بسم الله الرحمن الرحيم

Parasitology - 306 ح

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Chapter 1

Introduction to Parasitology.

Parasitology: is the science dealing with organisms living in or on other organisms.

Parasitism:

Most plants and animals are able to live independently and are largely self-sufficient in obtaining and metabolizing the nutrients they require for their growth and reproduction. A small group of plants and animals however, are not so independent and some of these have evolved a more or less intimate relationship with another organism of a different species so that both partner benefit from the relationship. This relationship in which neither partner is harmed is called commensalisms. If, however, one partner benefits from the relationship and is unable to complete all its development and reproductive processes without the aid of the other partner, the relationship is then one of parasitism.

Parasitic relationship:

In the parasitic relationship, the half of the partnership which benefits from the relationship is the **Parasite**, and that which provides the benefit is the **Host**.

Endoparasite and Exoparasite:

When a parasite lives within its host (malaria), it is referred to as an endoparasite and is said to cause infection. A parasite however which lives on the outer surface or in the superficial tissues of its host (flea) is called exoparasite, and is said to cause infestation.

Life cycles of Parasitism:

All parasites pass through a series of developmental stages before a stage is reached when the organism reproduces and a new cycle of development begins. There may be few or several developmental stages, with at least one stage occurring in a host organism.
Within the developmental cycle (life cycle) they may b several phases or parasite multiplication or only one. According to species the phase of multiplication may be sexual or asexual. In some parasites sexual multiplication is followed almost immediately by asexual multiplication.

Direct and indirect life cycle:

When a parasite requires only one species of host in which to complete its development it is said to have a direct life cycle, e.g. the life cycle of the parasite that cause the amoebiasis in human (Entamoeba histolytica) requires only a human host for its completion.

When two or more hosts are required, the life cycle is referred to as indirect, e.g. the filarial worms that parasitized humans required both human host and insect host in which to complete their development.

Classification of hosts:

Definite host is either:

-The host in which sexual reproduction takes place, for example human is the definite host for Schistosoma haematobium.

Or

- The host in which the mature or the most highly developed form of the parasite occurs. Human is the definite host for trypanosome.

Intermediate host:

This term is used to describe the species of host or hosts, other than the definite host, that are essential to complete the indirect life cycle of the parasite, e.g. tsetse fly is an intermediate host for the trypanosome species that cause African trypanosomiasis.

In the life cycle of the parasitic worms, intermediate hosts harbour the larval forms.

In the indirect parasitic life cycle, the term vector is usually applied only to blood-feeding arthropod intermediate hosts such as mosquitoes, tsetse flies and sandflies.

The term mechanical vector is used, to describe a vector which assist, in the transfer of parasitic form between hosts but is not essential in the life cycle of the parasite, i.e. no parasitic development occur in such a vector. An example of the mechanical vector, is the fly that transfers the amoebic cysts from infected faeces to the food that is eaten by the humans. A non arthropod mechanical vector is called transporter, or paratenic host. In such host the parasite remains viable but does not develop.

Reservoir host (carrier):

A reservoir host is an animal in which a parasite usually resides or one in which a parasite which infects the humans is able to be maintained in the absence of the human host.

A parasitic infection in which the normal host is an animal, but can produce disease in human is called a zoonosis. E.g. leishmaniasis, and African trypanosomiasis.

Transmission and diseases caused by parasites:

Routs of transmission:

1- By ingesting the parasite in the food, water, or from hands that have been contaminated by faeces that contain the infective form of the parasite. Example: Entamoeba histolytica.
2- By ingestion the parasite in the raw or under-cooked meat. Example: Taenia saginata.
3- By ingestion the parasite in the raw or under-cooked fish, crab. Example: Fasciolopsis buski.

4- By contact with the water contaminated with parasite as in Schistosoma species.
5- By parasite entering the blood and tissues through the bite of an insect, as occur in Loa loa.
6- By inoculation of the parasite into the blood by an insect as occur with: plasmodium species.
7- By sexual contact as occur with Trichomonas vaginalis.
8- By infected faeces from an insect being rubbed into the site of the insect bite as occur in Trypanosoma cruzi.

Parasitic disease:

Not all parasitic infections cause disease of clinical significance. Many factors influence whether an infection causes disease including:

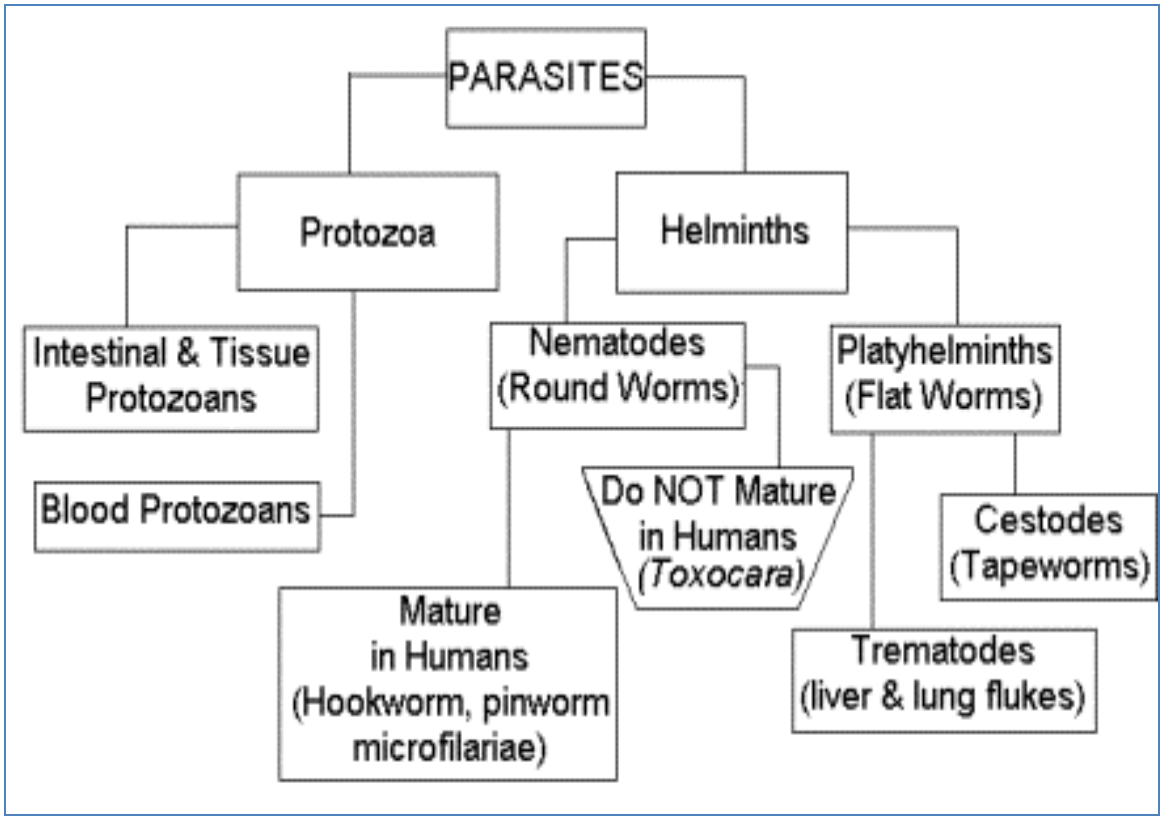
Parasitic factors:

- Number and strain of parasite.
- Strain of the parasite.

Host factors:

- Age and level of natural immunity at the time of infection.
- Immune responses to the infection.
- Presence of co-existing disease or condition which reduces the immune responses, e.g. pregnancy.

General classification:



Genus Entamoeba.

General characteristics:

Intestinal parasites amoeba of man.
Infectious stage to man the cystic stage.
Transmission is by ingestion.
Movement is by pseudopodia called amoeboid movement.

Entamoeba Histolytica.

Geographical Distribution:

Allover the world, tropical and subtropical countries. High numbers found among the countries with high temperature, or communities with bad hygiene.

Habitat :

Trophozoites (adult): found in the large intestine.
Occasionally in liver causes liver abscess.
Occasionally in lungs causes lungs abscess.
Cyst: in intestinal lumen.
No cyst is found in abscess.

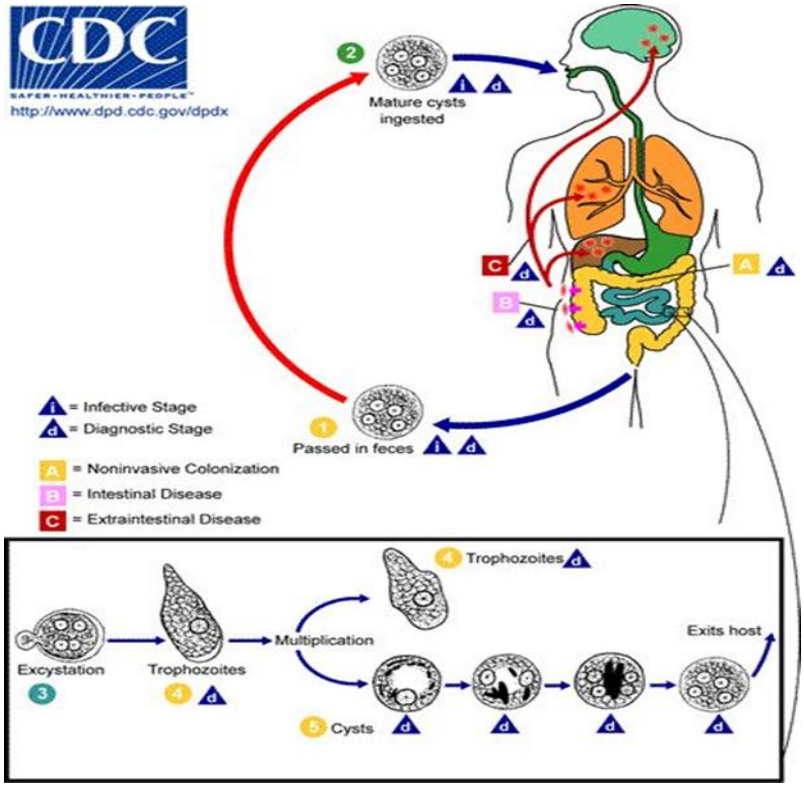
Note : trophozoites and cysts are found in stool liquid fresh from a carrier, especially stool that contains blood and mucus.

Transmission and Life cycle:

Food and drinks, contaminated with feacea containing cysts of E. hystolytica

Life cycle:

1- After ingestion every cyst excysts in the large intestine to produce amoeba which multiply repeatedly.
2- The amoeba form single nucleated cyst which develop into infective cyst which have 4 nuclei. Once cysts are formed, they do not become amoeba again in the same host.
3- The infected cysts are execrated in the faeces. They can survive and remain infective for several weeks in sewage and water.



Pathogenesis:

1. Causes ulceration of the large intestine.
2. Amoebic hepatitis and Lung abscess.
3. Rare cases of brain abscess.

Lab diagnosis:

Specimens: 1- Stool (direct or concentrated method)

2- Serum for serological tests (cellulose acetate precipitin CAP) for liver abscess.

3- Biopsy for liver, lung and intestine.

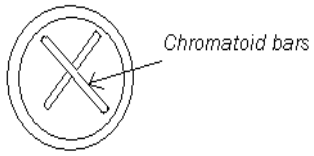
Macroscopic examination:

1- Colour.
2- Smelling (very strong bad smelling).
3- Acidic.
4- Bloody and mucous.

Microscopic examination:

1- Look for cyst.
2- Look for trophozoites

Morphology of the cyst under the microscope:

1- Wet preparation. "saline"	2- Iodine preparation.
	
We can see the chromatoid bodies (small or big, fine or thick). Sometimes we can see the stained nucleus. Glycogen mass can not be seen.	Purpose of Iodine: To see the glycogen mass clearly, and the nucleus (one to 4). We do not see the chromatoid bodies

Important points:

1- E. hystolytica cyst never shows more than 4 nucleus, if more so it will be another species of entamoeba (E. coli with up to 8 nuclues). The cyst measures about 10-15 µm.
2-Amoeba measures 25x 20 µm, and it is moving actively in the fresh worm specimen. It may contain digested RBCs .
.

Commessal Amoebe

EX: Entamoeba Coli.

Entamoeba Coli.

