Phylum: **Sarcomastigophora**

Sub phylum**:** **Mastigophora**

Class: **Zoomastighphora**

Mastigophora, the flagellates, have one or more whip-like flagella and, in some cases, an undulating membrane (eg, trypanosomes). These include intestinal and genitourinary flagellates (*Giardia, Trichomonas, Dientamoeba, Chilomastix*) and blood and tissue flagellates (*Trypanosoma, Leishmania*).

Giardia intestinalis (*syn. *Giardia lamblia, G. duodenalis)

Giardia sp., a flagellate, is the only common pathogenic protozoan found in the duodenum and jejunum of humans. It is the cause of giardiasis.

Giardia duodenalis is another name commonly ascribed to the parasite that causes human giardiasis; the term *Giardia intestinalis* is frequently used in Europe and *Lamblia* in the former Russia. Much of the confusion is due to merging of species names now that human giardiasis is recognized as a zoonosis and species based on supposed single-host parasitism have been synonymized. Pending further taxonomic clarification, the name of the species first described, *G lamblia*, will be retained.

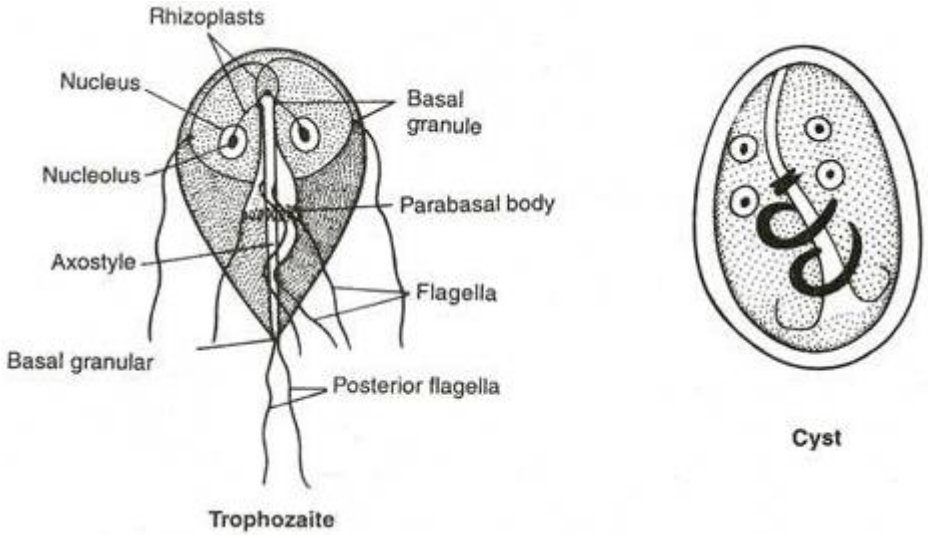
Giardia lamblia has a worldwide distribution with prevalence rates of 2–5% in industrialized countries (الدول الصناعية) and very high rates, up to 50%, in developing countries (الدول النامية). Children up to the age of five are frequently infected.

Morphology & Identification

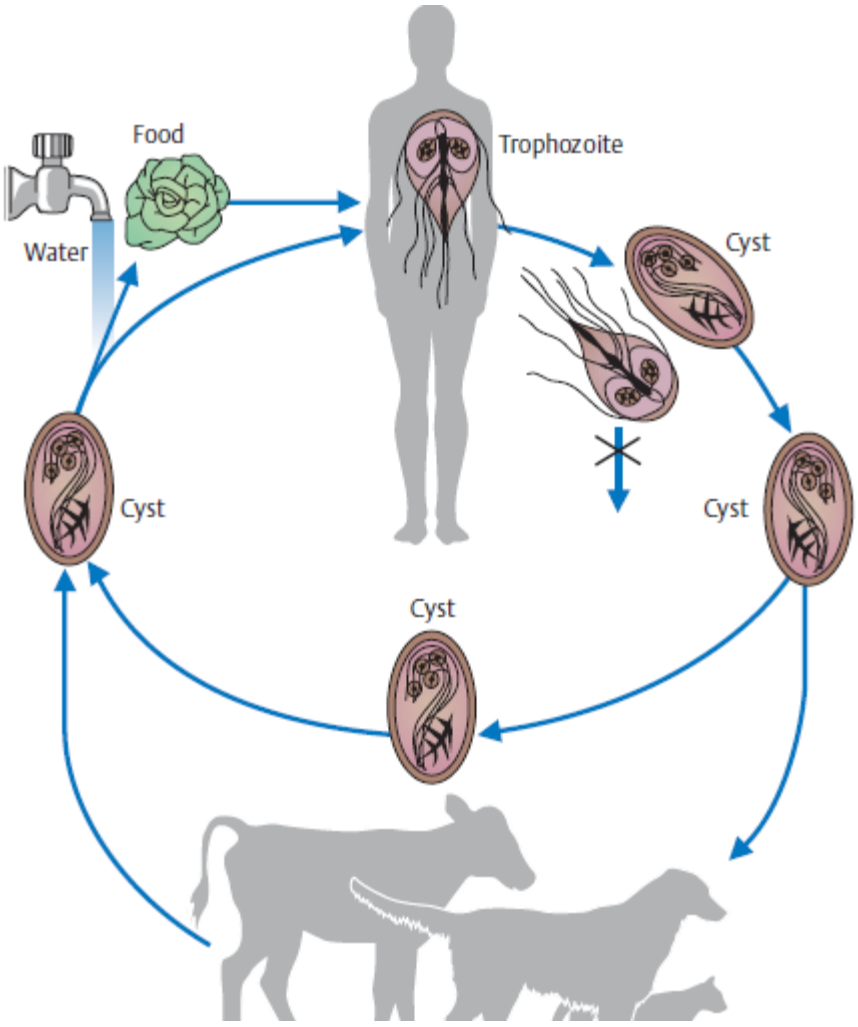
Giardia exists in two morphological forms: a motile vegetative stage, the trophozoite, and a cyst stage. The trophozoite is a pear -shaped, symmetric organism 9-21 µ m in length and 5–12 µm wide. There are four pairs of

flagella (8), two nuclei with prominent central karyosomes, and two axostyles (rod-like supporting organelles). Their dorsal side is convex (محدب); the anterior part of the ventral side forms a concave adhesive disk. Reproduction is by means of longitudinal binary fission of the trophozoites, which are able to produce variant specific surface proteins. The trophozoites live on the small intestine mucosa (less frequently on the gallbladder mucosa as well).

Cysts are oval (8–18 × 7–10 µm) with four nuclei, flagella, and claw-shaped median bodies. The cysts (and, less frequently, trophozoites) are excreted in stool.



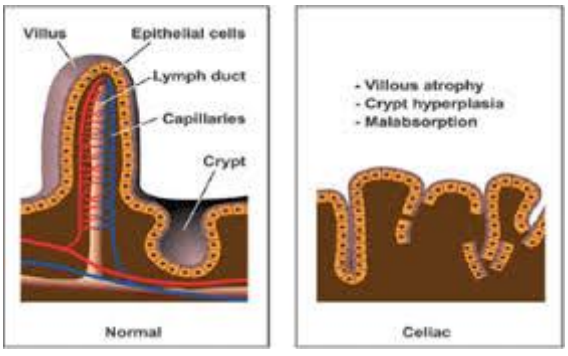
The cysts excreted in stool are responsible for spreading the infection. They remain viable for up to three weeks in moist surroundings at 21 °C and up to about three months in cool water (8 °C). The trophozoites, by contrast, die off soon outside the host. Cysts are transmitted by the fecal-oral route from person to person (within families, kindergartens, etc.) or in food and drinking water. The incubation period for giardiasis is **1 to 3 weeks** after exposure to the parasite.



Life cycle of *Giardia*

Pathogenesis & Clinical Findings

Cysts may be found in large numbers in the stools of entirely asymptomatic persons. In some persons, however, large numbers of parasites attached to the bowel wall may cause (1) irritation (تهيج), (2) low-grade inflammation of the duodenal or jejunal mucosa, (3) acute or chronic diarrhea associated with *crypt hypertrophy, villous atrophy or flattening, (4) epithelial cell damage. The stools may be watery, semisolid, greasy and bad-smelling. Malaise (شعور بالضيق), weakness, weight loss, abdominal cramps (تقلصات بالبطن). Children are more liable to clinical giardiasis than adults. Immunosuppressed individuals are especially liable to massive infection with severe clinical manifestations. Symptoms may continue for long periods.



*للاطلاع

Diagnosis

Diagnosis depends upon finding the dicysts in stools, or cysts and trophozoites in liquid stools. An ELISA kit (enzyme-linked immunosorbent assay) is now also available to detect Giardia-specific structural and soluble antigens in stool samples.

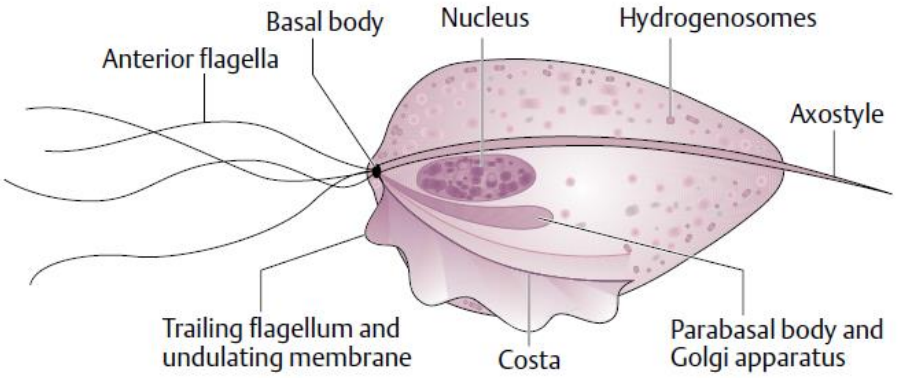
Treatment

Metronidazole (Flagyl) will clear over 90% of *G. lamblia* infections. Recently (nitrothiazole compound) are used for chemotherapy of infections.

Trichomonas vaginalis

Trichomonas vaginalis is a frequent flagellate species that occurs worldwide and is transmitted mainly by sexual intercourse. Causative agent of trichomonosis, it causes vaginitis in women and urethritis in men.

Parasite: *Trichomonas vaginalis* is a pear shaped protozoon about 10–20 µm long and 2–14 µm wide. Five flagella emerge from a basal body at the anterior pole, four freely extend forwards and one extends backwards, forming the outer edge of the undulating membrane, which reaches back only just beyond the middle of the cell. An axial rod made up of microtubules (the axostyle) protrudes with its free tip from the posterior end of the cell. The oval cell nucleus lies near the upper pole of the protozoon. Trichomonads are anaerobic protozoa, it colonizes the mucosa of the urogenital tract and reproduces by longitudinal binary fission. Trichomonads do not encyst.



Morphology of *Trichomonas vaginalis* (schematic).

The nonpathogenic trichomonads, *Trichomonas hominis* and *Trichomonas tenax,* cannot readily be distinguished from *T. vaginalis* when alive. For all practical purposes, trichomonads found in the mouth and lung of many vertebrate are *T. tenax;* in the intestine [caecum and colon] are *T. hominis.*

Pathogenesis and Pathology

The incubation period between exposure and infection is unknown, but it's thought to range from **five to 28 days**. *T. vaginalis* is capable of causing low-grade inflammation. [The pH, physiologic status of the vaginal and the bacterial flora are among the factors affecting pathogenicity]. The organisms do not survive at normal vaginal acidity of pH 3.8–4.4.

In females, the infection is normally limited to vagina and cervix; it does not usually extend to the uterus. Vaginitis can develop after an incubation period of two to 24 days, and then colonizes the vaginal mucosa, infection results in production of yellow or cream-colored discharge. In males, the prostate, seminal vesicles, and urethra may be infected. About 10% of infected males have a thin, white urethral discharge.

Diagnosis

A fresh specimen of vaginal or urethral secretion is mixed with physiological saline solution and examined under a microscope for trichomonads. Dried smears may be stained with hematoxylin or Giemsa. Other special methods are based on detection of antigen (ELISA) or DNA (PCR).

Treatment

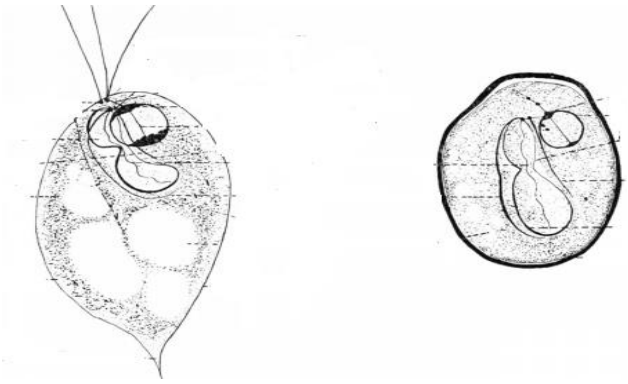
Metronidazole (Flagyl) is given at dose of 500 mg orally twice daily for 7 days. It is the only drug that should be used to treat trichomoniasis if there is any chance of pregnant.

Dientamoeba fragilis [Lec 3]

Long classified with the amebas, this occasionally pathogenic organism is now recognized as an ameboflagellate in the same order as Trichomonas. In it ameba stage it measures 4–18µ m, has one or two nuclei. It is commonly found in the human colon along with the true amebas, but it contains a flagellate structure (the parabasal body) near the nuclei and, like *Trichomonas*, lacks a cyst stage. It is mildly pathogenic in about 25% of infected individuals, who may experience abdominal pain, diarrhea and vomiting similar to giardiasis.

Chilomastix mesnili

This parasite can be confused with trichomonas in the laboratory. It is found throughout the world. The trophozoite is pear-shaped 10-20 µm. It has 1 large nucleus with a small karyosome. The oral groove (cytostome) is sometimes seen near the nucleus. It has 3 flagella which extend from the nucleus at the anterior end of the parasite like Trichomonas, but the spiral motion of the trophozoite is unlike that of Trichomonas. The cyst is lemon-shaped, uninucleate, and 7–10 µm long, excystation releases trophozoites. It normally lives in the cecum and/or colon; it is generally considered a commensal whose contribution to pathogenesis is uncertain and no treatment is recommended. Infection occurs by the ingestion of cysts in contaminated water or food or by the fecal-oral route



Common protozoa) infections of humans

Organism	Disease and site of infection	Mode of transmission	Geographic distribution
Amoebae			
Entamoeba histolytica	Amoebiasis: gut and occasionally liver	Faecal-oral ingestion of cysts	World-wide
Acanthamoeba	Chronic encephalitis in immunocompromised host	Haematogenous spread from skin or lung	World-wide
	Keratitis (infection of cornea)	Contaminated contact lenses or eye trauma	World-wide
Naegleria fowleri	Acute meningoencephalitis	Nasal instillation whilst swimming	World-wide
Flagellates			
Trichomonas vaginalis	Trichomoniasis: vagina and urethra	Sexually transmitted (usually asymptomatic in males)	*World-wide*
Giardia lamblia	Giardiasis: gut	Faecal-oral ingestion of cysts	World-wide

LEC 5 **PARASITOLOGY**

The Hemoflagellates

The hemoflagellates of humans include the genera *Trypanosoma* and Leishmania.

Phylum: **Sarcomastigophora**

Sub phylum**:** **Mastigophora**

Order: **Kinetplastida**

Family: **Trypanosomatidae**

One feature of this family is that various forms develop during the life cycle in vertebrates and vectors (insect) involved. The morphologically differentiated forms include spindly, uniflagellate stages (trypomastigote, epimastigote, promastigote) and a rounded, amastigote form.

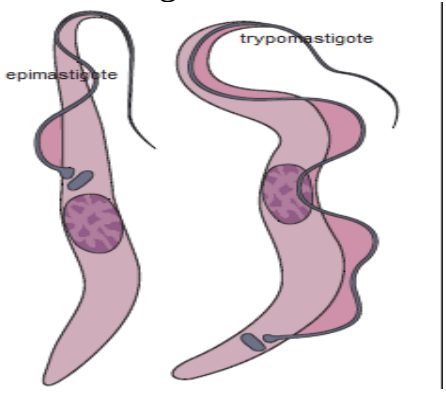
Trypanosoma

There are two distinct types of human trypanosomes:

1- African, causative agents of African trypanosomosis (sleeping sickness) and is transmitted by tsetse flies (*Glossina*): *Trypanosoma brucei rhodesiense* [occurs in eastern and southeastern Africa and *Trypanosoma brucei gambiense* [the disease occurs mainly in western and central Africa].

2- American, which causes Chagas' disease and is transmitted by conenose bugs (*Triatoma*): *Trypanosoma cruzi*.

Trypomastigote has: a central nucleus, an elongated mitochondrion containing the kinetoplast in its posterior section. Close to, but outside of the mitochondrion is the base of the flagellum, which originates in the plasmatic basal body. The flagellar adheres locally to the cell surface so that an "undulating membrane" is folded out during movement—visible under a light microscope. Special organelles of the kinetoplastids are the membrane-enclosed glycosomes, which contain glycolytic enzymes. Spiral microtubules forming a cytoskeleton are arranged along the inner cell membrane, in the epimastigote form, the kinetoplast and base of the flagellum are near the nucleus or more toward the anterior end.



