MALARIA

Etiology
Four *Plasmodium* species are responsible for human malaria These are

1-*P. falciparum*, 2-*P. vivax*,3-*P. ovale* and 4-*P. malariae*.

Epidemiology
There are an estimated 200 million global cases of malaria leading a mortality of more than one million people per year.

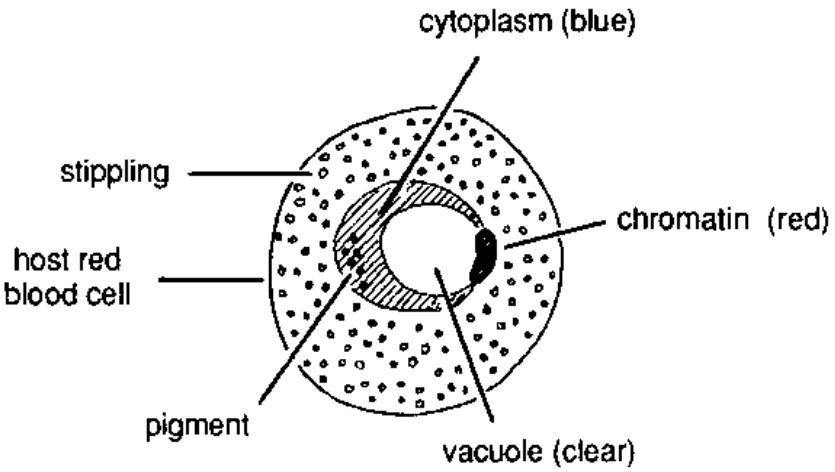
P. falciparum and *P. malaria*are the most common species of malarial parasite and are found in Asia and Africa. *P. vivax* in Latin America, India whereas, *P. ovale* is almost found in Africa

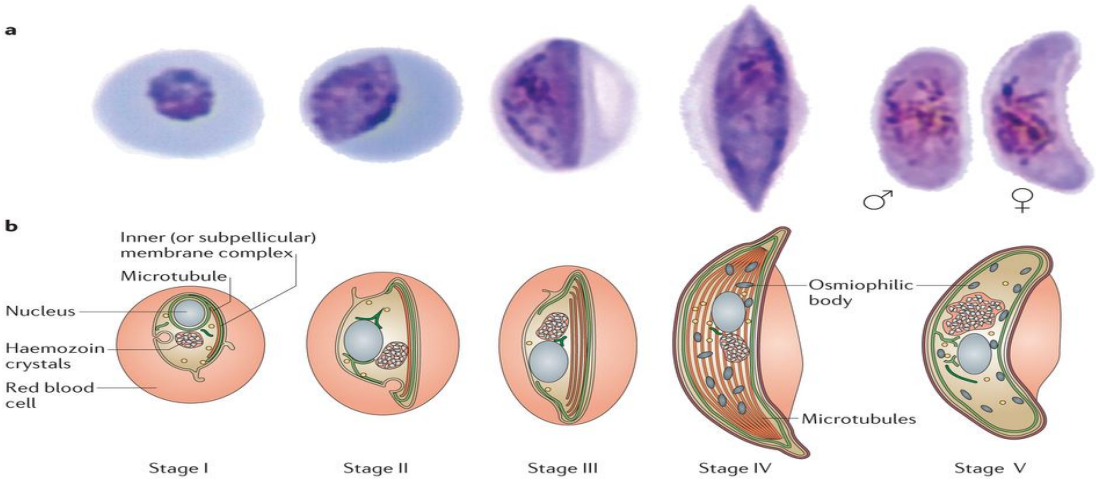
Morphology
Malarial parasite **trophozoites** are generally **ring shaped**, 1-2 microns in size, although other forms (**ameboid and band)** may also exist.

The sexual forms of the parasite**(gametocytes)** are much larger and 7-14 microns in size.

