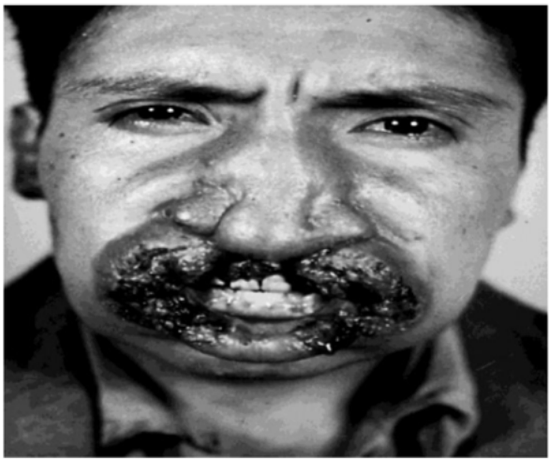
Clinical Manifestations:-

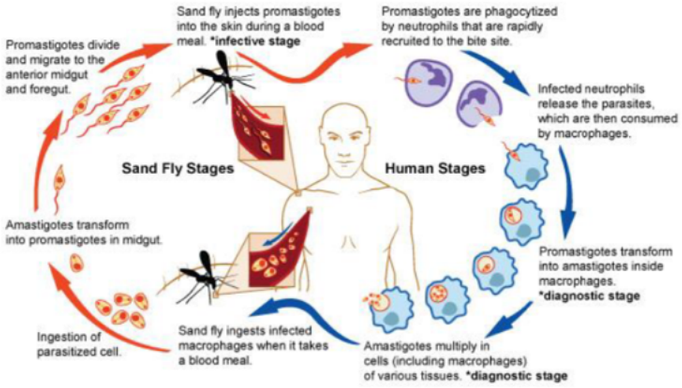
- inital symptoms are similar to that of cutanous leishmaniasis
- single or multpile lesions and ulcers develop at the mucosal regions (nose, mouth, throat cavities) and in the adjacent tissue
- extensive disfiguring of the nasal septum, lips, and palate (does not include the bones)
- extensive disfiguring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

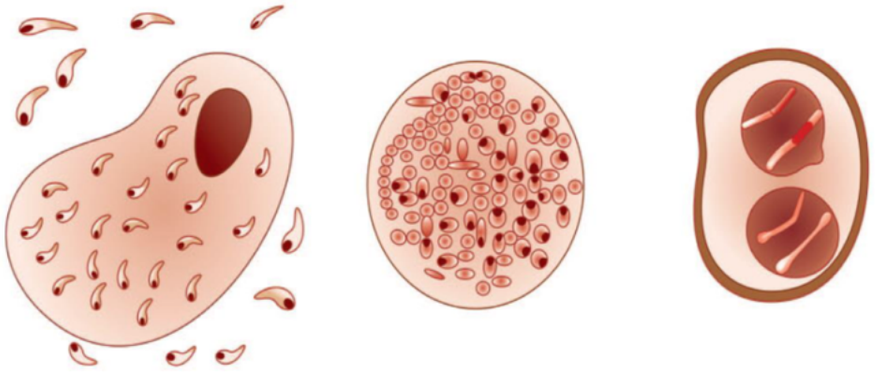
The rapidly proliferating trophozoites in acute infection are called **tachyzoites**

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).

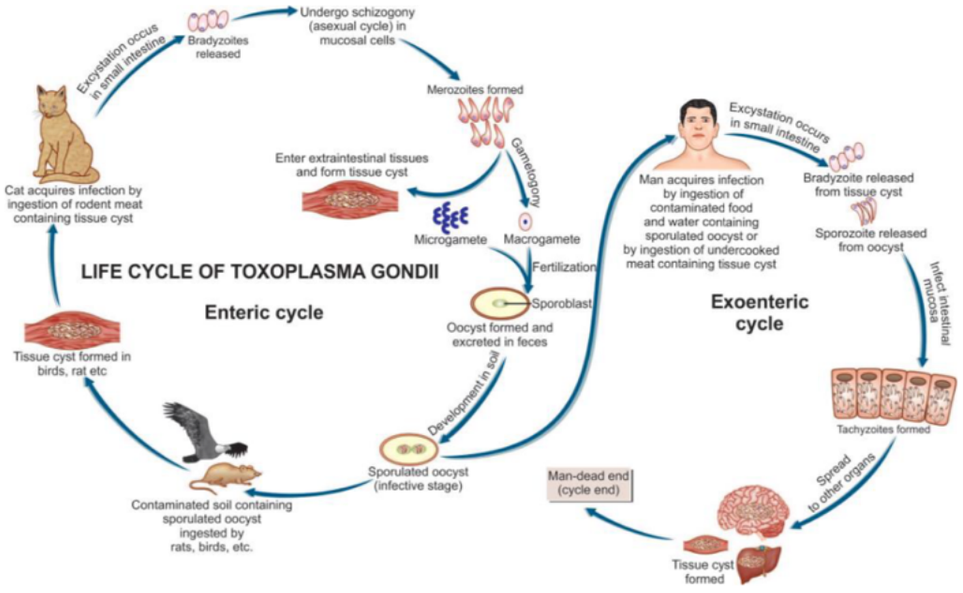
Defi nitive host: Cats and other felines, in which both sexual and asexual cycle takes place.

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