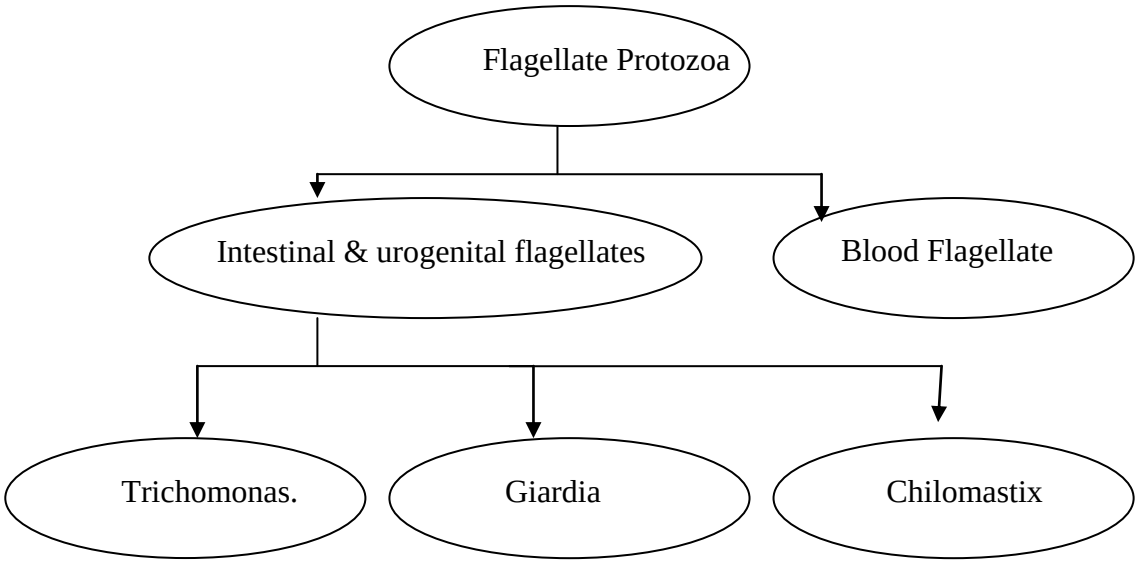
Geographical distribution: all over the world.
Habitat: large intestine of the man.
Morphology: trophozoite measures 15-30 µm, the cytoplasm contains food and bacteria, no red cells.
Cyst : measures 15-30 µm, the cytoplasm contains up to 8 nuclei
In saline: chromatoid body is rare and needle-like.
In Iodine: Glycogen mass faint or diffused.

Life cycle: the same of E. histolytica but not pathogen.

Infection/ pathogenicity:
No pathogenicity.

Flagellate protozoa.

There are called flagellates because they move with flagella.



Some are causative organisms for dysentery.
1) Genus: Trichomonas.
T. hominis (intestinal).
T. vaginalis (genital organs).
T. tenax (human mouth)

General characteristics of trichomonas:

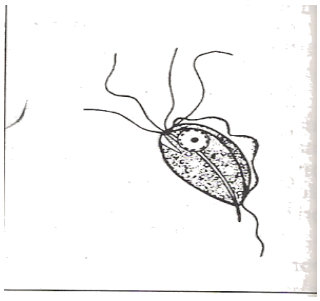
1. Pear shape or ovoid.
2. Have 5 flagella, 4 directed anterior and free posterior enclosing undulating membranes.
3. Have Costa, achromatic basal red.
4. Have exosyle, a structure in the centre.

Transmission:
By ingestion, infective stage trophozoite from flagella form (flagella form).
Note: there is no cystic form.

1-Trichomonas Hominis

Geog. Distribution: all over the world, more in worm areas.
Habitat: in the large intestine especially caecum of human.

Morphology:
Trophozoite: Same as T. vaginalis.



Movement: Jerky.
Cyst: No cyst stage.
Transmission: perhaps by ingestion of the flagella form.
Infection/pathogenesis: not known.
Diagnosis:

1. Wet preparation.
2. Seroimmnunological investigations.
3. Culture.

Specimens: blood or stool.

1- Wet preparation of stool in saline to see the active motile forms (we should examine it quickly).
2- Iodine preparation to differentiate of the morphology characters.
3- Seroimmnunological investigations (agglutination & haemoagglutination).

2-Trichomonas vaginalis

Geog. Distribution: all over the world.
Habitat: In the genital tract of women and man (prostate gland).
Morphology:

1- Average length 13µm
2- 4 flagella anterior.
3- The 5^{th} passing back word to enclose.
4- Undulating membrane.
5- Has exostyle Costa & cleft (mouth).
6- Movement: twist and rotate (likened to a falling leaf).

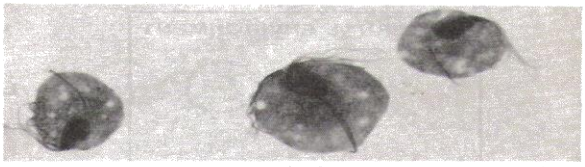


Plate 14.7 ***Trichomonas vaginalis*** **stained preparation.**
Courtesy of V. Zaman.

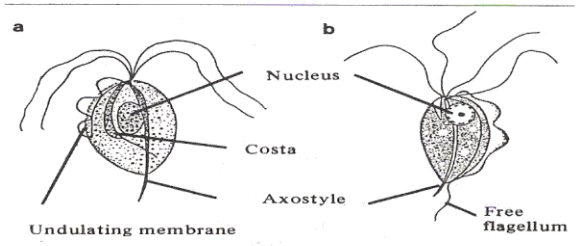
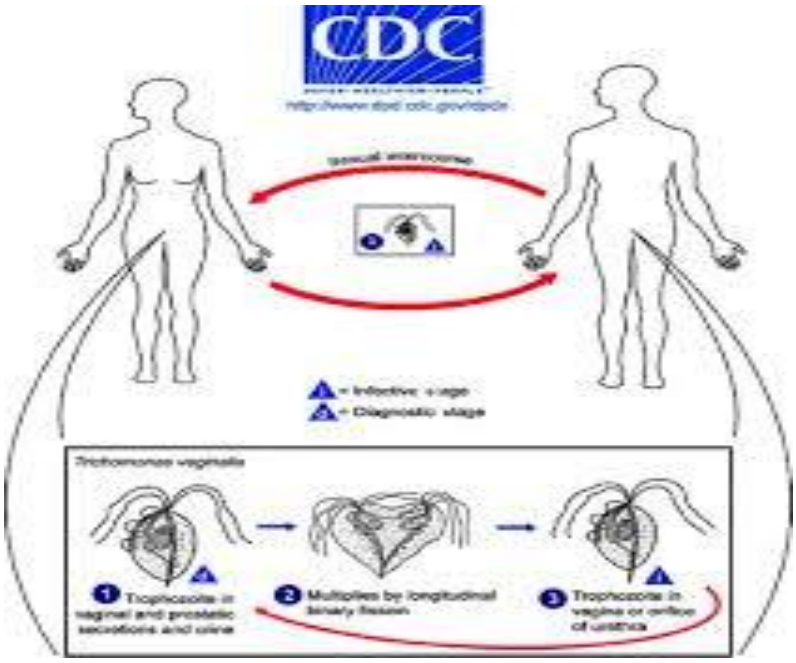


Fig. 14.4 *a* ***Trichomonas vaginalis.***

Life cycle: flagella are found in the genital tract.
Transmission: during dealings with contaminated matters (clothes, cotton..etc.



Trichomonas vaginalis resides in the female lower genital tract and the male urethra and prostate ❶, where it replicates by binary fission ❷. The parasite does not appear to have a cyst form, and does not survive well in the external environment. *Trichomonas vaginalis* is transmitted among humans, its only known host, primarily by sexual intercourse ❸.

Pathogenesis:
In women: Vaginal discharge and vaginal scratching and itching, vaginal creamy discharge and dysuria.
In men: affects prostate gland in man and can cause urethritis and discharge which lasts 4 weeks. The discharge is mainly asymptomatic and the male can serve as carrier.
Diagnosis:
1-Specimens:

- From male: urine, vaginal swap, or urethral discharge.
- From female: urine, or prostate secretions.

2-Lab diagnosis:

1- Examination of vaginal discharge and vaginal scraping (swap).
2- Examination of urine sediment after centrifugation.
3- Stained smear.
4- Culture.

Genus: Giardia

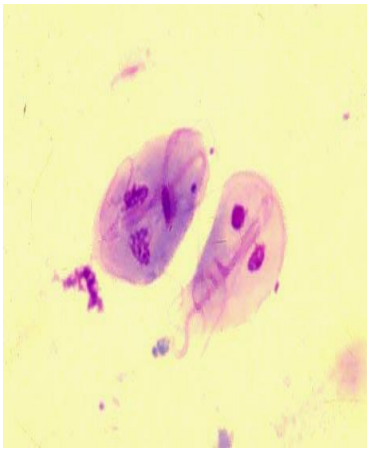
G. lambelia or G. intestinal.
Geographical distribution: all over the world.
Habitat:
Trophozoite: in the upper part of the small intestine, especially in the children.
Also in faesec after laxative and diarrhea.

Morphology:

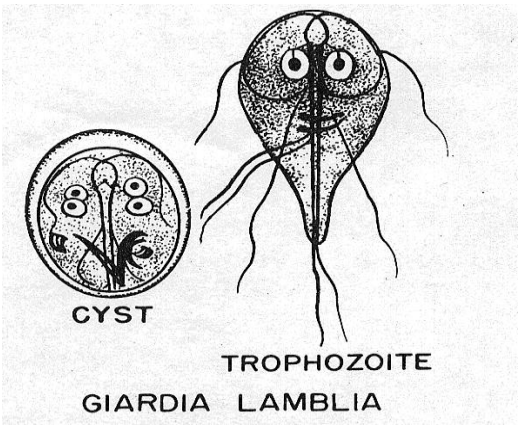
occurs as both a flagellated trophozoite and a non-flagellated cyst form

- trophozoite (9-21 μm long), motile, with 8 long flagella, ventral sucker which attaches to duodenal mucosa; lives only in small intestine; non invasive.

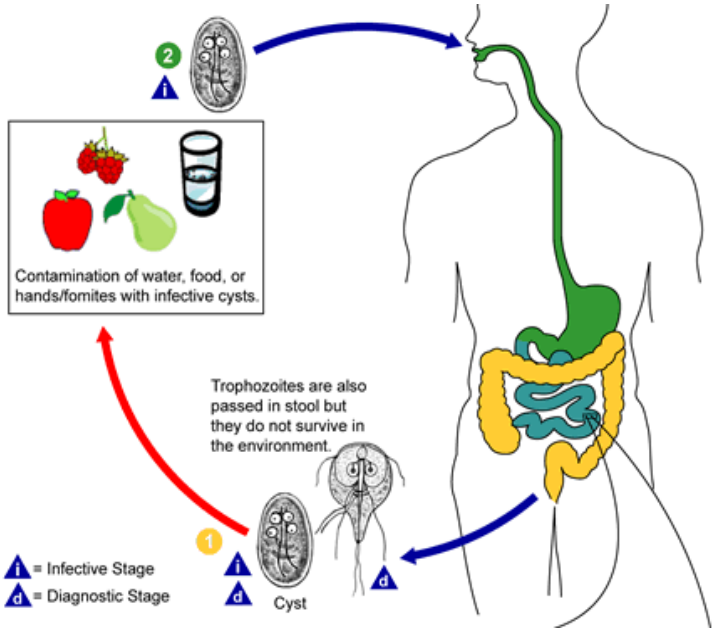
- cyst (8-12 μm); resistant to external environment, to municipal chlorination; intermittently expelled in stool.



Giardia trophozoites



Giardia cyst



Transmission and Life cycle:

It is transmitted by ingestion of the infected form (cyst) in contaminated food or drink. encystment occur in the intestine. Ingestion of contaminated food containing cyst of Giardia. Within 30 minutes cyst hatches out 2 trophozoites. From stomach to the duodenum where cyst divides giving rise to 2 trophozoites. In the duodenum, .multiplication of trophozoites occurs and colonization occurs

Pathogenesis: the disease is called giardiasis, and it affects the wall of the intestine. This can lead to:

1. Nausea, flatulence and some times constipation.
2. It can result in significant morbidity among children.

Lab Diagnosis:
Specimens: stool, serum (immunodiagnosis), duodenal biopsy (histological examination).
Macroscopical examination:
The color of the stool is yellow, fattish, and with foul smelling.
Microscopical examination:
Using the saline or the iodine preparations for stool, we can detect the trophozoite and the cyst forms. The cyst is easy to be found in the stool under the high power (X40).

CILIATE.

Blantidium coli (B. coli).
It is a protozoa moving by the cilia.
It is the only pathogenic ciliate in the digestive tract in man.
Geographical distribution: world wide, more in worm climate areas.

Habitat: Trophozoite in the large intestine of wild and domestic animals. Not common in human.

Infective form: the cyst is the infective form, and can be seen in the stool.

Morphology:

- Trophozoite: largest protozoa in man, measures about 50- 200µm in length and has two nucleolus (macro+micro).

