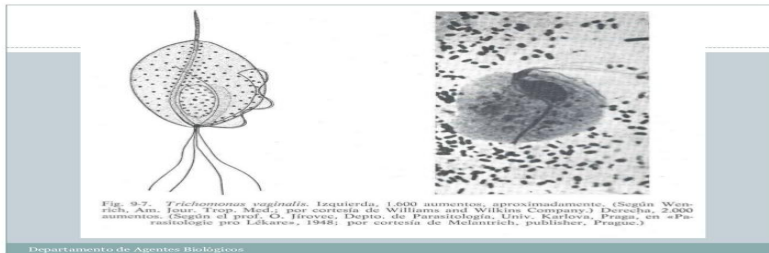


TISSUE PARASITES

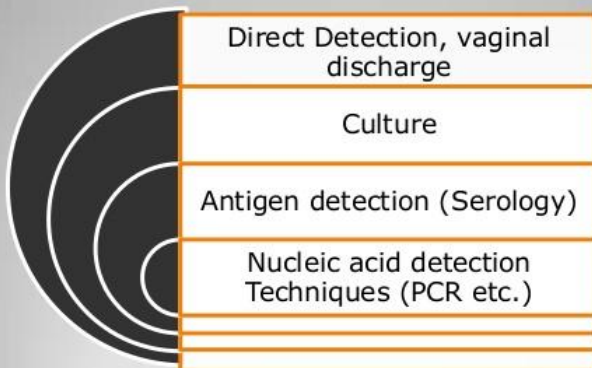
Trichomonas vaginalis

- **Trichomonas vaginalis** :is an anaerobic flagellated protozoan a form of microorganism.
- The parasitic microorganism is the causative agent of trichomoniasis and is the most common pathogenic protozoan infection of humans in industrialized countries.



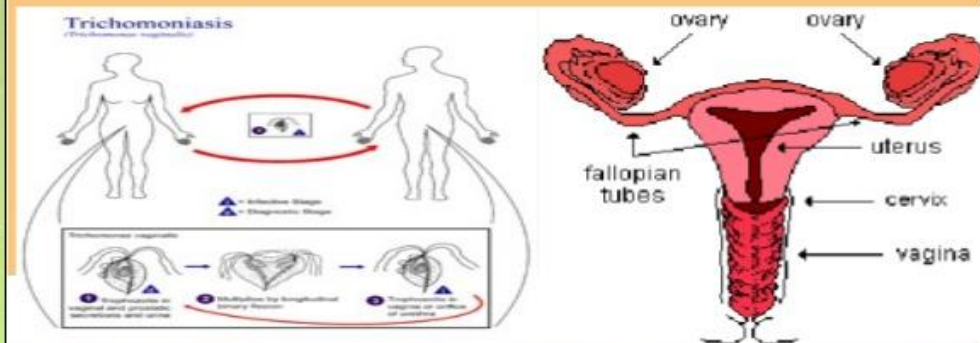
- The human genital tract is the only reservoir for this species. Trichomonas is transmitted through sexual or genital contact.
- Cause by the single-celled protozoan parasite, *Trichomonas vaginalis*

LABORATORY DIAGNOSIS



Mechanism of infection ;

- Produces mechanical stress on host cells and then ingesting cell fragments after cell death.



LEISHMANIA

INTRODUCTION

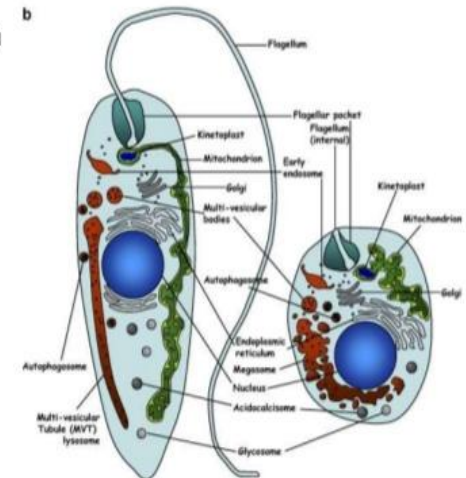
- **Leishmania:**
 - Is a genus of trypanosomatid protozoa, which causes a fatal vector-borne parasitic disease called Leishmaniasis.
 - It is spread by the bite of sandflies of the genus Phlebotomus in the Old World, and of the genus Lutzomyia in the New World.
- **Leishmaniasis:**
 - is the **second-largest parasitic killer** in the world (after malaria) and is endemic in many parts of Africa, Asia and South America.



MORPHOLOGY

(same in all species)

- The parasite exists in 2 forms;-
- 1. **Amastigotes** – aflagellar stage
- 2. **Promastigotes**- flagellar stage



IMPORTANT SPECIES

- *L. donovani*
- *L. tropica*
- *L. mexicana*
- *L. braziliensis*
- *L. major*
- *L. guyanensis*
- *L. lainsoni, etc*

MODE OF TRANSMISSION

(*L. donovani*)

1. Mainly by the bite of sand fly (vector) Phlebotomus argentipus
 2. Less frequently by:
 - blood transfusion,
 - congenital infection,
 - accidental inoculation of cultured promastigotes in the lab. Workers.
 - sexual intercourse.
- ☐ Males are affected more (due to increased exposure to sand flies through the occupation and leisure activities).