P. falciparum is the largest and is **banana shaped** while others are smaller and **round.**

P. vivax causes stippling of infected red cells





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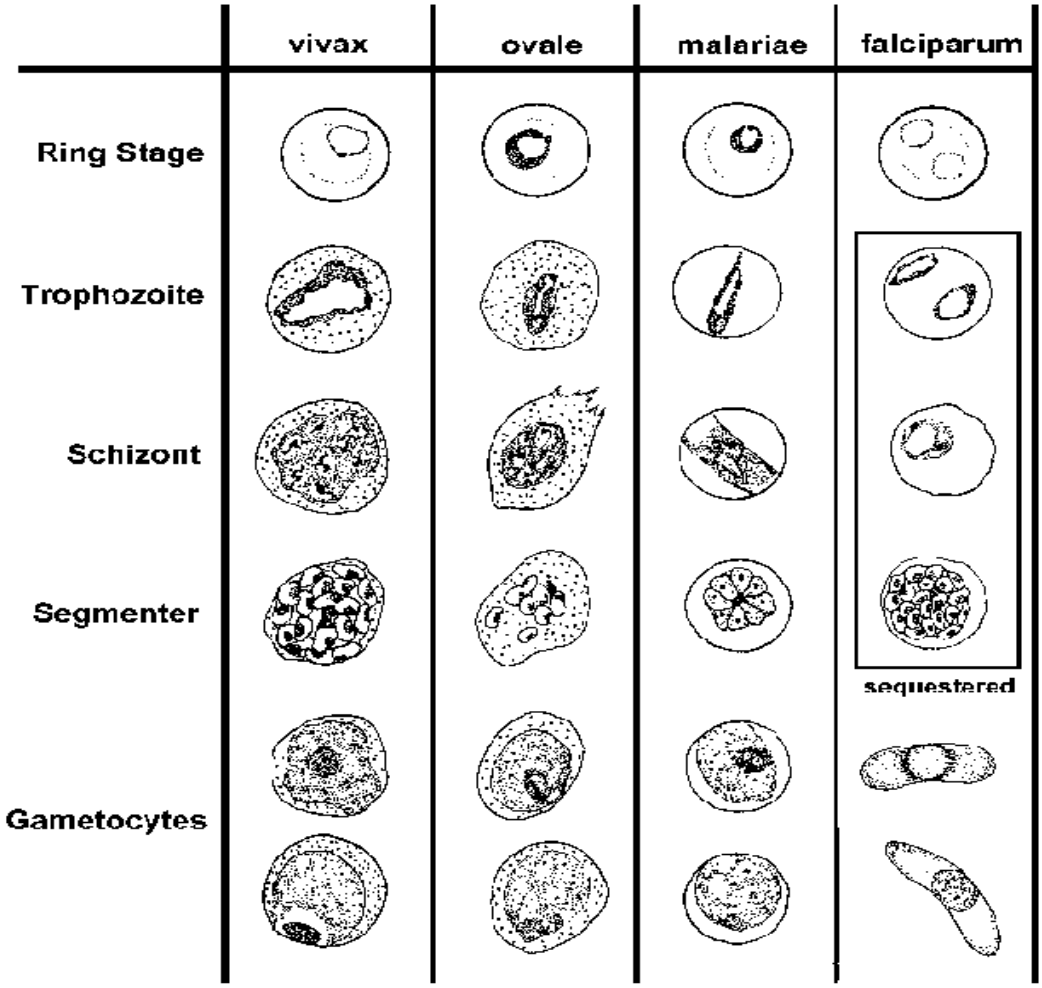


Life cycle

Malarial parasites are transmitted by the infected female anopheline mosquito which Injects**sporozoites** present in the saliva of the **insect** Sporozoites infect the

-**liverparenchymal cells**where they may remain dormant or undergo stages of **schizogony** to produce **schizonts** and **merogony** to produce merozoites (meronts). When parenchymal cells rupture, thousands of **meronts** are released into blood and infect the red cells

. In red cells,the parasites mature into **trophozoites**. These trophozoites undergo schizogonyand merogony in red cells whichburst and release **daughter merozoites**.

Some of themerozoites transform into**male and female gametocytes** while others enter red cells tocontinue the erythrocytic cycle.