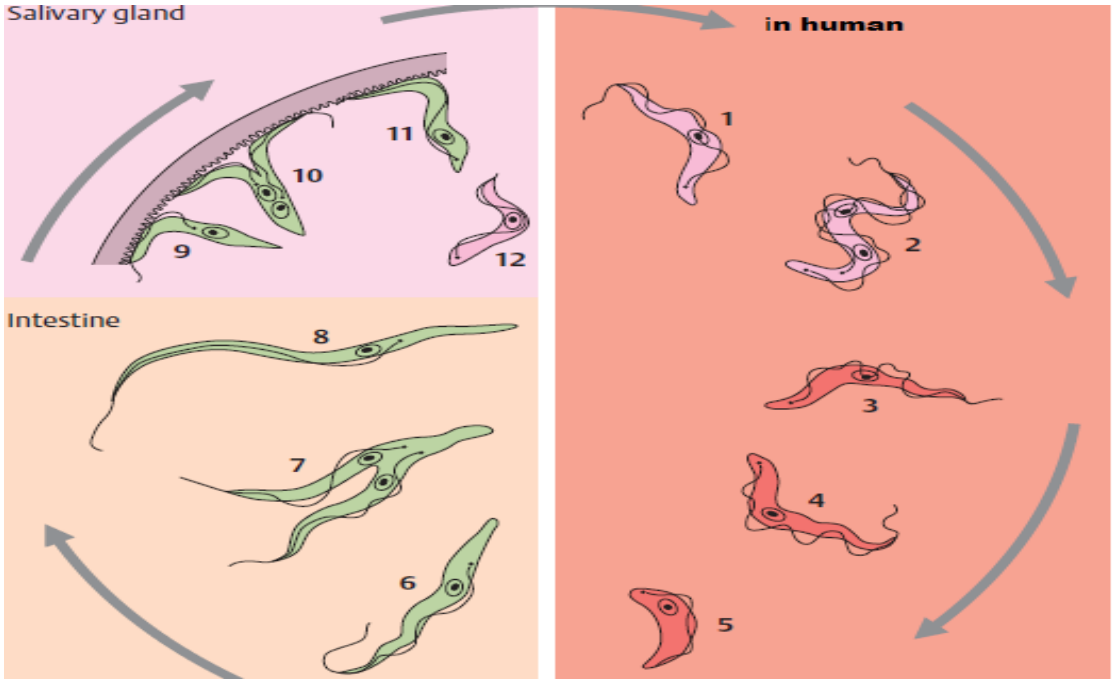
Life cycle

T. gambiense and T. rhodesiense parasitize extracellular in the blood plasma or in other body fluids of vertebrates.

1-The trypomastigote forms are pleomorphic in human blood they transform to slender, 25–40 µm-long forms with the flagellar tip extending outside the anterior end, reproduce by longitudinal binary fission.

2-With decreasing parasitemia, they appear short, 12–25 µm long forms without a free flagellar end. These forms do not divide in blood but are infective for Glossina (tsetse flies). The cell surface of the bloodstream forms is covered with a uniform layer (about 10–15 nm thick) of a specific glycoprotein, which can be replaced by another glycoprotein. These are variant specific surface antigens (VSSA),

3-The trypanosomes taken up by Glossina (tsetse flies) when they suck blood from an infected host go through a complex developmental and reproductive cycle in the insects lasting 15–35 days. The resulting (metacyclic) stages can then be inoculated into the skin of a host with the fly's saliva. Infected Glossina can transmit the trypanosomes throughout their entire lifespan (up to six months).



Life cycle of *Trypanosoma brucei* Human blood: 1 trypomastigote, slender form with variant specific surface antigen (VSSA); 2 binary fission form; 3, 4 slender forms with other VSSA type; 5 short (**Stumpy forms**: thick and short, typically no free flagellum, but a short one may be present) . Glossina intestine: 6–8 procyclic forms without VSSA (reproduction by longitudinal fission). Salivary gland of Glossina: 9, 10 epimastigote forms on epithelium; 11 trypomastigote form without VSSA; 12 trypomastigote form with VSSA (metacyclicform). (According to Parasitic Protozoa, Vol. 2, San Diego: Academic Press; 1992: 94.)

In *T. cruzi* the (reduviid) bugs ingest trypomastigote in bloodmeals from infected hosts (vertebrate animals, humans).

1-The bug usually bites round the edges of the mouth and eyes. The trypomastigotes are either rubbed into the skin or penetrate the conjunctiva or membranes of the nose and mouth.

2- Trypomastigotes become amastigotes in localized reticuloendothelial cells and multiply. The amastigotes develop into trypomastigotes which are released into the blood when the cell ruptures.

3- The trypomastigotes reach tissue cells especially heart muscle, nerves, skeletal muscle and smooth muscle of the gastrointestinal system by way of the blood and lymphatic system.

4-The trypomastigotes become amastigotes and multiply forming pseudocysts. Within the pseudocyst some amastigotes become elongated and develop first into epimastigotes and then trypomastigotes. When the cell ruptures the trypomastigotes are released into the blood and continue to circulate while others invade further tissue cells. The life cycle completes when a (reduviid) bugs vector ingests circulating trypomastigotes. In the vector the trypomastigotes transform and develop into epimastigotes, multiply by binary fission in the gut of the bug. After about 10-15 days, metacyclic trypomastigotes are formed and can be found in the hindgut of the bug.

Pathogenesis, Pathology, & Clinical Findings

1-Infective trypanosomes of *T bruci* are introduced through the bite of the tsetse fly and multiply at the site of inoculation to trypanosomal chancre. 2-Invade the lymph glands. Invasion of the lymph glands is usually accompanied by a high irregular fever with shivering (رجفة), sweating and an increased pulse (نبض) rate. The lymph glands near the bite often become swollen, in *T. b.* gambiense the glands at the back of the neck and *T. b.* rhodesiense usually the glands under the jaw are affected. As the disease progresses, edema of the eyelids, face and sleeplessness are features along with increasing lethargy (خمول).

Trypanosomes may invade the central nervous system giving symptoms of meningoencephalitis, confusion, apathy, excessive sleeping and incontinence. At this stage, the cerebrospinal fluid (CSF) usually contains mononuclear cells and a few trypanosomes may be detected. If untreated, character changes, coma develops, finally resulting in death. Such signs are

more commonly seen with *gambiense* than in *rhodesiense* in which patients often die before these symptoms develop fully.

T cruzi entry of the parasite into the skin or conjunctiva with a local, inflammatory dermal reaction (chagoma) or conjunctivitis with eyelid edema. In acute phase: fever, edema, lymph node swelling, hepatomegaly, splenomegaly, myocarditis, and, less frequently, meningoencephalitis.

Differential Diagnosis

T b rhodesiense and *T b gambiense* are morphologically identical but may be distinguished by their geographic distribution, vector species, and clinical disease in humans. Examination of blood for the parasites, examination of aspirates from enlarged lymph glands for the parasites, examination of the CSF for the parasite and detection of trypanosomal antibodies in the serum.

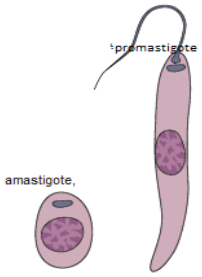
Treatment

There is no effective drug treatment for American trypanosomiasis. African trypanosomiasis is treated principally with suramin sodium (Germanin).

Leishmania

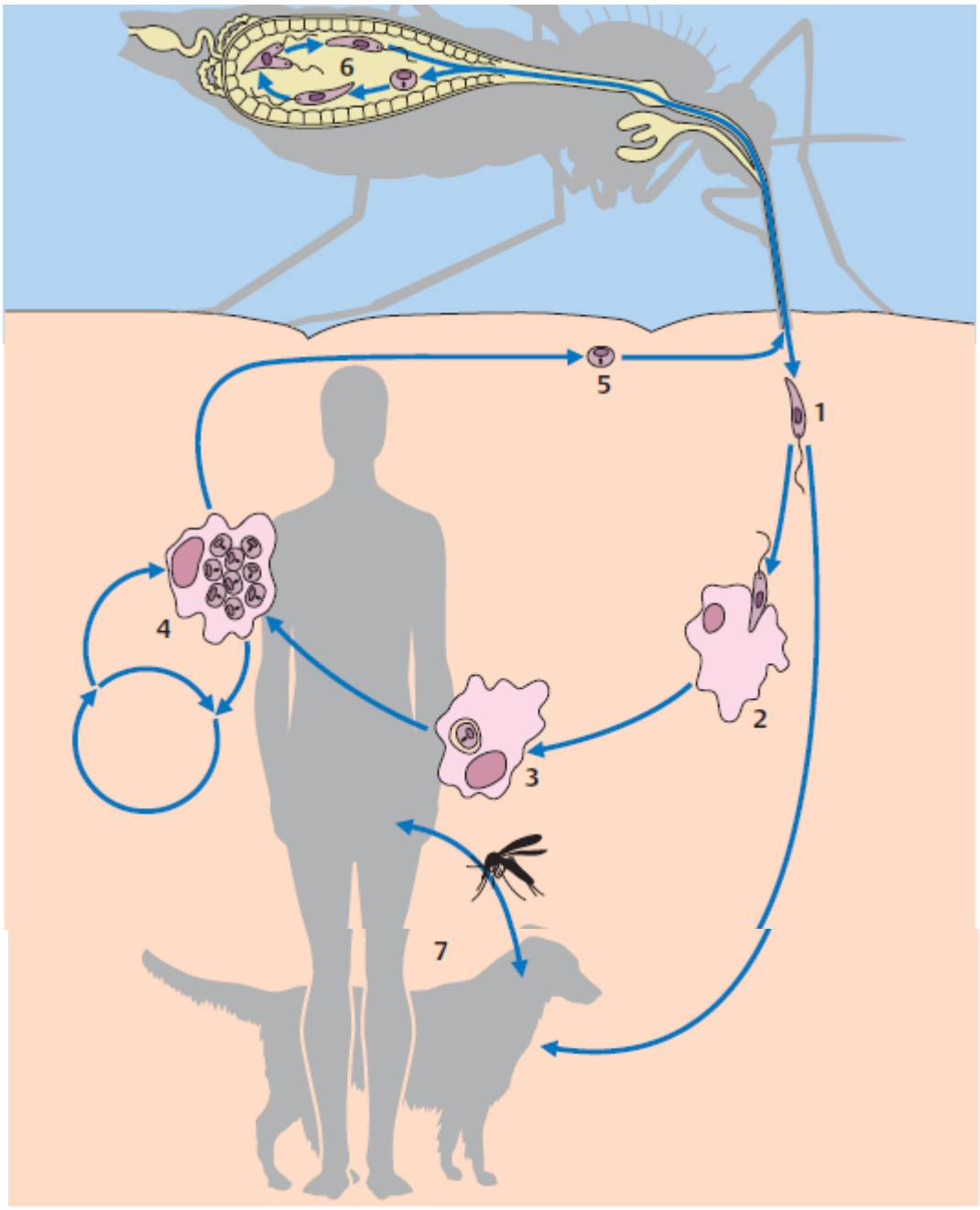
The genus *Leishmania* are parasitic protozoa responsible for the leishmaniases, a group of diseases affecting human and various animal populations throughout much of the tropics and subtropics. Over 30 species of *Leishmania* have been named to date, and of these 10 or so are of significant medical and veterinary importance. Leishmanias are transmitted by sandflies (Phlebotomidal) and cause the following main forms of leishmaniasis in warm regions. The major clinical syndromes found in human beings (1) visceral leishmaniasis (kala-azar [VL]), (2) cutaneous leishmaniasis[CL] (Oriental sore, Baghdad boil, wet cutaneous sore, dry cutaneous sore, (3) mucocutaneous or naso-oral leishmaniasis [MCL] (espundia).

The sand fly genera Phlebotomus (Old World) and Lutzomyia (New World) transmits the infective promastigotes by bite. The promastigotes rapidly change to amastigotes after phagocytosis by macrophages, and then multiply, filling the cytoplasm of the macrophages. The infected cells burst, the released parasites are again phagocytosed, and the process is repeated, producing a cutaneous lesion or visceral infection depending upon the species of parasite and the host response. The amastigotes are oval, 5-8 µm, with a laterally placed oval vesicular nucleus and a dark-staining, rod-like kinetoplast.



Life cycle

In humans and other vertebrates, leishmanias parasitize in mononuclear phagocytic cells(macrophages, monocytes, Langerhans cells) in the amastigote form. The leishmania species are transmitted by female of "sandflies." The amastigote stages of the parasite ingested by the insect with a blood meal are transformed in its intestine into slender, flagellate promastigote forms 10–15 µm long, which reproducing asexually by longitudinal fission then again migrate back into the proboscis. At tropical temperatures this process takes five to eight days. When infected sandflies take another blood meal the promastigote forms are inoculated into a new host (humans or other vertebrates). The promastigotes quickly (within 12–14 hours) transform into amastigote stages, which are finally surrounded by a parasitophorous vacuole within the phagolysosome and reproduce by binary fission. The amastigote forms are then released and can infect new cells.



Life cycle. 1 Inoculation of promastigote stages by sandfly; 2 ingestion of parasites by phagocytes (Langerhans cells, dendritic cells, macrophages); 3 amastigote form in parasitophorous vacuole of a macrophage; 4 reproduction of amastigote forms in a macrophage; 5 ingestion of amastigote forms by sandfly with blood meal; 6 transformation into promastigote form and multiplication in insect; 7 dog as reservoir host.

Type of leishmaniasis

Visceral leishmaniasis: Also known as kala- azar or black fever [a symptom of hyperpigmentation of hands, feet and abdomen. The disease is second largest killer in the world after Malaria. Several species of leishmania to give rise to the visceral leishmaniasis.

L. donovani: Asia: India, Bangladesh. Mainly in adults. Reservoir hosts: humans. Vectors: Phlebotomus species.

L. donovani: Africa: Mainly Sudan, Ethiopia, And Kenya. Reservoir hosts: humans dogs (Felidae, rodents?)2. Vectors: Phlebotomus species.
L. infantum: Mediterranean region (Turkey, northern Africa), Middle East and central Asia, China. In children and adults; in adult's cutaneous manifestations as well: Reservoir hosts: humans, dog.

Pathogenesis, Pathology, & Clinical Findings
1-Irregular fever, sometimes hectic.2- Hepatosplenomegaly (usually, the spleen is more prominent than the liver), anemia, leukopenia and 3-a high total protein level and a low albumin level.
Even with recovery, kala -azar does not always leave its host unmarked. Sometimes after successful treatment, a secondary form of disease may set in, called post kala-azar dermal leishmaniasis.

Cutaneous leishmaniasis is endemic in 88 countries on five continents. There are 1–1.5million cases of cutaneous leishmaniasis reported yearly worldwide. The common name are (Baghdad sore, oriental sore, Delhi boil, chiclero ulcer, etc) *L tropica, L major, L mexicana, L braziliensis,* and other dermotropic forms induce a dermal lesion at the site of inoculation by the sandfly**:**
Main localization: Skin. Incubation: Weeks to months.
Primary symptoms: On skin accessible by Phlebotomus species, development of solitary or multiple, dry, later possibly ulcerating papules; rarely spread to lymph vessels and nodes.
L. major: Northern Africa, Middle East, causing moist type cutaneous lesion (ulceration type).
Incubation: up to 2 months. Rapid growth of cutaneous lesion, later ulceration and healing within (6) months. Reservoir hosts: rodents.
L. tropica: Mediterranean region, India, causing dry type cutaneous lesion (non- ulceration type).
Incubation: 2–24 months. Development of lesions and persistence longer than *L. major*. Reservoir hosts: humans.
L.chagasi: Central and northern South America. Mainly in youths. Cause chiclero ulcer. Reservoir hosts: humans, dogs, fox species. Vectors: Lutzomyia species.

Therapy and prevention. Treatment of VL is usually done with pentamidine.
In March 2014, FDA approved the oral agent **miltefosine** [antitumor alkylphospholipid for oral application] for treatment of cutaneous, mucosal, and visceral leishmaniasis.

Phylum: Ciliophora

Class: Kinetofragminophora

Order: Trichstomastida

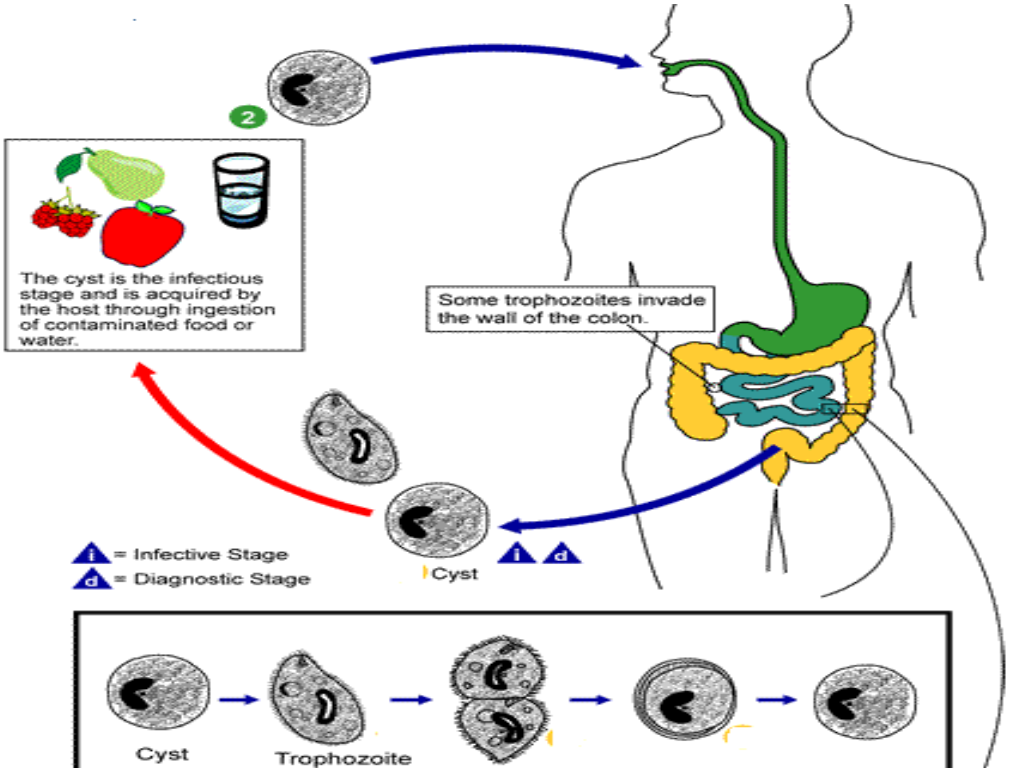
Ciliophora: are complex protozoa bearing cilia distributed in rows or patches, with two kinds of nuclei in each individual. *Balantidium coli,* a giant intestinal ciliate of humans and pigs, is the only human parasite representative of this group.