In *L. tropica*

- amastigotes reside in the large mononuclear cells of the skin



In *L. mexicana*

- Amastigotes found in reticuloendothelial cells and lymphatic tissues of skin

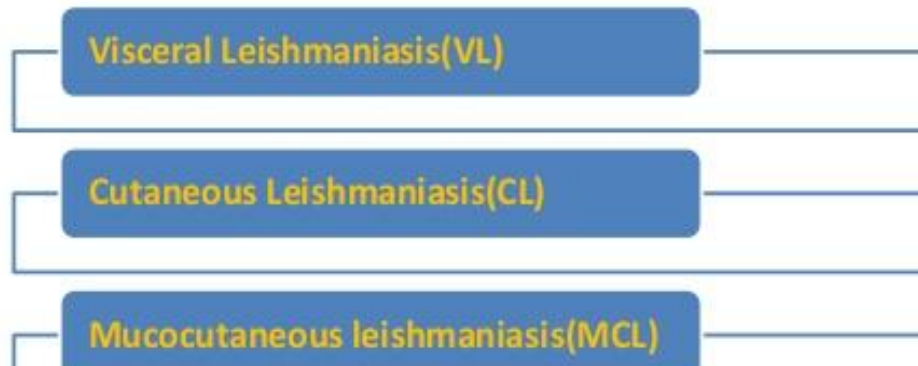


In *L. braziliensis*

- amastigotes are found in reticuloendothelial cells and lymphatic tissues of skin and mucus membrane

TYPES OF LEISHMANIASIS

Leishmaniasis is divided into clinical syndromes according to what part of the body is affected



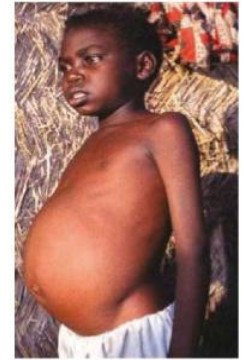
3. Mucocutaneous leishmaniasis(MCL)

- Caused by *L. braziliensis* and occasionally by *L. panamensis*
- Part of the body affected most is **skin and mucous membrane of nose and pharynx**



1. Visceral Leishmaniasis (VL) or Kala-azar

- caused by *L. donovani*
- part of the body affected most is **internal organs**



Splenomegaly

2. Cutaneous Leishmaniasis (CL)

(most common type)

- a) **Old world CL**:- caused by *L. tropica*, *L. aethiopia*
- b) **New world CL**:- caused by *L. mexicana*, *L. braziliensis*, *L. guyanensis*
- c) **Dermal leishmanoid or Post kala-azar dermal leishmaniasis (PKDL)**:- caused by *L. donovani*



VECTOR

(Sand fly)

