# MALARIA

## Etiology

Coccidians: *Plasmodium* species are responsible for human malaria. These are *P. falciparum, P. vivax, P. ovale and P. malariae.*

## Epidemiology

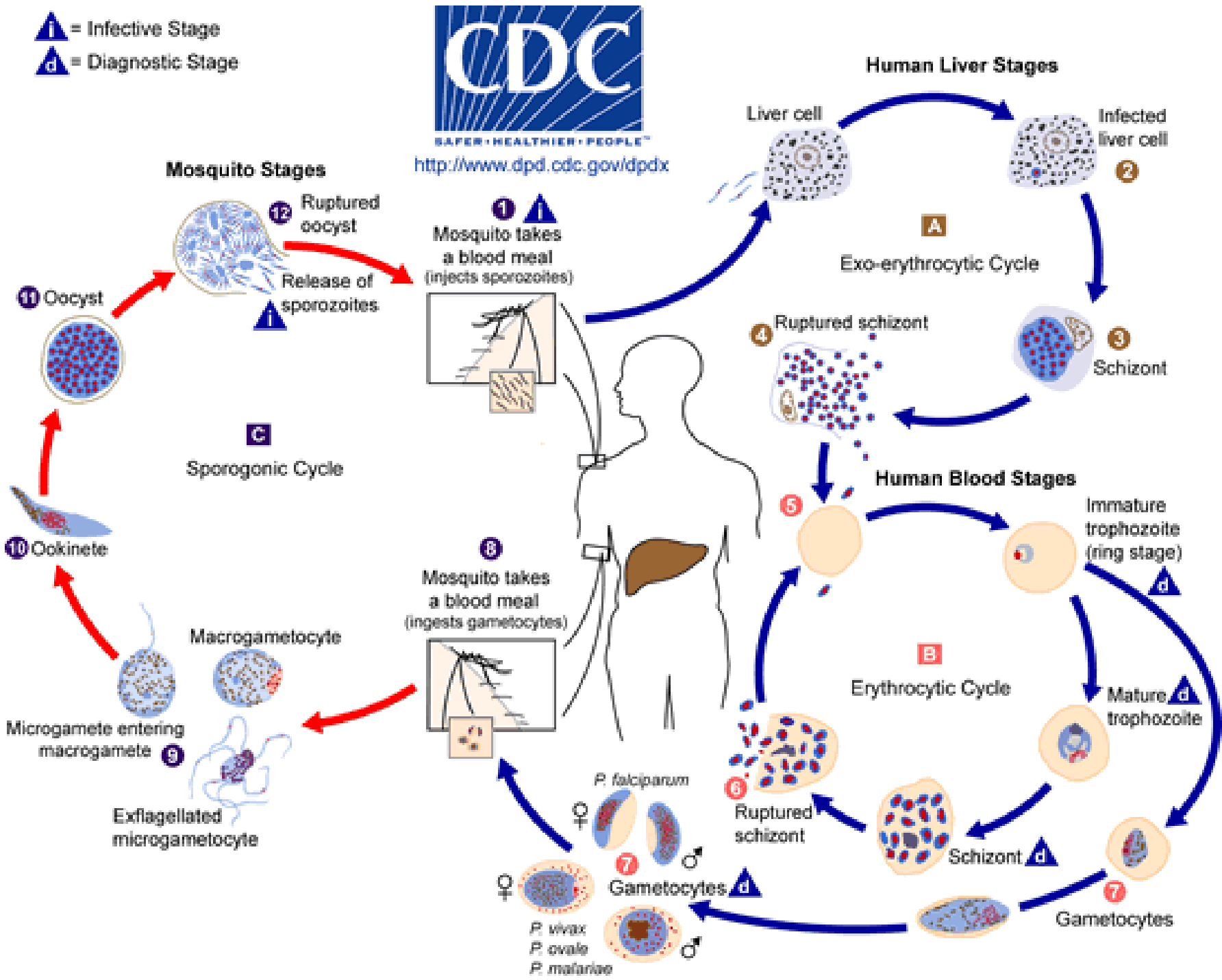
There are an estimated **200 million** global cases of malaria leading to a mortality of more than **one million people per year**. *P. falciparum* (malignant tertian malaria) and *P. malariae (*quartan malaria) are the most common species of malarial parasite and are found in Asia and Africa. *P. vivax* (benign tertian malaria) predominates in Latin America, India and Pakistan, whereas, *P. ovale* (ovale tertian malaria) is almost exclusively found in Africa.