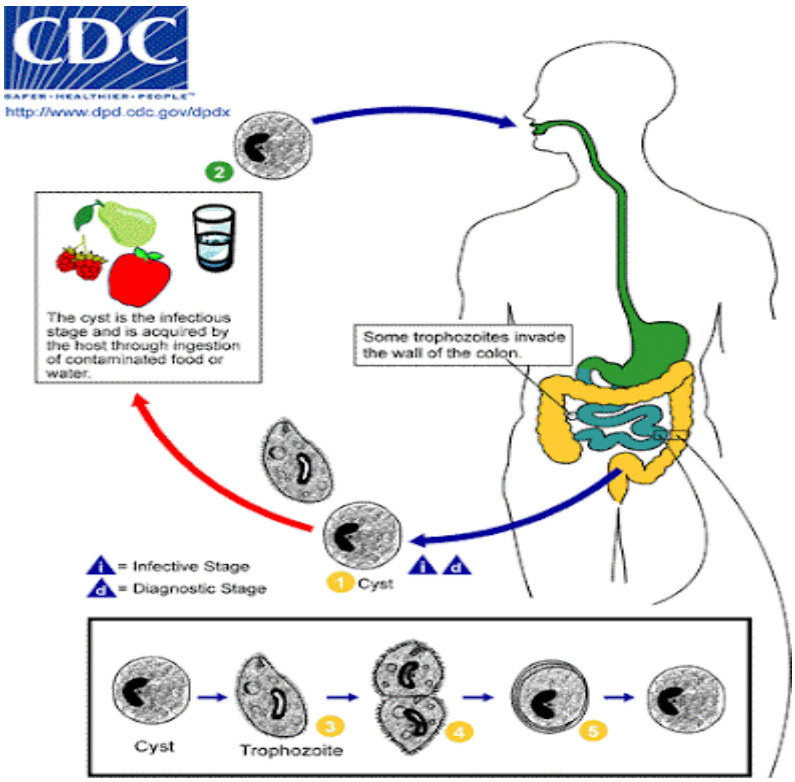
Transmission and Life cycle:

By ingestion of infective form from contaminated food or water or contaminated hands.

- ❖ Following ingestion, the cyst excyst in the intestine, each cyst producing a single ciliated.
- ❖ The ciliates multiply in the colon by binary division often following conjugation during which nuclear particles are exchanged between individuals.
- ❖ Thick walled cyst are formed which are excreted in the faeces. The cysts are infective when passed.

Look for large oval shaped ciliates which have rapid revolving motility. The ciliates are easily seen, measuring 50-200X40-70µm. by focusing the whipping cilia can be seen especially in the cytosome (mouth). In the dysentery specimens the ciliated usually contain ingested red cells.

Note: B. cloi ciliates degenerate rapidly in faeces, therefore sample should be examined while fresh.



Cysts are the parasite stage responsible for transmission of balantidiasis 1. The host most often acquires the cyst through ingestion of contaminated food or water 2. Following ingestion, excystation occurs in the small intestine, and the trophozoites colonize the large intestine 3. The trophozoites reside in the lumen of the large intestine of humans and animals, where they replicate by binary fission, during which conjugation may occur 4. Trophozoites undergo encystation to produce infective cysts 5. Some trophozoites invade the wall of the colon and multiply. Some return to lumen and disintegrate. Mature cysts are passed with feces 1.

Pathogenesis: the disease is called Blantidiasis, Blantidiasis dysentery.
It can cause ulcer in the intestinal mucous membrane, nausea, vomiting.

Diagnosis: specimen is stool.
Macroscopically we see mucous and blood.
Microscopic examination:
We can see both the cyst and\or the trophozoite.

The blood tissue flagellates

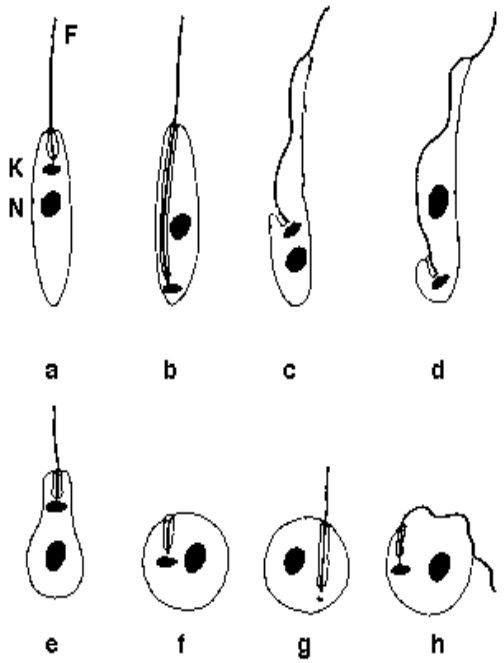
Leishmania and Trypanosoma.

Descriptive terms:

Nucleus:
- Kinetoplast: arises from axoneme.
- Undulating membrane: is not present in the non flagellate stage. It is ectoplasm of the body extending to surround the flagellum ranging along the side of the body.

Developmental stage in zoo-mastigophora:

Life-cycle stages of trypanosomatidae
a, promastigote;
b, ophistomastigote;
c,epimastigote;
d, trypomastigote;
e, choanomastigote;
f, amastigote;
g, paramastigote;
h,....K, kinetoplast; N, nucleus; F, flagellum.



A. Amastigote.
B. Promastigote.
C. Epimastigote.
D. Trypomastigote.

Promastigote form: it is the infective form of the Leishmania.
Transmission to human occurs when the infected sand fly feed from man.

Genus: Lieshmania.

Classification of Leishmania :

It can be classified into:

1. L. Donovani Complex.
2. L. Braziliensis Complex.
3. L Mexicana.
4. L Tropica.
5. L. Major.
6. L. aethiopica

It can be classified als according to the diseases which it causes into:

(1) Cutaneous Leishmaniasis (in the skin).
(2) Mucocutaneous Leishmaniasis (in mucocutaneous tissues and skin).
(3) Visceral Leishmaniasis (in the internal organs- Kalazar).

Lieshmania Donovani.

Geographical distribution:

India and also where, the disease is called Kala-azar.

Pathogencity:

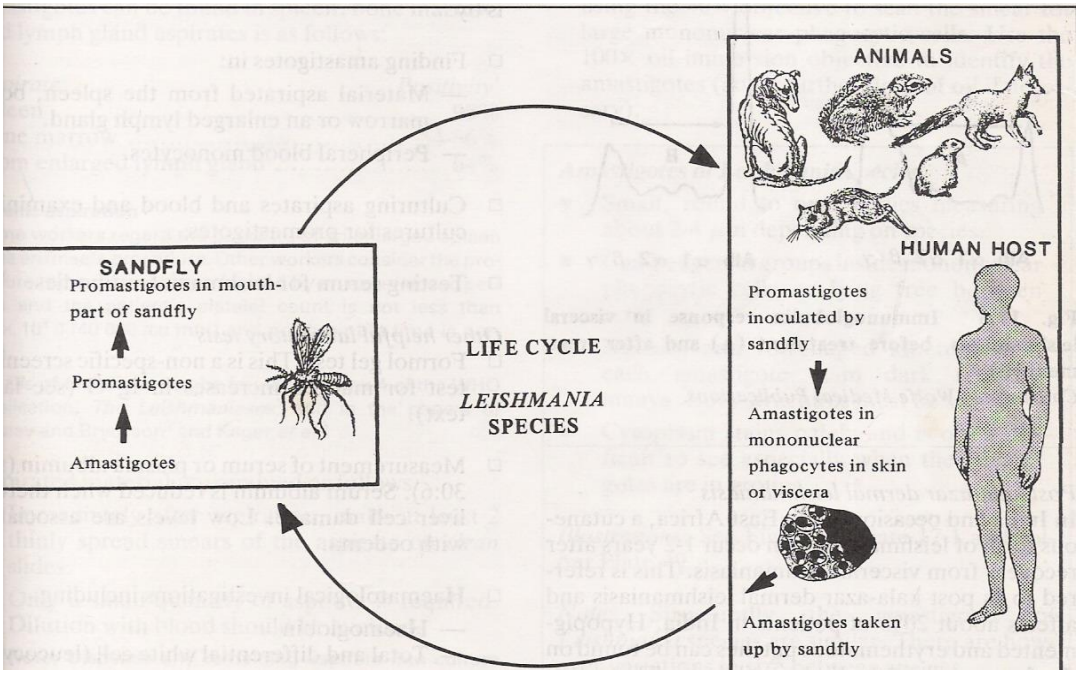
Cause the visceral leishmaniasis or Kala azar, called the death fever, anaemia, dysentery and loosing weight.

Habitat:

Found in RES (Reticuloendothelial System) Visceral organs, specially spleen, liver, bone marrow, intestinal mucosa.
Also in: Kidney, lungs, CSF.

Stages in life cycle:

(1) amastigote in man and reservoirs, e.g. dog and rodents.
(2) Promastigote: in sand fly vector and in lab culture "infective stage".

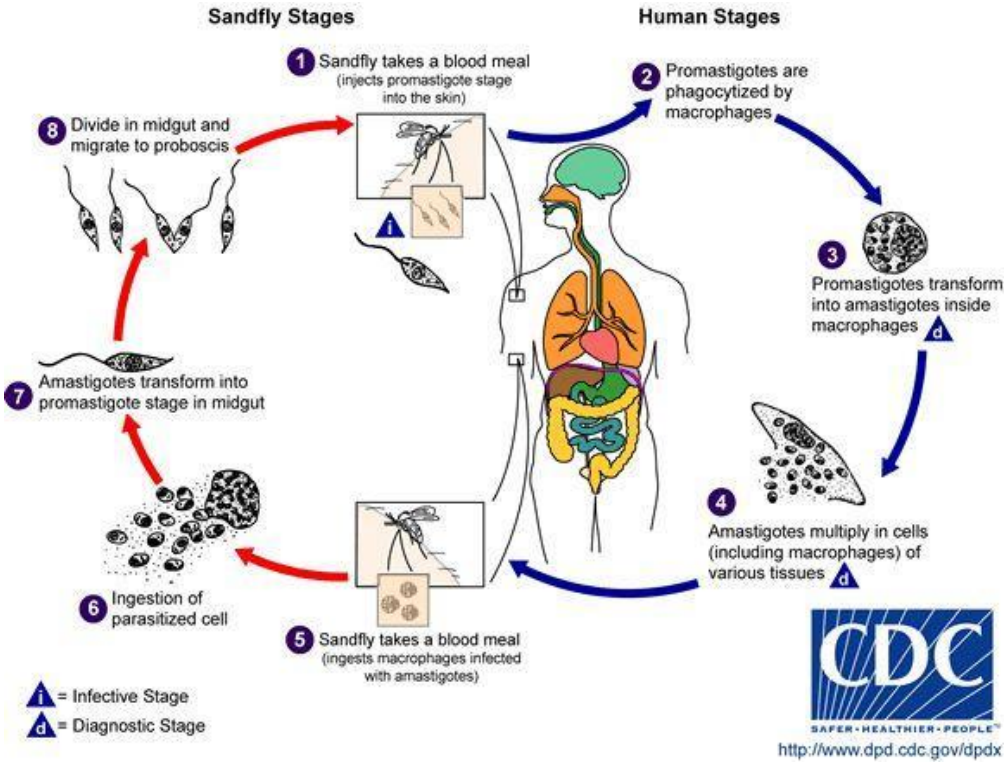


In man:

2. Following inoculation, the promastigotes are taken up by phagocytic cells and develop into amastigotes.
3. Amastigotes are spread in the blood and multiply in the macrophage of the RES, e.g. liver spleen, BM…
4. Intracellular and free amastigotes are ingested by the female of sandfly vector when it sucks blood.

In sand fly vector (Phalebotomus):

5. Amastigotes become flagellated promastigotes in the midgut of the sand fly.
6. Promastigotes multiply and fill the lumen of the sand fly. After several days these move foreword to the head and the mouth of sand fly ready to be inoculated when the vector next takes blood meal.



2- Leishmania causing Cutaneous (skin) Leishmaniasis:

1. L Tropica.
2. L. Major.
3. L. aethiopica

Geographical distribution:

Meddle east, Afghanistan, India, ethiopica and also where.

Life cycle:

The parasites multiply in the skin macrophages.

Pathogenicity:

Causes ulcer in the skin, healing.

Lab diagnosis of Leishmaniasis:

1. By finding amastigotes in:
    a) Material aspirated from spleen, liver, BM and enlarged lymph nodes.
    b) Peripheral blood monocytes.
2. Culturing aspirates for promastigotes
3. Testing serum for leishmanial antibodies.

4. Biopsy.
5. Detection of amastigotes in smear taken from infected ulcer.
6. Animal inoculation.

Genus: Trypanosoma.

Species of medical importance:
T. brucei complex (gambiense and rhodesiense) which cause the African trypanosomiasis.
T. cruzi which causes the American trypanosoiasis.

1. African trypanosomiasis.

Geographical distribution:
African trypanosomiasis occurs in tsetse fly areas of Africa (tropical, east and west).