• *Phlebotomas*

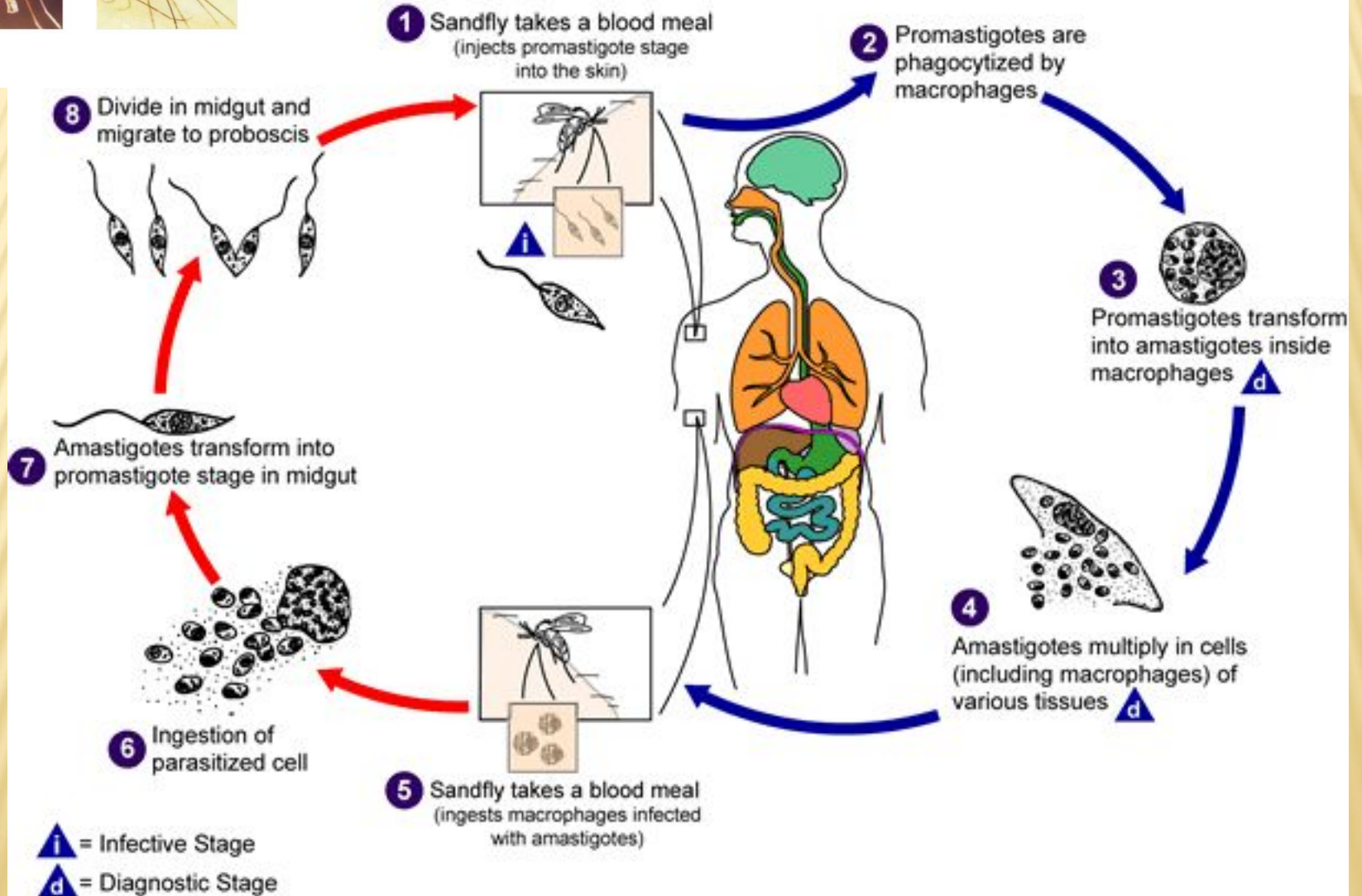
• *Lutzomyia*

دورة



Sandfly Stages

Human Stages



SYNONYMS OF LEISHMANIASIS

Visceral
leishmaniasis

Kala-azar,

Black fever

Dum-Dum fever,

Sahib's disease

Kala Dukh

White leprosy

Cutaneous
leishmaniasis

Aleppo boil,

Baghdad boil,

Delhi boil,

Kandahar sore,

Lahore sore,

Oriental sore,

Mucocutaneous
Leishmaniasis

Breda's disease

bosch yaws,

bush yaws

forest yaws

- **Clinical Features:**

Human leishmanial infections can result in 2 main forms of disease, cutaneous leishmaniasis and visceral leishmaniasis (kala-azar). The factors determining the form of disease include leishmanial species, geographic location, and immune response of the host. Cutaneous leishmaniasis is characterized by one or more cutaneous lesions on areas where sandflies have fed. Persons who have cutaneous leishmaniasis have one or more sores on their skin. The sores can change in size and appearance over time. They often end up looking somewhat like a volcano, with a raised edge and central crater. A scab covers some sores. The sores can be painless or painful. Some people have swollen glands near the sores (for example, in the armpit if the sores are on the arm or hand).

- **Laboratory Diagnosis:**

Examination of Giemsa stained slides of the relevant tissue is still the technique most commonly used to detect the parasite.

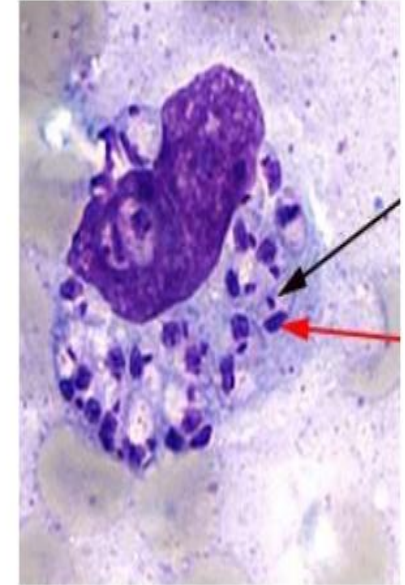
- **Diagnostic findings**

- Microscopy

- Isolation of the organism in culture (using for example the diphasic NNN medium) or in experimental animals (hamsters) constitutes another method of parasitologic confirmation of the diagnosis, and in addition can provide material for further investigations (e.g., isoenzyme analysis).
- Antibody detection can prove useful in visceral leishmaniasis but is of limited value in cutaneous disease, where most patients do not develop a significant antibody response
- molecular (PCR) approaches. Such techniques, however, are not readily available in general diagnostic laboratories.

Direct Evidences (contd.....)

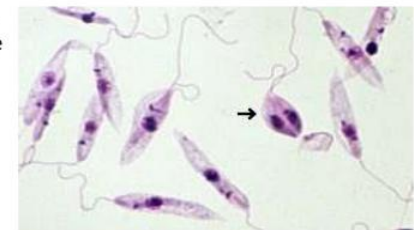
1. Peripheral blood by thick film method.(Amastigote form)



Amastigotes in a macrophage

Direct Evidences (contd.....)

2. Blood culture in N.N.N. Medium. (Promastigote form)



Promastigote from culture in NNN medium

TOXOPLASMA GONDII

- **Causal Agent:**

Toxoplasma gondii that infects most species of warm blooded animals, including humans, . A single-celled parasite (a protozoan parasite) causes a disease known as toxoplasmosis. While 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.

- **Geographic Distribution:**

Serologic prevalence data indicate that toxoplasmosis is one of the most common of humans infections throughout the world. A high prevalence of infection in France has been related to a preference for eating raw or undercooked meat, while a high prevalence in Central America has been related to the frequency of stray cats in a climate favoring survival of oocysts and soil exposure. The overall seroprevalence in the United States among adolescents and adults, as determined with specimens collected by the third National Health and Nutrition Examination Survey (NHANES III) between 1988 and 1994, was found to be 22.5%, with a seroprevalence among women of childbearing age (15 to 44 years) of 15%.

TOXOPLASMOSIS

☐ Widely-distributed zoonosis caused by *T. gondii* protozoa.

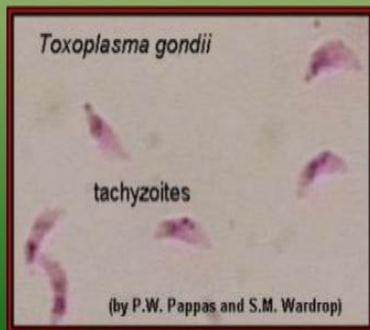
☐ The definitive host is the domestic cat and other felines.

☐ Humans and other mammals are intermediate hosts.

MORPHOLOGY

☐ The intracellular parasites (tachyzoite) are $3 \times 6 \mu$ and crescent shaped organisms that are enclosed in a parasite membrane to form a cyst measuring $10-100 \mu$ in size.