Balantidium coli

Causative agent of balantidiosis

Balantidium coli are the largest protozoan and the only ciliate parasite that infects humans. Trophozoite size (30–150 µm long) with two nuclei are clearly visible macronucleus is long and kidney- shaped and spherical micronucleus nestled next to it. It is frequently found as an inhabitant of the large intestine of monkeys, rats, and in pigs. Transmission of balantidiasis occurs via ingestion of spherical cysts (40–60 µm) from host to host on the fecal-oral route. Following ingestion, excystation occurs in the small intestine and the trophozoites colonize the large intestine. Trophozoites undergo encystation to produce infective cysts. Some trophozoites invade the wall of the colon and multiply; some return to the lumen and disintegrate. It occasionally causes intestinal necrosis and inflammation with ulceration. Mature cysts are passed in the stool. Diagnosis involves detection of cysts or vegetative forms in fecal samples. Drugs recommended for treatment are **tetracyclines** and alternatively **metronidazole**.

*In the cyst only macronucleus and contractile vacuoles are visible.



Phylum: Apicomplexa [have a unique organelle, a type of plastid called an apicoplast].

Class: Sporozoea [they are unicellular spore- forming].

Order: Eucoccida

Sporozoans

→ This is a unique group because all members are parasitic (most are intracellular).

→ They lack any visible means of locomotion.

→They have complex life cycles involving sexual and asexual reproduction.

→They are capable of producing many stages among which are sporocysts or oocysts are producing the infectious sporozoites.

→ The class **Coccidia** contains the human parasites *Isospora*, *Toxoplasma*, and others. One of these, *Cryptosporidium*, has been implicated as a cause of intractable diarrhea among the immunosuppressed. Within the class **Haematozoa** (blood sporozoans) are the malarial parasites (*Plasmodium* species) and members of the order Piroplasmida, which includes *Babesia* species.

Blood Sporozoans

[The Plasmodia]

P. vivax (Benign Tertian Malaria)

P. malariae (Quartan Malaria)

P falciparum (Malignant Tertian Malaria)

P ovale (Ovale Tertian Malaria)

Life cycle. The life cycle of malaria plasmodia includes phases of asexual multiplication in the human host and sexual reproduction and formation of sporozoites in the vector, a female Anopheles mosquito.

In human host**:**

1. Humans are infected through the bite of an infected female Anopheles mosquito that inoculates sporozoites into the bloodstream. Only a small number of sporozoites are needed to cause an infection in humans (about 10 *P. falciparum*). Within about 15–45 minutes of inoculation, the sporozoites of all *Plasmodium* species reach the liver in the bloodstream and infect hepatocytes, in which asexual multiplication takes place.

2. The sporozoite develops into a multinuclear, large (30–70 μm) schizont described as a tissue schizont. Following cytoplasmic division 2000 (*P. malariae*) to 30 000 (*P. falciparum*) merozoites are produced. This development takes six (*P. falciparum*) to 15 (*P. malariae*) days.

3. Schizonts release the merozoites, which then infect red blood cells. [**In infections with *P. vivax* and *P. ovale*, some sporozoites remain hidden as so-called hypnozoites, which may develop into schizonts following activation after months or years].

4. A *Plasmodium* that has recently infected an erythrocyte (<12 hours) appears ring-shaped with a thin cytoplasmic border in a Giemsa-stained blood smear. This stage is very similar in all four *Plasmodium* species.

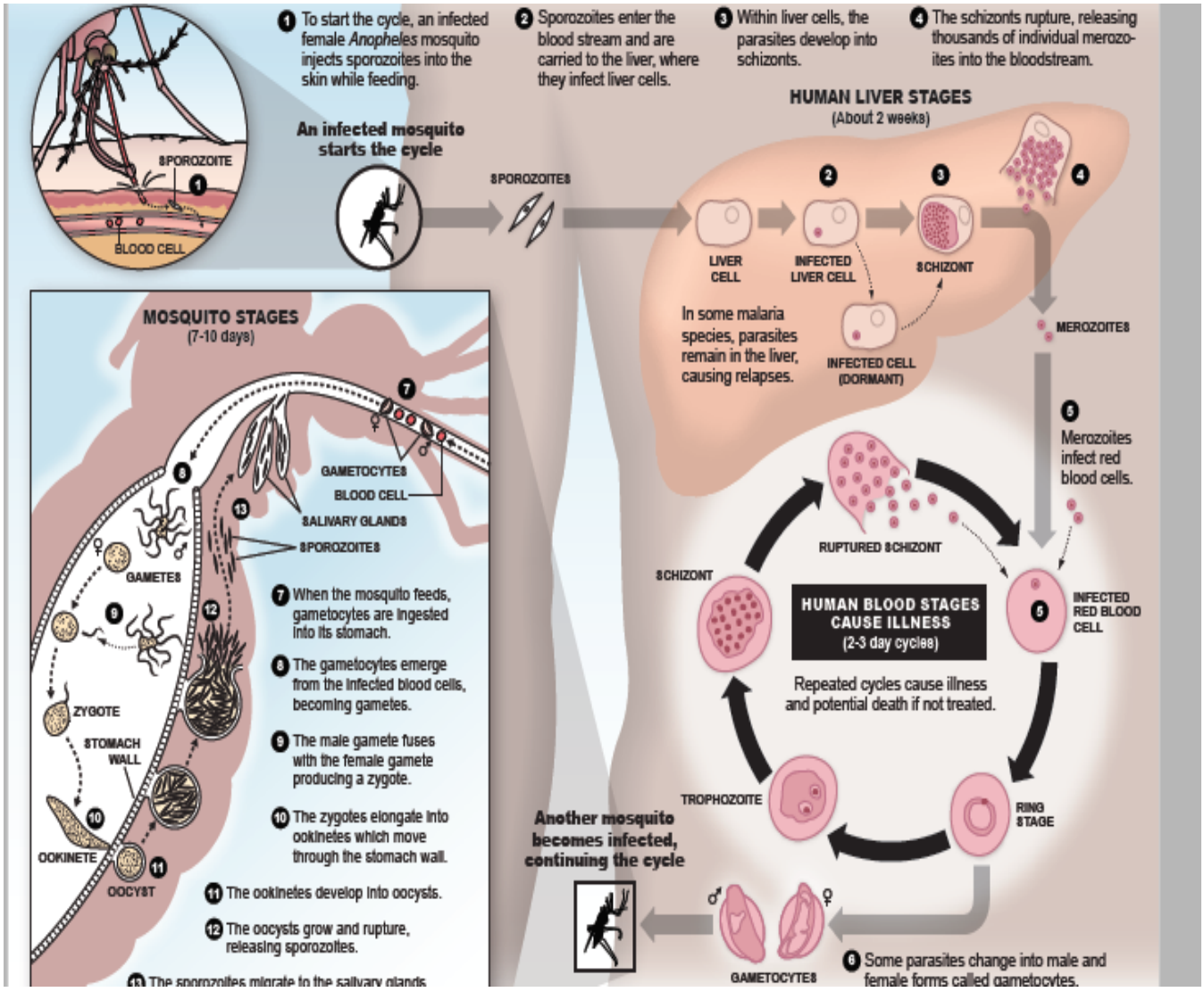
5. The ring forms develop into schizonts, which feed on glucose and hemoglobin. The schizont undergoes multiple divisions to produce merozoites, in different numbers depending on the Plasmodium species.

6. The merozoites enter the blood plasma when the erythrocyte is destroyed; they infect other erythrocytes and begin a new asexual cycle. A cycle takes 48 hours

with *P. vivax, P. ovale*, and *P. falciparum* and 72 hours with *P. malariae*. In Erythrocytic stage [Trophozoites, Gametocytes].

Life cycle in mosquito host:

7. When the mosquito feeds, gametocytes are ingested into its stomach. The gametocytes emerge from the infected blood cells, becoming gametes.
8. The male gamete fuses with the female gamete producing a zygote.
9. The zygotes elongate into ookinetes which move through the stomach wall.
10. The ookinetes develop into oocysts. The oocysts grow and rupture, releasing sporozoites. The sporozoites migrate to the salivary glands, ready to be injected and renew the cycle.



Signs and Symptoms

Initial symptoms non-specific 1-Headache 2-Muscle aches 3- Nausea, vomiting .
Then malarial paroxysms begin .Shaking chill (10-15 min) • High fever (typically 10

h; up to 36 h) -Cycle repeats every 36-72 hours (species specific) - Primary attack lasts 2-24 weeks (spp. specific) then, Splenomegaly -Hepatomegaly and Anemia.

Diagnosis • There are two ways to diagnose malaria in humans: – Clinical diagnosis n detection of parasites in the blood.

Treatment • Most drugs used in treatment are active in the blood stage: – Chloroquine, sulfadoxine pyrimethamine (Fansidar) and mefloquine.

Tissue Sporozoans

Toxoplasma gondii [was first discovered in 1908 in desert rodent]

Causative agent of toxoplasmosis

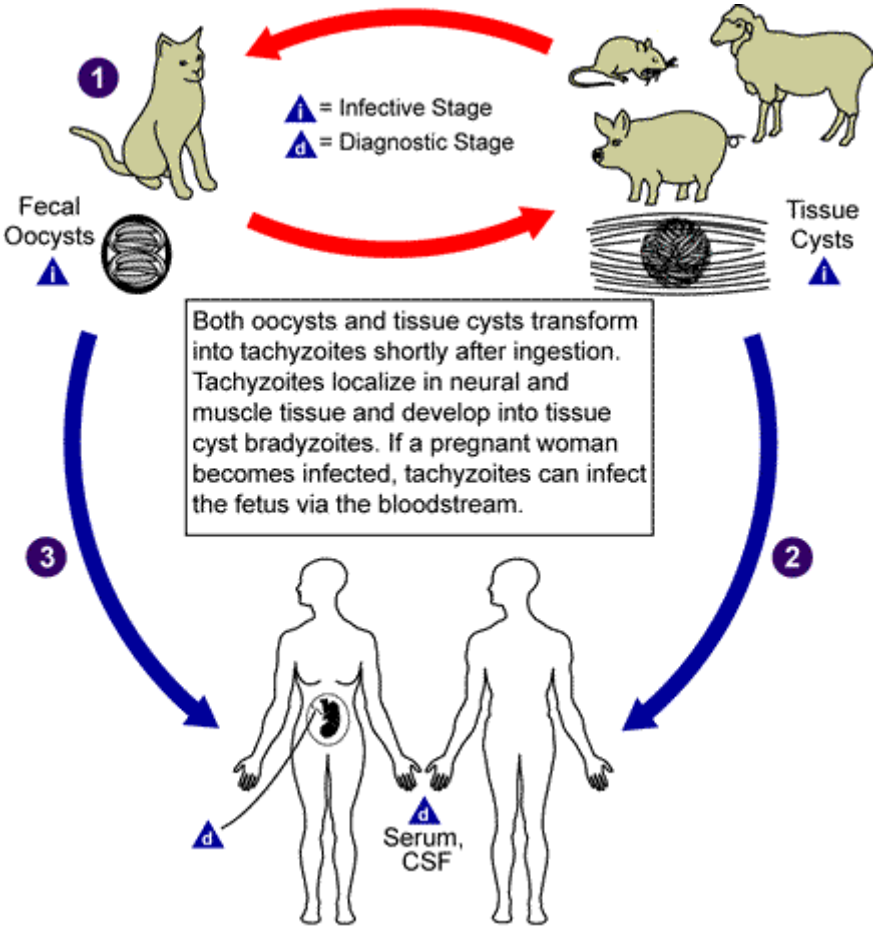
Toxoplasma gondii is the causative agent of a zoonosis that occurs worldwide with high prevalences (up to 80% depending on region and age). Humans are infected by ingesting oocysts excreted by the definitive hosts (cats) or by eating unprocessed meat containing Toxoplasma cysts. If a women contracts toxoplasmosis for the first time during pregnancy, via placental transmission of the pathogen to the fetus is possible with potential severe consequences (for example malformations(تشوهات) or eye damage. Transmission can occur in 3 ways (1)-Fecal-oral (2)-Eating contaminated meat (3)Transplacental.

The life cycle of *T. gondii* includes various stages:

Development in the definitive host (cat):

1- Cat becomes infected by eating infected rodent (either sporozoites from Oocysts or Bradyzoites from tisse cysts) invade the muscle cells of cats samll intestine where they from Schizonts or Gametocytes after sexual fusion of Gametes, oocysts develop exit from the host cells into the gut lumen of the cat and pass out via feces.
2- The Oocyst are contaminated with water, food and soil are ingested by intermediate host.
   The protozoa reproduce asexually in the intermediate host (mammals, birds, humans).
3- Ingested oocyst goes to the digestive tract and releases eight sporozoites which pass through the gut wall circulate in body and invade various cells.

4- It is here that they are engulfed by macrophages .
5- In the macrophage tachyzoites [are sickle shaped]develop and travel to various parts of the body via blood stream (Heart, spleen, liver and brain).
6- Then tachyzoites encyst into zoitocysts and pseudocysts which contain bradyzoites (inactive).



Pathogenicity and clinical manifestations

1-Primary infection in immunocompetent persons: This is the most frequent form without clinical manifestations, recognizable by the specific serum antibodies. The infection can persist for the life of the host.

2-Primary infection during pregnancy. This may cause prenatal infection of the fetus and thus become a significant threat**.**

3-Primary infection in immunosuppressed persons.

The infection gives rise to febrile generalized illness with hepatosplenomegaly, myocarditis, meningoencephalitis, eye damage, and other manifestations. There is a high rate of lethality if left untreated.

Diagnosis

► Biopsy of humans.
► (ELISA) Enzyme-Linked Immunobsorbent Assays.
► (IFAT) Indirect Fluorescent Antibody tests.
► In prenatal diagnostics, PCR is seeing increasing use for direct detection of pathogens in the amniotic fluid. Detection of Toxoplasma DNA with this method is a reliable sign of fetal infection and demands a chemotherapeutic or other response accordingly

Treatment

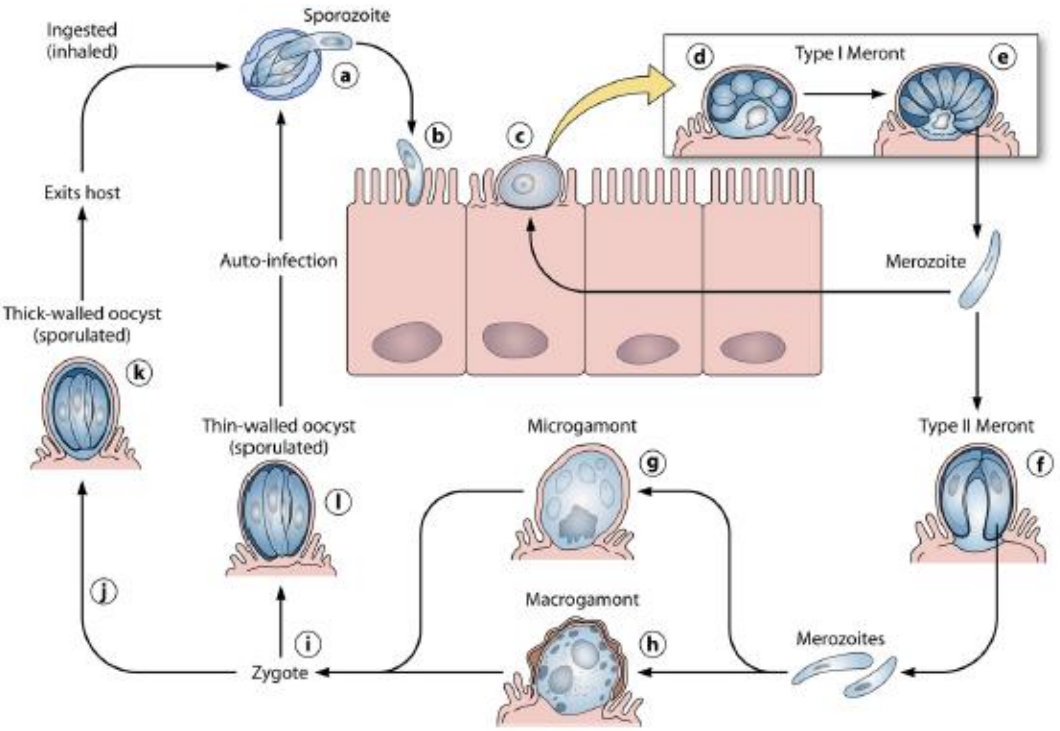
Spiromycin, Sulfonamide and clindamycin.