## 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.

Life cycle

Malarial parasites are transmitted by the infected female anopheline mosquito *(Anopheles*) which injects sporozoites present in the saliva of the insect. Sporozoites infect the liver parenchymal cells where they may remain dormant (hypnozoites) 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. P. ovale and P. vivax infect immature red blood cells whereas P. malariae infects mature red cells. P. falciparum infects both. In red cells, the parasites mature into trophozoites. These trophozoites undergo schizogony and merogony in red cells which ultimately burst and release daughter merozoites. Some of the merozoites transform into male and female gametocytes while others enter red cells to continue the erythrocytic cycle. The gametocytes are ingested by the female mosquito, the female gametocyte transforms into ookinete, is fertilized, and forms an oocyst (figure 20) in the gut. The oocyte produces sporozoites (sporogony) which migrate to the salivary gland and are ready to infect another host. The liver (extraerythrocytic) cycle takes 5-15 days whereas the erythrocytic cycle takes 48 hours or 72 hours (P. malariae). Malaria can be transmitted by transfusion and transplacentally.


= Infective Stage
= Diagnostic Stage
CDC
SAFER · HEALTHIER · PEOPLE™
http://www.dpd.cdc.gov/dpdx
Human Liver Stages
Liver cell
Infected liver cell
2
A
Exo-erythrocytic Cycle
3
Schizont
4
Ruptured schizont
Human Blood Stages
5
Immature trophozoite (ring stage)
d
Mature trophozoite
d
B
Erythrocytic Cycle
6
Ruptured schizont
Schizont
d
7
Gametocytes
d
Gametocytes
P. falciparum
P. vivax
P. ovale
P. malariae
8
Mosquito takes a blood meal
(ingests gametocytes)
Macrogametocyte
Microgamete entering macrogamete
9
Exflagellated microgametocyte
10
Ookinete
11
Oocyst
12
Ruptured oocyst
Release of sporozoites
Mosquito Stages
1
Mosquito takes a blood meal
(injects sporozoites)
C
Sporogonic Cycle

**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.

In parasitemia: headache, pains in the bones and joints, chilly sensations and fever.

As the disease progresses, the chills and fever become more prominent. The chill and fever follow a cyclic pattern (paroxysm) with the symptomatic period lasting 8-12 hours.

The duration of which depends upon the species of the infecting parasite. This interval is about 34-36 hours in the case of P. vivax and P. ovale (tertian malaria), and 58-60 hours in the case of P. malariae (quartan malaria). Classical tertian paroxysm is rarely seen in P. falciparum and persistent spiking or a daily paroxysm is more usual.

Within a few hours the patient feels exhausted but symptom-less and remains asymptomatic until the next paroxysm. Each paroxysm is due to the rupture of infected 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 progressively and results in death. This organism causes sequestration of capillary vasculature in the brain, gastrointestinal and renal tissues. Chronic malaria results in splenomegaly, hepatomegaly and nephritic syndromes. (COMPLICATIONS)

Pathology
Symptoms of malaria are due to the release of massive number of merozoites into the circulation.

Diagnosis
Diagnosis is based on symptoms and detection of parasite in Giemsa stained blood smears. There are also antibody tests and PCR.

Treatment and Control

Treatment is effective with various quinine derivatives (quinine sulphate, chloroquine, meflaquine and primaquine, etc.). Drug resistance, particularly in P. falciparum and to some extent in *P. vivax* is a major problem.