☐ Cysts in cat feces (oocysts) are $10-13 \mu$ in diameter



- Definitive hosts for *Toxoplasma gondii* are members of family Felidae (domestic cats and their relatives).
- Unsporulated oocysts are shed in the cat's feces. Although oocysts are usually only shed for 1-2 weeks, large numbers may be shed. Oocysts take 1-5 days to sporulate in the environment and become infective. Intermediate hosts in nature (including birds and rodents) become infected after ingesting soil, water or plant material contaminated with oocysts.
- Oocysts transform into tachyzoites shortly after ingestion. These tachyzoites localize in neural and muscle tissue and develop into tissue cyst bradyzoites. Cats become infected after consuming intermediate hosts harboring tissue cysts. Cats may also become infected directly by ingestion of sporulated oocysts.
- Animals bred for human consumption and wild game may also become infected with tissue cysts after ingestion of sporulated oocysts in the environment. Humans can become infected by any of several routes:

Toxoplasma gondii exists in four forms
All parasite stages are infectious.

1. TACHYZOITES
2. TISSUE CYSTS
3. BRADYZOIT
4. OOCYSTS



How is Toxoplasmosis Transmitted?

A. Oocysts:



- Contact with cats
- Dirty hands
- Contaminated food
- Water



B. Tachyzoites (Tg):

- Meat
- Blood and its products
- Tissue transplants



C. Bradyzoite:

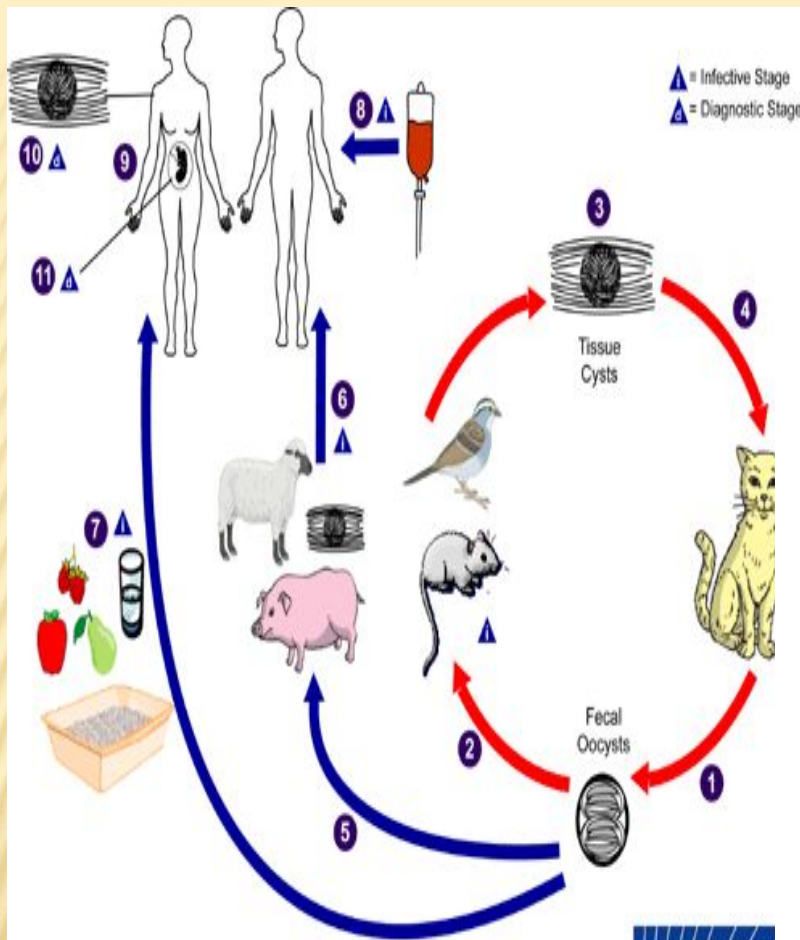
- Meat



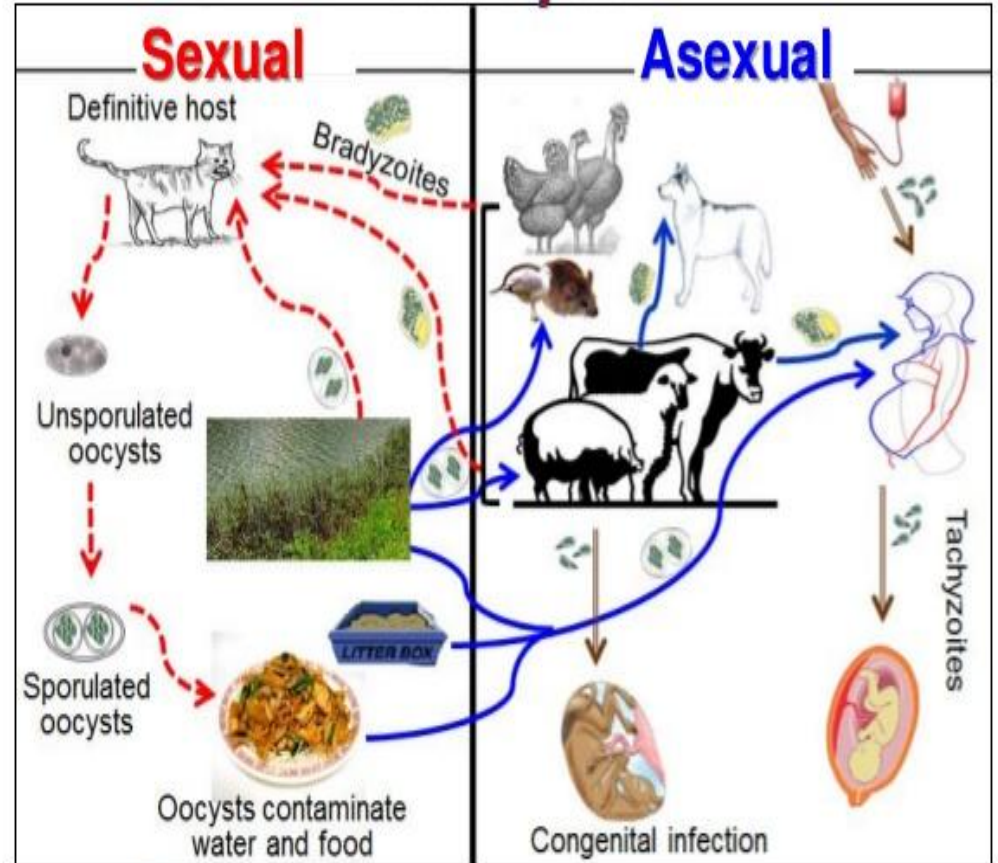
Cats are infected by hunting and eating their prey (Bradyzoites), or by coming into contact with oocysts in feces from infected cats