Habitat:

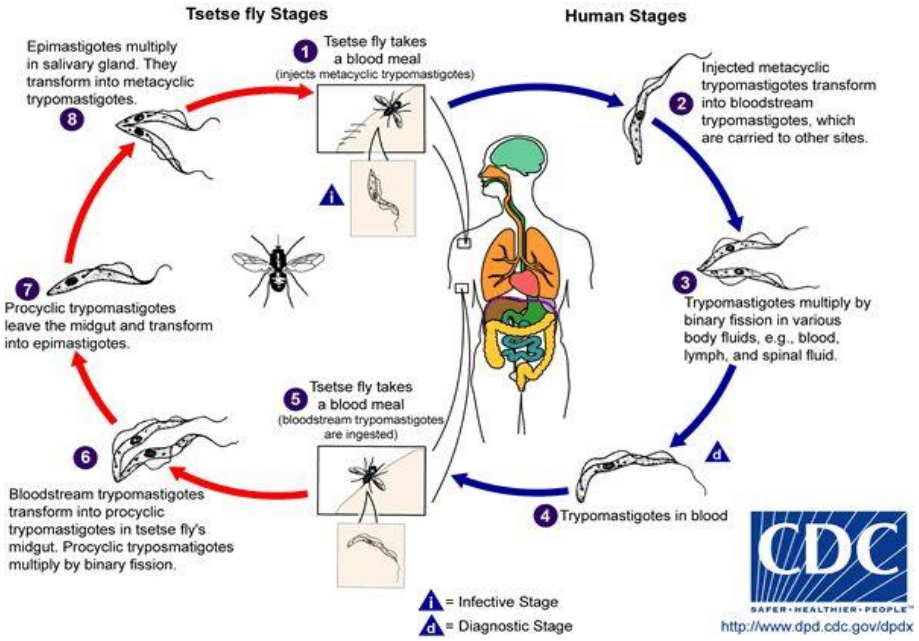
1) trypomastigotes form in man (blood, CSF).
2) epimastigotes and metacyclic forms: in the midgut of the tsetse.

Transmission:

1. By bite of tsetse fly vector (intermediate host).
2. Mechanical inoculation.
3. Blood transfusion.

Life cycle:

During a blood meal on the mammalian host, an infected tsetse fly (genus *Glossina*) injects metacyclic trypomastigotes into skin tissue. The parasites enter the lymphatic system and pass into the bloodstream ❶. Inside the host, they transform into bloodstream trypomastigotes ❷, are carried to other sites throughout the body, reach other blood fluids (e.g., lymph, spinal fluid), and continue the replication by binary fission ❸. The entire life cycle of African Trypanosomes is represented by extracellular stages. The tsetse fly becomes infected with bloodstream trypomastigotes when taking a blood meal on an infected mammalian host (❹, ❺). In the fly's midgut, the parasites transform into procyclic trypomastigotes, multiply by binary fission ❻, leave the midgut, and transform into epimastigotes ❼. The epimastigotes reach the fly's salivary glands and continue multiplication by binary fission ❽. The cycle in the fly takes approximately 3 weeks. Humans are the main reservoir for *Trypanosoma brucei gambiense*, but this species can also be found in animals. Wild game animals are the main reservoir of *T. b. rhodesiense*.



Pathogenecity:

the disease is called Sleeping sickness.

1. Painful Chancre at site of inoculation
2. High Irregular fever, headache.
3. Lymphadenopathy.
4. Loss of weight, sweating.
5. Long time infection can show long time sleeping -central nervous system (CSF) involved and can lead to death.

Life cycle:

1. Metacyclic trypomastigotes are inoculated through the skin (tsetse), they develop into trypomastigotes, which multiply.
2. The trypomastigotes are carried to the heart, and various organs of the body.
3. Trypomastigotes are ingested by tsetse when it sucks blood. In the midget, the parasite develops and multiplies.
4. After 2-3 weeks, the trypomastigotes migrate to the salivary glands where they develop into epimastigote, which in turn develop into metacyclic trypomastigotes.

Morphology: spindle shape body

1- Pleomorphic trypanosomiasis: showing variety of forms measuring from 18-35µm in length.
2- Single flagella arising from kinitoplast, and extend forewords, and undulating membrane.
3- Small dot-like kinetoplast.
4- Dark centrally nucleus, with pale cytoplasm.

Diagnosis:

Specimens: blood, CSF, Lymph gland aspiration.

1- Blood examination: to detect the trypomastigotes.
- ❖ Thick stained blood film.
- ❖ Capillary tube centrifugation concentration technique.
- ❖ Test tube centrifugation concentration technique.

2- CSF and Lymph node aspirate: to detect motile trypomastigote.
3- Serological tests.

4- Inoculation in lab animals.

2- American trypanosomiasis.

Geographical distribution:

South of North America, Central America and South America.

Habitat:

1. Trypomastigotes form in blood of man.
2. Amastigote: in man tissues (liver, spleen, lungs, BM, brain and lymph glands).
3. Epimastigotes and metacyclic forms: in the midgut of the Bug.

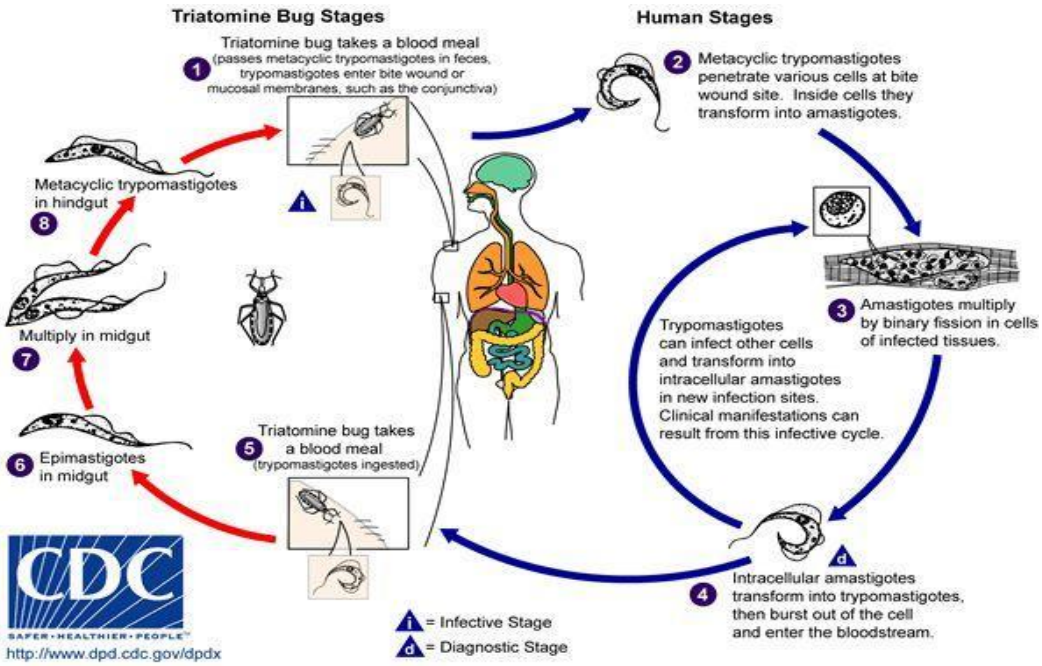
Transmission:

The infective form is the metacyclic trypomastigote, which is contained in the bug faeces, and then rubbed in wound-cut made by insect on the skin.

Life cycle:

- ❖ Metacyclic trypomastigote contained in bug faeces, penetrate the skin after insect bite.
- ❖ Develop into trypomastigotes which infect the RE cells near the site of bite, and multiply intracellularly as amastigotes.
- ❖ Amastigotes develop to form trypomastigote, which released into the blood when the cell is ruptured. NO MULTIPLICATION OCCURS IN THE STAGE OF BLOOD TRYPOMASTIGOTES.
- ❖ By the way of blood, Trypomastigotes reach tissue cell, and become amastigotes and multiply forming masses called pseudocycts.

- ❖ In the pseudocyctes amastigotes develop into epimastigotes, and then trypomastigotes and continue. Some of them taken by the bug through another bite.
- ❖ In the vector, it develops into epimastigotes, which multiply to give metacyclic trypomastigote in the hindgut of the bug.



Pathogenecity:

American trypanosomiasis, or Chaga's disease, Where the patient suffer of inflamed swelling, if it was in near the eyes, this is Romana signs oedema of eyelids. In the acute stage, amastigotes multiply and spread in the tissues, it causes fever, malaise, enlargement of lymph node, liver, and lesser in spleen. Acute attack for the heart can cause death.

Laboratory diagnosis:

1- By finding the trypomastigotes in the blood during early acute infection.
2- Detection of epimastigotes in the blood culture.
3- By finding amastigotes in lymph node aspirate.
4- Serum for serology.
5- Inoculation in lab animals.

Genus: MALARIA PARASITES.

The malaria parasites are protozoan parasites, belong to the family plasmodium, and classified into many species. The plasmodium which infects human are:

Widespread species	Plasmodium falciparum.
	Plasmodium vivax.
Less widespread specie	Plasmodium malariae.
	Plasmodium ovale.

The term Benign malaria is used to describe the P. Vivax and P. Ovale.
The term Malignant malaria is used to describe the P. falciparum. Quart

Transmission and Life cycle:

1- By the bite of the female of the anopheles mosquitoes.
2- By transfusion of infected blood.
3- By injection through the use of needle and syringes contaminated with infected blood.
4- Very occasionally, congenital transmission occurs.

* Two hosts involved: man and mosquito.
* The malaria of man has two stages:

1. In the anopheles a sexual, extracellular cycle of development occurs which called sporogony.
2. In human host, malaria parasite has an asexual intracellular cycle of development called schizogony, the human is the intermediate host.

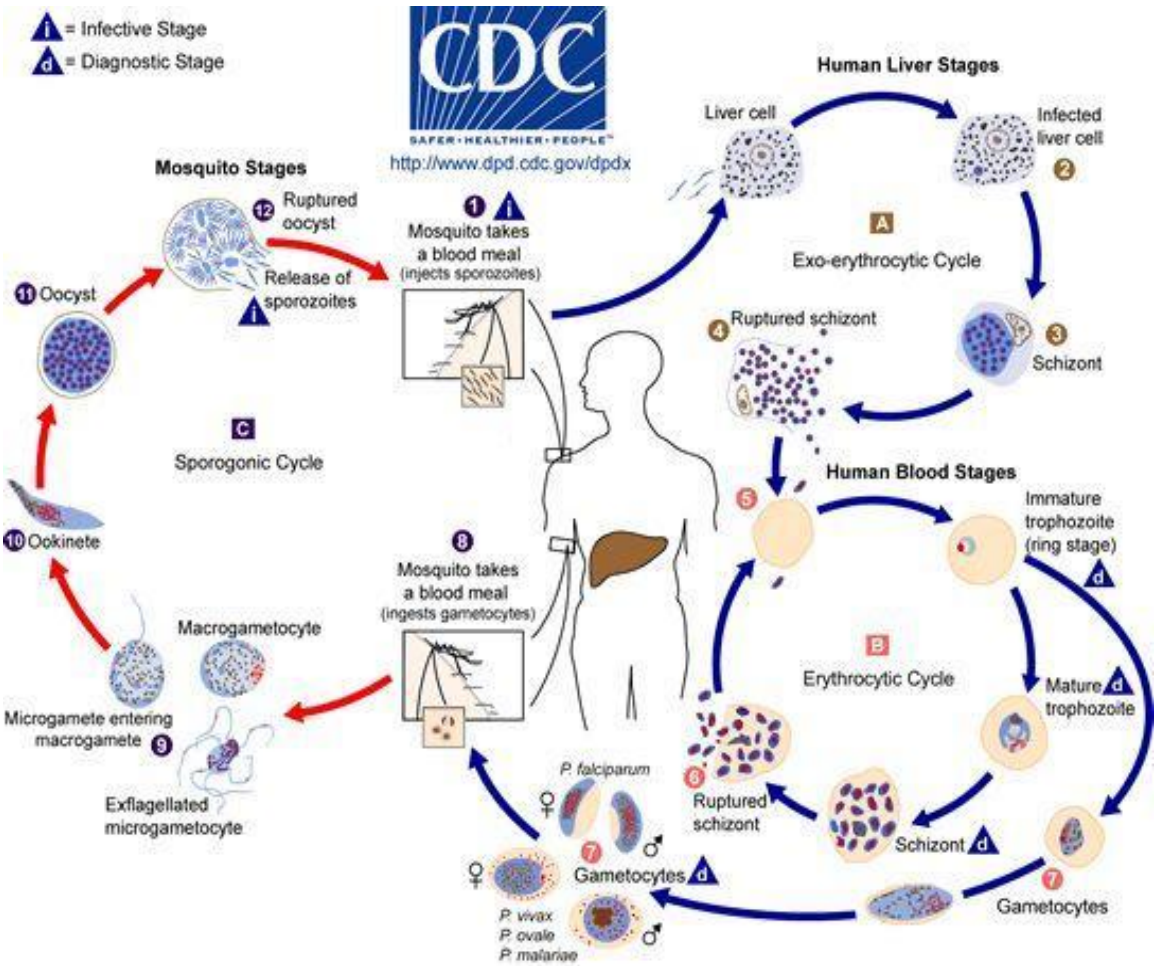
Life cycle:

The infective stage is the sporozoites.