-**The gametocytes are ingested** by the female mosquito,

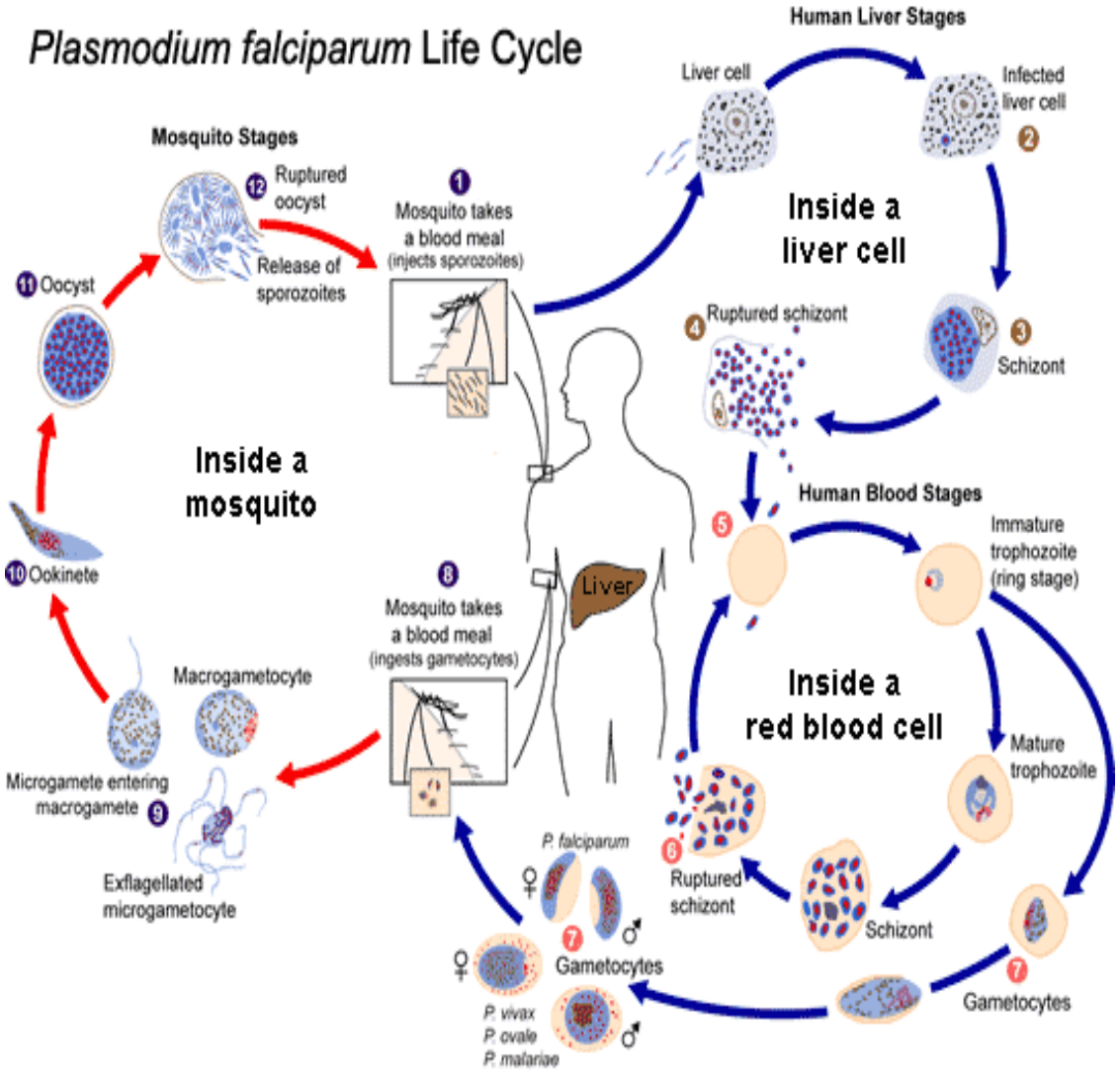
The gametocytes, male (microgametocytes) and female (macrogametocytes), are ingested by an *Anopheles* mosquito during a blood meal ❽. the microgametes penetrate the macrogametes generating **zygotes**

-The **zygotes** in turn become **motile** and elongated **(ookinetes)**❿which invade the midgut wall of the mosquito where they develop into **oocyst**⓫ The **oocysts** grow, rupture, and release **sporozoites**⓬,which make their way to the mosquito's salivary glands.

Inoculation of the sporozoites into a new human host perpetuates the malaria life cycle

❶ . The liver (extraerythrocytic) cycle takes 5-15 days whereas the erythrocytic cycle
❶ takes 48 hours or 72 hours

(*P. malariae*). Malaria can be transmitted by transfusion a nd transplacentally.