Transmission to man:

1. Ingestion of **sporulated oocysts** from contaminated soil, food or water
 2. Ingestion of tissue cyst containing **bradyzoites** from undercooked meat
 3. By blood transfusion, needle stick injuries, organ transplantation
 4. Transplacental transmission
 5. Laboratory accidents
- (**Tachyzoites** are the infective form in blood)



Life Cycle



- *T. gondii* transmission via tachyzoites
- - - *T. gondii* transmission via oocysts
- *T. gondii* transmission via bradyzoites (tissue cysts)



In the human host, the parasites form tissue cysts, most commonly in skeletal muscle, myocardium, brain, and eyes; these cysts may remain throughout the life of the host

Healthy people (nonpregnant)

Healthy people who become infected with *Toxoplasma gondii* often do not have symptoms because their immune system usually keeps the parasite from causing illness. When illness occurs, it is usually mild with "flu-like" symptoms (e.g., tender lymph nodes, muscle aches, etc.) that last for weeks to months and then go away. However, the parasite remains in their body in an inactive state. It can become reactivated if the person becomes immunosuppressed.

Mother-to-child (congenital)

Generally if a woman has been infected before becoming pregnant, the unborn child will be protected because the mother has developed immunity. If a woman is pregnant and becomes newly infected with *Toxoplasma* during or just before pregnancy, she can pass the infection to her unborn baby (congenital transmission). The damage to the unborn child is often more severe the earlier in pregnancy the transmission occurs. Potential results can be

- a miscarriage

- a stillborn child

- a child born with signs of toxoplasmosis (e.g., abnormal enlargement or smallness of the head)

Infants infected before birth often show no symptoms at birth but may develop them later in life with potential vision loss, mental disability, and seizures.

SYMPTOMS

Common symptoms of *T. gondii* infection in cats includes:

- Fever;

- Ocular inflammation;

- Anorexia;

- Lethargy;

- Abdominal discomfort; and

- Neurologic abnormalities (Vollaire).

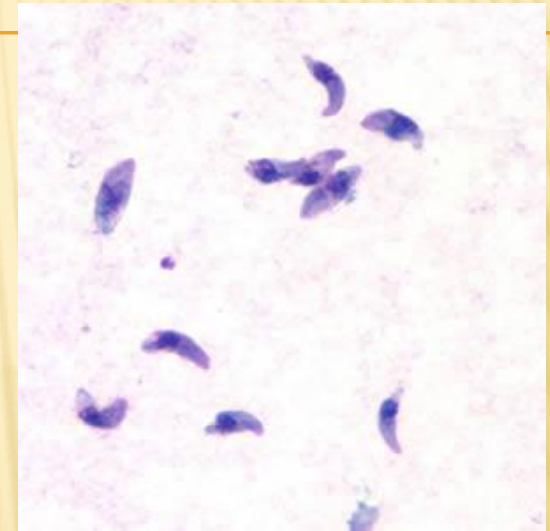
How can *Toxoplasma* affect my unborn child?

If you are newly infected with *Toxoplasma* while you are pregnant, or just before pregnancy, then you can pass the infection on to your baby. You may not have any symptoms from the infection. Most infected infants do not have symptoms at birth but can develop serious symptoms later in life, such as blindness or mental disability. Occasionally infected newborns have serious eye or brain damage at birth

- **Laboratory Diagnosis:**

The diagnosis of toxoplasmosis may be documented by:

- Observation of parasites in patient specimens, such as bronchoalveolar lavage material from immunocompromised patients, or lymph node biopsy.
- Diagnosis
- A *Toxoplasma*-positive reaction, stained by immunofluorescence (IFA). (CDC Photo)
- The diagnosis of toxoplasmosis is typically made by **serologic** testing. A test that measures immunoglobulin G (IgG) is used to determine if a person has been infected. If it is necessary to try to estimate the time of infection, which is of particular importance for pregnant women, a test which measures immunoglobulin M (IgM) is also used along with other tests such as an avidity test.
- Diagnosis can be made by direct observation of the parasite in stained tissue sections, cerebrospinal fluid (CSF), or other biopsy material. These techniques are used less frequently because of the difficulty of obtaining these specimens.
- Parasites can also be isolated from blood or other body fluids (for example, CSF) but this process can be difficult and requires considerable time.
- Molecular techniques that can detect the parasite's DNA in the amniotic fluid can be useful in cases of possible mother-to-child (congenital) transmission.
- Isolation of parasites from blood or other body fluids, by intraperitoneal inoculation into mice or tissue culture. The mice should be tested for the presence of *Toxoplasma* organisms in the peritoneal fluid 6 to 10 days post inoculation; if no organisms are found, serology can be performed on the animals 4 to 6 weeks post inoculation.
- Detection of parasite genetic material by PCR, especially in detecting congenital infections in utero.