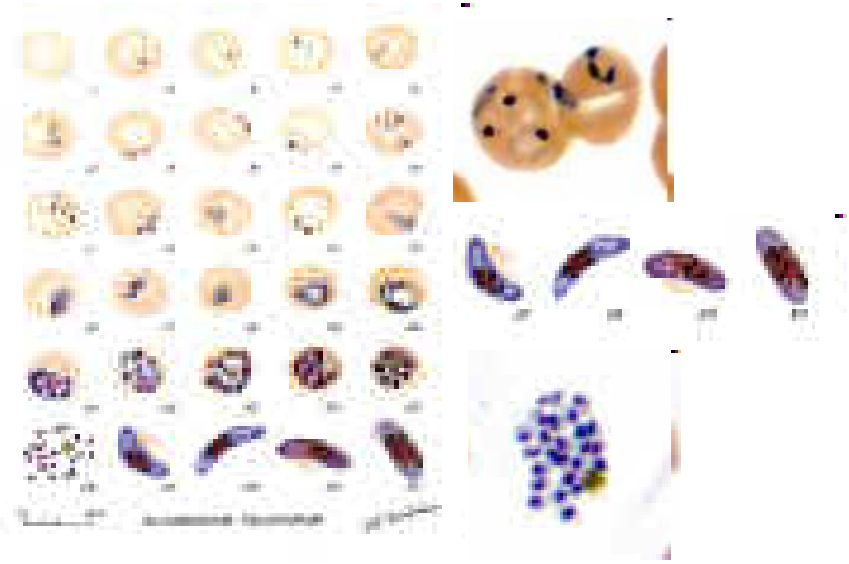
Control measures are:

eradication of infected anopheline mosquitos.