Plasmodium falciparum Life Cycle
Human Liver Stages
Liver cell
Infected liver cell
2
Inside a liver cell
Ruptured schizont
4
3
Schizont
Human Blood Stages
5
Immature trophozoite (ring stage)
Inside a red blood cell
Mature trophozoite
Ruptured schizont
6
Schizont
7
Gametocytes
P. falciparum
7
Gametocytes
P. vivax
P. ovale
P. malariae
8
Mosquito takes a blood meal (ingests gametocytes)
Liver
Macrogametocyte
Microgamete entering macrogamete
9
Exflagellated microgametocyte
10 Ookinete
11 Oocyst
Inside a mosquito
Mosquito Stages
12 Ruptured oocyst
Release of sporozoites
1
Mosquito takes a blood meal (injects sporozoites)

Symptoms

The symptomatology of malaria depends on the parasitemia, the presence of the organism in different organs and the parasite burden. The incubation period varies generally between 10-30 days., the patient develops headache , pains in the bones and joints, chilly sensations and fever.

As the disease progresses, the chills and fever The chill and fever follow (paroxysm) with the symptomatic period lasting 8-12 hours.

In between the symptomatic periods, there is a period of relative normalcy, that progresses to teeth chattering, cyanotic lips and nails (cold stage).

This lasts for about an hour. At the end of this period, The patient experiences profuse perspiration andthe temperature begins to drop.

. Each paroxysm is due to the rupture ofinfected erythrocytes and release of parasites.

Without treatment, all species of human malaria may ultimately result in spontaneous cure except with *P. falciparum* which becomes more severe and results in death.Chronic malaria results in splenomegaly, hepatomegaly .

Diagnosis

Diagnosis is based on symptoms and detection of parasite in Giemsa stained blood smears.

There are also antibody tests

Treatment and Control

Treatment is effective with various quinine derivatives

. Control measures are eradication of infected anopheline mosquitos.

Vaccines are being developed and tried but none is available yet for routine use.

TOXOPLASMOSIS

Etiology

Toxoplasma gondii is the organism responsible for toxoplasmosis

T. gondii infection occurs worldwide with about 5% to 50% of adults in the United States of America having toxoplasma antibodies. About 1% to 6% of domestic cats in Europe excrete oocyst of toxoplasma gondii in their feces and about 1% of cats in the United States shed the Toxoplasma cysts. Sheep, Goats, Cattle and Pigs are frequently infected. Toxoplasma infection is usually sporadic, but outbreaks associated with ingestion of raw meat or contaminated water supply.

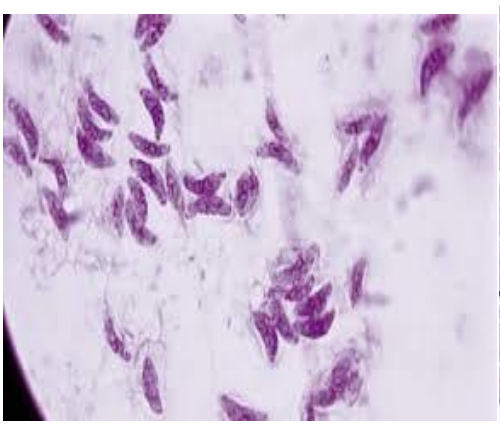
1. **Epidemiology :** the parasite is found throughout the world, more than 60 million people in the United States may be infected with the *Toxoplasma* parasite. Of those who are infected, very few have symptoms because a healthy person's immune system usually keeps the parasite from causing illness. However, pregnant women and individuals who have compromised immune systems should be cautious; for them, a *Toxoplasma* infection could cause serious health problems **(CDC , 2017)**

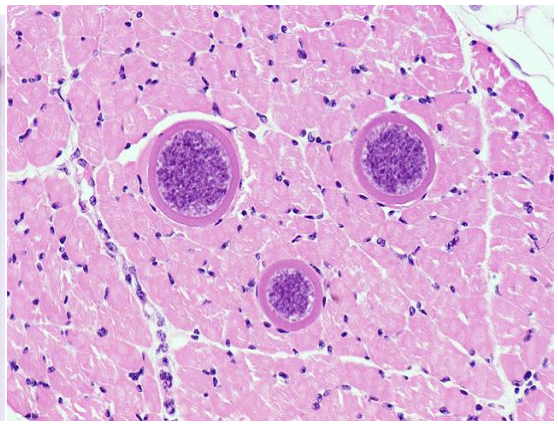
Morphology

Tachyzoites – these are the proliferative forms of toxoplasma gondii that reproduce rapidly within macrophages by endogenous budding. Tachyzoites are sickle-shaped cells that are about 4 to 7 micrometer long and 2 to 4mm wide.