1- Sporozoites contained in saliva of an infected mosquito are injected into the blood of human host.
2- After circulating in the blood stream they enter the liver cells (hepatocytes).
3- They grow, multiply and develop into schizonte (pre erythrocytic schizonte), which when mature contains about 30 000 merozoites.
4- When mature, the schezonte and the liver cell rupture and merozoite enter the blood. To survive it should enter red blood cells within minutes. This stage takes 36-48 h. to complete.
5- Merozoites become trophozoites, and when fully developed will become schizontes (Schizogony) which contains 8-32 merozoites (Erythrocytic schizonte). When mature it will rupture, and release merozoites.

6- After many cycles of invading RBCs, some of the merozoites develop into schizonts which follow a sexual development and become a gametocyte which will be ingested by a female of anopheles.

7- In the mosquito, they develop into male and female gametocytes, which when fertile a zygote is formed.
8- Zygote develop multiple times to form the sporozoites which spread in the saliva of the mosquito and when bite a human start again.



Clinical features:

- The patient Feels cold, rigor and headache.
- Fever, sweating.
- Anaemia with enlarged spleen.
- Diarrhoea and vomiting.

Diagnosis:

Before starting, remember you should ask the patient if he or she has taken any anti-malaria dugs for the last 24 hours.

Specimen:

Blood, serum.

Lab diagnosis:

a- Examining the stained thick blood film to detect the parasites.
b- Examining the stained thin blood film to identify the species of the parasite.
c- Examining the Buffy coat.
d- Serological examination.

Chapter 3

Phylum Platyhelminthes

Flatworms are acoelomate, triploblastic animals. They lack circulatory and respiratory systems, and have a rudimentary excretory system. The digestive system is incomplete in most species. There are four classes of flatworms, the largely free-living turbellarians, the ectoparasitic monogeneans, and the endoparasitic trematodes and cestodes. Trematodes have complex lifecycles involving a molluscan secondary host and a primary host in which sexual reproduction takes place. Cestodes, or tapeworms, infect the digestive systems of primary vertebrate hosts.

Trematodes

The phylum Platyhelminthes contains the class Trematoda, more commonly known as the trematodes. Most of the common zoonotic trematodes are digenetic, which means these worms require a minimum of two hosts to develop into the adult stage (Nithiuthai et al., 2004). Asexual and sexual reproduction normally occur in an invertebrate and vertebrate host, respectively (Roberts et al., 2013a). Trematodes are primarily hermaphroditic with the family Schistosomidae being a notable exception (Mone and Boissier, 2004). Adult worms live in a variety of locations in the definitive host, including the lungs, liver, and stomach (Bowman, 2009). Regardless of location, operculated eggs are shed in the feces of the definitive host. After a designated time period, which is dependent on temperature, oxygen tension, and pH, the eggs will develop into a miracidium. The miracidium is a ciliated free-living organism, which upon hatching from the egg, enters a mollusk – the intermediate host (Roberts et al., 2013e). The hatching process can occur in water or after ingestion by the requisite intermediate host. In the case of certain species such .(as Fasciola hepatica, light facilitates hatching (Wilson, 1968

Once inside the intermediate host, usually a snail, the miracidium undergoes a complex series of morphological changes to the saclike sporocyst. Inside this sporocyst, other sporocysts, known as daughter sporocysts, may develop. Alternatively, another intermediate stage, the redia or germinal sac, may develop. Rediae normally exit the sporocyst in a dramatic manner, rupturing

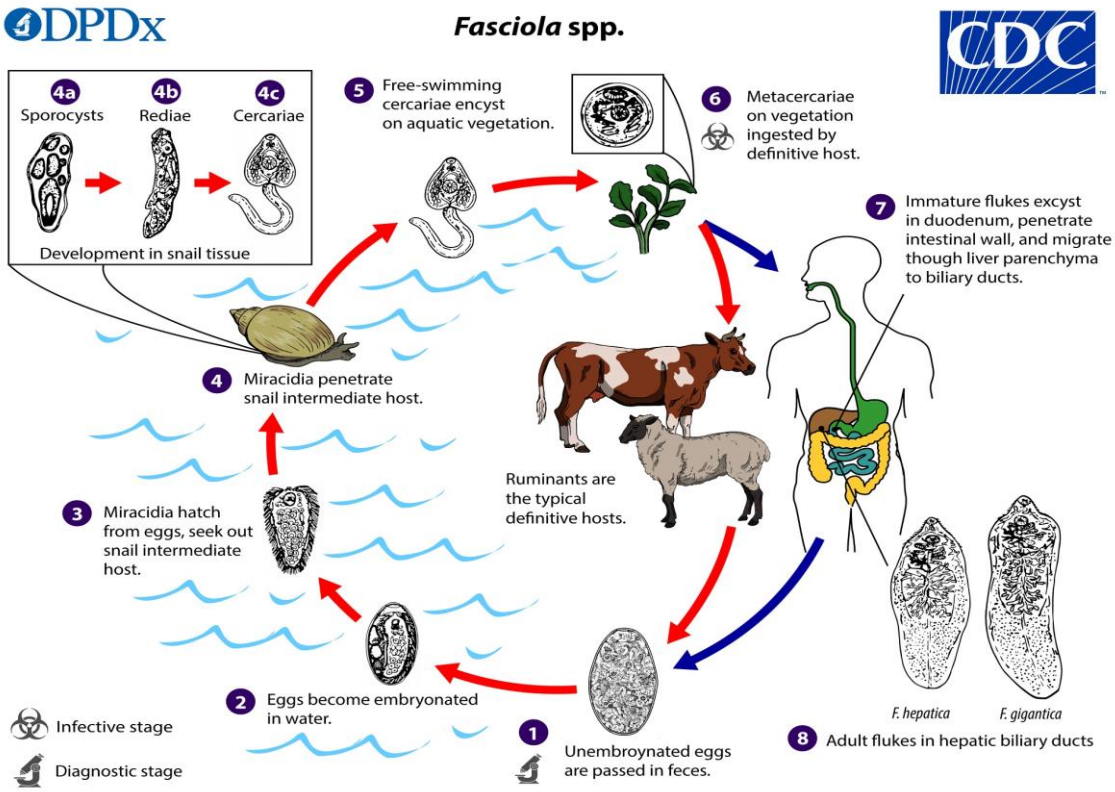
the membrane, after which they begin a wandering migration around the host. Rediae can then either develop into daughter rediae or into the next stage, which is the cercaria. The cercariae leave the host and can infect a definitive host in the case of the family Schistosomidae. In other trematodes, the cercariae will either infect a second intermediate host, where they may enter a dormant stage known as the metacercariae (Roberts et al., 2013e). Alternatively, the cercariae of some trematodes, such as the family Fasciolidae, will remain in the environment where they encyst as metacercariae (Olsen, 1947). The life cycle is completed when the definitive host ingests either the second intermediate host containing the metacercariae or the encysted metacercariae in the external environment. As with nematodes, the trematodes undergo complex migrations within the host, after which they arrive at the designated tissue (Nithiuthai et al., 2004; Roberts et al., 2013e). The life cycle and biology described above is what would be considered a classic trematode life cycle. As with all organisms, there are notable exceptions

Liver fluke
Fasciola hepatica
order
Echinostomida
family
Fasciolidae
taxonomy
Fasciola hepatica Linnaeus, 1758, "in aquis dulcibus ad radices lapidum, inque hepate pecorum. Diss. de Ovibus;" Europe.
other common names
English: Sheep liver fluke; French: Grande douve du foie, douve du foie de mouton; German: Großer Leberegel.
physical characteristics
Adult liver flukes may reach 1.7–2.2 in (4–5 cm) in length and 0.6 in (1.5 cm) wide. They are typically about 1.3 in (3 cm) long, 0.4 in (1 cm) wide, and have a spiny tegument. They taper toward the rear. The front end bears an oral sucker and a cone-shaped tip. The sucker on the fluke's ventral (lower) surface is larger than the oral sucker. The ventral sucker is about a third of the body length behind the oral sucker. The branched ovary is situated behind and to the side of the ventral sucker about a third of the way back in the body. The testes are also branched and extend throughout the body behind the ovary.
distribution

Worldwide, but found most often in Europe and Latin America in habitats congenial to their freshwater snail and definitive hosts.

Life Cycle

Immature eggs are discharged in the biliary ducts and passed in the stool 1. Eggs become embryonated in freshwater over ~2 weeks 2; embryonated eggs release miracidia 3, which invade a suitable snail intermediate host 4. In the snail, the parasites undergo several developmental stages (sporocysts 4a, rediae 4b, and cercariae 4c). The cercariae are released from the snail 5 and encyst as metacercariae on aquatic vegetation or other substrates. Humans and other mammals become infected by ingesting metacercariae-contaminated vegetation (e.g., watercress) 6. After ingestion, the metacercariae excyst in the duodenum 7 and penetrate through the intestinal wall into the peritoneal cavity. The immature flukes then migrate through the liver parenchyma into biliary ducts, where they mature into adult flukes and produce eggs 8. In humans, maturation from metacercariae into adult flukes usually takes about 3–4 months; development of *F. gigantica* may take somewhat longer than *F. hepatica*.



Diagnosis

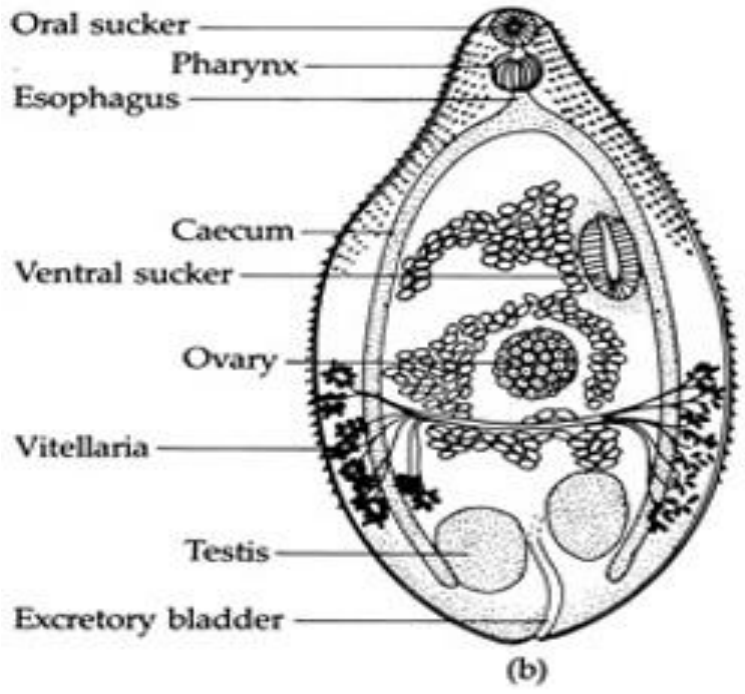
The standard way to be sure a person is infected with *Fasciola* is by seeing the parasite. This is usually done by finding *Fasciola* eggs in stool (fecal) specimens examined under a microscope. More than one specimen may need to be examined to find the parasite. Sometimes eggs are found by examining duodenal contents or bile.

Infected people don't start passing eggs until they have been infected for several months; people don't pass eggs during the acute phase of the infection. Therefore, early on, the infection has to be diagnosed in other ways than by examining stool. Even during the chronic phase of infection, it can be difficult to find eggs in stool specimens from people who have light infections.

Certain types of blood tests can be helpful for diagnosing *Fasciola* infection, including routine blood work and tests that detect antibodies (an immune response) to the parasite.

Intestinal trematodes

The trematode Heterophyes heterophyes, a minute intestinal fluke.