Cryptosporidium

Causative agent of cryptosporidiosis

Cryptosporidiosis in humans is predominantly caused by *Cryptosporidium hominis* (= human genotype of *C. parvum*) and the bovine genotype of *C. parvum.* Humans are infected by peroral ingestion of infective oocysts. In immunocompetent persons, the infection remains inapparent or manifests as a self-limiting diarrhea. Persistent, choleralike, life-threatening diarrheas are observed in AIDS patients.

Life cycle



Isospora

Causative agent of isosporosis

Isospora belli is coccidian protozoan parasite which infected different animal such as human, cat, pig, and dog.

1- Unsporulated oocysts passed in the feces and become sporolated after 24 hours.
2- Then infected human or dog and release of sporozoites, further development (schizogony) takes place in the epithelium of the upper small intestine then release merozoite to invade another cell and some of them developed to macre and micro gametocyte (sexual cycle).
3- , leading finally to oocyst formation.

In AIDS patients, encysted sporozoites have been found in various extraintestinal organs (lymph nodes, liver, gallbladder, spleen). In those patient can cause severe clinical symptoms persistent diarrhea, weight loss, and fever.

Diagnosis is made by detection of unsporulated oocysts (20–30 µm long) in stool or of developmental stages in intestinal biopsies.

Platyhelmintha (syn. Platyhelminthes)

Trematoda (Flukes)

1-Most of the trematode species that parasitize humans are dorsoventrally flattened with an oval to leaf-shape, unsegmnted body except Schistosoma sp. which is cylindrical.

2-The body is equipped with 2 muscular suckers for attachment; an oral one and a ventral one except Heterophyes which has thired genital sucker.

3- Most species are hermaphroditic, only the schistosomes have separate sexes

4-All require one or more intermediate host(s) for completion of their life cycles.

a- The 1st intermediate host of all flukes is snail where asexual reproduction occurs
b- The adult worm develops in secondary intermediate host except schistosmes which do require only on intermediate host.

5- The eggs are in general oval equipped with a lid at the top called operculum, through which the larval worm comes out to find its appropriate snail host.

6-The mode of transmission is by ingestion of cyst-contaminated food, except in schistosomes, where cercariae penetrate the skin directly.

Kigdom :Animelia

Phylum: Platyhelminthes

Class: Trematoda

Subclass: Digenea

***Fasciola* species**

Fasciola hepatica (Common Liver Fluke) and *F. gigantica* (Large or Giant Liver Fluke)

Causative agents of fasciolosis

Fasciola hepatica and *F. gigantica* are frequent bile duct parasites of domestic animals. In their life cycle freshwater snails act as intermediate hosts. Humans become accidentally infected when they eat plants (e.g., watercress الجرجير) to which infectious parasite stages (metacercariae) adhere.

{للقراءة *Fasciola hepatica* occurs worldwide as an important parasite in domestic ruminants (حيوانات مجترة) that can also infect other animal species. Sporadic or endemic *F. hepatica* infections in humans have been reported from about 50 countries (WHO, 1999). In Asia and Africa, human infections with the 7.5cm long giant liver fluke (F. gigantica) are also reported.

Parasites, life cycle, and epidemiology.

F. hepatica is a flattened, leaf-shaped parasite about [2–5cm] long and at most [1cm] wide. The cephalic is cone shape with the oral sucker. The definitive hosts are sheep, human, cattle.

1-Adult liver flukes parasitize in the bile ducts. They produce large (approx.

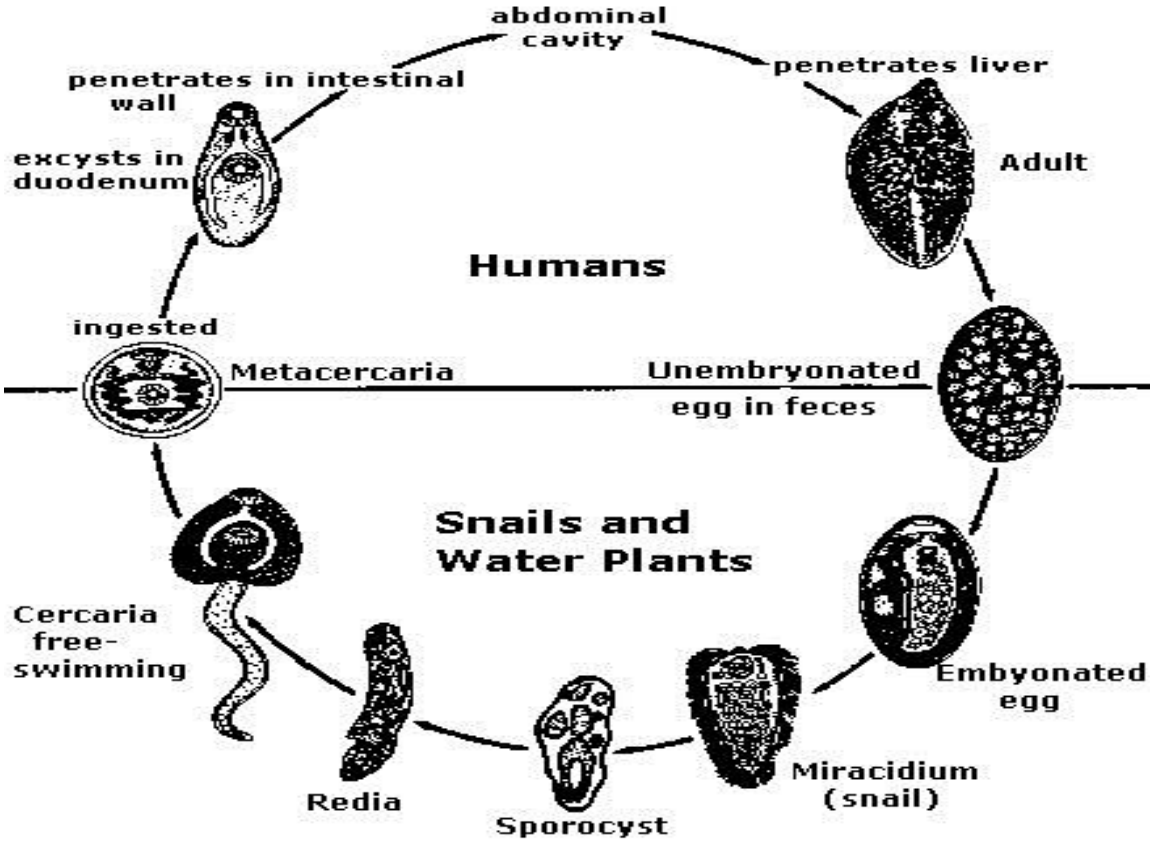
130×85 µm), golden-brown, eggs that are shed by the bile duct-intestinal tract route.

2- Miracidium, develops in the egg within a few weeks. The miracidia then hatch and penetrate into freshwater snails (Lymnaea truncatula)

3-Then they transform into sporocysts. After formation of further asexual reproductive stages (rediae), then tailed cercariae develop and swarm out of the snails into water.

4-They soon attach to plants and encyst, transform into infective stage [encysted metacercariae], which are then ingested with vegetable food of their definitive hosts.

Eating watercress contaminated with metacercariae is one of the sources of infection for humans. They enter the intestinal wall, and migrate through the peritoneal cavity to the liver. After migrating through the hepatic parenchyma for about six to seven weeks, the parasites finally reach the bile ducts, in which they develop to sexual maturity. Egg excretion begins two to three months post infection [p.i.].



Clinical manifestations. The infection appear after an incubation period of four to six weeks, become symptomatic with abdominal pain, hepatomegaly, fever, leukocytosis

and eosinophilia (acute phase) and anemia. Occasionally, the parasites also migrate into other organs than the liver.

Diagnosis. 1- Rise in liver specific serum enzymes. 2-Detection of operculated eggs in stool after at least two to three months p.i.

Therapy. The drug of choice is **Triclabendazole**, originally developed as a veterinary drug and is recommended by the WHO.

Dicrocoelium

Dicrocoelium dendriticum (Lancet Liver Fluke)

Causative agent of dicrocoeliosis

The lancet liver fluke (0.5–1.0 × 0.2 cm) a bile duct parasite in sheep, cattle, and other herbivores, Its life cycle includes two intermediate hosts (terrestrial snails and ants). Humans become infected accidentally when they ingest ants containing infective metacercariae of this liver fluke. Such infections are rare with mild abdominal and hepatic symptoms.

Diagnosis is based on detection of eggs in stool (about 40 × 25 µm, oval, dark brown, containing.

Treatment Praziquantel has been shown to be effective against *Dicrocoelium* in animals.

Schistosoma (Blood Flukes)

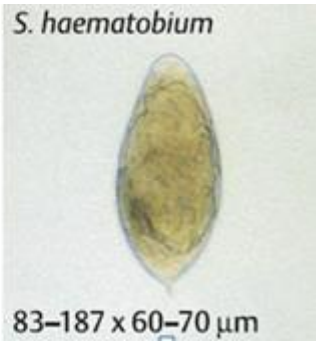
Causative agents of schistosomosis or bilharziosis

Schistosomosis is also known as bilharziosis after the German physician Theodor Bilharz, who discovered Schistosoma hematobium in human blood vessels in 1851. One of the most frequent tropical diseases with about 200 million infected persons. The occurrence of schistosomosis depends on the presence of suitable intermediate hosts (freshwater snails).

Human infections result from contact with standing or slow-moving bodies of water (freshwater) when Schistosoma cercariae enter the skin.

Schistosoma hematobium causes **urinary schistosomosis***; S. mansoni* and *S. japonicum*, are the causative agents of **intestinal schistosomosis** and other forms of the disease. Schistosomosis occurs endemically in 74 tropical and subtropical countries of Africa, South America, and Asia

Schistosoma species and length (mm)	**Main location of adult stages**	**Eggs: characteristics, dimensions, and excretion**	**Intermediate hosts (snails), Animal reservoir hosts**
S. haematobium M: 7–15 F: 9–20	Venis of urinary (bladder, etc.)	Ovoid, with terminal spine, 83–187×60–70µm E: urine, rarely stool	I: Bulinus species R: (Monkeys)
S. mansoni M: 6–10 F: 7–15	Mesenteric veins Venis of intestine	Ovoid, with lateral spine, 112–175 ×45–70µm E: stool	I: Biomphalaria species. R: (monkeys, dog, rodents)
S. japonicum M: 7–20 F: 10–26	Mesenteric veins Venis of intestine	Elliptical, spine tiny lacking 70–100 ×50–65µm E: stool	I: Oncomelania species. R: Cattle, buffalo, pig, dog, rodents,



Schistosoma mansoni Pair 

1-The adult parasites live in the **lumen of veins**. Sexually mature Schistosoma females lay about 100–3500 eggs a day, depending on the species, each containing an immature miracidium (= ciliate larva)

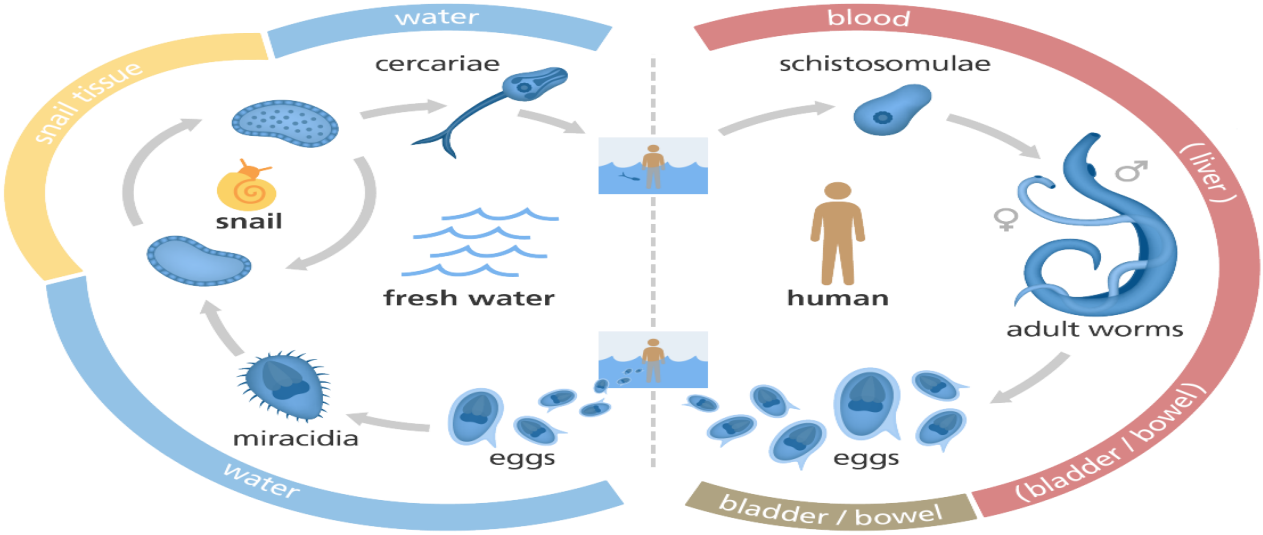
2- The eggs are deposited into freshwater, the miracidia hatch from the eggshell and begin their search for a suitable intermediate host.

3- miracidia enter suitable freshwater snails serve as intermediate hosts in which the reproduce asexually, producing sporocysts, and finally numerous cercariae,

4- The cercariae swim freely about or cling to the surface of the water. Upon contact with a human host, enzyme secretion and dynamic movements enable them to enter the skin within a few minutes. During the infection process, the cercaria loses its tail, sheds the surface and transforms into the schistosomulum.

*****Migration of Schistosomes in the Human Body**

Infection → schistosomula penetrate subcutaneous tissues → find venous capillaries or lymph vessels → migrate through the venous circulatory system into the right ventricle of the heart and the lungs → travel hematogenously → hepatic vein then development into adult worms and migration into mesenteric veins or urinary vein depend on species. [Life cycle of ***Schistosoma mansoni***]



Clinical features

1-The 1st sign is itching and rash (cercarial dermatitis) at penteraion site.

2-Acute intestinal schistosomiasis *(S. mansoni, S. japonicum*) is characterized by fever, headache, hepatosplenomegaly and eosinophilia.

3-Acute urinary schistosomiasis *(S. haematobium*) is accompanied by fibrosis of bladder.

4-Manifestations of chronic schistosomiasis incloud formation of bladder and urinary stones, bleeding oesophagal varices, hepato and splenomegaly, diarrhea [hematuria may progress to cancer].

Diagnosis

1-If no eggs appear, demonstration in rectal biopsy (for all species), or in biopsy of bladder.2-X-ray of urinary tract or intestine.

3-Serogical test.

Treatment

Praziquantel is drug of choice for infections by all schistosoma.

Phylum: Nemathelminthes

Class: Nematoda

General feature

1-Nematodes (or round worms) are elongated, cylindrical, smooth and non-segmented helminthes. Their cylindrical non-segmented bodies allow them to be easily distinguishable from other helminthes. Vary greatly in size.

2- They have complete digestive tract with oral, gut and anal openings. The body is tapered to a relative point at both ends.

3-They are also found to have separate sexes, with the male (being smaller than the female) and have a curved or coiled posterior end with copulatory spicules; some species show a copulatory bursa.

4-The adult anterior - may have hooks, teeth, or cutting plates in the buccal cavity. These are used for attachment.

5-They can be found in fresh water, in the sea and the soil, successfully invading both animals and plants.

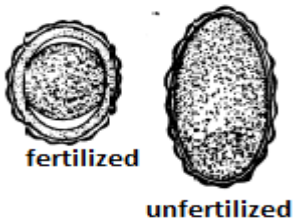
6-Nematodes invade the body fluids such as the blood or lymph channels [e.g.***Trichinella***] and also the intestine [***Ascaris***] result in an increase in **IgE and eosinophilia.**

7-Nematodes are classified into two main categories according to their primary location: [Intestinal nematodes and Tissue nematodes].

Intestinal Nematodes	Tissue Nematodes
Most adult worms live in the intestinal tract.	Live either lymph vessel, skin or subcutaneous tissue, connective tissue.
Enterobius vermicularis *Ascaris lumbricoides* Hookworm [*Ancylostoma duodenale* and *Necator americanus*] *Trichuris trichiura*	Lymphatic filarial *Wuchereria bancrofti*. *Brugia malayi* Cutaneous filariae *Loa loa, Onchocerca volvulus*
The Diagnostic Stages of Helminths Found in Humans*: Eggs.*	The Diagnostic Stages by demonstrating Microfilariae in blood or tissue.