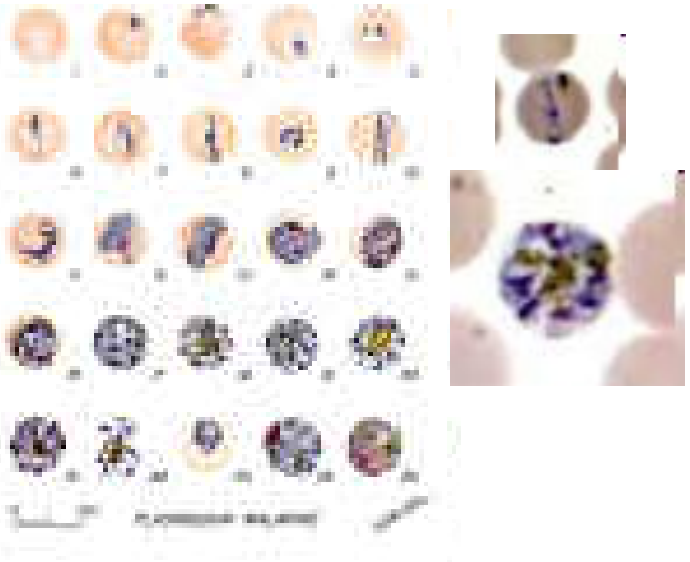
Chemoprophylaxis

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

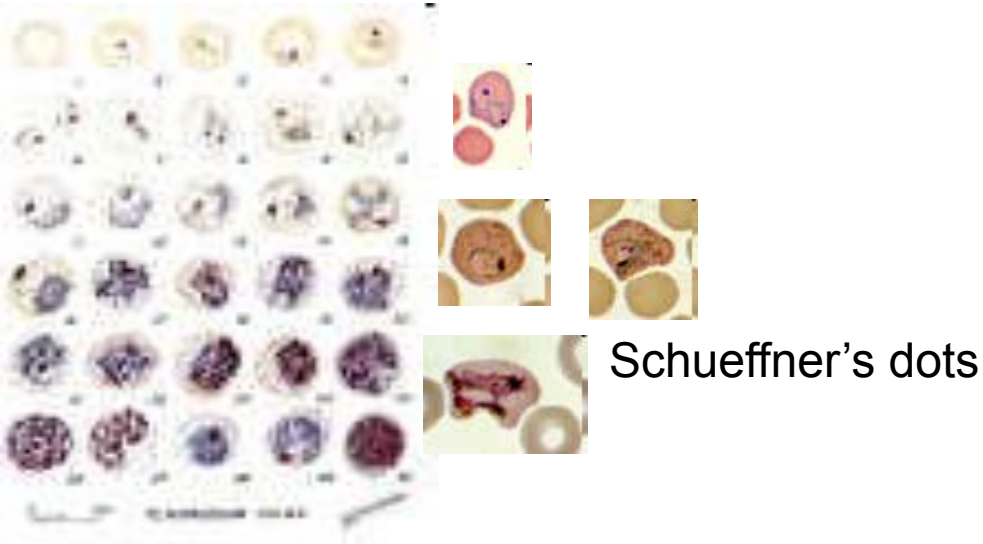
P.falciparum



P. malariae

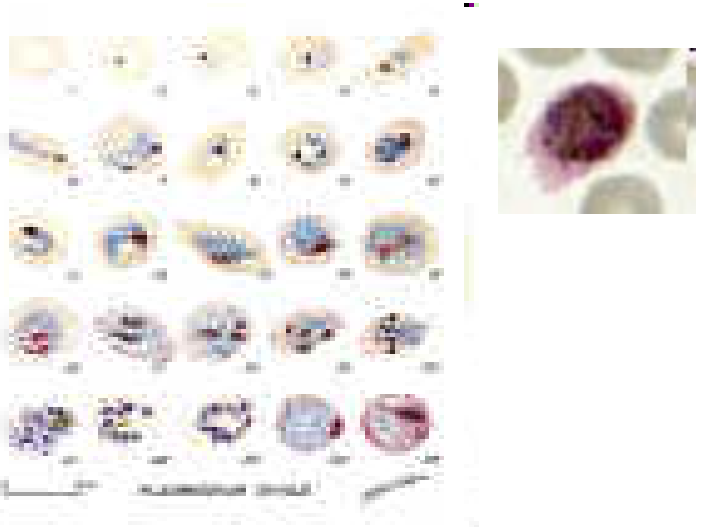


P. vivax



Schueffner’s dots

P. ovale



# TOXOPLASMOSIS

## Etiology

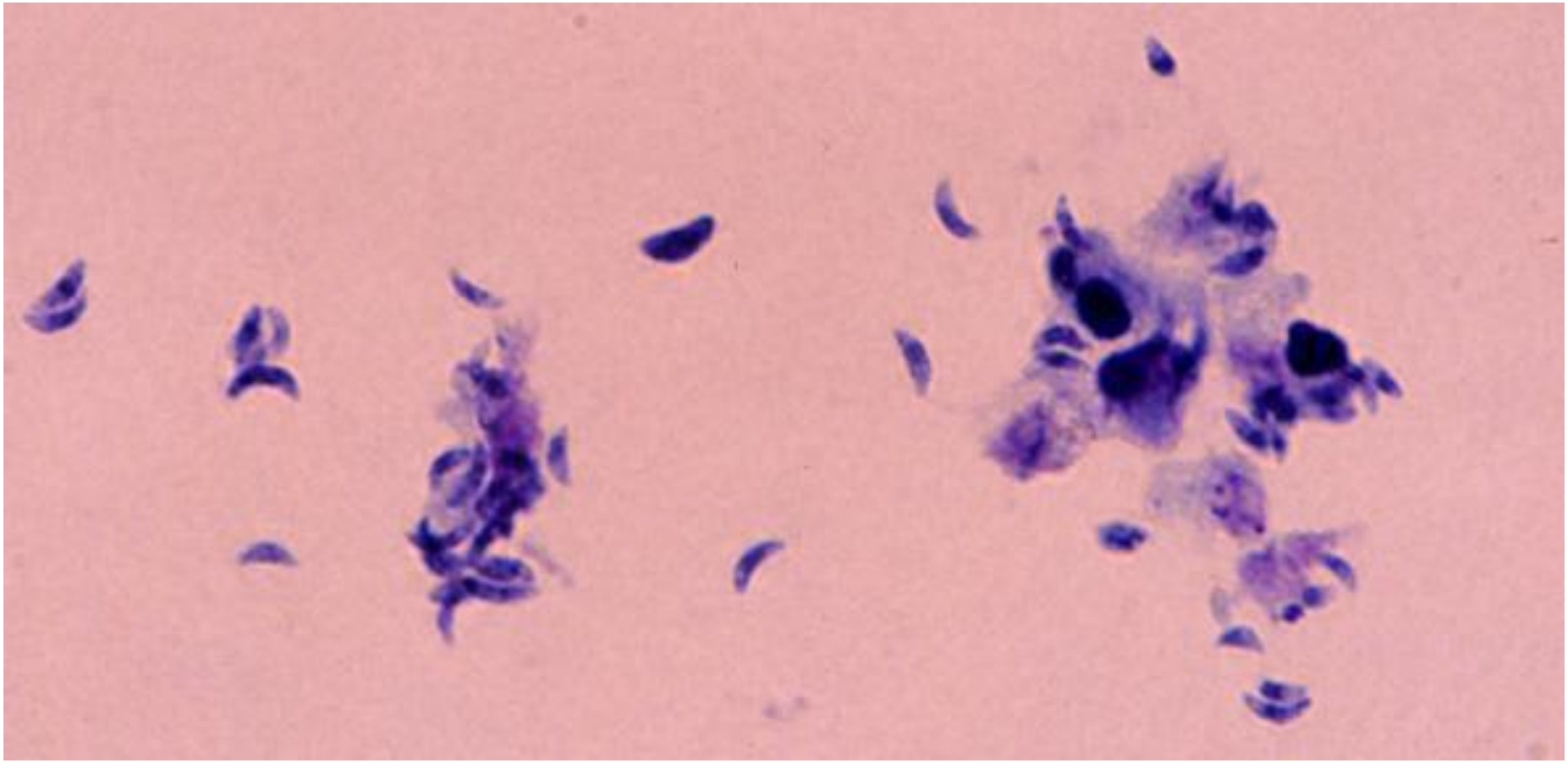