2. **Bradyzoites** (also called **cystozoites**) – these are the forms that undergo slow reproduction within the cysts. The cysts have relatively resistant walls and can grow as large as 150 micrometer – containing up to several thousand bradyzoites.

3. **Oocysts** are the rounded and encysted stages of toxoplasma parasite that have resistant cyst walls – measuring about 9 by 14 micrometer in size.





Life cycle

The natural life cycle of *T. gondii* occurs in cats and small rodents, although the

parasite can grow in the organs (brain, eye, skeletal muscle, etc.) of any mammal or birds

Cats gets infected by -**ingestion of cysts in flesh.**

- Decystation occurs in the small intestine, and the organisms penetrate the submucosal epithelial cells where they undergo several generations of mitosis,

finally resulting in the - development of micro- (male) and macro- (female) gametocytes.

- Fertilized macro-gametocytes develop into oocysts that are discharged into the gut

lumen and excreted.

- Oocysts sporulate in the warm environment and are infectious to a variety of animals including rodents and man.

- Sporozoites released from the oocyst in the small intestine penetrate the intestinal

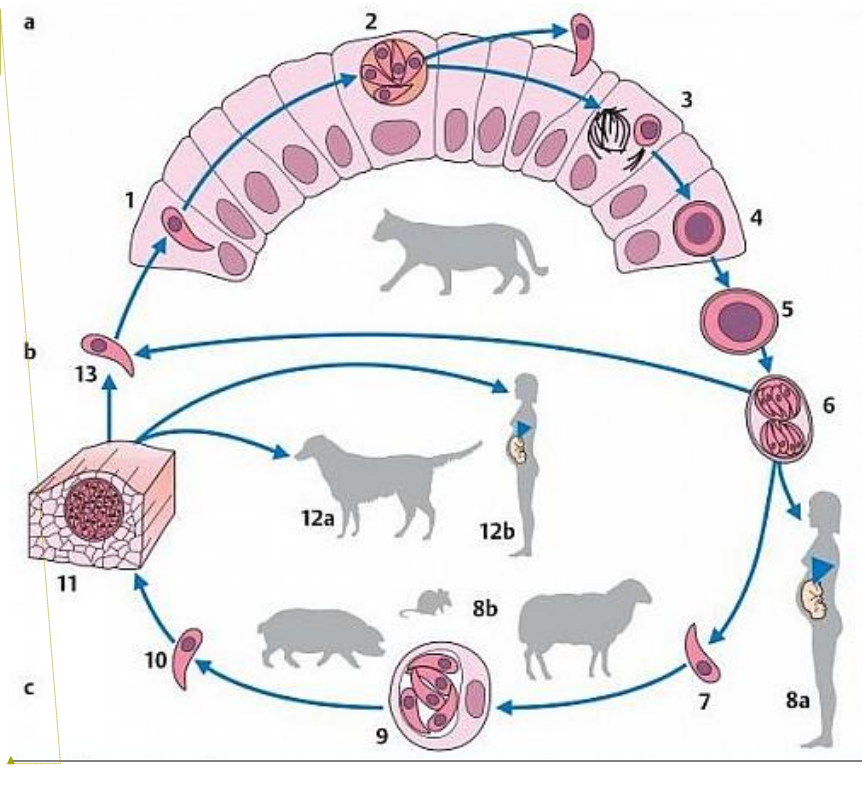
mucosa and find their way into **macrophages** where they divide very rapidly) and form

a cyst which may occupy the whole cell.

- The infected cells burst and release the tachyzoites to enter other cells, including

muscle and nerve cells, where they are protected from the host immune system and **multiply slowly (bradyzoites).**

منسّق:الخط: (افتراضي) Arial، ٩ نقطة، مائل، خط اللغة العربية وغيرها: Arial، ٩ نقطة، مائل



- **Symptoms**
  - Most people who become infected with *Toxoplasma gondii* are not aware of it.
- Some people who have toxoplasmosis may feel as if they have the “flu” with swollen lymph glands or muscle aches and pains that last for a month or more.
- Severe toxoplasmosis, causing damage to the brain, eyes, or other organs, can develop from an acute *Toxoplasma* infection or one that had occurred earlier in life and is now reactivated. Severe cases are more likely

in individuals who have weak immune systems, though occasionally, even persons with healthy immune systems may experience eye damage from toxoplasmosis.