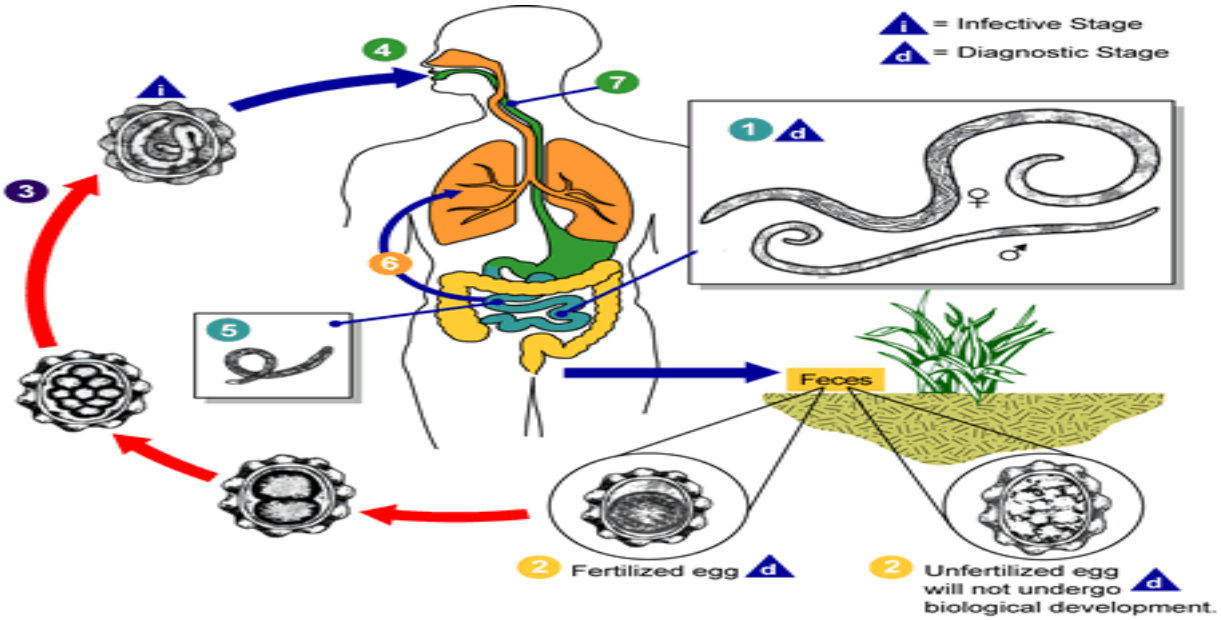
Intestinal Nematode

Ascaris lumbricoides is one of most common human parasites, it is the largest of the intestinal nematodes, which adult worm parasitize in the intestinal tract of human, and cause **Ascariasis.**

Common name	Round worm
Length of adult worm	females length of 40 cm while male worms may reach 20～ 30 cm.
Shape	In both sexes, the mouth is surrounded by one dorsal and two ventrolateral lips. The posterior end of the female is straight while that of the male curves ventrally.
Infective stage	The fertilized [embryonated] eggs with a thick and bumpy outer wa stained golden brown with bile
Mode of transmission	Ingestion of egg in food contaminated with human feces
Site of infection	Small intestine, lung
Clinical findings	Heavy worm burdens cause vomiting and abdominal pain Intestinal blockage, The patient may have symptoms of pneumonitis with cough and low grade fever during the migrati larvae through the liver and lungs.
Diagnosis	Identification of eggs in feces.[oval, brown with colorless shell].
Treatment	 Lay 200,000 eggs **Treatment: Mebendazole**
Epidemiology	worldwide, but it is most prevalent in the areas with warmer climates, moister and poor sanitation

Life Cycle

The life cycle of *Ascris* consist of two parts, one is eggs *development in the soil, another adult worms inhabit humans body. When fertilized eggs are deposited, the zygote is uncleved, and it remains in this state until the egg reach soil. Eggs deposited in soil are very sensitive to environmental temperature at this stage of development. The zygote within the eggshell develops at a soil temperature of about 21～30℃. After 2-4weeks in moist soil at optimal temperatures and oxygen levels, the embryo molts at least once in the shell and develops to an infective larva. Eggs containing infective larvae may remain viable in the soil for two years or longer.[see figure below]



Hookworm Species

Ancylostoma duodenale is an Old World hookworm.*[have two pairs of curved teeth on the ventral wall.

Necator americanus is a New World hookworm.*[have semilunar cutting plates on the dorsal wall

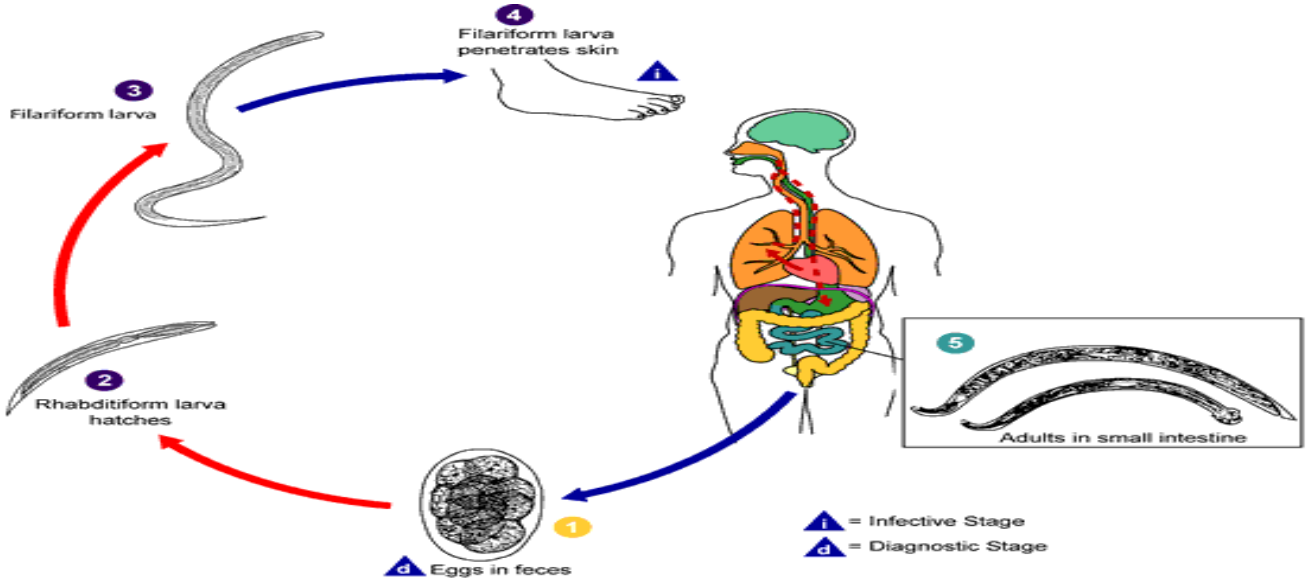
Length of adult worm	Grayish white and slightly curved worm, females 10–13µm And[males 8–11µm has an umbrella-shaped bursa with riblike rays at the posterior end].Anterior end is depicted showing cutting teeth.
Infective stage	Filariform larva
Mode of transmission	Filariform larva in moist soil penetrate skin
Site of infection	Small intestine, heart, lung
Eggs	The ova are oval and transparent with a smooth thin shell With the 4-8 cell inside. Lay 20,000 eggs
Clinical finding	Skin inflammation [ground itching], intestinal necrosis and blood Loss cardiac problem, tissue damage, pneumonitis, brain damage or Respiratory failure. Chronic infections may lead to iron deficiency and anemia
Diagnostic stage	Colorless oval shape egg, Concentration of stool by formalin-ether or simple salt floatation stool is essential to detect light hookworm infection.
Treatment	Mebendazole

Life Cycle

1-Eggs are passed in the stool, and under favorable conditions (moisture, warmth, shade), larvae hatch in 1 to 2 days.

2-The non-infective first stage (rhabditiform) larvae develop into free living adults in the soil within 2–5 days and produce infective third stage or filariform larvae which can penetrate exposed skin.

3-On contact with the human host, the larvae penetrate the skin and are carried through the veins to the heart then to the lungs. They penetrate into the pulmonary alveoli, then to the pharynx, and are swallowed. The larvae reach the small intestine, where they reside and mature into adults. Adult worms live in the lumen of the small intestine. [See figure below]



Trichuris trichiura

Trichuris trichiura, more commonly known as the Whip Worm, due to the whip-like form of the body. These nematodes are most commonly seen in tropical climates and in areas where sanitation is poor. It causes Trichuriasis. [it is an intestinal infection caused by invasion of the mucosa of the colon by the adult worm].

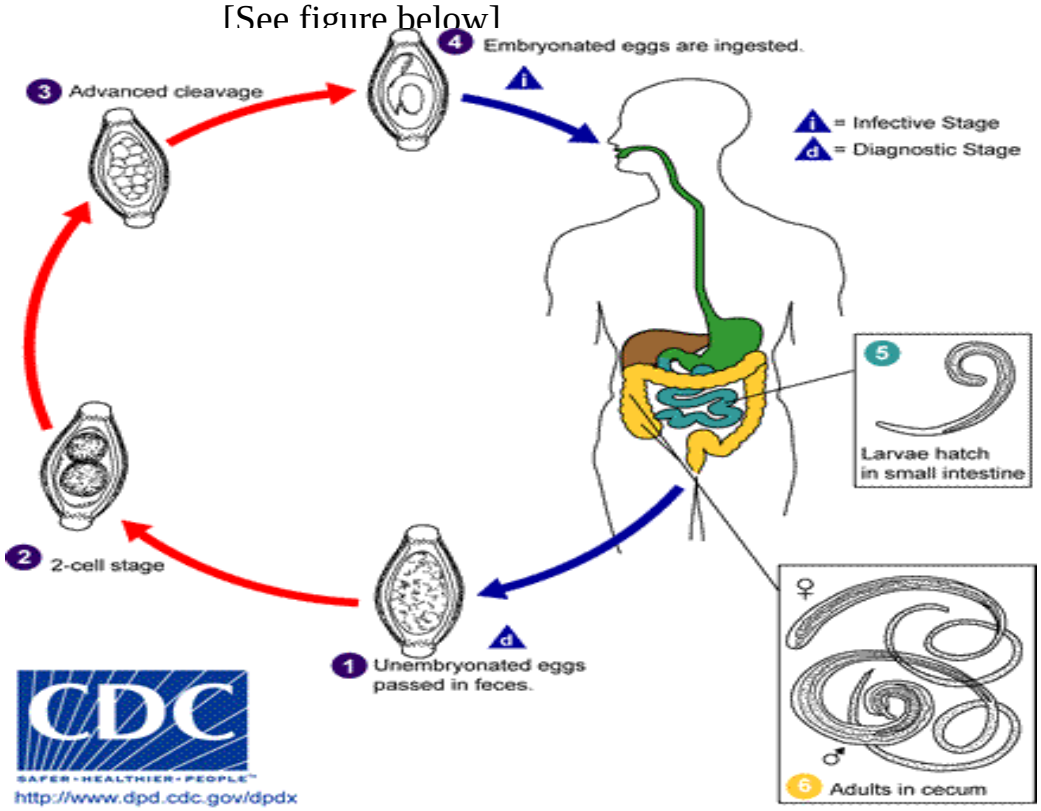
Common name	Whipworm
Morphology	Adult worms are whip-shaped, the anterior being long, thin and hair- like and the posterior h being short, thick and stout. Males are slightly smaller than females, the latter measuring 35～50 mm in length
Disease	
Infective stage 	The egg is typically barrel(برميل)-Shaped with two polar plugs. Its yellowish brown and double shelled
Mode of transmission	Contaminated food with eggs
Site of infection	Colon
Clinical findings	Damage of the intestinal mucosa. Abdominal cramps, dysentery and **prolapsed rectum**
Diagnosis	Egg in feces
Treatment	Mebendazole or albendazole

Life cycle

1-The unembryonated eggs are passed with the stool . In the soil, the eggs develop into a 2-cell stage, then they embryonate .2- eggs become infective in 15 to 30 days. After ingestion (soil-contaminated hands or food), the eggs hatch in the small intestine, and release larvae.

[See figure below]

3-The larva mature and become adult. in the colon then live in the cecum and ascending colon.



Enterobius vermicularis

Commonly known as pinworm, is parasitic only to humans. It is familiar to parents of young children worldwide. The infection of *E.vermicularis* may cause Enterobiasis.

Common name	Pinworm, oxyuris, seatworm
Morphology	Small, female pinworms, measuring 8-13 mm, Males are 2-5mm long and posses a curved tail.
Eggs	The eggs are ovoid but asymmetrically flattened on one side, a colorless, thick shell covers the larva. 
Disease	Enterobiasis
Infective stage	The embryonated eggs
Mode of transmission	eggs are picked up on the hands from bed-clothes or beneath fingernails contaminated when the host scatches the perianal zone.
Site of infection	Large intestine
Clinical findings	itching and irritation caused by the migration of gravid females around the perianal, weight loss, hyperactivity and grinding of teeth. Gravid females may also migrate up the female reproduc become trapped in the tissues, and cause vaginitis and endometritis.
Diagnosis	Female worms emerge at night and are frequently visible observed c well. For eggs observation, a strip of cellophane tape to the perianal skin, remove the tape, and place it on a clean microscop examination.
Treatment	Pyrantel pamoate to all members of the household in a single dose and repeated once after 2 weeks.

Life cycle

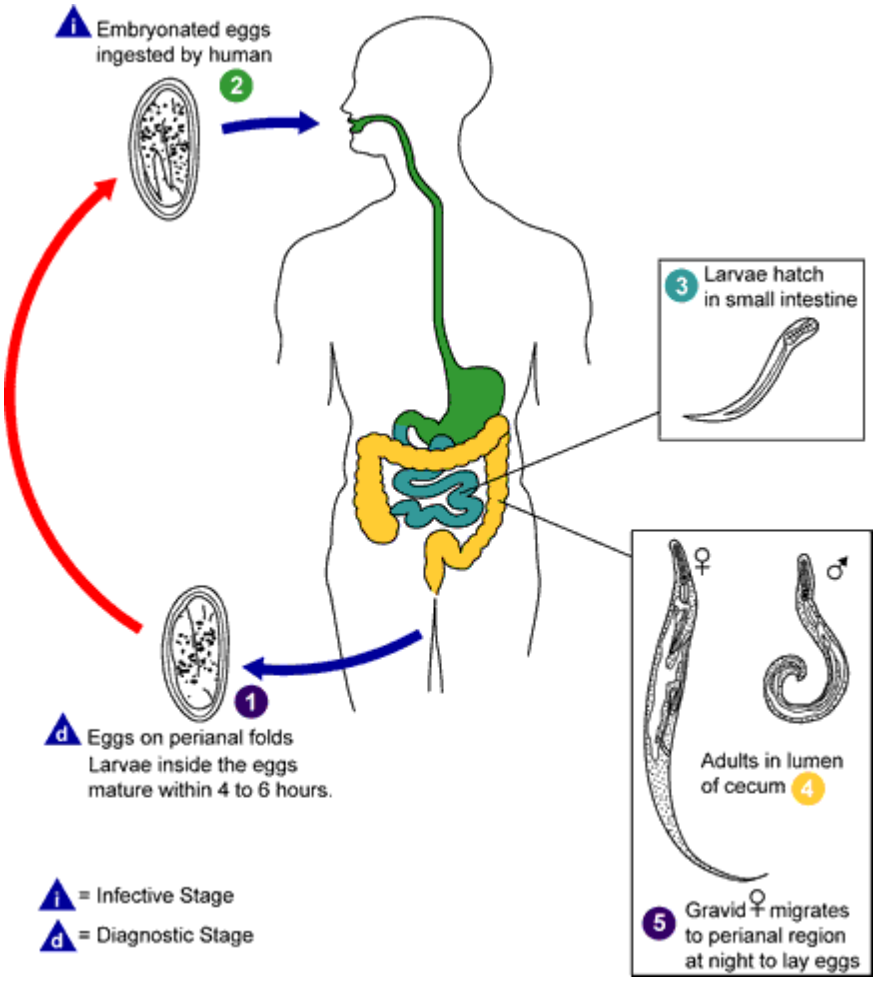
1-Infection and reinfection occur when eggs containing the infective larvae are ingested by the host.

2-Ingested eggs usually hatch shortly after reaching the duodenum. Then larvae develop as they migrate, reaching sexually maturity by the time they arrive at the colon.

3-Adhering to the mucosa, the worms feed on bacterial and epithelial cells. Males die following copulation, while females, with up to 15,000 eggs in their uteri, migrate to the perianal regions, they deposit their eggs and then also die.

4- Each egg contains an immature larva. The infective, third-stage larva completes development within the egg several hours after leaving the body of the female worm.

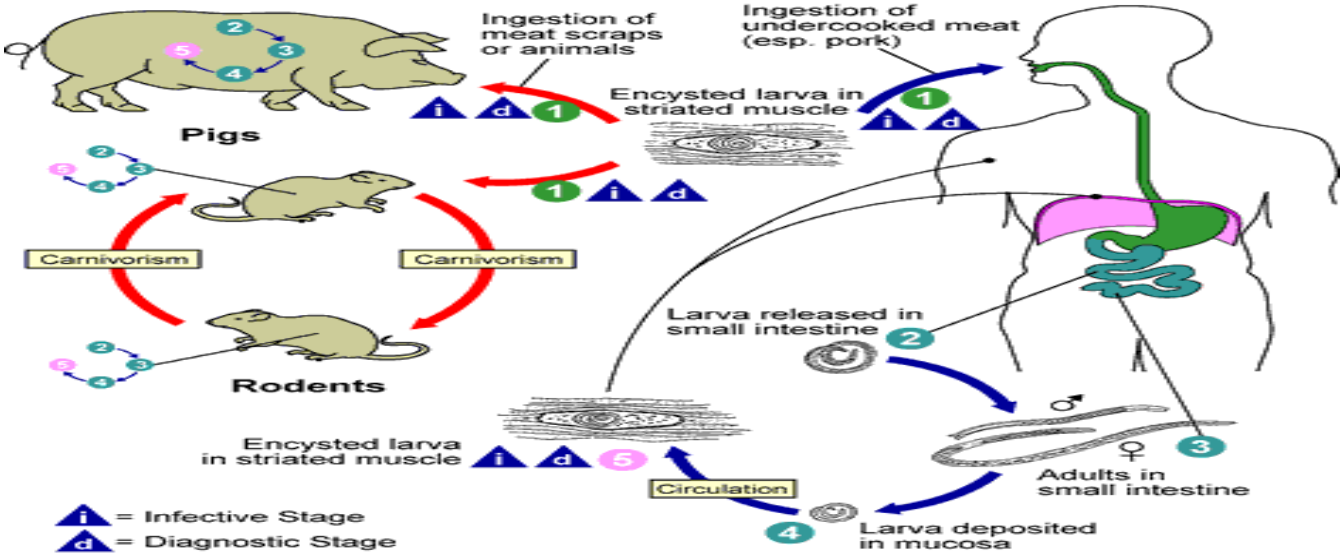
[See figure below].