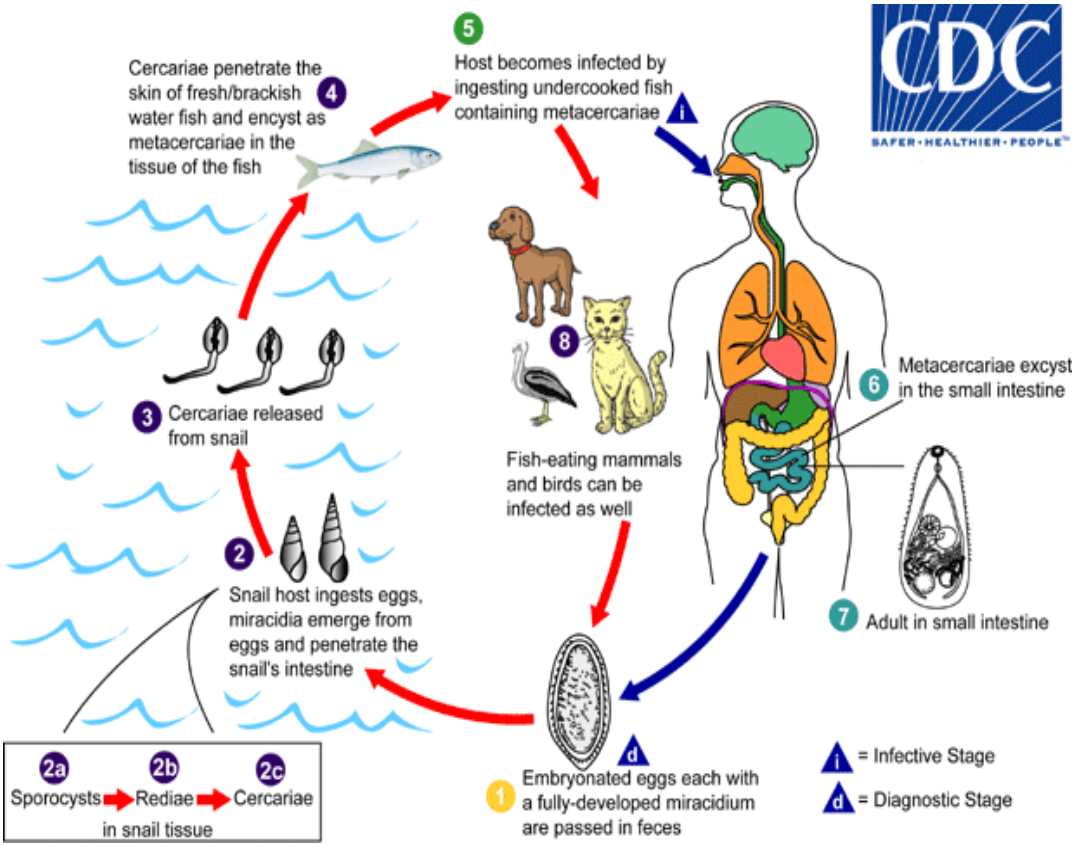
Geographic Distribution

Egypt, the Middle East, and Far East.

Heterophyiasis is acquired by eating infected raw or undercooked fish from freshwater or brackish water containing **metacercariae (encysted stage)**. After ingestion, metacercariae excyst and attach to the mucosa of the small intestine. There, they develop into adults, growing to about 1.0 to 1.7 mm by 0.3 to 0.4 mm.

Life Cycle

Adults release embryonated eggs each with a fully-developed miracidium, and eggs are passed in the host's feces[1]. After ingestion by a suitable snail (first intermediate host), the eggs hatch and release miracidia which penetrate the snail's intestine[2]. Genera *Cerithidia* and *Pironella* are important snail hosts in Asia and the Middle East respectively. The miracidia undergo several developmental stages in the snail, i.e. sporocysts[2a], rediae[2b], and cercariae[2c]. Many cercariae are produced from each redia. The cercariae are released from the snail[3] and encyst as metacercariae in the tissues of a suitable fresh/brackish water fish (second intermediate host)[4]. The definitive host becomes infected by ingesting undercooked or salted fish containing metacercariae[5]. After ingestion, the metacercariae excyst, attach to the mucosa of the small intestine[6] and mature into adults (measuring 1.0 to 1.7 mm by 0.3 to 0.4 mm)[7]. In addition to humans, various fish-eating mammals (e.g., cats and dogs) and birds can be infected by *Heterophyes heterophyes* [8].



symptoms

The main symptoms are diarrhea and colicky abdominal pain. Migration of the eggs to the heart, resulting in potentially fatal myocardial and valvular damage, has been reported from the Philippines. Migration to other organs (e.g., brain) has also been reported.

Laboratory Diagnosis

The diagnosis is based on the microscopic identification of eggs in the stool. However, the eggs are indistinguishable from those of Metagonimus yokogawai and resemble those of Clonorchis and Opisthorchis.

Schistosomes.

Schistosomes are trematodes (flukes) that live in the blood.

Classification of medical importance schistosomes:

1- S. Haematobium (urinary).
2- S. Mansoni (intestinal).
3- S. Japonicum (intestinal).

Features of human schistosomes:

- They develop in the venous system of the intestine and bladder.
- Sexes are separated.
- They are not like the other flukes, which are flattened but they are long and worm-like.
- Human is the definite host of S. Haematobium. S. Mansoni.
- Transmission is by contact with water containing the infective form of the parasite which is the cercariae. These develop in the snail and are able to penetrate the unbroken skin.

Geographical distribution:

S. Haematobium: Africa, M. East and India.
S. Mansoni : Africa, M. East and S. America.
S. Japonicum: Far East.

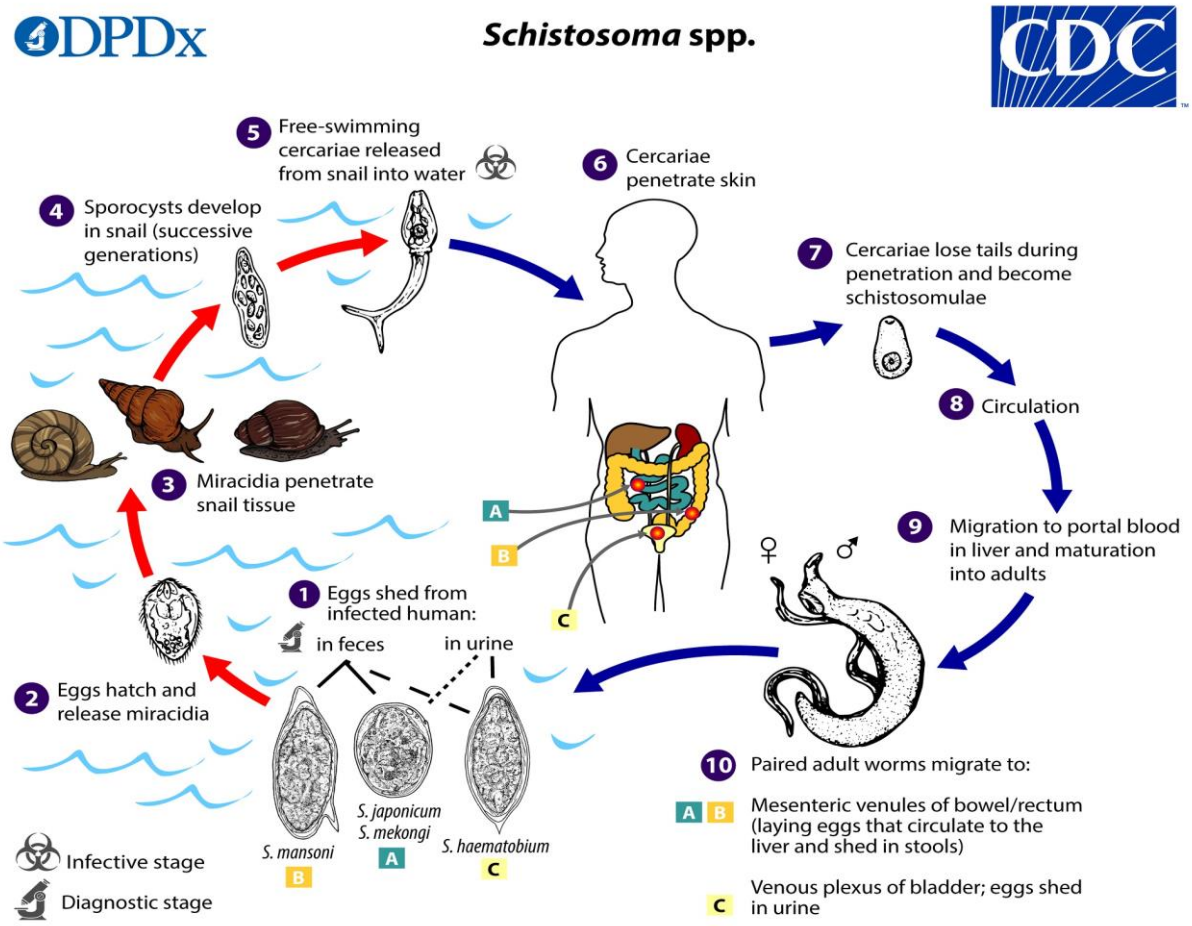
Habitat:

1- S. Haematobium:
- Adult in the venous system of the bladder.
- Egg: in urine and occasionally in stool (contamination).

2- S. Mansoni:
- Adult in the venous system of the large intestine and mesenteric vein.
- Egg: in stool and occasionally in urine.

3- S. Japonicum:
- Adult in the venous system of the large intestine and gastric vein.
Egg: in stool only.

Transmission and Life cycle:



Life Cycle

1- S. Haematobium infection is caused by an infected person passing urine containing the egg of the parasite into water which is used by the others for bathing, washing, agricultural purposes, and fishing.
2- S. Mansoni infection: is caused by an infected person passing stool containing the egg of the parasite into water which is used by the others for bathing, washing, agricultural purposes, and fishing.
3- S. Japonicum infection: is caused by an infected person or animal passing stool containing the egg of the parasite into water which is used by the others for bathing, washing, agricultural purposes, and fishing.

Schistosoma eggs are eliminated with feces or urine, depending on species (1). Under appropriate conditions the eggs hatch and release miracidia (2), which swim and penetrate specific snail intermediate hosts (3). The stages in the snail include two generations of sporocysts (4) and the production of cercariae (5). Upon release from the snail, the infective cercariae swim, penetrate the skin of

the human host [6], and shed their forked tails, becoming schistosomulae [7]. The schistosomulae migrate via venous circulation to lungs, then to the heart, and then develop in the liver, exiting the liver via the portal vein system when mature, [8] [9]. Male and female adult worms copulate and reside in the mesenteric venules, the location of which varies by species (with some exceptions) [10]. For instance, *S. japonicum* is more frequently found in the superior mesenteric veins draining the small intestine [A], and *S. mansoni* occurs more often in the inferior mesenteric veins draining the large intestine [B]. However, both species can occupy either location and are capable of moving between sites. *S. intercalatum* and *S. guineensis* also inhabit the inferior mesenteric plexus but lower in the bowel than *S. mansoni*. *S. haematobium* most often inhabitsin the vesicular and pelvic venous plexus of the bladder [C], but it can also be found in the rectal venules. The females (size ranges from 7–28 mm, depending on species) deposit eggs in the small venules of the portal and perivesical systems. The eggs are moved progressively toward the lumen of the intestine (*S. mansoni,S. japonicum, S. mekongi, S. intercalatum/guineensis*) and of the bladder and ureters (*S. haematobium*), and are eliminated with feces or urine, respectively [1].

Clinical features:

1- S. Haematobium:

- The disease is called "Bilharzias or Bilharsiasis", eggs classified in the bladder.
- Within 24 h. an intensive irritation may occur at the site of penetration, this is called "swimmer's itch".
- Haematuria: blood in the urine.
- Proteinuria: protein in urine.
- In heavy infections: Liver and spleen enlargement.
- Deposition of eggs in many organs in heavy and old infections.

2- S. Mansoni and S. Japonicum:

- Within 24 h. an intensive irritation may occur at the site of penetration, this is called "swimmer's itch".
- Host reaction to eggs logged in the intestinal mucosa leading to the formation of Granulomata which can cause colonic and rectal polyps.
- Ulceration and thichness of the wall of bowel.
- In heavy infections: Liver and spleen enlargement.
- Deposition of eggs in many organs in heavy and old infections.

Laboratory diagnosis:

1- S. Haematobium:

Sample: urine.

- Finding the eggs or occasionally the hatched miracidia in urine.
- Detecting eggs in the rectal or bladder mucosal biopsy.

2- S. Mansoni:

- Finding the eggs in faeces and may also be found in urine.
- Detecting eggs in the rectal in biopsy if they are not found in faeces.

3- S. Japonicum:

- Finding the eggs in faeces.
- Detecting eggs in the rectal in biopsy if they are not found in faeces.

Other finding:

- Mucous and blood in the stool in the urine.
- Blood eosinophili .
- Bacteriuria.

Chapter 4

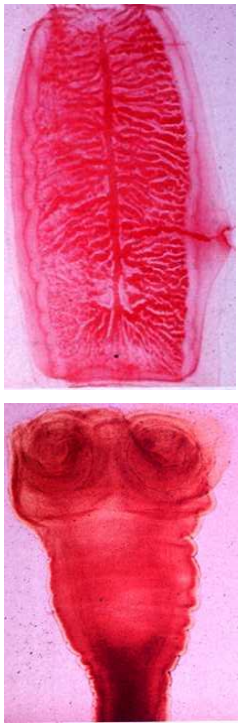
TAPEWORM (CESTODES).

Taenia (cysticercus), Echinococcus (hydatid), Diphyllobothrium, Hymenolepis, Dipylidium

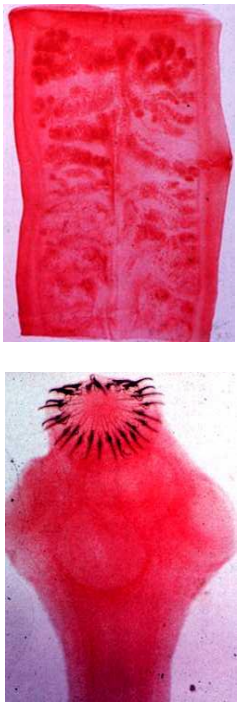
General Structure of Tapeworm

Head region (scolex) : contains suckers (1) and hooks (2) used to attach to a host organism.