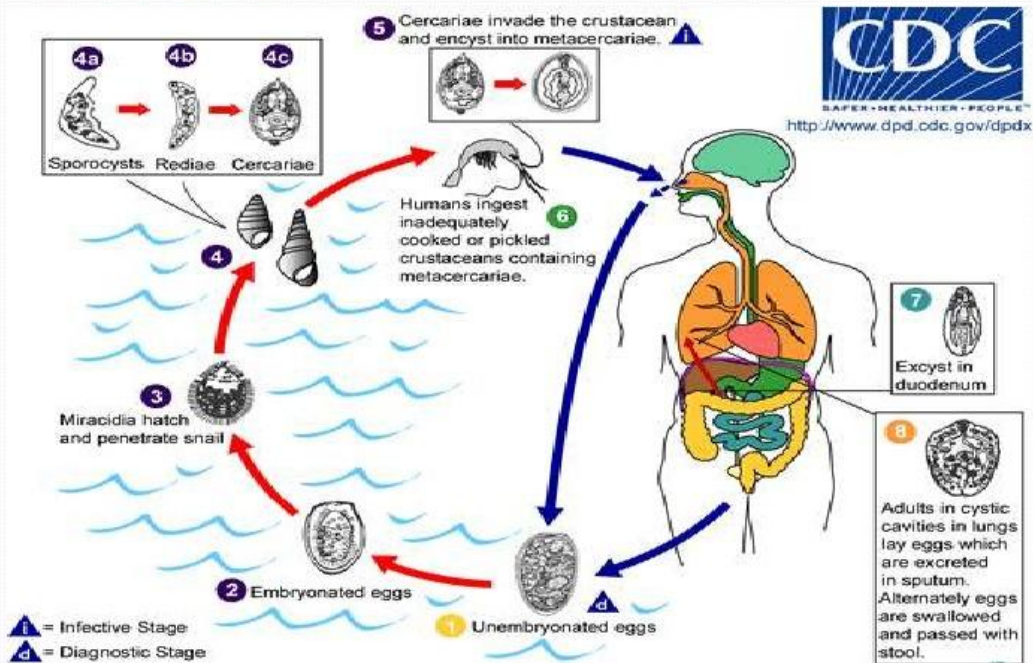


Paragonimus

Paragonimus westermani westermani

major species of lung fluke that infects humans, causing **paragonimiasis**. The species sometimes is called the Japanese lung fluke or oriental lung fluke. Human infections are most common

Life cycle



Adult worm



Lung Flukes: *Paragonimus westermani*

- *Paragonimus westermani* ova
 - Yellowish brown
 - Thick-shelled
 - Operculated with a thickened abopercular egg
 - May be seen in the sputum or in feces if the sputum is swallowed



LIFE CYCLE

1. Infective stage: Metacercariae
2. Infective mode: eating raw fresh water crabs and crayfish with metacercariae
3. Infective route: by mouth
4. Site of inhabitation: lungs
5. Intermediate hosts: 1st int. host is melania snail. 2nd int. hosts are crab and crayfish.
6. Reservoir hosts: carnivores such as tiger, lion, wolf, fox, dog, leopard, cat and etc
7. Life span: 5-6 years

DR. T.V.RAO MD

7

Lung Flukes: Pathogenesis and Clinical Manifestations

- Paragonimiasis
 - Cough
 - Hemoptysis
 - Symptoms consistent with pulmonary tuberculosis
 - Misdiagnosed as PTB



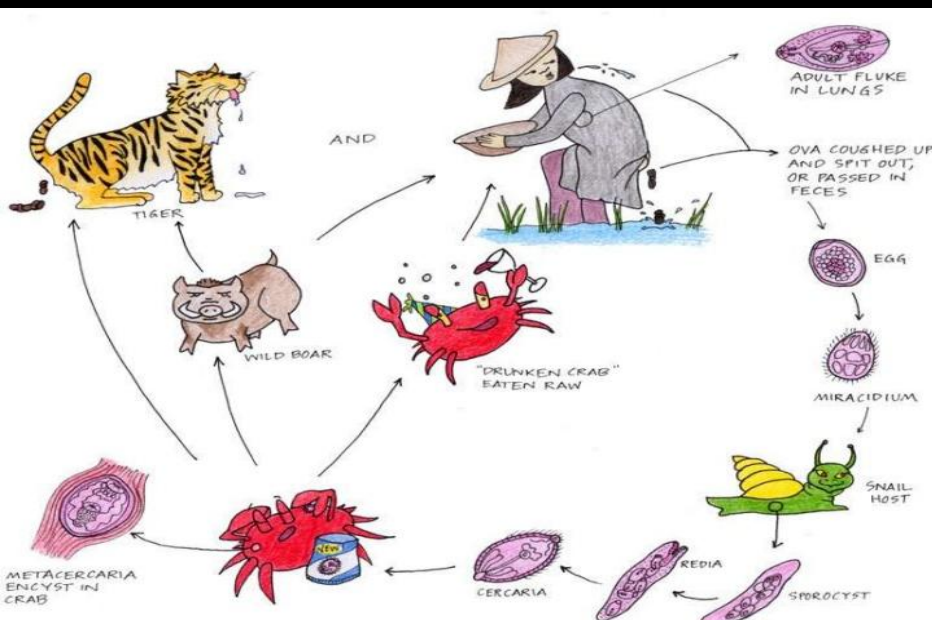
Prepared by FZHapan

Lung Flukes: Diagnosis of Paragonimiasis

- Radiographs aid in diagnosis
- Definitive diagnosis is based on the finding of ova in the sputum, stool or less frequently in aspirated material from abscesses or pleural effusions
- Multi-dot ELISA



Prepared by FZHapan



clonorchis sinensis

Pathology of Clonorchiasis

Adult worms of *C. sinensis* mainly live in bile ducts of definitive host

Humans or carnivorous animals are infected due to the ingestion of raw or undercooked fish