Trichinella spiralis

The adult worms are very small and slender with slightly tapered anterior ends, white and just visible to the naked eye. The male measures 1.4～1.6 mm in length. The female size is 3～4mm. The infective stage is encysted larva and the transmission rote via ingestion of larvae in undercooked pork meat. The site of infection is striated muscles. Adult worms live in small intestine, and larvae live in skeletal muscle.

Life cycle [See figure below].



Cestoda

Kingdom: Animalia
Phylum:Platyhelminths
Class: Cestoda
Order: Cyclophyllidea
Family: Taeniidae [genus :*Taenia.* Species *(1) sagenata. (2) solium.*]
[genus :*Echinococcus granulosis*.]

There are many types of tapeworms. The most popular tapeworms associated with infection are pork tapeworms, beef tapeworms, fish tapeworms, and dwarf tapeworms.

General Feature

1-All tapeworm contain of long, multi-segmented body.
2-Cuticle of the body is provided with pores through which the worm takes it nutrient.
3- They are hermaphroditic (male and female reproductive organs are present in each mature segment).

Morphology

The cestodes are long, segmented and tape-like worms. They differ trematodes **in many ways.**

1-Adult worm is flat, long, white or milk white in color. It consists of **scole**x, **neck** and **strobilus** (strobilus is a specific structure of tapeworm ((الجزء الطويل بعد العنق, consisting of a linear series of sets of reproductive organs of both sexes; which one is called **immature proglottid**, then mature **proglottid**. As it becomes crowded with eggs, this is **gravid proglottid.**

2-Most tapeworms bear a "head", or scolex, may be provided with sucker, groove, hook, spines, glands, or combinations of these. The neck may be long or short. At contains germinal cells that apparently are responsible for giving rise to new proglottids.

Life Cycle

Cestodes complete their life cycle in two or their different hosts (excep *Hymenolopis nana* complete the life cycle in a single host only). Taenia species are the most common cestode parasites of humans. More than 60 million people are infected with *T. saginata* (beef tapeworm) worldwide and about four million are infected with *T. solium* (pork tapeworm).

1- Adult worms live in the intestine of vertebrates. Eggs or gravid proglottids are passed with feces [the eggs can survive for days to months in the environment].

2- Cattle (*T. saginata*) and pigs (*T. solium*) become infected by ingesting plants contaminated with eggs or gravid proglottids.
3- In the animal's intestine, the **oncospheres** [embryogenesis within the egg to result in a larva] hatch, invade the intestinal wall, and migrate to the striated muscles, where they develop into **cysticerci.**
4- A cysticercus can survive for several years in the animal. Humans become infected by ingesting raw or undercooked infected meat. In the human intestine, the cysticercus develops over 2 months into an adult tapeworm, which can survive for years. The adult tapeworms attach to the small intestine by their scolex and reside in the small intestine.

****** Autoinfection may happen in *T.solum* [humans is the definitive host as well as intermediate host when egg ingestion by human, the egg become oncospheres].**

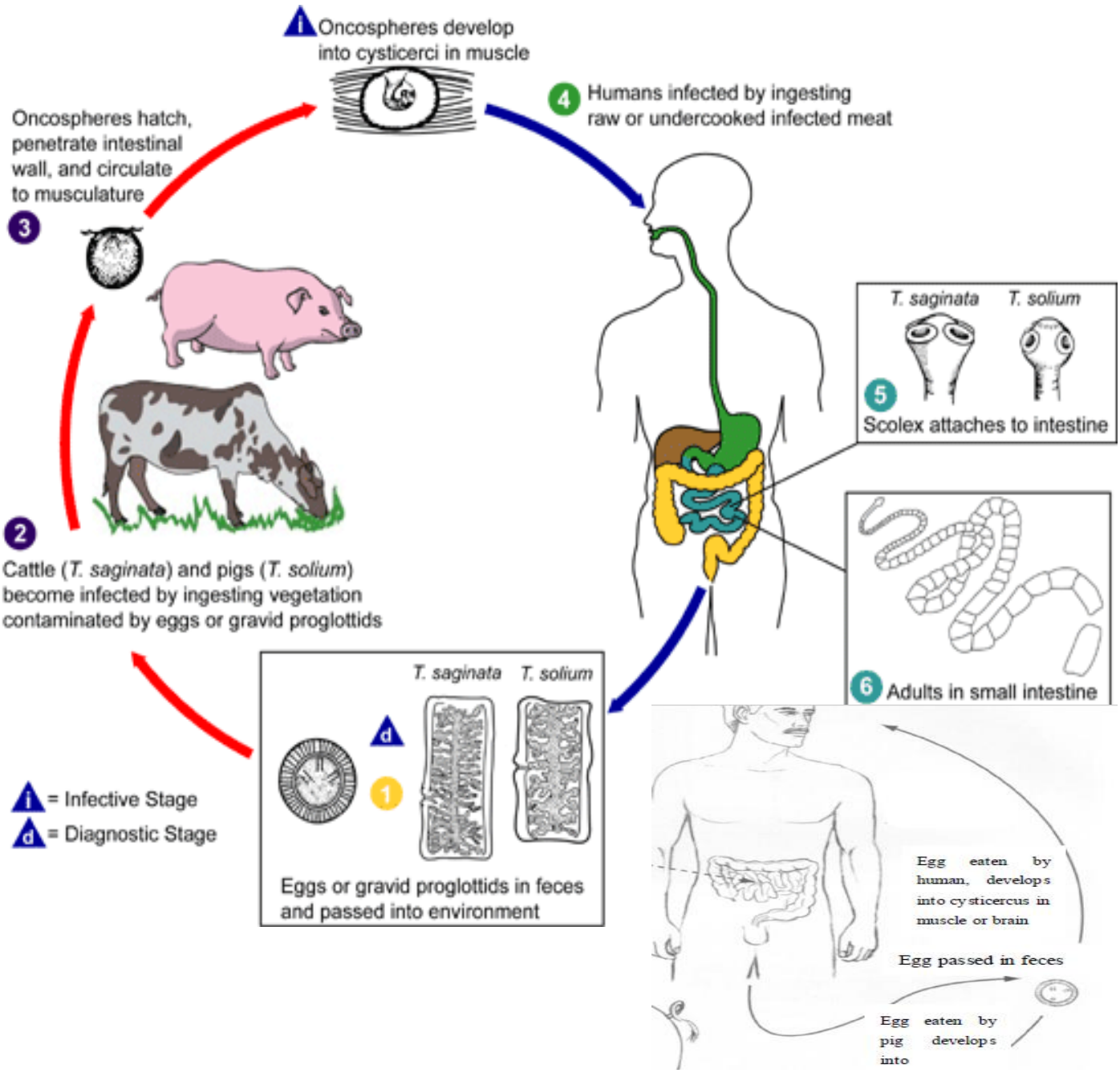
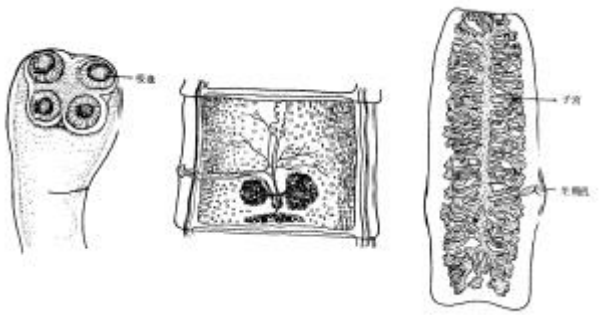
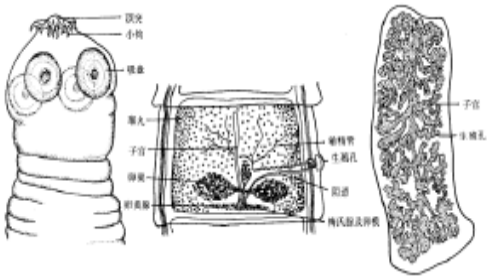


Table. Some characteristics differentiating *T. saginata* from *T. solium*

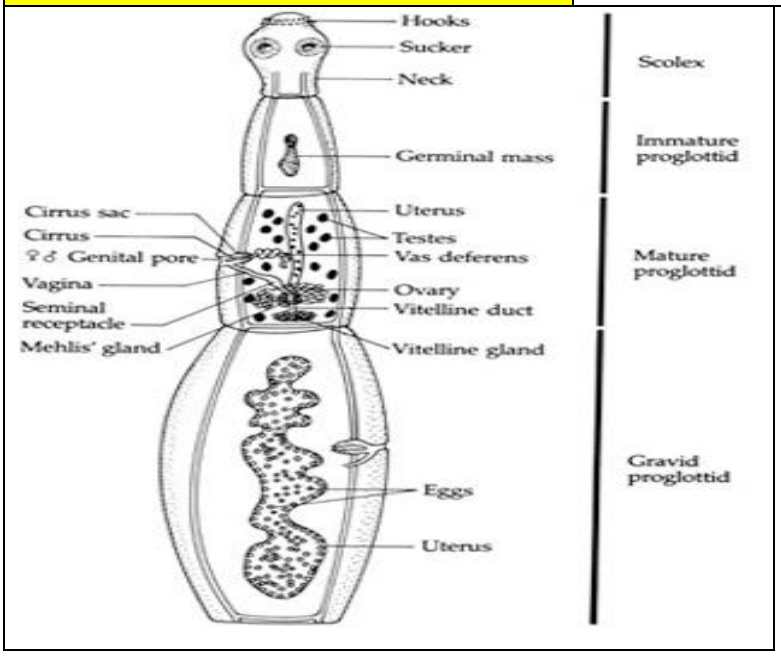
Characteristic	*Taenia saginata*	*Taenia solium*
Common name	Beef tapeworm	Pork tapeworm
Geographical distribution	World wide	World wide
Disease	Taeniasis	Taeniasis, ,Cysticercosis
Intermediate Host	Cattle, reindeer	Pig
Mode of infection	Ingestion of undercooked	Ingestion of undercooked pork or by autoinfection
Site of Development	small intestine	Muscle, small intestine,brain
Infective stage	Man	Man
Scolex: adult worm	4 sucking dick no hooks	4 sucking dick with hooks
Scolex: cysticercus	No rostellum	Rostellum & hooks
Proglottids: uterine branches	23 (14 – 32), ovary: Two lob	8 (7 –11), ovary: Three lob
		

Sign and symptoms		Laboratory diagnosis	Treatment
<u>Taeniasis</u> Mild abdominal symptoms, Vomiting, Anemia, loss of appetite	<u>Cysticercosis</u> It is dangerous if in brain causing epileptic convulsion	<u>Microscopic identification</u> 1-Eggs 2-Scolex and proglottid in feces 3-Cysticercosis by x-rays 4-Serological diagnosis: **ELISA**	Taeniasis: Paraziquntal

Echinococcus granuolosus

Echinococcus granuolosus is causative agent of cystic hydatid diseases. The **dogs** and other canids (e.g. foxes, wolves, etc.) as **final hosts**. *Echinococcosis* is a zoonosis [a disease of animals that affects humans].

Commen Name	Hydatid Tapeworm
Length of adult worm	a tiny tapeworm just a few millimeters long [3-8mm]
Geographic distribution	Worldwide particularly in area where dog Ingest organ from infected animals
Definitive hosts	Dog, foxes, wolves etc.
Intermediate hosts for larval tapeworms	Man, Deer, domestic cattle, domestic sheep,
Mode of transmission	By ingestion or by hand contaminated with dog feces
Site of infection	Liver, lung, bone [extraintestinal]. Direct contact with dogs is the most common way people get *Echinococcus granulosus*
Number of proglottids	3 immature, mature and gravid
Scolex	4 suckers + armed with 28-48 hooklets



Life cycle

The adult *E.granulosus* lives in the intestine of dogs and other canine hosts. Its intermediate hosts include sheep, cattle, and humans. Ovoid eggs containing single oncosphere are shed with the feces of infected definitive host. When the eggs are ingested by a suitable intermediate host such as man many development stage occur.

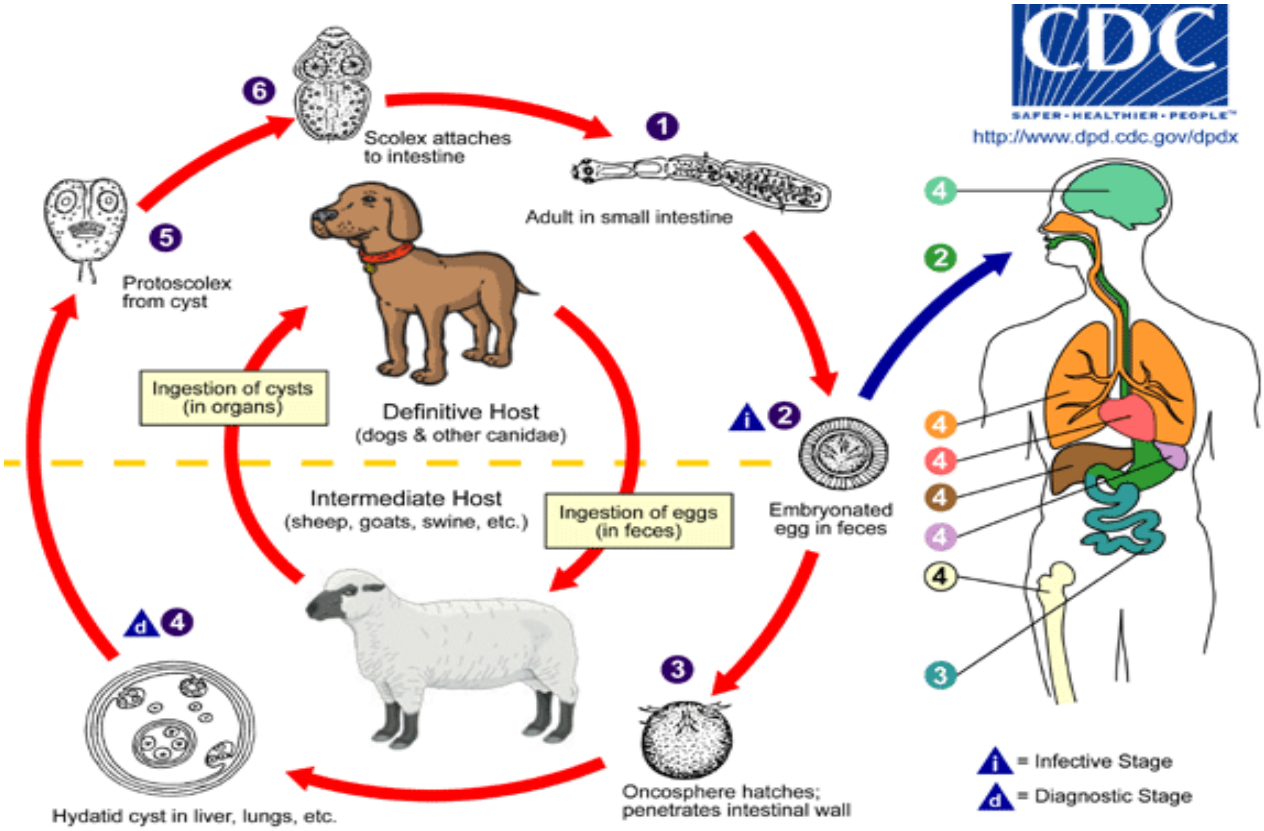
1- Echinococcus eggs, when swallowed by man, produce embryos (oncosphere) that penetrate the small intestine,

2-Then enter the circulation and form cysts in liver, lung, bones, and sometimes, brain.

3-The cyst is round and measures 1 to 7 cm in diameter, although it may grow to be 30 cm. The cyst consists of an outer a nuclear hyaline and an inner nucleated germinal layer which can bud many protoscoles also containing clear yellow fluid.

4-Daughter cysts attach to the germinal layer, although some cysts, known as brood cysts, may have only larvae (hydatid sand). Man is a dead end host.