Identification of scolex:
It is very small measuring only one mm in diameter.
The scolex has four suckers and crown of hooks.
Taenia saginata and T. solium:
Scolex And Proglottids



(1)



(2)

Zone of Proliferation - Undifferentiated area behind the scolex (neck region)(
Strobilia - Chain of segments (proglottids)
Proglottids : square body segments used for reproduction.
Immature proglottids - developing reproductive
Mature proglottids: mature reproductive organs .
Gravid proglottids: contain eggs in the uterus.

Tapeworms are of considerable medical and economic importance. About 50 million people are infected with Taenia saginata or T. solium, and about 50,000 people die annually of cysticercosis, an infection with larval Taenia. The morphology, life cycles and medical importance of tapeworms (Platyhelminthes, Eucestoda) are discussed. Particular attention is paid to Taenia solium (which causes cysticercosis in humans), Taenia saginata, Echinococcus granulosus and Echinococcus multilocularis (which cause cystic and multilocular=alveolar hydatid disease in humans, respectively), Diphyllobothrium latum, and some other tapeworms of man and domestic animals

Main features of the cestodes:

1. Adults of most live in the small intestine.
2. Human is the definite host of the T. Saginata and T. Solium.
3. Transmission of Taenia species is by ingestion of cysticercus larvae in undercooked beef (T. Saginata) or pork (Solium).
4. Laboratory diagnosis:
5. Is by finding gravid segments and eggs in the faeses. The eggs of T. Saginata can also be found in the perianal area.
6. Body is divided into segments.

1- *Taenia Saginata*.

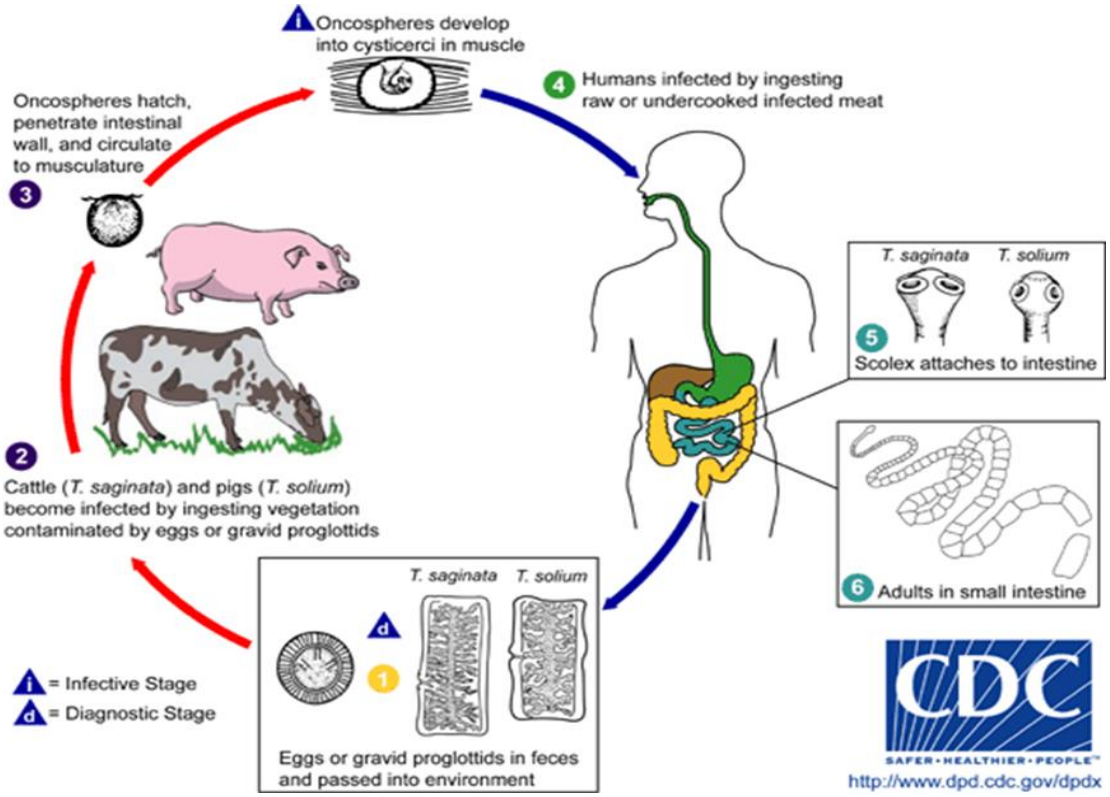
Distribution:

T. saginata has a wide world distribution.

Life cycle:

1. After ingestion of egg, the scolex (head) is freed and become attached to the wall of the small intestine by its suckers. Segments are formed from the neck region and within 2-3 months grow into adult tapeworm with gravid (egg-filled) segments being found at the tail end. Usually one worm present but multiple can occur.
2. When fully developed, the graved become attached and eggs passed in stool.

3. To continue, eggs must reach the place of cattle food, after ingestion by a cow, pass through the intestine wall to the muscles, where the egg grows into infective larvae.



Clinical features:

Usually infection rarely produces serious features. They may be abdominal pain with intestinal disturbances and loss of appetite. Very occasionally appendicitis can occur.

Identification of *T. saginata* scolex:

It is very small measuring only two mm across. The scolex has four suckers and no hooks. The absence of hooks distinguish it from T. Solium.

2 - *Taenia Solium*.

Distribution:

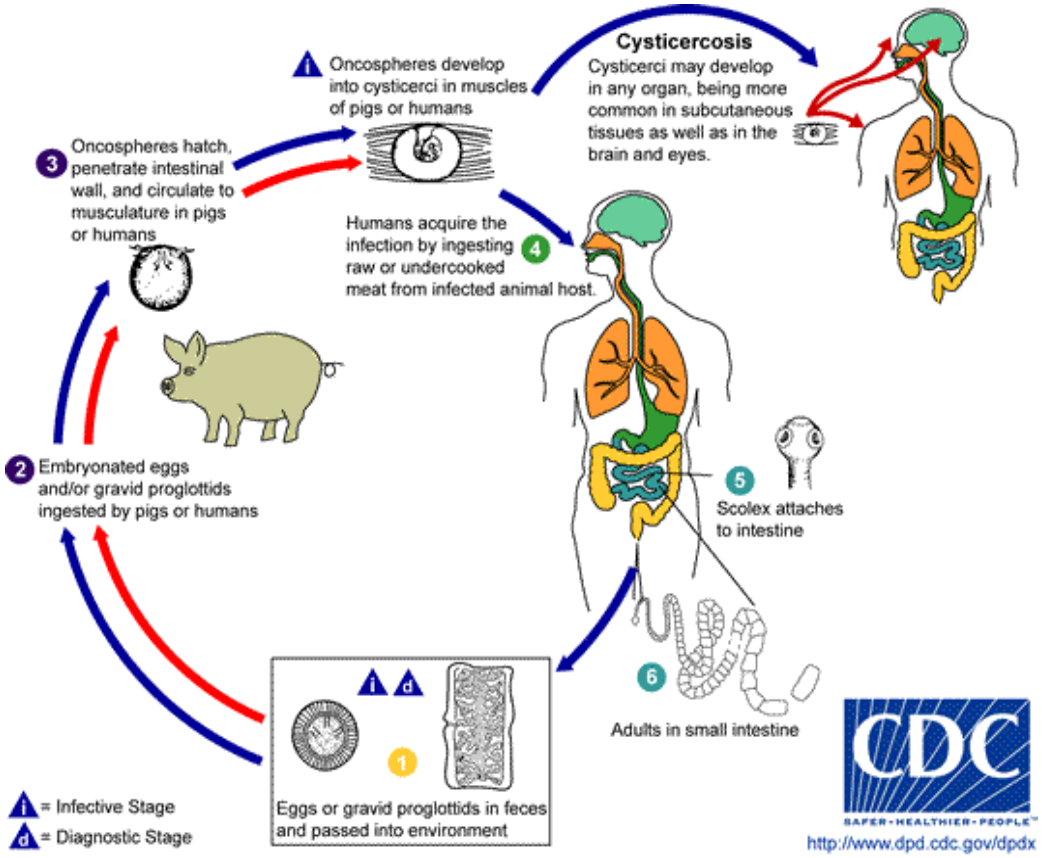
T. solium present mainly in Ethiopia, southern Africa and china .

Life cycle:

4. After ingestion of egg, the scolex (head) is freed and become attached to the wall of the small intestine by its suckers. Segments are formed from the neck region and

within 2-3 months grow into adult tapeworm with gravid (egg-filled) segments being found at the tail end. Usually one worm present but multiple can occur.

5. When fully developed, the graved become attached and eggs passed in stool.
6. To continue, eggs must reach the place of cattle food, after ingestion by a pig, pass through the intestine wall to the muscles, where the egg grows into infective larvae.



Diagnosis

Diagnosis of Taenia tapeworm infections is made by examination of stool samples; individuals should also be asked if they have passed tapeworm segments. Stool specimens should be collected on three different days and examined in the lab for Taenia eggs using a microscope. Tapeworm eggs can be detected in the stool 2 to 3 months after the tapeworm .infection is established

Tapeworm eggs of T. solium can also infect humans, causing cysticercosis. It is important to diagnose and treat all tapeworm infections.

Cysticercosis

Cysticercosis is a parasitic tissue infection caused by larval cysts of the tapeworm Taenia solium. These larval cysts infect brain, muscle, or other tissue, and are a major cause of adult onset seizures in most low-income countries. A person gets cysticercosis by swallowing eggs found in the feces of a person who has an intestinal tapeworm. People living in the same household with someone who has a tapeworm have a much higher risk of getting cysticercosis than people who don't. People do not get cysticercosis by eating undercooked pork. Eating undercooked pork can result in intestinal tapeworm if the pork contains larval cysts. Pigs become infected by eating tapeworm eggs in the feces of a human infected with a tapeworm.

Chapter 4

Helminthes worms.
Nemathelminthes (Round or Cylindrical worm).

Intestinal Nematodes.
Genus: Ascaris.
Species: A. Lumbricoides.

Major features of the intestinal round worms:

- Long cylindrical, non segmented.
- Adult worms live in the intestinal tract.
- Have alimentary canal with mouth and anus.
- Separated sexes (male and female).
- Some have free living while others are saprophytic.
- Female is longer than male, and some have spicules.
- Adult worms are oviparous, egg to larvae then adult worm.
- Some are laviparous: larvae then adult. Example: Strongyloides.
- The term filariform is used to describe the infective larva form that cause infection by penetrating the skin, i.e. S. stercoralis and Hook worm.
- The term rhabditiform is used to describe the non infective larva form that hatch from the egg in the intestine.

Geographical distribution:
World wide distribution, common among children and poor living standard.

Transmission and life cycle:
By ingestion contaminated food, water, or from hands that have become faecally contaminated.

Morphology:

A. Egg:

1. Fertilized egg: yellow-brown and the shell is covered by an albuminous coat. Oval or round measures 60x40 µm. contains a central granular mass which is an unsegmented fertilized ovum.

2. Unfertilized egg: occasionally found. Darker in colour, more elongated.

B. A. lumbricoides worm:

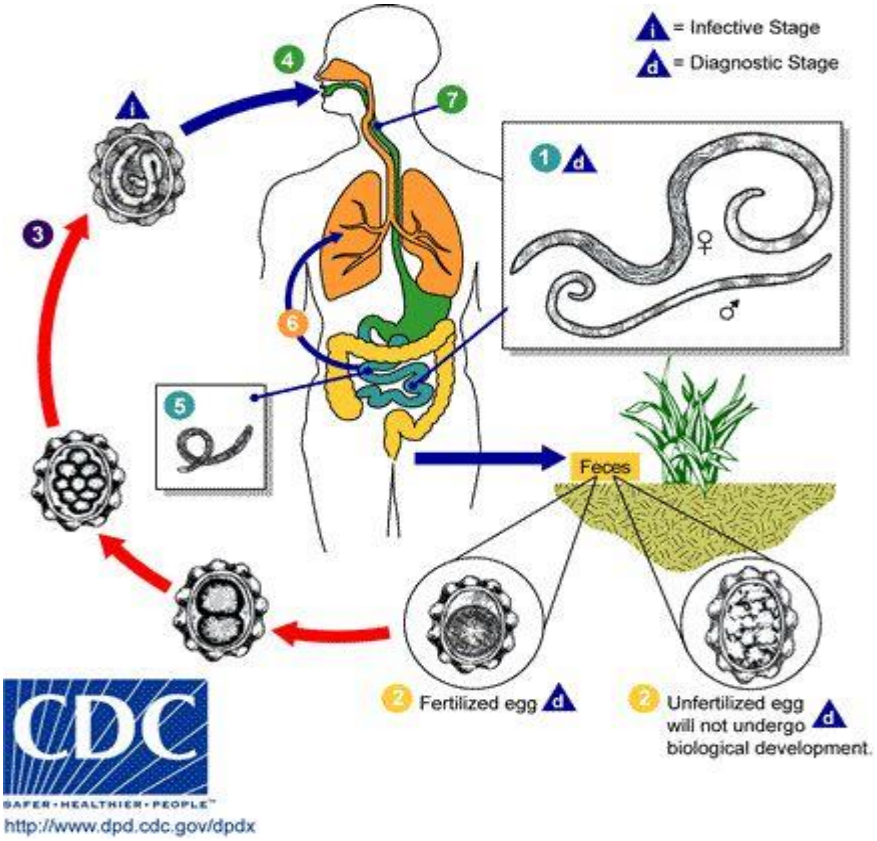
Large, female measures 20-35 cm long.

