebrate occurs through bite of the nymphal stage of *Ixodid* ticks.

Incubation period is 1–6 weeks Babasiosis can also be transmitted via blood transfusion. Transovarian transmission in ticks also occurs.

In their life cycle, merogony takes place in vertebrate hosts and sporogony in the invertebrates.

Man acquires infection by bite of the infected ticks

(**definitive host**).

Sporozoites present in the salivary glands of tick are introduced in man or other mammals (**intermediate host**).

Sporozoites change to trophozoites in the circulation, which then invade the RBCs and multiply asexually by binary fi ssion or schizogony to form 4 or more trophozoites. Newly formed trophozoites are released by rupturing erythrocytes and invade new erythrocytes.

Some of the sporozoites grow slowly inside red cells and become folded like an accordion. These are thought be gametocytes.

Female ticks become infected by feeding the host blood.

In the digestive tract of tick, the gametocytes multiply sexually and later migrate to the salivery glands where they divide by multiple fi ssion into smaller forms known as '**vermecules'**.

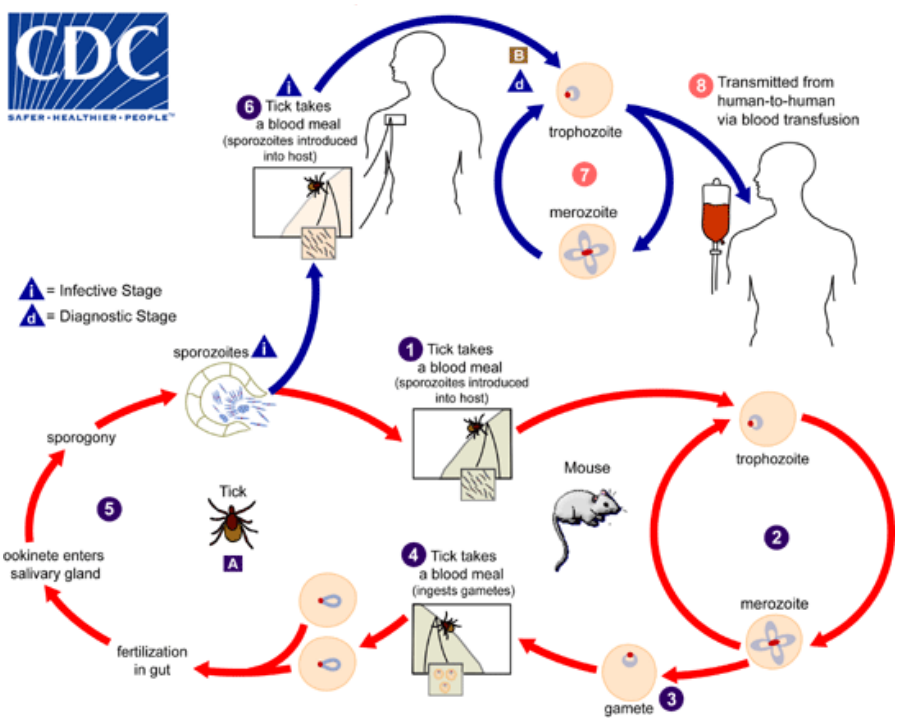
Vermecules undergo secondary schizogony to produce sporozoites, which are the infective forms for human.

Pathogenecity and Clinical Features

Hemolysis of the infected erythrocytes is primarily responsible for many clinical manifestations.

There is accumulation of parasites in the capillaries of liver, spleen, and kidneys which leads to cellular degeneration and necrosis.

In acute disease, there is malaise, fatigue, fever, myalgia,arthalgia, dry cough and anorexia. Fever exceeds 38°C and can reach 40.6°C accompanied by chill and sweat.



Diagnosis:

Molecular techniques, including qPCR on EDTA blood

Serological testing, microscopic examination for blood film

Prevention:

Avoidance of known tick areas, particularly during “tick seasons”, use of an effective anti-tick product and daily checking for/effective removal of ticks may help reduce the risk of disease transmission.

Differences between the human species of plasmodium

	Plasmodium vivax	Plasmodium malariae	Plasmodium falciparum	Plasmodium ovule
1. Geographical distribution	Tropical and temperate region. Very common.	Tropical and temperate region. Not very common.	Tropical region. Very common.	West Africa and South America.
2. Age of erythrocytes preferred	Young	Old	All ages	Unknown
3. No. of parasites per cubic mm. of blood.	20-40,000	5-10,000	50-1,00,000	-
4. Trophozoite	Ring like form is large and grows into a highly amoeboid form with prominent vacuoles.	Ring like form is large and grows into a slightly amoeboid band like form with inconspicuous vacuoles.	Ring like form is rather small and grows into a compact amoeboid form. There may be 2 or more parasites in one erythrocyte.	Ring like form is large and grows into amoeboid form.
5. Schizont	Large than a normal erythrocyte.	Slightly smaller than a normal erythrocyte.	Much smaller than a normal erythrocyte.	Smaller than a normal erythrocyte.
6. Infected erythrocyte	Becomes enlarged and frequently distorted.	Does not enlarge.	Does not enlarge may get slightly distorted.	Becomes enlarged and oval.
7. Haematin	Light brown in fine granules evenly	Dark brown in coarse granules	Dark in one or two solid masses.	Light brown scattered.