The life cycle

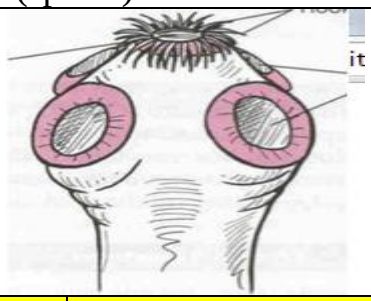


Laboratory diagnosis	Treatment
Mainly by ultrasonography Serological precipitation test **Casoni test:** is intradermal reaction egg thick walled with radial striation (contain hexacanth embryo).	Albendazole for Echinococcosis Surgery for hydatid cyst

Family: Hymenolepididae

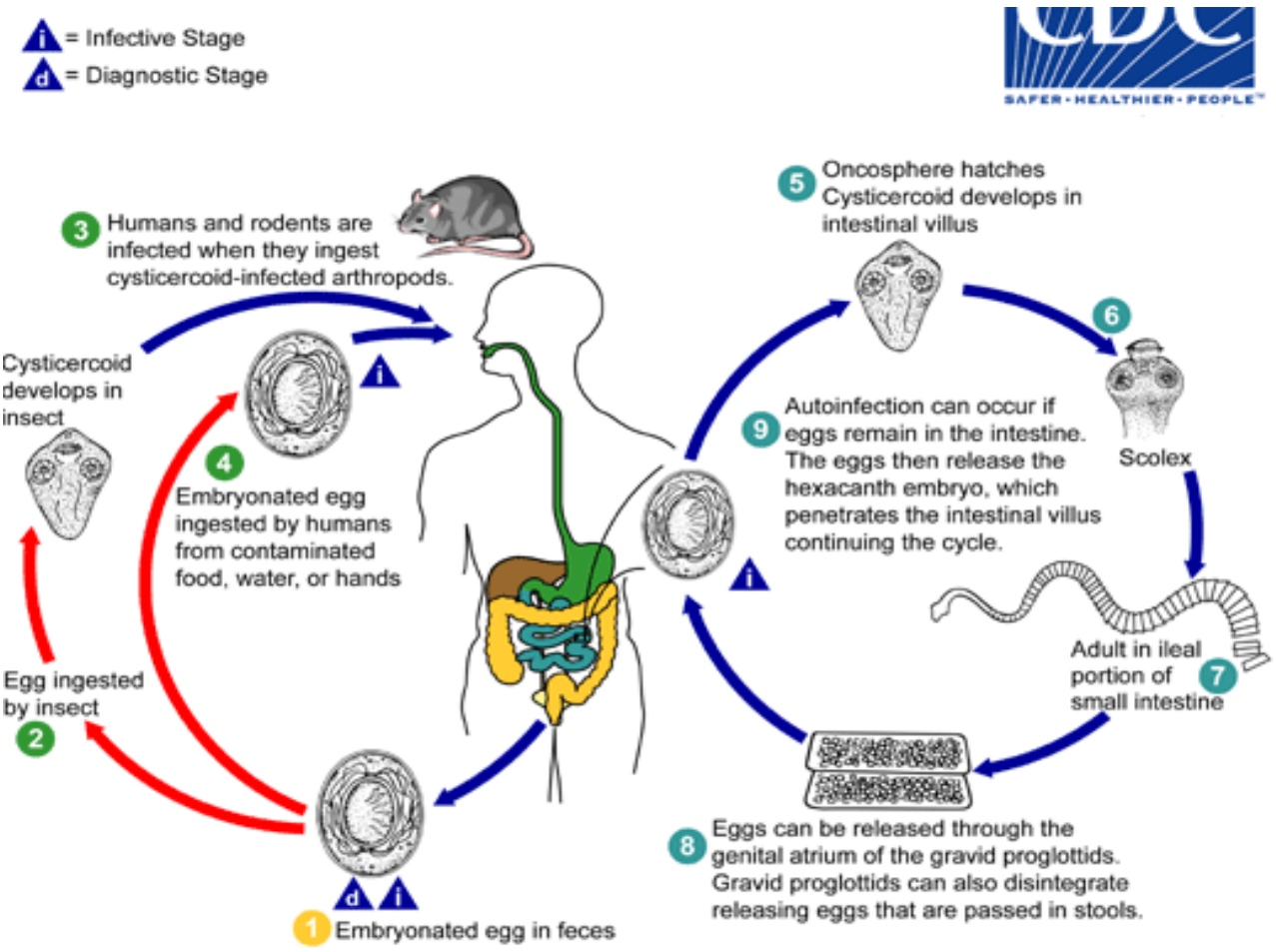
Hymenolepis nana

Hymenolepis nana, the dwarf tapeworm, is the smallest tapeworm to infect humans. The infection is more frequently seen in children although adults are also infected, causing hymenolepiasis.

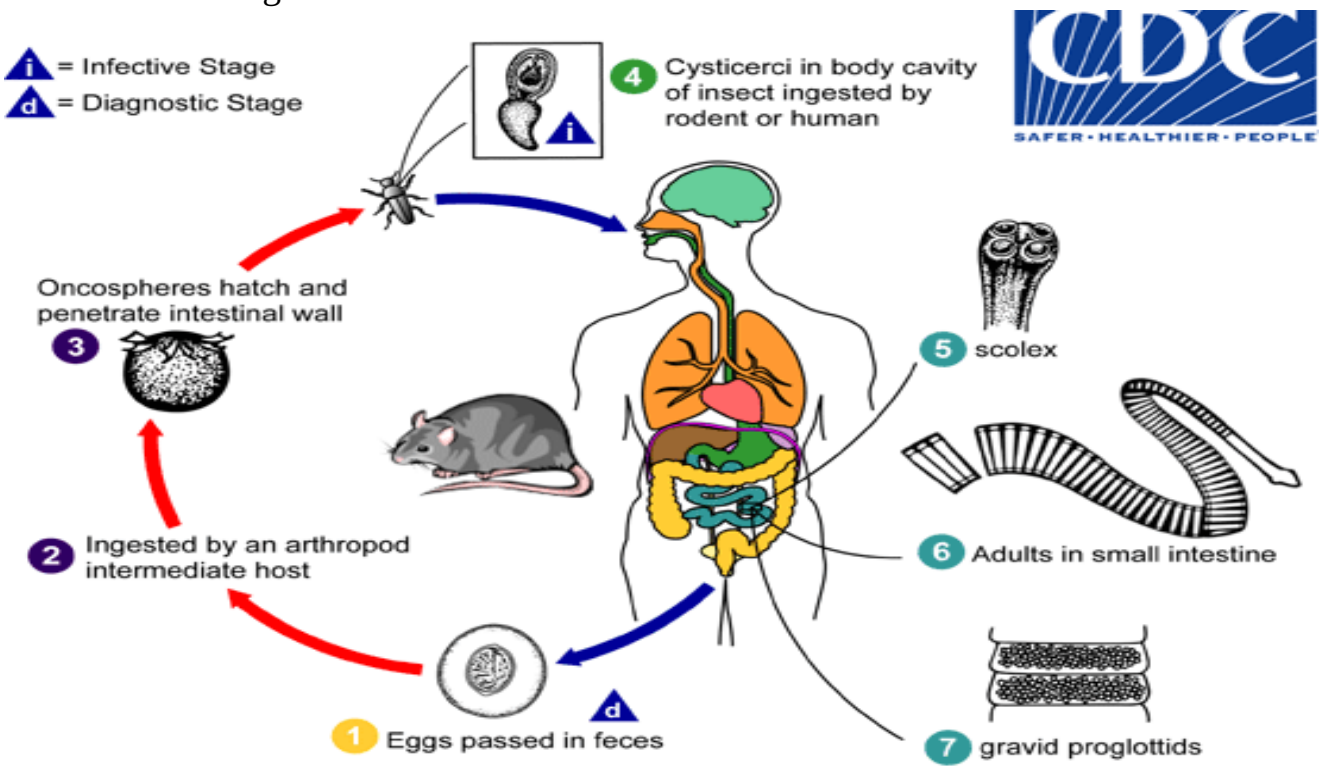
	H. nana	*H. diminuta*
Common name	Dwarf tapeworm	Rat tapeworm
Length adult worm	15-40 mm	20-60 cm
Geographical distributi	Worldwide (the most common human cestode)	Worldwide
Disease	Hymenolepiasis or dwarf tapeworm	Hymenolepiasis
Mode of infection	By egg ingestion or by hands contaminated with man s Feces (autoinfection)	Swallow of insects Infected with eggs
Site of infection	Small intestine	Small intestine
Infective stage	Cysticercoid (tailed larva) The oncosphere, or larval stage, has six hooks	Cysticercoid (tailed larva)
Definitive host	Man , mice	Rodents , man
Intermediate host	None (in autoinfection)	Fleas , beetles , cocroaches
scolex	A rostellum with 4 suckers and a crown of hooks (spines)	4 suckers and a rostellum <u>without hooks</u>
		 Scolex

Sign and symptoms	Laboratory diagnosis	Treatment
Infections due to *H. nana* may cause no symptoms even with heavy worm burdens. However, symptoms are, irritability, anorexia, abdominal pain and diarrhea. Heavy worm burdens may be caused by auto-infection	identification of the characteristic ova formol-ether concentrate of feces *H. nana*: [30-50µm] with Internal cover+6 hook *H. diminuta*: larger [70-80µm]	Praziquntal

Life cycle of *H. nana* (please read figure below)



Life cycle of *H. diminuta*: The life cycle of *H. diminuta* requires intermediate arthropod host e.g. larval fleas and various beetles. Human infection occurs by the accidental ingestion of an infected arthropod, which contains the cysticercoids**.** Please read the figure below.



Phylum: Nemathelminthes

Class: Nematoda

The Blood Nematodes [Tissue (filarial) Nematode].

These nematodes are known as filariae and consist of a group of nematodes which have successfully invaded the blood stream, connective tissue or serous cavities of vertebrates**.**

Lymphatic Nematodes	Cutaneous Nematodes
Wuchereria bancrofti. *Brugia malayi* .	*Loa loa*, *Onchocerca volvulus*

Wuchereria bancrofti

Wuchereria bancrofti is a nematode causing lymphatic filariasis. There are two strains of *W. bancrofti*;

1. The nocturnal periodic strain which is widely distributed in endemic regions (Africa, India and the Far East) with the microfilariae being in their highest concentrations between the hours of 10pm and 2am.

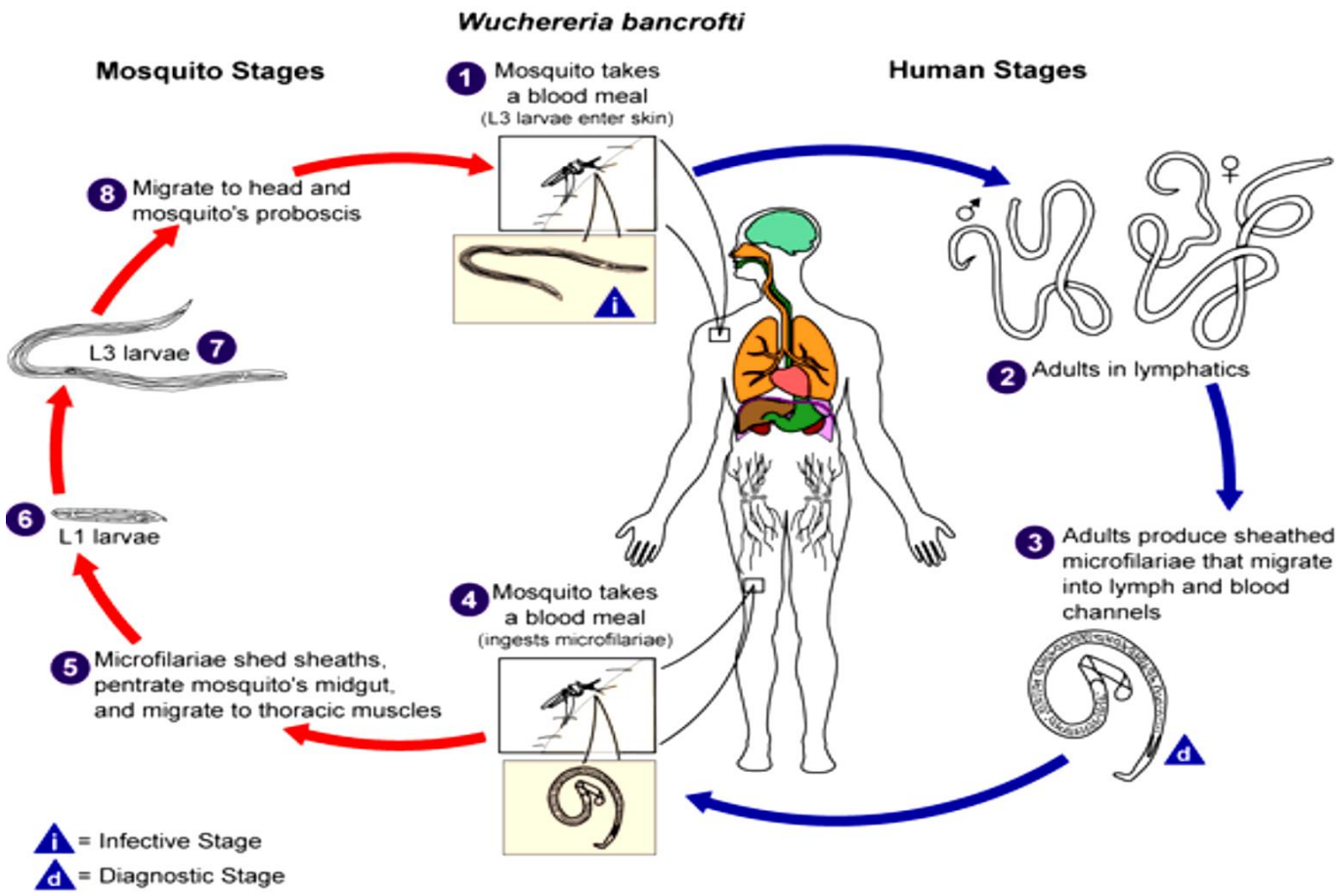
2. The sub-periodic strain which is found in the Pacific region, and has a microfilaremia all the time with the highest numbers being detected between noon (12) and 8pm.

Disease	lymphatic filariasis, Elephantiasis
Geographical distribution	Tropical and subtropical countries (الاستوائية وشبه الاستوائية)
Shape	The adult worms are white and threadlike. The male measures between 2.5–4cm the males have spirally coiled tails whereas the female is larger, measuring between 8-10cm.;. Sexually mature female worms release microfilaria, which are pre-larval stages. These are released into the bloodstream. Some have sheathed' microfilaria.
Infective stage	Motile microfilaria (sheathed)
Mode of transmission	Mosquito *Culex quinquefasciatus*,
Clinical findings	Fever , ***Elephantiasis***: lymphadenitis (inflammation in lymph node) Inflammation lymph vessels in legs, arm and testis, thickening of Tissue fibrosis. Some develop tropical pulmonary eosinophilia, wheeze and cough.
Diagnosis	Identification of microfilaria in blood by staning methods
Treatment	Diethylcarbamazine, Ivermectin, DEC, Mosquito control

Life Cycle

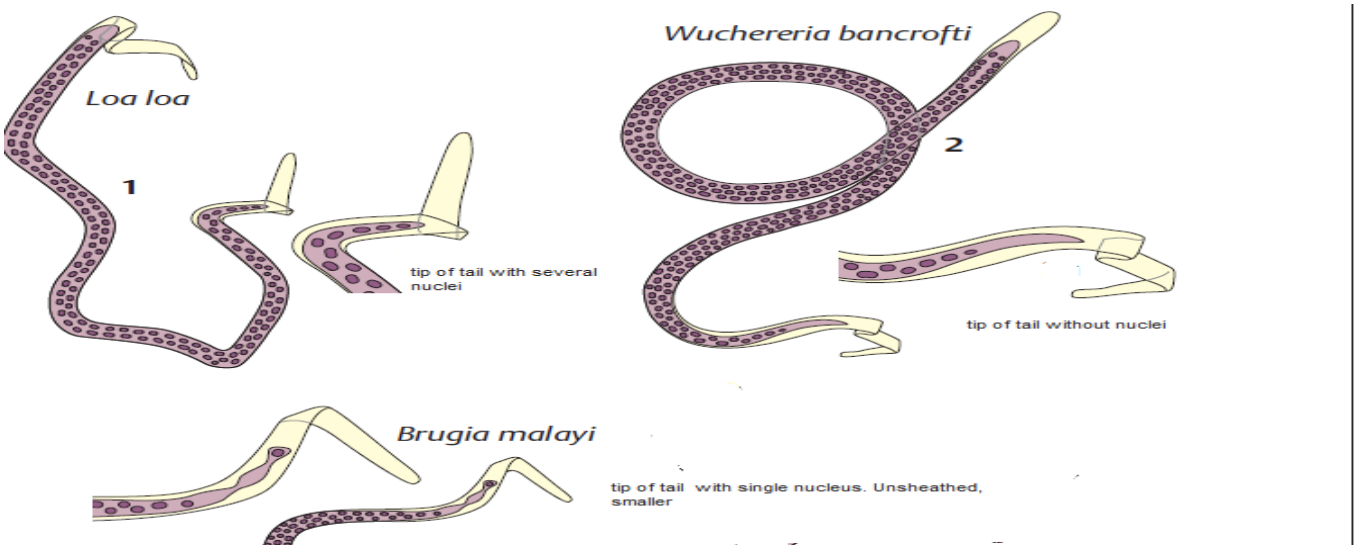
Microfilariae enter the host during a blood meal when the vector, a mosquito, punctures the skin. The infective larvae enter through the wound and migrate to the peripheral lymphatics where they grow to mature male and female worms. They can live there for several years. After mating, the gravid females release sheathed microfilariae into the peripheral blood where they can be detected 8-12 months after the initial infected bite.