By 3-6 mm wide and male worms' measure 15-30 cm long by 2-4 mm wide, it is pink-brown or yellow-white in colour. Tale of male has two small spicules (rod-like projections). When examined with magnifying lens, three small lips can be seen around the mouth.

THE INFECTIVE FORM IS THE EGG.

Life cycle:

- Following ingestion of infective egg, the larvae hatch in the small intestine and penetrate the wall toward the blood vessels.
- In the circulation, larvae migrates to the lung-heart circulation during which they develop.
- After migration up to the trachea, they remain in the small intestine and grow into mature worm.
- After mating, the female produces large numbers of eggs which are passed in faeces.
- In soil and under special situations, eggs become infective by containing infective larvae.



(Life cycle of Ascaris Lumbricoides).

Clinical features and pathology:

- During their heart-lung migration, cause inflammatory and hyper sensitivity reactions.

- When they in the intestine, they cause abdominal pain, nausea, diarrhoea and vomiting.
- If heavy infections, cause obstructions in the intestine, bile duct, and pancreatic duct.

Laboratory diagnosis:

1- By finding eggs in faeces by the direct saline preparation.
2- Identifying A. lumbricoides worms expelled through the anus or mouth.

Morphology:

C. Egg:

3. Fertilized egg: yellow-brown and the shell is covered by an albuminous coat. Oval or round measures 60x40 µm. contains a central granular mass which is an unsegmented fertilized ovum.

4. Unfertilized egg: occasionally found. Darker in colour, more elongated.

D. A. lumbricoides worm:

Large, female measures 20-35 cm long.

By 3-6 mm wide and male worms' measure 15-30 cm long by 2-4 mm wide, it is pink-brown or yellow-white in colour. Tale of male has two small spicules (rod-like projections). When examined with magnifying lens, three small lips can be seen around the mouth.

2- Enterobius Vermicularis.

Called the thread worm and pinworm, causes enterobiasis.

Distribution:

World wide.

Habitat:

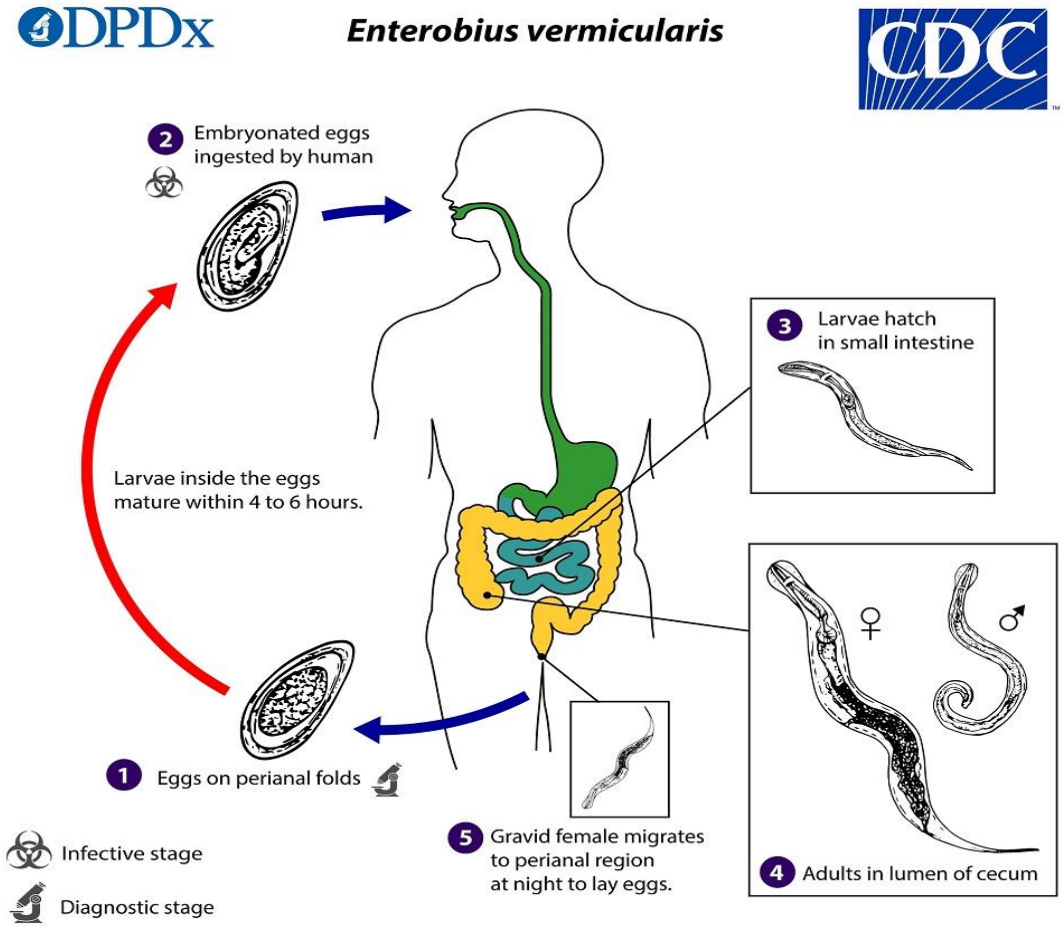
Adult worm in small intestine.
Egg deposited in the peri-anal skin.
Infective larvae: during night when eggs hatch on the buttock.

:Morphology

.Adult: male measures about 4 mm in length with curved tail and spicules
.Female: measures about 10 mm in length, with straight and pointed tail
Egg: asymmetric colorless and flattened from one site, measures about 55-25 µm.

Transmission and life cycle:

1- Ingestion of infective egg.
2- Infection is easily transmitted by contaminated bed or clothes.
3- Autoinfection when egg hatches on the buttocks.
4- Infective larvae migrate back to the intestine.



Gravid adult female *Enterobius vermicularis* deposit eggs on perianal folds 1 . Infection occurs via self-inoculation (transferring eggs to the mouth with hands that have scratched the perianal area) or through exposure to eggs in the environment (e.g. contaminated surfaces, clothes, bed linens, etc.) 2 . Following ingestion of infective eggs, the larvae hatch in the small intestine 3 and the adults establish themselves in the colon, usually in the cecum 4 . The time interval from ingestion of infective eggs to oviposition by the adult females is about one month. At full maturity adult females measure 8 to 13 mm, and adult males 2 to 5 mm; the adult life span is about two months. Gravid females migrate nocturnally outside the anus and oviposit while crawling on the skin of the perianal area 5 . The larvae contained inside the eggs develop (the eggs become infective) in 4 to 6 hours under optimal conditions 1 .

Rarely, eggs may become airborne and be inhaled and swallowed. Retroinfection, or the migration of newly hatched larvae from the anal skin back into the rectum, may occur but the frequency with which this happens is unknown.

Pathology:

Rarely causes serious disease, usually intense irritation around the anus. In female infection of urinary and genital tract may occur. Worms in appendix may cause appendicitis.

Diagnosis:

1- By finding the egg in samples collected from perianal skin using adhesive tape, or recovered from clothing during the night.
2- Egg can also be found in stool but this is less commonly.
3- By finding the adult (female only) worm in faeces or during clinical examination (occasionally less that 10% of cases).

3-Hook worm.
Ancylostoma duodenale.
Necator americanus.

Distribution:

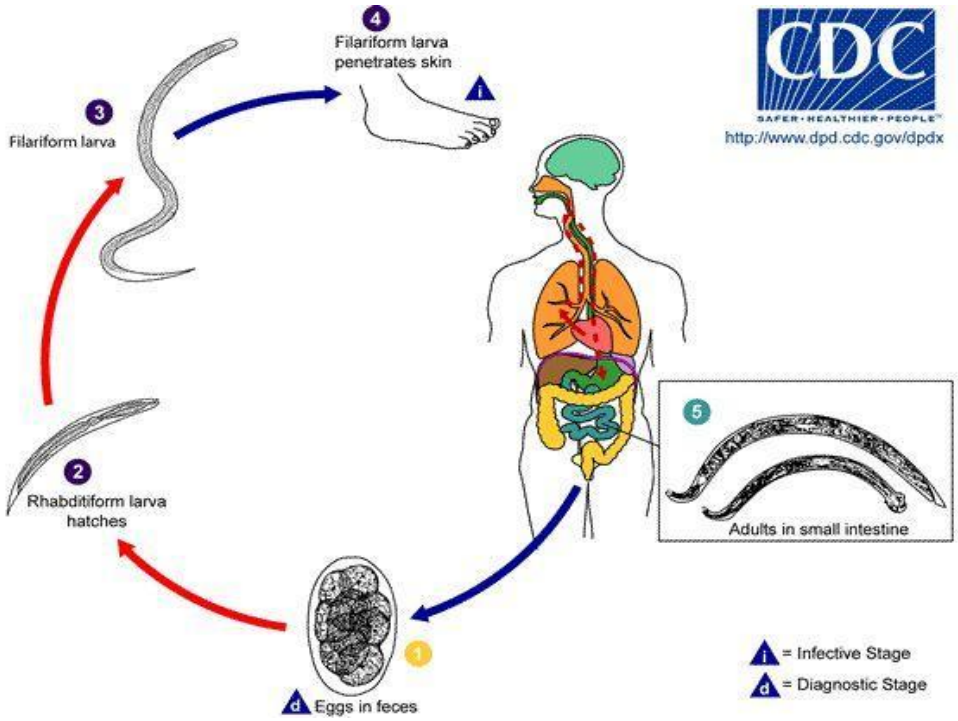
Tropics and sub-tropics, worm areas. Necator americanus is more common than Ancylostoma duodenale.

Habitat:

Adult worm in small intestine.
Egg in faeces but not infective.
Infective larvae: free in soil and water.

Transmission and life cycle:

1- Infection occurs when infective filariform larvae penetrate the skin.
2- Then larvae follow heart-lung migration.
3- Adult in small intestine.
4- Egg passed in faeces.
5- Larvae hatches from egg under favorable condition.
5- Develop into rhabditiform larvae, which develop into infective filariform larvae.



(life cycle of Ancylostoma duodenale.)

Eggs are passed in the stool ❶ , and under favorable conditions (moisture, warmth, shade), larvae hatch in 1 to 2 days and become free-living in contaminated soil. These released rhabditiform larvae grow in the feces and/or the soil ❷ , and after 5 to 10 days (and two molts) they become filariform (third-stage) larvae that are infective ❸ . These infective larvae can survive 3 to 4 weeks in favorable environmental conditions. On contact with the human host, typically bare feet, the larvae penetrate the skin and are carried through the blood vessels to the heart and then to the lungs. They penetrate into the pulmonary alveoli, ascend the bronchial tree to the pharynx, and are swallowed ❹ . The larvae reach the jejunum of the small intestine, where they reside and mature into adults. Adult worms live in the lumen of the small intestine, typically the distal jejunum, where they attach to the intestinal wall with resultant blood loss by the host ❺ . Most adult worms are eliminated in 1 to 2 years, but the longevity may reach several years.

Some *A. duodenale* larvae, following penetration of the host skin, can become dormant (hypobiosis in the intestine or muscle). These larvae are capable of re-activating and establishing patent, intestinal infections. In addition, infection by *A. duodenale* may probably also occur by the oral and the transmammary route. *A. ceylanicum* and *A. caninum* infections may also be acquired by oral ingestion. *A. caninum*-associated eosinophilic enteritis is believed to result following oral ingestion of larvae, not percutaneous infection. *N. americanus* does not appear to be infective via the oral or transmammary route.

Pathology:

1- The first sign is skin reaction at the site of penetration.
2- Mild respiratory symptoms.
3- Adult hookworm causes chronic blood loss leading to developing iron deficiency anaemia in prolonged infection.

Morphology:

Diagnosis:

1- Finding hookworm egg in faeces by direct or concentration technique.

Egg: oval 60X40 µm. colorless with thin shell.
Adult:

2- In old stool sample larvae may hatch.

Books

Doss, Mildred A. *Index Catalogue of Medical and Veterinary Zoology: Trematoda*, Parts 1–8. Washington, DC: U. S. Government Printing Office, 1966.
Olsen, O. Wilford. *Animal Parasites: Their Biology and Life Cycles.* Minneapolis: Burgess Publishing Co., 1967.
Schell, Stewart C. *How to Know the Trematodes.* Dubuque, IA: William C. Brown Co., Publishers, 1970.