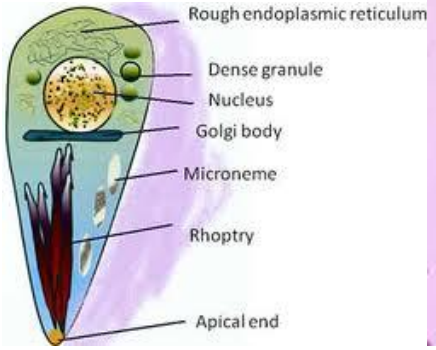
- Signs and symptoms of ocular toxoplasmosis can include reduced vision, blurred vision, pain (often with bright light), redness of the eye, and sometimes tearing. Ophthalmologists sometimes prescribe medicine to treat active disease. Whether or not medication is recommended depends on the size of the eye lesion, the location, and the characteristics of the lesion (acute active, versus chronic not progressing). An ophthalmologist will provide the best care for ocular toxoplasmosis.
- Most infants who are infected while still in the womb have no symptoms at birth, but they may develop symptoms later in life. A small percentage of infected newborns have serious eye or brain damage at birth.

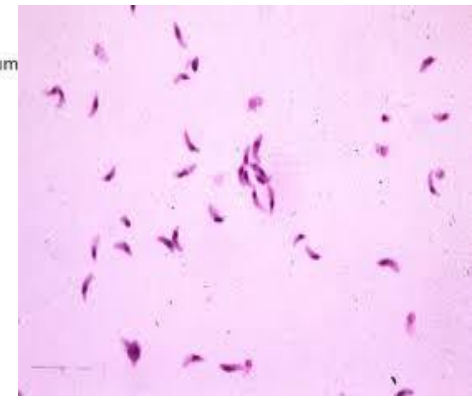
Diagnosis

Suspected toxoplasmosis can be confirmed by isolation of the organism from tonsil or lymph gland biopsy.

Treatment

Acute infections benefit from pyrimethamine or sulphadiazine. Spiramycin is a successful alternative. Pregnant women are advised to avoid cat litter and to handl e uncooked and undercooked meat carefully.





What is Toxoplasma gondii?

Toxoplasma gondii is a protozoan parasite that infects humans as well as many other animals such as pig, sheep and cattle; it is the causative agent of an infectious disease known as Toxoplasmosis. Toxoplasma gondii is one of the organisms that cause the **TORCH infections** - Toxoplasmosis, Others (such as syphilis, varicella-zoster, parvovirus B19), Rubella, Cytomegalovirus (CMV) and Herpes (common infections that can cause congenital anomalies).

Toxoplasma gondii transmission

1. Humans are commonly infected by eating undercooked meat containing the cyst or by eating food or drinking water that is contaminated with the feces of an infected cat containing the oocyst.
2. A fetus in the womb can be infected when the parasite in an infected pregnant mother crosses the placenta to infect the baby. The earlier the infection in pregnancy, the worse the prognosis
3. Someone who has had toxoplasma gondii infection in the past and the immune system was able to control the infection can still get reinfected whenever the immune system is suppressed. This occurs by reactivation of the oocysts that were in the body issues
4. A rare mode of transmission of *t. gondii* is through organ transplants where an infected organ is transplanted to an uninfected recipient

Toxoplasma gondii Epidemiology

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supply.

Toxoplasma gondii Characteristics

1. The definitive host of this parasite is the domestic cat and other members of the feline family
2. Humans and other mammals are the intermediate hosts
3. The rapidly multiplying forms of Toxoplasma gondii are called **Tachyzoites** – these reproduces by asexual reproduction inside macrophages and infects more cells
4. The slowly multiplying forms of Toxoplasma gondii are called **Bradyzoites** – these form are cyst that have been controlled by the immune system and they remain in tissues without causing problems in a competent immune system but become reactivated and serve as source of infection when the immune system becomes suppressed by any disease such as tuberculosis, diabetes, HIV/AIDs, chemotherapy etc. The bradyzoites are important for diagnosis of the disease when seen in tissue specimens
5. The cyst of Toxoplasma gondii can still be viable for up to 3 weeks in meat when stored at a temperature of 48 ^{o}C and can still be infectious at this stage; but deep-freezing of the meat to –208 ^{o}C kills the bradyzoites within 3 days. Toxoplasma gondii oocysts are resistant to harsh environmental conditions but are rapidly killed when heated to 708 ^{o}C.