The mosquito acquires the infection by ingestion of the microfilaria in the blood meal. The microfilariae lose their sheath on arrival in the stomach of the mosquito due to gastric juices. The larvae migrate to the thoracic muscles and develop into infective larvae over a period of 6-14 days. The larvae then migrate to the mouthparts of the mosquito which infects the host during a blood meal. [See figure below]



Brugia malayi

Brugia malayi is a nematode causing lymphatic filariasis in South East Asia. It transmitted by various *Anopheles* species of mosquitoes and also by *Mansonia* species, a mosquito that usually bites during the night. This species like *W. bancrofti* also parasitizes the lymph nodes and lymphatics; causing Malayan filariasis.



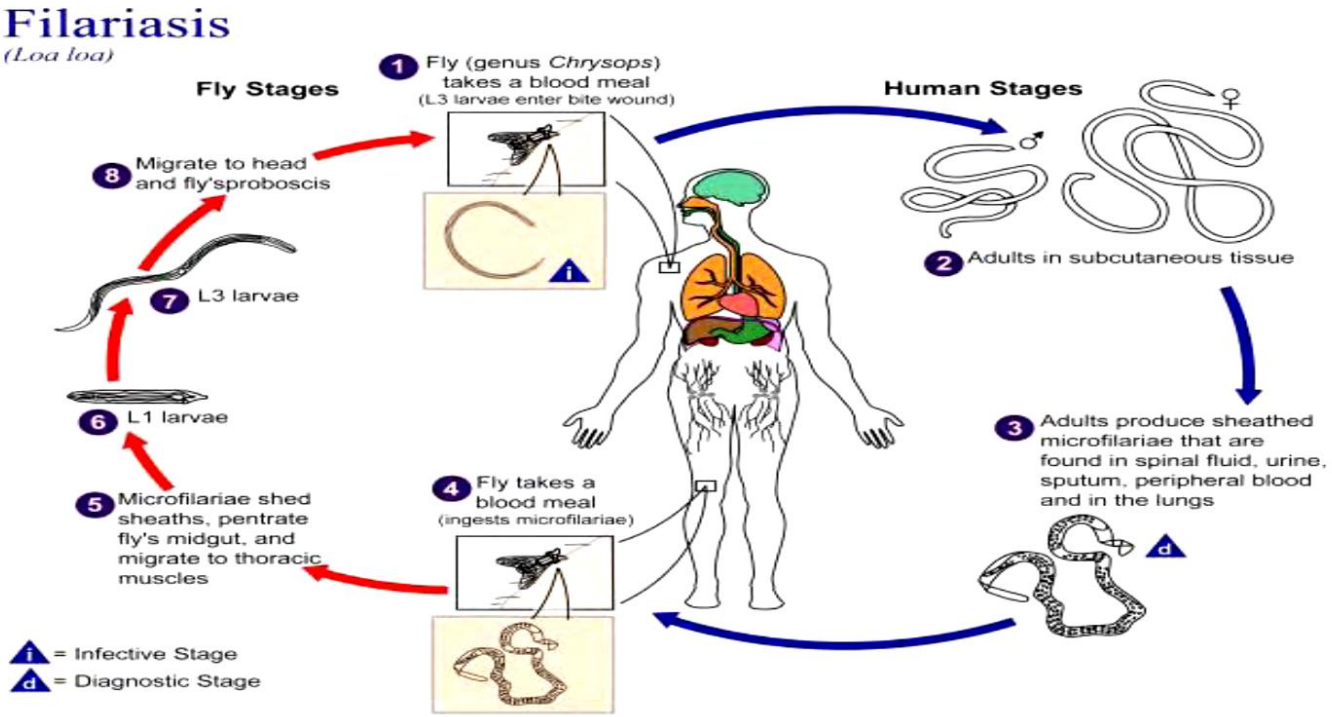
Loa loa

Loa loa, also known as the African eye worm, is a filarial nematode endemic in the rain forests of West and Central Africa. It is transmitted by fly *Chrysops* species, also known as mango flies or horse flies and **humans are the only known reservoir**. Adults migrate in the subcutaneous tissues of man, with them eventually migrating across the eyeball under the conjunctiva.[Loiasis or Calabar swelling disease].

Life Cycle

1- The adult worms live in the subcutaneous and deep connective tissues and the microfilariae are found in the peripheral blood.

2- The microfilariae have been taken up by the Chrysops during a blood meal they develop within the fat body. They develop through to L3 within 10–12 days. The microfilariae, L3 reenter the hosts blood stream when the fly takes another blood meal. They reach adult worms within 4- 6 months living in the subcutaneous and deep connective tissues. [See figure below]



Clinical Disease

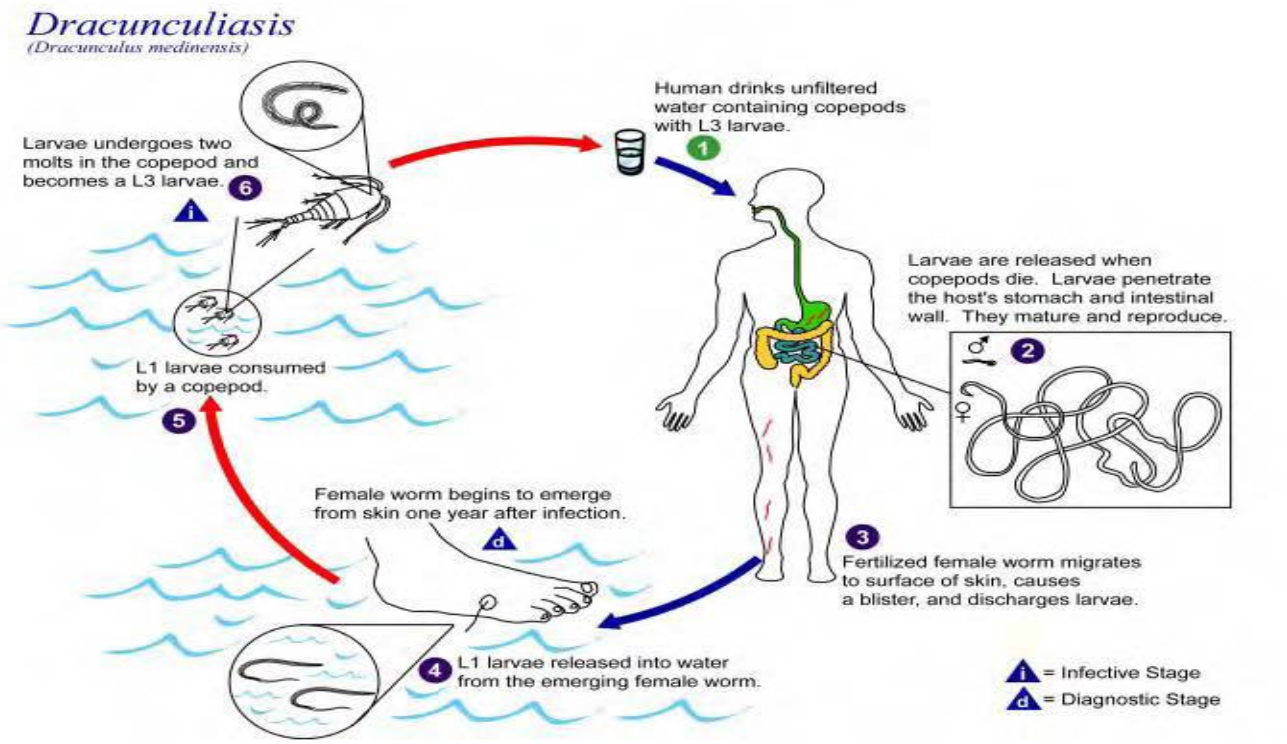
The most common pathology associated with Loa loa infections are Calabar swellings, which are inflammatory swellings resulting in a localized subcutaneous edema. These swellings are due the host's response to the worm or its metabolic products and localized pain.

Dracunculus medinensis

Dracunculus medinensis is a non-filarial parasite as it only has one uterus whereas filaria have two. It is usually associated with places where there is a lack of clean drinking water e.g. step wells in India, Yemen, Africa, Iran, and ponds in Ghana. The life cycle usually involves copepods ((مجدافيات الأرجل) نوع من القشرياتج)intermediate host. They are parasitic in the connective tissue or wall of intestine. The disease is known as Dracunculiasis.
The female over 100cm and the male 2cm.

Life Cycle

Female worms which are gravid with microfilariae migrate to the layers of human's skin, especially foot, arms and shoulders. Here the worms secrete a substance (substance is unknown) which causes a blister to rise over its anterior end where it has punctured the lower layers. The blister eventually forms into an ulcer which on contact with water, the uterus is projected out of the ulcer cavity, and a cloud of milky white secretion, containing a lot of active larvae, is released.



Clinical Disease

Skins ulcer. The patient can also exhibit vomiting, diarrhea, asthmatic attacks. Symptoms usually subside when the lesion erupts. If the worm is removed, healing usually occurs without any problems. If the worm is damaged or broken during removal results in an anaphylactic reaction.

Diagnosis

Demonstration in local lesion or fluid discharged of adult worm, then rolling the worm around a small stick and slowly pulling it out of the skin. With this method you must be careful not to pull apart the worm as it will recoil back into the skin and cause secondary infections. Treatment by [Diethylcarbamazine, niridazole].

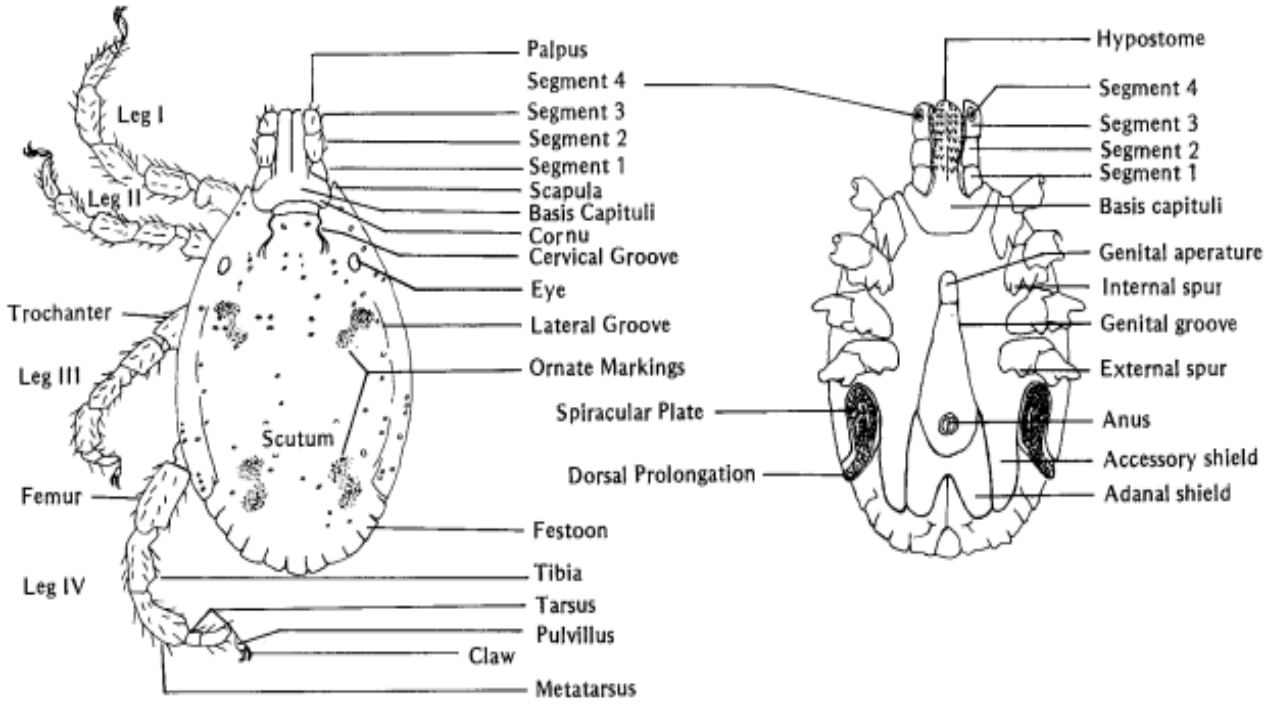
Onchocerca volvulus

It causes onchocercosis, a disease that manifests mainly in the form of skin alterations, lymphadenopathy, and eye damage, which latter is the reason for the special importance of the disease. *Onchocerca volvulus* is endemic in 30 countries in tropical Africa (from the Atlantic coast to the Red Sea) and Yemen. Adult filariae in fibrous nodule, usually 0.5–2cm sometimes up to 6 cm in diameter in the subcutis along the iliac crest, ribs, scalp, etc., more rarely in deeper tissues. Nodulation occurs about one to two years after infection and is either asymptomatic or causes only mild symptoms.

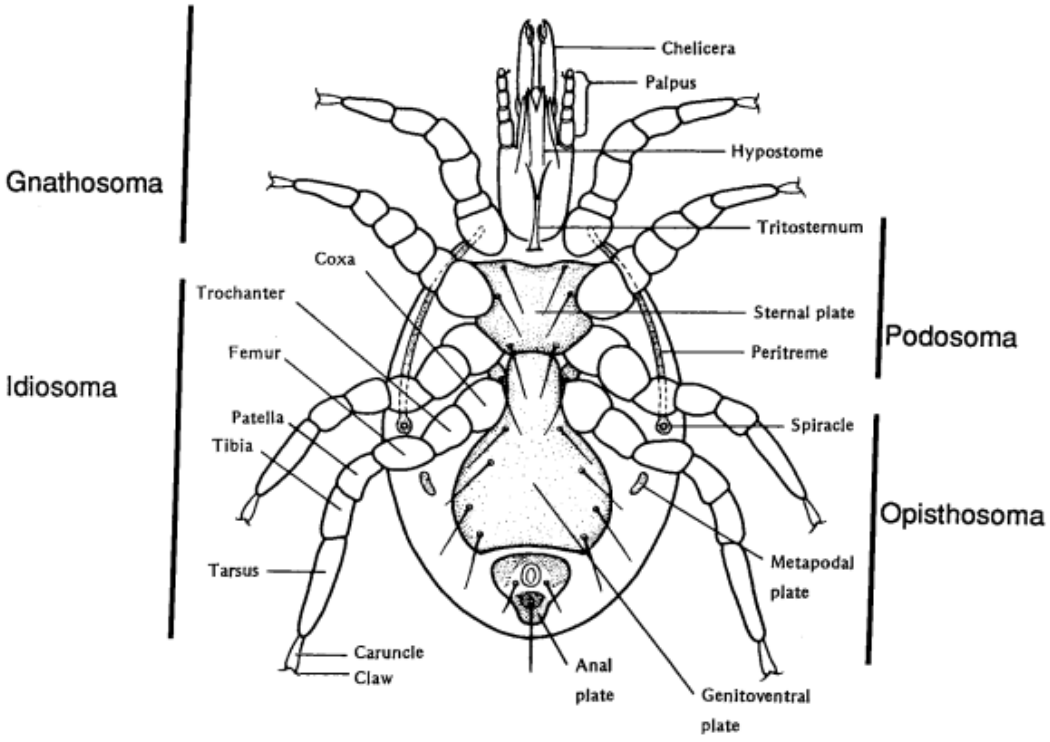
Species and length (cm)	Distribution	Vector	Localization of adults	Microfilariae: characteristics and periodicity	Pathology
Onchocerca volvulus ♂♂: 2.0–4.5 ♀♀: 23–50	Africa, Central and South America	**Black flies:** *Simulium*	Subcutaneous connective tissue	221–358 µm, unsheathed, in skin, not periodic	Skin nodules, dermatitis, eye lesions

Arthropods

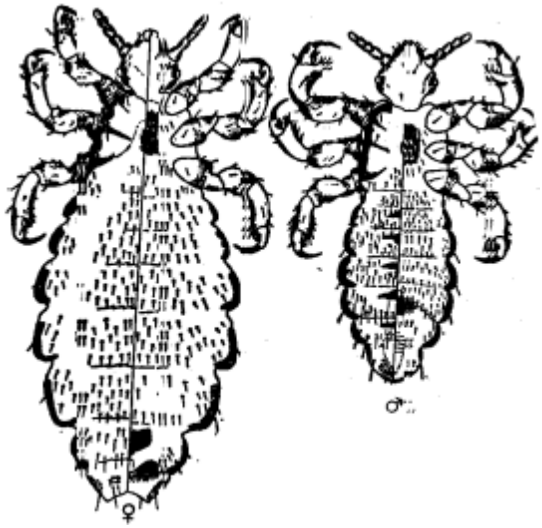
Parasitic arthropods are ectoparasites that have a temporary or permanent association with their hosts. Their considerable medical significance is due to their capability to cause nuisance or skin diseases in humans and to act.



Dorsal and venter view of hard tick



External anatomy of a mite



Male Female Lice

Arthropod	Disease
Hard tick/ Ixodidae	Forest encephalitis
	Xingjing haemorrhagic fever, Lyme disease. Q fever
Soft tick/Argasidae	Tick-borne recurrent fever (Q)
Chigger/Trombiculid mites	Scrub typhus
Itch mite/Sarcoptidae mite	Scabies
Demodicidae mite	folliculitis
Dust mite/Pyroglyphidae	Asthma, Allergic rhinitis, Allergic dermatitis
Mosquito	Malaria , Filariasis(, Japanese B encephalitis, Dengue fever,Yellow fever
Fly	Dysentery, Typhoid fever, Cholera, Poliomyelitis, Amebic dysentery
Sand fly	Kala-azar disease/ visceral leishmaniasis
Flea	Plague, Murine typhus , Hymenolepiasis diminuta
Lice	Epidemic typhus, Lice-borne relapsing fever