*Toxoplasma gondii* is the organism responsible for toxoplasmosis

## Epidemiology

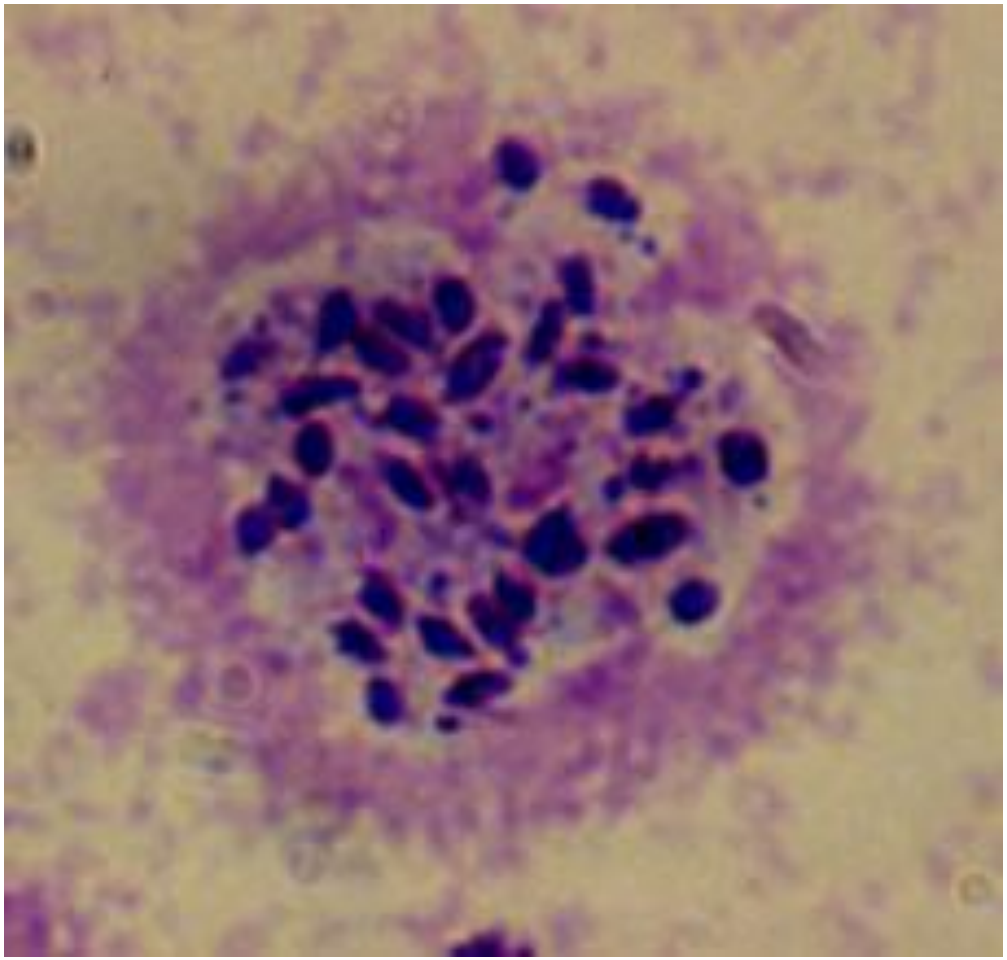
*Toxoplasma* has worldwide distribution and 20%-75% of the population is seropositive without any symptomatic episode. However, the infection poses a serious threat in immunosuppressed individuals and pregnant females.