Definitive host: Human, cat, dog, mice etc

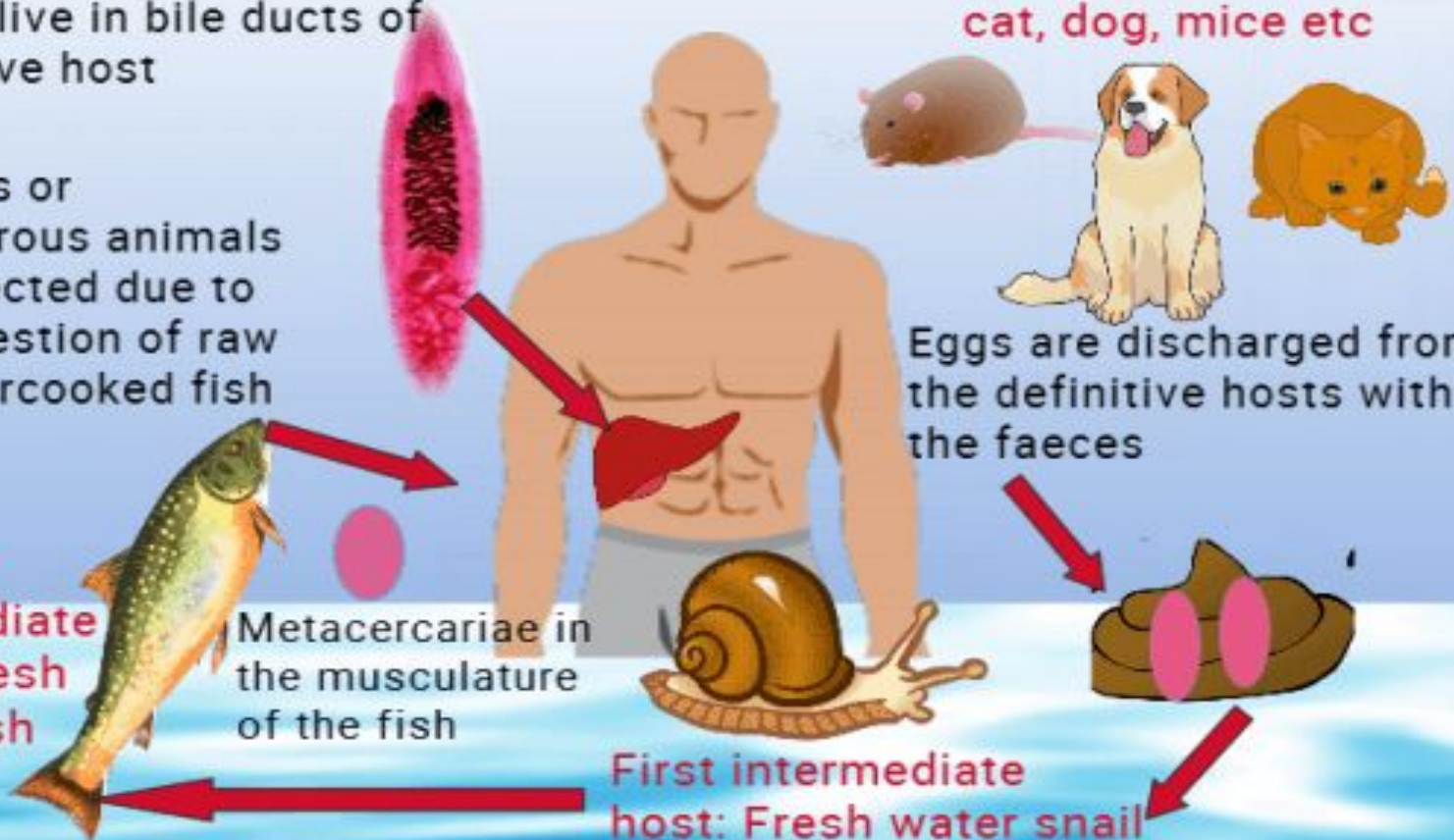
Eggs are discharged from the definitive hosts with the faeces

Second intermediate host: Fresh water fish

Metacercariae in the musculature of the fish

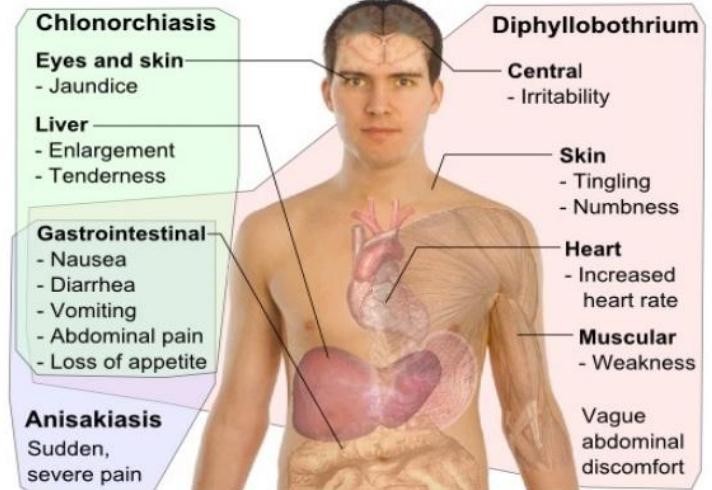
First intermediate host: Fresh water snail

Mainly prevalent in Asian countries and regions, including South Korea, China, Northern Vietnam, and far eastern Russia.