Anatomy of the different life cycle stages of Toxoplasma gondii

4. **Tachyzoites** – these are the proliferative forms of toxoplasma gondii that reproduce rapidly within macrophages by endogenous budding. Tachyzoites are sickle-shaped cells that are about 4 to 7 micrometer long and 2 to 4mm wide.
5. **Bradyzoites** (also called **cystozoites**) – these are the forms that undergo slow reproduction within the cysts. The cysts have relatively resistant walls and can grow as large as 150 micrometer – containing up to several thousand bradyzoites.
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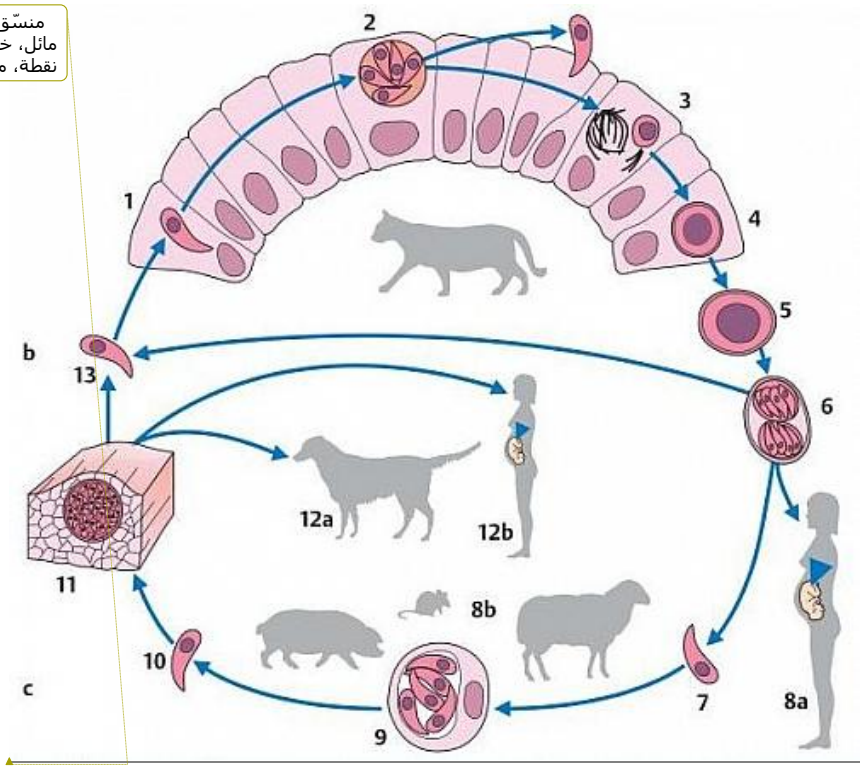

Toxoplasma gondii Life cycle

Toxoplasma gondii Life cycle

1. The Life cycle of Toxoplasma gondii begins within the intestine of the cat as it ingests cysts of toxoplasma gondii in raw meat of mice
2. After ingestion, bradyzoites are released from the cysts in the small intestine - the parasite gains entry into the mucosal cell of the epithelial tissue of the small intestine of the cat
3. In the mucosal cells, they bradyzoites differentiate into male and female gametocytes (the gametes fuse to form **oocysts**)
4. Oocyst are then excreted in cat feces
5. Development of the parasite in an intermediate host (mammals, birds, humans etc)
6. Infection of man occurs following ingestion of oocyst or consumption of meat of an infected animal containing the cyst.

Birth defects caused by Toxoplasma gondii

1. Cleft lip

2. Hydrocephalus (abnormally large head due to accumulation of cerebrospinal fluid
3. Microcephaly (some babies may have small head instead of large head)
4. Microphthalmia (abnormally smallish eyes)
5. Blindness

Toxoplasma gondii Tests for diagnosis

1. Immunofluorescence assay for IgM antibody is used
2. Culture of the organism can be done
3. Microscopic examination of Giemsa-stained specimens will identify presence of trophozoites in acute infections. The trophozoites are crescent in shape.

Toxoplasma gondii Prevention

1. Avoid eating undercooked meat - properly cook meat before eat
2. Pregnant women should avoid disposing cat litter