## Morphology

The intracellular parasites (tachyzoite) are 3x6 microns, coma-shaped organisms
Bradyzoite in tissue cysts.
Cysts in cat feces (oocysts) are 10-13 microns in diameter.



Tachyzoites

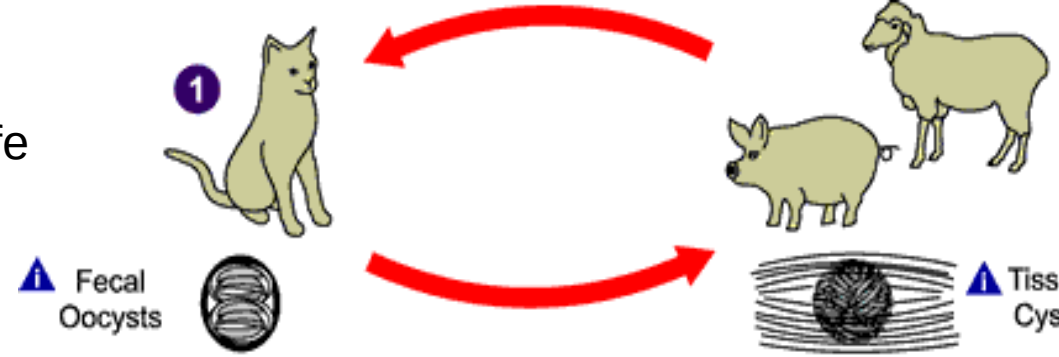


Bradyzoites

## 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 bird. 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 (hence the name tachyzoites) and form a cyst which may occupy the whole cell. The infected cells ultimately 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). These cysts are infectious to carnivores (including man). Unless man is eaten by a cat, it is a dead-end host.

All stages exist in cat.
*T gondii* can complete life cycle in cats alone.
Also, gametogony and schizogony take place