- **Source of infection:** patients, infected reservoir hosts: cats, dogs, mice, pigs
- **Route of transmission:** the infection is acquired by eating raw or inadequately cooked freshwater fish or shrimp, which are previously infected
- **Susceptibility:** human is generally susceptible, related with the dietary habits

Symptoms of Raw fish infection



Laboratory Findings

- Blood routine test: eosinophilia, anemia in severe infection
- Eggs examination:
 - simple smear feces to find eggs
 - Stool concentration technique may increase the positive rate
 - Duodenal aspiration: raise the chance of finding eggs



DRACUNCULUS MEDINENSIS



Dracunculus medinensis

DRACUNCULIASIS :

- Also known as guinea worm disease.
- Vector borne parasitic disease.
- Involves subcutaneous tissues (leg and foot).
- Caused by nematode parasite, *Dracunculus medinensis*.
- Its not lethal but disable its victim temporarily.

- Transmitted exclusively when people drink stagnant water contaminated with parasite infected water fleas.
- It affects people in rural, deprived and isolated communities who depend mainly on open surface water sources such as ponds and wells.

LIFE CYCLE:::

*The adult female emerges from the skin (90% legs and feet)

*Person with protruding worm enters water, and female releases larvae

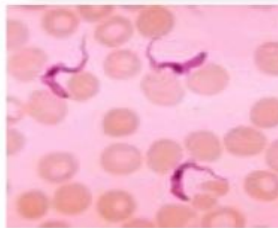
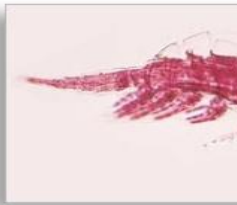
*Copepods ingest larvae and within 10-14 days they reach the infective stage



LIFE CYCLE:::

(continued)

- *Once ingested, larvae travel to the small intestine
- *Penetrate wall of small intestine and pass into the body cavity
- *Over 10-14 months, the adult females grow to full size (2-3 feet)
- *The mature female then migrates to site where she will emerge (usually lower limbs)
- *A blister will develop at the emerging site, and within 1-3 days it will rupture
- *Worm will emerge from ulcerated skin

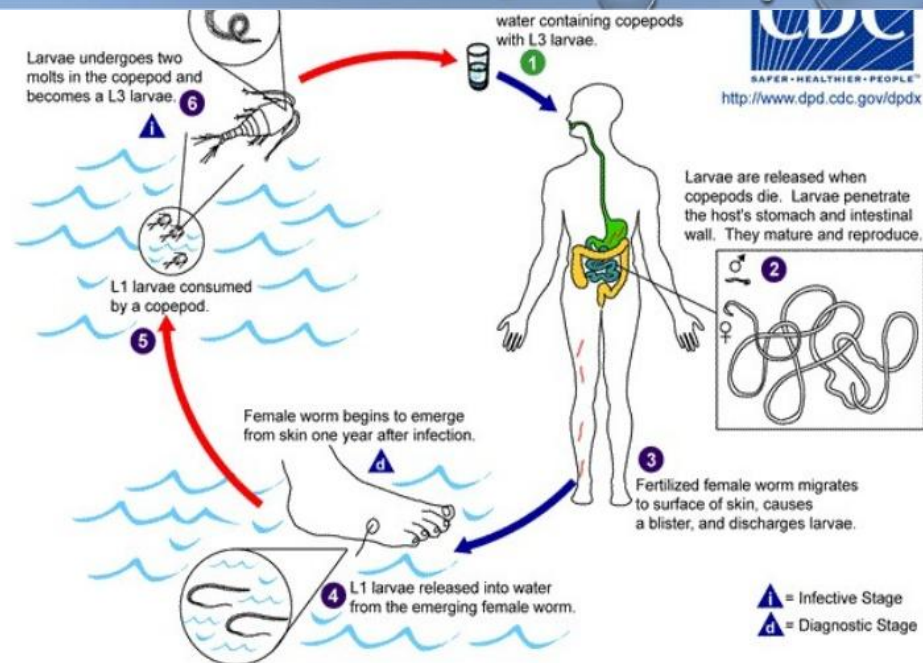


- DETECTION OF LARVA
- CONTACT WITH WATER - LARGE AMOUNT OF LARVA DISCHARGES
- MICROSCOPIC EXAMINATION
- SEROLOGY
- ANTIBODY SEEN IN SERUM BY ELISA & FLUORESCENTS ANTIBODY TEST
- SKIN TEST
- ANTIGEN IS INJECTED INTRADERMAL TO SEE WHEEL REACTION



SIGN/SYMPTOMS :

- Intense burning pain localized to path of travel of worm (the fiery serpent).
- Fever
- Nausea
- Vomiting
- Allergic reaction



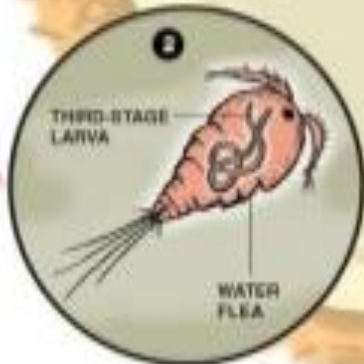
The Life Cycle of Guinea Worm Disease

1 The cycle starts...

Seeking relief from pain, sufferer soaks a blister with exposed worm in nearby water source. On contact with water, the worm bursts, releasing hundreds of thousands of immature first-stage larvae into the water.



2 Tiny water fleas ingest the larvae which, molt twice, becoming mature third-stage larvae. The process takes about 2 weeks.



3 Another person drinks the water containing the water fleas with the infective larvae. The water fleas are digested, releasing the larvae in the stomach.



4 The larvae, which resist digestion, migrate to the small intestine and penetrate the intestinal wall into the body cavity, where they grow into worms and mate.



5 Fertilized female worms, up to 3 feet long, move through connective tissue to various areas of the body, usually the lower limbs.

6 Approximately a year later, after the larvae were ingested, the worm forms a painful blister near the skin surface. The blister bursts, exposing the worm.

10-14 MONTHS

7 The cycle continues...



Thanks
wish you all the best