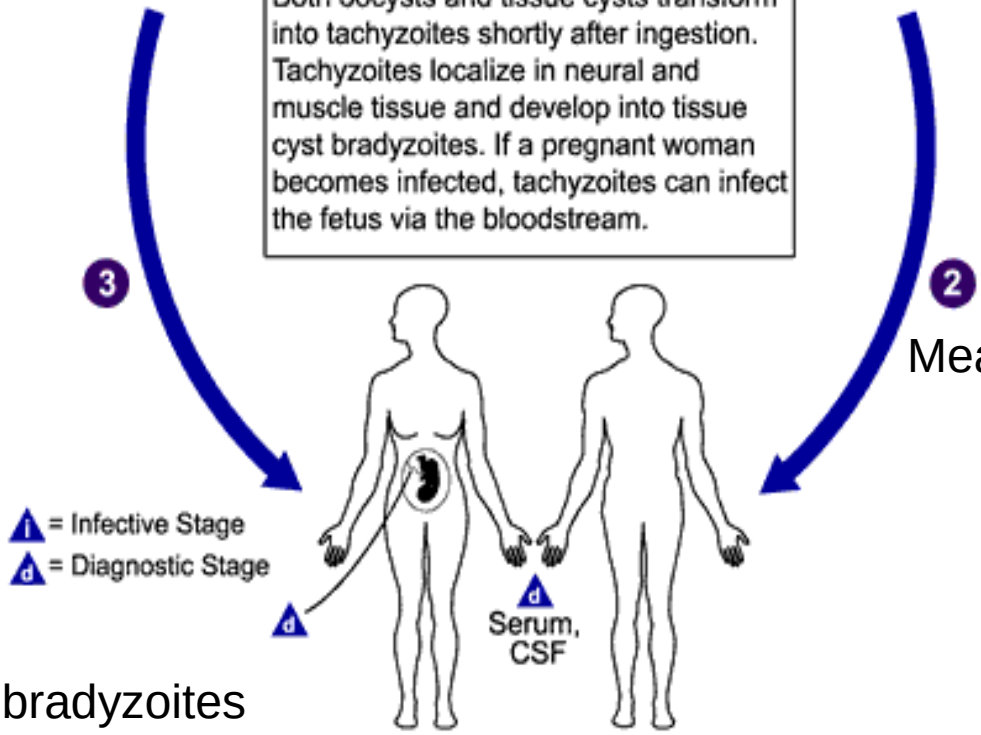



Bradyzoites

Sporozoite

Both oocysts and tissue cysts transform into tachyzoites shortly after ingestion. Tachyzoites localize in neural and muscle tissue and develop into tissue cyst bradyzoites. If a pregnant woman becomes infected, tachyzoites can infect the fetus via the bloodstream.

Tachyzoites: intracellular in all hosts




Meat (bradyzoite) or Milk (tachyzoite)

In human: tachy- and bradyzoites

**Diagnostic Stage**

1) Serological diagnosis.
or
2) Direct identification of the parasite from peripheral blood, amniotic fluid, or in tissue sections.

Symptoms
Toxoplasma infection is common

It rarely produces symptoms in normal individuals.

Its serious consequences are limited to pregnant women and immunodeficient hosts.

Congenital infections occur in about 1-5 per 1000 pregnancies of which 5-10% result in miscarriage and 8-10% result in serious brain and eye damage to the fetus. 10-13% of the babies will have visual handicaps. Although 58-70% of infected women will give birth to a normal offspring, a small proportion of babies will develop active retino-chorditis or mental retardation in childhood or young adulthood.

In immunocompetent adults, toxoplasmosis, may produce flu-like symptoms, sometimes associated with lymphadenopathy.

In immunocompromised individuals, infection results in generalized parasitemia involvement of brain, liver , lung and other organs, and often death.

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

Serodiagnosis:ELISA, IFAT.

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

PNEUMOCYSTIS PNEUMONIA

*Pneumocystis jiroveci* (formerly known as *Pneumocystis carinii*)

*Pneumocystis jiroveci* was formerly thought to be a protozoan but is now known to be a fungus.

Pneumocystis pneumonia is an infection of immunosuppressed individuals and is particularly seen in AIDS patients.
*It spreads from person to person in cough droplets. Infection in immunosuppressed individuals results in pneumonia associated with fever, tachypnea, hypoxia, cyanosis and asphyxia.*

Diagnosis is based on isolation of organisms from affected lungs. Stained with silver staining (Gomori methanamine. Trimethoprim-sulphamethoxazole is the treatment of choice .